The impact of Stress and Anxiety on the neurocognitive performance of Australian Nurses:

An electroencephalographic and psychometric assessment

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Submitted in partial fulfilment of the requirements for the degree of Doctor of Philosophy (Science) at the University of Technology Sydney.

I. Declaration

I certify that the work in this thesis has not been previously submitted for a degree nor has it been submitted as part of requirements for a degree except as fully acknowledged within the text.

I also certify that the thesis has been written by me. Any help that I have received in my research work and the preparation of the thesis itself has been acknowledged. In addition, I certify that all information sources and literature used are indicated in the thesis.

Signature: Production Note: Signature removed prior to publication. Date: 12/07/2018

Ty Lees

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III. Publications and Presentations

Publications relevant to thesis

Journal Articles

1. Maharaj, S., **Lees, T.,** and Lal, S., 2018. Negative Mental States and Their Association to the Cognitive Function of Nurses. *Journal of Psychophysiology*, doi: 10.1027/0269-8803/a000223
2. **Lees, T**., and Lal, S., 2017. Stress and its impact on the cognitive performance of Australian nurses. *Stress and Health*, **33**(1), 45-54 doi: 10.1002/smi.2672
3. **Lees, T**., Khushaba, R., and Lal, S., 2016. Electroencephalogram Associations to Cognitive Performance in Clinically Active Nurses. *Physiological Measurement*, **37**(7):968-980, doi: 10.1088/0967-3334/37/7/968

Conference Abstracts

1. **Lees, T**., Kalatzis, G., and Lal, S., 2015. Examining negative mental states and their association to psychometric and electroencephalographic measures of cognitive performance in Australian Nurses. *Psychophysiology*, **52**(S24), doi: 10.1111/psyp.12495
2. **Lees, T**., Maharaj, S., and Lal, S., 2015. Electroencephalographic markers of subjective cognitive performance: Implications towards electrophysiological prediction of early cognitive decline. *Frontiers in Human Neuroscience*. doi: 10.3389/conf.fnhum.2015.219.00057

Invited Presentations

1. **Lees, T**., Stress and Cognitive Performance in Nurses: An example of research in the NRU. Oral Presentation: Warfighter Effectiveness Research Centre (WERC) United States Airforce Academy Brownbag 2015, Colorado Springs, USA.

Conference Presentations

1. **Lees, T.,** Maharaj, S., Kalatzis, G., Nassif, N., Newton, P, and Lal, S. The neurocognitive relationship between stress and anxiety, and memory and decision making performance of Australian Nurses. Poster presentation: 58th

Annual meeting of the Society for Psychophysiological Research 2018, Quebec City, Canada

2. Maharaj, S., **Lees, T.,** Zaslawski, C., Fatima-Shad, K., and Lal, S. Assessing the link between negative mental states and cognitive performance in health professionals. Poster Presentation: 2017 Australasian Cognitive Neuroscience Society (ACNS) Conference, Adelaide, Australia.
3. Maharaj, S., **Lees, T**., and Lal, S., Electroencephalographic changes in beta & gamma reactivity associated with cognitive performance. Poster Presentation: Inter-University Neuroscience & Mental Health Conference 2016, Sydney, Australia
4. **Lees, T**., Kalatzis, G., and Lal, S. Examining negative mental states and their association to psychometric and electroencephalographic measures of cognitive performance in Australian Nurses. Poster presentation: 55th Annual meeting of the Society for Psychophysiological Research 2015. Seattle, USA
5. **Lees, T**., Maharaj, S., and Lal, S. Electroencephalographic markers of subjective cognitive performance: Implications towards electrophysiological prediction of early cognitive decline. Oral Presentation: 25th Annual Conference of the Australasian Society for Psychophysiology 2015, Sydney Australia
6. **Lees, T**., and Lal, S. Negative mental states, their association to frontal lobe brain activity and the effect coping. Oral Presentation: 2nd Inter-University Neuroscience & Mental Health Conference 2015, Sydney, Australia.
7. Maharaj, S., **Lees, T**., and Lal, S. Cognitive function associations to mental states in nurses. Poster presentation: 32nd Combined Health Science Conference; New Horizons 2015, Sydney, Australia.
8. Kalatzis, G., **Lees, T**., Nassif, N., and Lal, S. Investigating Cognitive Function in Clinical and Healthy Samples using Electroencephalography and Psychometric Assessment: A Comparative Study. Poster Presentation 32nd Combined Health Science Conference; New Horizons 2015, Sydney, Australia
9. **Lees, T**., and Lal, S. Electroencephalography and its associations to cognitive performance in nurses: An exploratory assessment. Poster Presentation: 30th Combined Health Science Conference; New Horizons 2013. Sydney, Australia.
10. **Lees, T**., and Lal, S. Occupational stress and cognitive performance in nurses. Poster Presentation: 29th Combined Health Science Conference; New Horizons 2012. Sydney, Australia.

Other Publications

Journal Articles

1. **Lees, T.,** Shad-Kaneez, F., Simpson, A.M., Nassif, N., Lin, Y., and Lal, S., 2018. Heart rate variability as a biomarker for predicting stroke, post-stroke complications and functionality. *Biomarker Insights* (In press)
2. **Lees, T**., Nassif, N., Simpson, A.M., Shad-Kaneez, F., Martiniello-Wilks, R., Lin, Y., Jones, A., Qu, X., and Lal, S., 2017. Recent advances in molecular biomarkers for diabetes mellitus: a systematic review. *Biomarkers*, 1-13, doi: 10.1080/1354750X.2017.1279216
3. Rothberg, L.J., **Lees, T**., Clifton-Bligh, R., and Lal, S., 2016. Associations between heart rate variability and blood glucose levels: implications for non-invasive glucose monitoring. *Diabetes Technology & Therapeutics*, **18**(6):366-376, doi: 10.1089/dia.2016.0010

Reports

1. Lal, S., **Lees, T**., Elliott, J.L., Rai, T., Gunning, S., and Newton, P., 2015. A review of current evidence about mental disorders and psychological and other wellbeing programs in the law enforcement workplace. Sydney, Australia. Commissioned by the NSW Police Force.

Invited Presentations

1. **Lees, T**., and Lal, S. Research and collaboration in the Neuroscience Research Unit (NRU). Oral Presentation: Warfighter Effectiveness Research Centre (WERC) United States Airforce Academy Brownbag 2015, Colorado Springs, USA.

Conference Presentations

1. Maharaj, S., **Lees, T.,** Zaslawski, C., Fatima-Shad, K., and Lal, S. The prevalence of depression in a cohort of Australian Nurses. Poster presentation: Inter-University Neuroscience & Mental Health Conference 2017, Sydney, Australia.
2. Kalatzis, G., **Lees, T.,** Nassif, N., Zaslawski, C., and Lal, S. Exploring cognitive function in diabetes and non-diabetes samples using electroencephalography

(EEG) and psychometric assessment: a comparative study. Oral presentation: 37th Annual Scientific Meeting of the Australasian Neuroscience Society 2017, Sydney, Australia

3. Hopkins, Z., **Lees, T.,** and Lal, S. Cardio vascular and Autonomic Associations to Post-traumatic stress disorder symptomatology. Oral Presentation: Inter-University Neuroscience & Mental Health Conference 2017, Sydney, Australia.
4. Vine, M., **Lees, T.,** Nassif, N., Simpson, A. M., and Lal, S. Investigating the associations between ADHD symptomology and chronic illness: cardiovascular disease and diabetes mellitus. Poster Presentation: Inter-University Neuroscience & Mental Health Conference 2017, Sydney, Australia.
5. Kalatzis, G., **Lees, T**., Nassif, N., Zaslawski, C., and Lal S. Investigating cognitive function in diabetes and healthy samples using electroencephalography (EEG) and psychometric assessment: a comparative study. Oral Presentation: Inter-University Neuroscience & Mental Health Conference 2016, Sydney, Australia
6. Jarman, L.R., Elliott J.L., **Lees, T**., Nassif, N., Simpson, A.M., Clifton-Bligh, R., and Lal, S. Associations between heart rate variability and blood glucose levels. Poster presentation: 33rd Combined Health Science Conference; New Horizons 2016, Sydney, Australia.
7. Ricciardiello, A., Elliott, J.L., **Lees, T**., and Lal, S. Emotion regulation and stress: physiological assessment. Poster presentation: 33rd Combined Health Science Conference; New Horizons 2016, Sydney, Australia
8. Gorval, M., **Lees, T**., Elliott, J.L., Nassif, N., Simpson, A.M., Clifton-Bligh, R., and Lal, S. Investigation of the association between blood glucose levels, anxiety and depression. Poster presentation: 33rd Combined Health Science Conference; New Horizons 2016, Sydney, Australia
9. Elliott, J., **Lees**, T., Nassif, N., and Lal, S. Stress and the New South Wales Police Force: The prevalence of various coping mechanisms. 2nd Inter-University Neuroscience & Mental Health Conference 2015, Sydney, Australia.
10. Kalatzis, G., **Lees, T**., and Lal, S. Investigating Cognitive Performance in Different Racial Groups: An Exploratory Study Using Electroencephalography and Psychometric Assessment. Poster presentation: 31st Combined Health Science Conference; New Horizons 2014. Sydney, Australia.

11. Elliot, J.L., **Lees, T.,** Nassif, N., and Lal, S. Cardiovascular measures and sleep health associations with shift work in police officers: A physiological assessment. Oral Presentation: 31st Combined Health Science Conference; New Horizons 2014. Sydney, Australia.

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VIII. List of Abbreviations

A – Auricular

ACTH – Adrenocorticotropic hormone

AD – Alzheimer's Disease

AIN – Assistant in Nursing

AUD – Australian Dollars

AVP – Arginine vasopressin

BMI – Body Mass Index

BP – Blood pressure

C – Central

CRH – Corticotropin releasing hormone

DASS – Depression, Anxiety, Stress, Scale

EEG – Electroencephalography

EN – Enrolled Nurse

EOG - Electrooculogram

ERP – Event Related Potential

F – Frontal

Fp – Frontal Pole

GAD – Generalised Anxiety Disorder

HPA – Hypothalamic Pituitary Adrenal Axis

HR – Heart Rate

HREC – Human research ethics committee

IIR – Infinite Impulse Response

LAQ – Lifestyle Appraisal Questionnaire

LASSO – Least Absolute Shrinkage Selection Operator

mmHg – Millimetres mercury

MCI – Mild Cognitive Impairment

MMSE – Mini-mental State Exam

MRI – Magnetic Resonance Imaging

NHP – Non-health Professional

O – Occipital

OCD – Obsessive Compulsive Disorder

P – Parietal

PCA – Principal Component Analysis

PD – Panic Disorder

RN – Registered Nurse

SNS – Sympathetic Nervous System

SSI – Standard Shiftwork Index

T – Temporal

UTS – University of Technology Sydney

WCCL – Ways of Coping Checklist

IX. Abstract

Stress and anxiety both have demonstrable impact, causing neuronal damage and death (Sapolsky, 1996, Conrad, 2006), functional connectivity changes (Bishop, 2009, Andreescu et al., 2014), and various cognitive impairments (de Quervain et al., 2000, Savage et al., 2000, Wetzel et al., 2006, Henderson et al., 2012, Nieuwenhuys et al., 2015). However, despite demonstrated quality of care reductions (Sveinsdóttir et al., 2006, Berland et al., 2008) and calls for further research (LeBlanc, 2009, Lees and Lal, 2017), a limited amount of research investigating the impact of stress and anxiety on the cognitive performance of health professionals has been conducted (LeBlanc, 2009). Therefore, the aim of the present study was to examine the relationships between stress, anxiety and cognitive performance in health professionals via comprehensive psychometric and electroencephalography (EEG) assessment; as well as assess the predictive capability of EEG in measuring cognitive performance.

Presently, this doctoral research reports on results obtained by analysing data from 118 nurses and 144 non-health professionals. The experimental protocol commenced by capturing participant demographic data, such as, blood pressure, heart rate, as well as hip and waist measurements, followed by the completion of pre-study questionnaires including the Lifestyle Appraisal questionnaire (Craig et al., 1996), the Depression, Anxiety, Stress scale (Lovibond and Lovibond, 1995b), and the Fatigue State Question (Lal and Craig, 2002). Following this, a two lead bipolar or 32 lead monopolar EEG was captured during a resting baseline and a Stroop test based active phase. After the electroencephalogram recording, psychometric cognitive performance was assessed by the Mini-Mental State Examination (Folstein et al., 1975) and the Cognistat (Mueller et al., 2007). Participants then completed the revised Ways of Coping Checklist (Vitaliano et al., 1985), and the Fatigue State Question, again for the latter. Nurse participants also

completed parts of the Standard Shiftwork Index (Barton et al., 1995). Lastly, participant's blood pressure was again recorded and the experiment concluded.

The results indicate that both non-health professionals and nurses experience stress and anxiety. In both groups, a stress level within the normal range was significantly associated ($p < 0.05$) with increased memory performance and delta activity, while theta and beta activity increases were similarly implicated for the nurse group only. However, with an increase in stress levels, stress was associated with increased judgement performance and fronto-temporal and parietal gamma activity, as well as reduced fronto-temporal delta activity in non-health professionals. Additionally, impaired memory performance as well as fronto-central delta, fronto-temporal and parietal gamma, and fronto-central and temporal beta activity increases were associated with this increased stress in nurses.

With respect to anxiety, it was associated with increased lifestyle risk factors, impaired global, attention, and memory domain performance, as well as delta, alpha and gamma activity changes in non-health professionals. Comparatively, in nurses anxiety was associated with improved Stroop test performance, global cognitive performance and delta and gamma activity, as well as impaired memory performance. Lastly, it was found that global cognitive performance could be predicted by a combination of fast wave EEG activity variables ($R^2 \geq 0.440$; $p \leq 0.013$). Similarly, unique combinations of EEG variables from the 5 investigated frequency bands predicted, in varying degrees, attention ($R^2 \geq 0.204$; $p \leq 0.014$), memory ($R^2 \geq 0.443$, $p \leq 0.010$) and judgement ($R^2 \geq 0.407$; $p \leq 0.001$) domain performance.

Collectively, these findings provide an insight into the cognitive impact of stress and anxiety, and determine a unique impact profile of stress and anxiety for both non-health

professionals and nurses. Additionally, they demonstrate the multifaceted nature of the relationship between stress, anxiety and cognitive performance, where both improvements and impairments are observed. Further understanding the impact of stress and anxiety on cognitive performance may enable the development and implementation of management and intervention strategies to preserve the cognitive health of health professionals, and in turn, ensure quality of patient care and reduce adverse medical event incidence. Further, it may be possible to use EEG activity to predict early cognitive impairment, which has strong implications for developing diagnostic measures for cognitive impaired states such as dementia and Alzheimer's disease.

Chapter 1 – Introduction

Modern medicine has and continues to evolve naturally alongside human culture, progressing from the simple shamanism and herbalism of the earliest men, through the ancient civilizations of Egypt, Babylon, and Rome, among others, into the alchemic societies of Medieval Europe and Arabia and finally into the Age of Enlightenment of the late 17th and early 18th centuries. It was during this time where a protoform of scientific method, making use of gradual rejection, replaced previous methodologies and became a prelude to the birth of a truer biomedical approach that searched for testable and reproducible results. The utilisation and expansion of this new method allowed human understanding of health, disease and medicine the opportunity to develop, expand and diversify into what we now know as modern medicine.

In the present day, all fields of medicine are rapidly innovating and expanding, and consistently providing novel technologies, treatment options and diagnostic tools that have the ability to improve the quality of human life. As this expansion occurs, the demand for people skilled in the provision of medical services and the use of these technologies also increases. These individuals are health professionals and their acquired skills and knowledge mark them as an important part of our society.

1.1 Health Professionals

Health professionals are individuals who, through their education and training, certification and/or licensure have acquired the specific knowledge and skillset that qualifies and enables them to provide health care services (Segen, 2006, Babbush, 2008, Mosby, 2013). They are typically registered by, or with, a government controlled department or registration board. Alternatively, in some instances, they may be certified

by a non-governmental independent organisation (Harris et al., 2009), where registration to such a group is often a legally enforced requirement that awards these individuals the ability to practice in their field.

1.1.1 Classification

The classification of the health professions and the individuals who are employed within such professions revolves largely around the typical services provided. Utilising such a service type division, and categorising the health professions (referring to all professions within the healthcare industry) and their employees using three main service groups (diagnostics, treatment and management) provides a strong basis from which a better understanding of the multifaceted nature of healthcare can be developed. By exploring these main service groups, we can define 'diagnostics' as the processes by which injury, illness and/or disease are identified within individuals (Harris et al., 2009). Further, 'treatment' refers to processes that directly follow diagnosis and are enacted in the immediate amelioration of diagnosed disease, illness or injury. Finally, management describes the amelioration processes provided after initial treatment and during the continuation towards recovery (Harris et al., 2009).

The present study selected nurses as the health professional sample group and this was a decision that was largely informed by two main considerations. Firstly, the multifaceted workload and duties undertaken by nurses, including providing support, care, and assistance to both patients and their families, participating in patient rehabilitation and promoting health education (DeLucia et al., 2009, American Nurses Association, 2017) involve both the treatment and management service areas. The implication of this is that, among health professionals, nurses are the professionals who spend the largest amount of time with patients (DeLucia et al., 2009). The second reason for selecting

nurses is that in Australia, nurses are numerically the largest group among all health professions (Nursing and Midwifery Board of Australia, 2016).

1.1.2 Health Professionals in Society

Every living organism values individual and group health in some manner and humans are no exception; as a collective, we view individual health as a significant and important contributor to quality of life. As society holds this value, the individuals that help restore and/or maintain individual health are rendered crucially important. Furthermore, it is important to note that the timely and accurate application of health services and technologies is vital, especially as these services and technologies are often administered in critical situations where errors can be fatal.

For the year of 2015-16, the Australian healthcare industry was staffed by a combined total of 657,621 registered health professionals (Australian Health Practitioner Regulation Agency, 2016a); indicating that 2.73% of Australia's population cares for the estimated 24.12 million Australians (Australian Bureau of Statistics, 2016). In this same year, the Australian nursing profession consisted of 346,387 personnel registered solely as nurses (both enrolled and registered nurses), and a further 29,699 registered as both a nurse and midwife, equating to a total of 376,086 individuals; an increase of 2.58% on the previous year (Australian Health Practitioner Regulation Agency, 2016b). These numbers indicated that as a collective, nurses represent 57.19% of all registered health practitioners in Australia and 1.44% of the country's total population (Australian Bureau of Statistics, 2016). Further, a report from the Nursing and Midwifery Board of Australia (2016) indicates that the Australian nursing profession is predominantly female, with 89.13% of nurses identifying as female.

1.1.3 Stress and Anxiety in the Health Professions

It is well established that the health professions are demanding and high responsibility occupations (Spector, 1999) and these characteristics are generated by the very nature of the professions which require individuals to diagnose, treat and manage the health of others. Therefore, it follows that the presence of these characteristics renders the health professions inherently stressful and research has indicated as such (Firth-Cozens, 2003, Thomas, 2004).

Whilst some research has demonstrated lower levels of stress in the health professions relative to other professions (Houtman and Kompier, 1995), other research has demonstrated the inverse, and some individual occupations (e.g. medicine and nursing) can have high stress, especially in particular circumstances (Spector, 1999). Interestingly, the work related stressors experienced in different health professions are often similar including, but not restricted to heavy workload, time on call, and patient relationships (Richardsen and Burke, 1991a, Richardsen and Burke, 1991b). However, a number of differences do exist (Menon et al., 1996), and are largely related to the contextual differences in the work performed (i.e. a nurse has different duties than a doctor) as well as the workload of the individual professions themselves. These differences, however, cannot be solely attributed to the profession as other variables do differ between the professions, for example, sex distribution. These experienced workplace stressors have been commonly associated with poor mental health in health professionals, which can impact patient care (Chopra, 2009, Chiang and Chang, 2012, Jones et al., 2015).

In the medical and dental fields, mental stress has been associated with 24% of all compensation claims, a value that is 4% above the industry average (Safework Australia, 2013), and indicates the prevalence of mental stress within the health

professions and further suggests stress to be inherent to the health professions. In the year of 2010-11, 7385 serious worker's compensation claims and five fatalities were attributed to mental stress (Safework Australia, 2013). Further, these claims possessed the highest median payment at $20,800 (a value that is double that of all other claims) and presented a visible cost of $153,600,000 AUD (Safework Australia, 2013). In the same year, the median time away attributed to mental stress was 13 weeks, a value triple the median for all other serious claims (Safework Australia, 2013). As such, there is an invisible cost associated with replacing staff, retraining and reskilling, supplementary to the $153 million in claim payouts that must also be considered when evaluating the economic impact of stress.

1.1.3.1 Nurses

Research has indicated that the nursing profession is inherently stressful (Lee and Wang, 2002, Sveinsdóttir et al., 2006, Golubic et al., 2009). This stress is generated from numerous sources mostly localised to daily activities including working with vulnerable and needy patients, working in critical care situations, provision of continuous care and other occupational stressors including long hours, collegial inexperience, and lack of support (Baldwin, 1999, Lee and Wang, 2002, Sveinsdóttir et al., 2006, Golubic et al., 2009, Howard et al., 2013). Indeed, the heavy workload and occupational demands placed upon nurses have previously been associated with poor mental health (Chopra, 2009). It has also been suggested that experiencing stress can have detrimental consequences upon nursing quality and the level of provided patient care (Sveinsdóttir et al., 2006), a conclusion, with obvious and serious consequences, that has been supported by additional research (Baldwin, 1999, Tarnow-Mordi et al., 2000, Berland et al., 2008).

1.2 Stress

Stress as a concept is vast and multidimensional in how it is defined, perceived and experienced, however, it can be generally defined as the disruption of an organism's homeostasis by any psychological, physical, social, economic or other factor(s), be they real or imagined (Harris et al., 2009, Conrad, 2011). Further, it is important to note, that this definition also includes the anxious, tense, frustrated and/or angry feelings that occur as a result of experiencing this homeostatic disruption (Herbert, 1999, Payne, 1999).

Lazarus and Folkman (1984) designed their transactional model of stress in an attempt to better understand the construct of stress, and see stress as the result of the interaction between the individual and their environment. The model describes four different appraisals that broadly apply to any circumstance and determines whether or not stress may arise from the experienced situation. In this model, a situation may be benign, or it may involve threats of future stress, harm, loss, and/or challenge (Lazarus and Folkman, 1984) and, as such, stress arises when environmental demands exceed the resources available to an individual to combat these demands, especially in personally significant situations (Lazarus and Folkman, 1984). Furthermore, the model indicates that individual differences and variations, for example, personality, coping method utilisation, and social group characteristics, may also have an impact on perception of stress (Figure 1.1) and hence, should be considered when conducting research on the effects of stress.

Figure 1.1 – Stress; a conceptual framework.

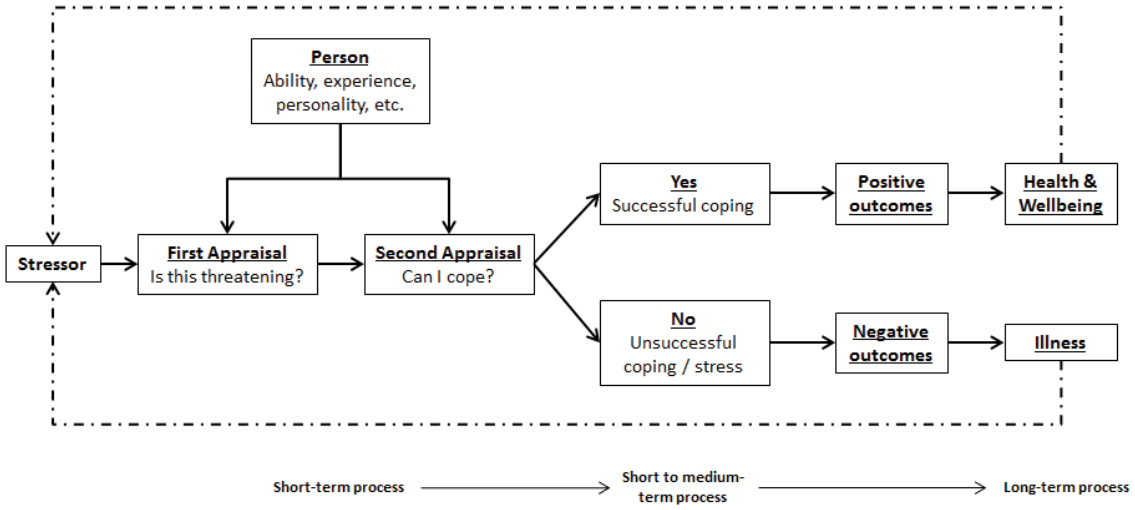

Figure 1 presents a conceptual flow diagram describing that describes the overall process of experiencing stress, as well as its individual components including the stressor/s, perceptual steps, cognitive steps and outcomes. Figure adapted from Payne (1999).

The first results of the Australian Health Survey published in 2012, indicated that approximately 1.8 million adult Australians reported having experienced stress that could be categorised as either high or very high (Australian Bureau of Statistics, 2012). The two major causes of this stress were indicated to be financial issues (reported in approximately 50% of respondents) and workplace stress (reported in 32% of respondents), indicating the importance of stress in Australia (Casey, 2012).

1.2.1 Eustress and Distress

Stress can be examined based on the value that the stressor and resultant strain may present to the individual. Although stress is thought to be a negative experience, the experience of stress naturally produces an adaptive response, and so, it is possible that in some instances it will be positive to the individual. Seyle (1975) coined the term eustress to describe this positive appraisal of stress, where it is defined to be positive if the adaptation response proves to be beneficial to the organism (Seyle, 1975, Simmons

et al., 2001). The positive nature of eustress has also been characterised using the transactional model of stress i.e. the individual's confidence in being able to successfully overcome their experienced stressors (Mohr, 1993). In contrast, any instance where experienced stressors are perceived as harmful, threatening or otherwise negative has been termed distress. Distress occurs when such a stressor places demands on the body that exceed its ability to maintain homeostasis, thus rendering the organism unable to master or overcome that stressor (Mohr, 1993, Conrad, 2011).

The existence of these constructs led to the development of an interesting quandary for stress research, in which it is possible that rather than being two ends of a singular continuum, both eustress and distress may represent two distinct states and as such may require separate measurement. Additionally, if eustress and distress are in fact two distinct states, it follows that it is not possible to confirm the presence of one simply by the absence of the other (Simmons et al., 2001), i.e. if an individual is not currently experiencing eustress, they do not immediately experience distress. Thus, it could be suggested that a third state of "non-stress" exists alongside eustress and distress, however, the construct of stress is most likely far more complex than this, and probably consists of a number of additional partially stressed and non-stressed states.

Finally, the passage of time alters our perception of experienced stressors, which then modulates our stress response (Herbert, 1999). This modulatory effect renders the distinction between acute and chronic stress an important consideration.

1.2.2 Acute and Chronic Stress

As mentioned, beyond the positive or negative nature of stress, the passage of time is equally important to the construct of stress. Acute stress is precipitated by the activation of the hypothalamic pituitary adrenal (HPA) axis and the sympathetic nervous system

(SNS) in response to experiencing a stressor (Cannon, 1929). The activation of these systems is normally time-locked and, as such, will only occur for a time period long enough to enable the responding organism to master the stressor (Conrad, 2011). However, overcoming challenges that are more prolonged relies on the adaptive processes of habituation and sensitisation.

Habituation is the process in which the stress response is completed regardless of the presence of the stressor, and occurs when an organism experiences benign homotypic stressors (Conrad, 2011). During this process, the organism retains the ability to respond to any new stressors and may become sensitised towards novel inputs, providing the ability to combat multiple stressors simultaneously. However, if the experienced stressor/s is of sufficient intensity, the organism may not be able to utilise habituation and sensitisation processes and hence would be unable to overcome the stressor. The inability to overcome experienced stressors can lead to significant dysfunction and chronic stress (Conrad, 2011) and possibly burnout; a psychological syndrome involving an ongoing stress response manifesting with cynicism, overwhelming fatigue, a sense of ineffectiveness and lack of accomplishment (Maslach, 1993), that is associated with cognitive declines (Linden et al., 2005, Sandström et al., 2005). Although, more recently, it has been suggested that such cognitive declines are reversible (Österberg et al., 2012). Burnout may develop gradually and can remain unnoticed; it is often the result of a misfit between intentions and the reality of the job (Schaufeli, 1999). Specific health profession related causes of burnout include, among other factors, long working hours, staff shortages, and provision of continuous care (Shimizu et al., 2005, İlhan et al., 2008).

Moreover, the distinction between acute and chronic stress is not the only way stress has been classified, and further specification can be based upon the nature of the experienced stressor, for example, financial.

1.2.3 Occupational Stress

Contextualising stress in the health professions requires a brief discussion of the concept of occupational stress. Research has developed a number of models that attempt to understand occupational stress, and its generation and impact. French and Kahn (1962) developed the Person-Environment Fit Model, which suggests stress is the result of an incongruence between an individual and their work environment i.e. stress will develop if the job skills, knowledge and/or training of an individual do not match or meet the demands of the stressor. Further, the inverse is also true, whereby, if the demands of a stressor are matched to the individual, they will be able to overcome that stressor. The model suggests that a greater misfit between the two components corresponds to a greater chance of stress developing.

A number of years later, Karasek (1979) further developed the Person-Environment Fit model and proposed the Demand-Control Model, which views stress as arising from the interaction between job demands and the individual's decisional freedom in meeting these demands and not necessarily ability only (Karasek, 1979, van der Colff and Rothmann, 2009). Delving further into this model, it is suggested that eustress occurs in active jobs which exhibit both high demands and high decisional latitude, as well as those that afford the individual the ability to deal with the problem in their own manner (Karasek, 1979). Conversely, opposing conditions i.e. high demands and low decisional latitude, or one that forces an individual into combating the situation in a predetermined (and possibly ill-fitting) manner, results in the development of distress (Karasek, 1979).

In addition, it should be noted that other categorisations of jobs do exist in this model, most important of which are passive jobs (those with low demands and low decisional latitude), as they have been correlated to poorer psychological health (Karasek, 1979). In the same year, Payne (1979) expanded on the Demand-Control model and added all forms of social support that an individual may receive, as research indicated that social support modulated stress (Gore, 1978). This updated model best predicts distress in jobs that present the individual with high demands, low control and low support.

Summarily, occupational stress is not absolutely defined, as a number of differing perspectives, models and theories exist, and the intervening years between the discussed models and now have seen a number of developments. However, most importantly, most proposed models/theories agree that the condition of the worker may be altered by their experience of occupational stress, and this may subsequently alter their capacity to work as normal (Engelhart et al., 1999); a change which could have far reaching consequences.

1.2.4 Physiological Basis of Stress

The complete human stress response is characterised by an increase in avoidance behaviour, as well as vigilance and arousal (Guthrie et al., 2010), and can be separated into two components based upon the physiological system activated. The first of these two systems is the sympathomedullary pathway, and the activation of the SNS (Figure 1.2). The somatosensory cortices and association areas send top-down input to the periventricular nucleus, which then synapses with sympathetic preganglionic neurons, which in turn synapse with adrenal Chromaffin cells (Bear et al., 2007, Martini and Nath, 2008). These cells secrete adrenaline and noradrenaline directly into the circulation (Porth, 2006), thereby stimulating a number of peripheral responses

including increased heart rate (HR), blood pressure (BP) (Raven et al., 2008), as well as central responses including improved reaction times (Martini and Nath, 2008).

Figure 1.2 – The sympathetic nervous system

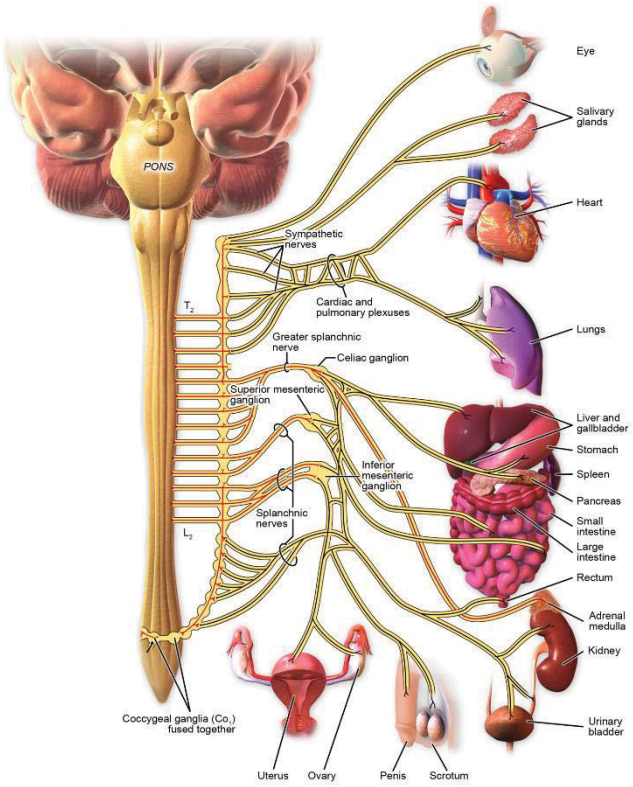

Figure 1.2 displays the sympathetic nervous system in its entirety, including the central components, as well as both the pre- and post-ganglionic neurons and their relevant effector organs (Adapted and modified from Blausen.com staff (2014)).

The second and hallmark component is the activation of the HPA axis (Figure 1.3), and the resultant production of cortisol (Seyle, 1975, Conrad, 2011). In the typical response, the paraventricular nucleus of the hypothalamus receives top-down input from the amygdala and sensory cortices, which stimulates the production and release of both corticotropin-releasing hormone (CRH) and arginine vasopressin (AVP) into the hypothalamopituitary portal circulation (Bear et al., 2007, Martini and Nath, 2008, Conrad, 2011). The release of CRH subsequently activates the anterior pituitary gland,

which then releases adrenocorticotropic hormone (ACTH) into the systemic circulation (Conrad, 2011).

Figure 1.3 – The hypothalamic pituitary adrenal axis

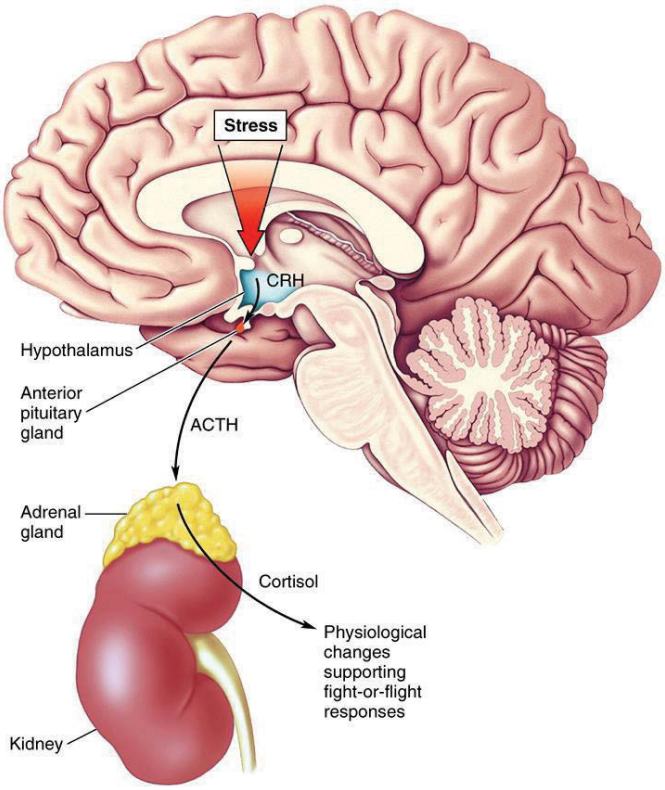

Figure 1.3 displays the hypothalamic pituitary adrenal axis. Stress activates the hypothalamus which communicates with the anterior pituitary gland via corticotropin-releasing hormone (CRH). In the presence of CRH, the anterior pituitary gland produces adrenocorticotrophic hormone (ACTH) which binds in the adrenal cortex and stimulates the synthesis and release of cortisol; a corticosteroid that generates a number of physiological changes characteristic of the stress response (Adapted from Bear et al., 2007 pg. 668).

In turn, ACTH binds in the adrenal cortex and stimulates the release of cortisol (Porth, 2006, Bear et al., 2007, Martini and Nath, 2008, Raven et al., 2008), which similarly enters the systemic circulation and subsequently triggers various physiological changes that complement those that result from SNS activation (Bear et al., 2007). Be that as it may, the exact activation pattern (particularly at the hypothalamic level) of the HPA

axis depends on the stressor experienced, for example, low blood glucose will activate the medial hypothalamus (Herbert, 1999).

1.2.4.1 Regulation of the HPA Axis

In addition to the activation pathways of the stress response, the regulation of the HPA axis is also a noteworthy component of the stress response. The amygdalae, the hippocampus, and the frontal cortex are the primary modulators of the HPA axis (Figure 1.4), although other brain regions and structures can also exhibit control (Bear et al., 2007).

Memory and emotion processes are tied strongly to the amygdalae, which receive processed sensory input from cortical association areas, and direct input from the olfactory bulbs, frontal and temporal polysensory areas as well as the sensory thalamus (Bear et al., 2007). Thus, the amygdalae contribute substantially to the emotional and memorial context of stressors, and largely does so by providing positive feedback to the HPA axis (Herbert, 1999). In contrast, the hippocampus functions as a negative feedback mechanism and inhibits the HPA axis, and does so through its characteristically large quantity of glucocorticoid receptors. However, this characteristic quality can also lead to the development of negative effects, as high concentrations of glucocorticoids, in particular cortisol, can inhibit the growth and development of neurons and, most importantly, cause atrophy of the pyramidal neurons of the cornu ammonis 3 region (Magarinos and McEwen, 1995, Sapolsky, 1996). In the instance that such pyramidal cell destruction does occur, it is possible for a circular decline to develop, whereby experienced stress is worsened over time as the inhibitory effect attributed to the hippocampus is reduced.

Figure 1.4 – Regulation of the Hypothalamic pituitary adrenal axis by other brain structures

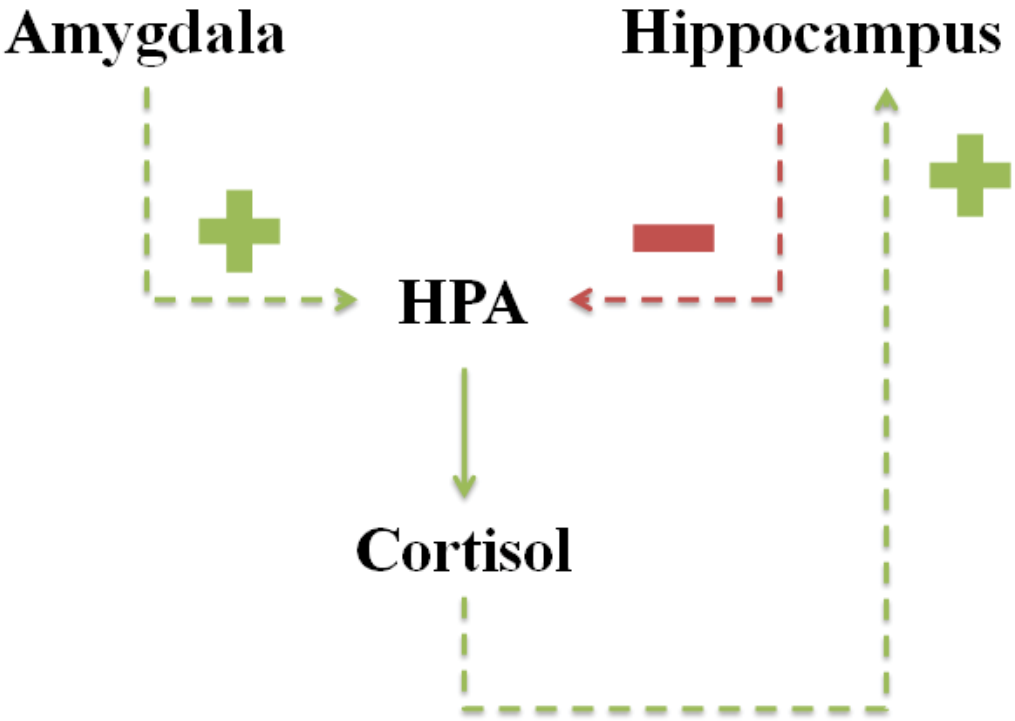

Figure 1.4 displays both the positive and negative feedback mechanisms of the hypothalamic pituitary adrenal axis. The amygdala provides processed sensory information to the hypothalamic pituitary adrenal axis to initiate the stress response, whilst, the hippocampus inhibits the HPA axis based on its interaction with serum cortisol concentration. Over time, excessive cortisol may impair hippocampal feedback (not shown).

Finally, the role of the frontal cortex in managing a large portion of the higher order functions of the brain, makes it a likely candidate as a centre for the neural processing of emotional responses associated to stress and thus may regulate the HPA axis and inform us of the consequences of both experiencing and managing stress (Herbert, 1999).

1.2.5 Coping with Stress

In their seminal work, Lazarus and Folkman (1984) broadly defined coping as '*the cognitive and behavioural efforts to manage internal or external demands that are taxing or exceeding the resources of the person*'. They also suggested that it was possible to separate coping strategies into two categories: (1) problem-focused (attempts to alter the stressor) and (2) emotion-focused (ways to accommodate the stressor), although the most common division is positive and negative, as determined by the impact on the individual.

Research indicates that positive coping mechanisms, for example, socialising, lead to increased job satisfaction (Arnetz, 2001) and reduced stress (Tattersall et al., 1999). Further, negative coping mechanisms, for example, alcohol consumption, are considered ineffectual (Tyler and Cushway, 1992, Koeske et al., 1993, Rout and Rout, 1994, Arnetz, 2001) and may lead to decreased job satisfaction as well as greater stress/anxiety (Tattersall et al., 1999, Arnetz, 2001).

Functioning in an inherently stressful profession, health professionals utilise a number of coping strategies to combat the stress that they experience. Research examining the coping strategies utilised has suggested that avoidance based strategies can predict higher levels of experienced stress (Firth-Cozens and Morrison, 1989, Tyler and Cushway, 1992) and chronic fatigue (Samaha et al., 2007). Conversely, problem focused coping strategies (Firth-Cozens and Morrison, 1989, Samaha et al., 2007), or those related to seeking social support (Tyler and Cushway, 1992, Moss and Paice, 1999) were more successful and predicted lower stress levels. However, measuring the success of coping strategies within health professionals is often difficult as there is often a reluctance to talk about any problems experienced (Arnetz, 2001).

1.2.6 Stress and Cognitive Performance

Early stress research conducted by Sapolsky et al. (1990) examined prolonged glucocorticoid exposure in the hippocampus because of its role in cognitive processes (Sapolsky, 2003). By implanting cortisol secreting pellets into the brains of adult male vervet monkeys these researchers were able to demonstrate soma shrinkage, darkening and dendritic atrophy of the cornu ammonis regions 2 and 3 following cortisol exposure (Sapolsky et al., 1990). Starkman et al. (1992) further established this relationship by utilising magnetic resonance imaging (MRI) to analyse the brains of 12 patients with Cushing's syndrome, and subsequently correlated this hormonal overexposure and subsequent physiological changes to memory dysfunction. A later imaging study from Gianaros et al. (2007) examined post-menopausal women and similarly concluded that chronic stress can cause hippocampal atrophy, as well as decreased volume in the right orbitofrontal prefrontal cortex, another brain region that supports cognitive processes (Lepage et al., 2000). Lupien and Lepage (2001), in their review, similarly indicate that the prefrontal cortex (amongst other areas) can be affected by stress and that structural changes within these regions may manifest as cognitive impairment.

Both acute and chronic distress may have a negative effect on an individual's performance (LeBlanc, 2009), and research has indicated that stress may broadly impair cognitive functions including cognitive flexibility, calculation, attention, visuospatial ability, executive functioning, and memory (Stokes and Raby, 1989, Kivimäki and Lusa, 1994, Mackenzie et al., 2007, Schwabe and Wolf, 2010, Henderson et al., 2012). Conversely, Duncko et al. (2007) used a virtual Morris water maze and cold pressor test to examine the effects of acute stress exposure on visuospatial learning and memory performance and found that their experimentally stressed group performed significantly better than their control counterparts. Additionally, Beste et al. (2013) also

demonstrated that stress can improve performance, specifically dual-tasking performance, and proposed this relationship was facilitated by an increase in processing efficiency and not cognitive flexibility. Moreover, Murray et al. (2010) found that acute stress caused neither an improvement nor an impairment in male pilots performance.

Focusing on specific cognitive domains, Kirschbaum et al. (1996) conducted two experiments using noun word lists to examine induced stress (via the Tier social stress test (Kirschbaum et al., 1993)), administered cortisone, and any memory related effects. Their results demonstrated an inverse relationship between cortisol levels and memorisation ability, suggesting that cortisol modulates declarative memory processes and concluding that stress can impair memory, a conclusion in line with more recent literature (Sandström et al., 2005, Tollenaar et al., 2008, Wolf, 2008, Comijs et al., 2010). de Quervain et al. (2000) also administered cortisone and examined memory recall to similar results; however, they suggested that only free recall is impaired by stress while the memory acquisition and consolidation was unaffected. Indeed, two studies from Domes et al. (2004) and Kuhlmann et al. (2005) also found that memory recall was impaired by stress, but indicated that this impairment only manifests when the individual is recalling emotional stimuli, be it positive or negative. In contrast, Lupien et al. (1999) found that working memory, or the capacity to store and manipulate information for brief periods of time (LeBlanc, 2009), rather than declarative memory was impaired when administering cortisone; a result support by work from Luethi et al. (2008), and an impairment which may lead to miscalculations or diagnostic errors and potentially subsequent fatalities.

Importantly, Lupien et al. (1999) also suggested that mild stress may improve working memory performance rather than causing impairment, providing evidence for the construct of eustress and an inverted U hypothesis (Yerkes and Dodson, 1908). Two

concepts that were more recently supported by an acute stress study from Human et al. (2013), however, their experiment specifically covered visuospatial memory. Similarly, a double blind placebo study from Buchanan and Lovallo (2001) administered hydrocortisone and found similar results indicating that elevated cortisol concentration during memory encoding enhanced long term recall performance of emotionally arousing pictures; a conclusion supported by Schwabe et al. (2008) who found that stress prior to learning could improve memory and recall. Cahill et al. (2003) provided further support and concluded that stress enhanced and improved memory encoding for emotionally arousing slides.

Henckens et al. (2009) examined memory encoding and retrieval using functional MRI, and suggested that acute stress drives a shift towards hyper-vigilant sensory processing and increases allocation of neural resources to noise reduction, which may account for improved memory performance when stressed. Most recently, Bos et al. (2014) used the social evaluative cold pressor test to determine that stress can modulate reconsolidation and hence lead to improvements in declarative memory. Interestingly, using a delayed recall test, Buchanan and Tranel (2008) found that elevated cortisol concentration was associated with reduced memory retrieval performance, whilst experience of stress without cortisol elevation enhanced retrieval of emotional stimuli, thus adding further ambiguity.

Moving to decision-making, early work from Keinan et al. (1987) demonstrated that experiencing distress led individuals to use non-systematic scanning strategies and not fully examine all alternatives, resulting in a greater rate of error. In the same year, a second study reaffirmed this result (Keinan, 1987) whereby stressed participants did not consider all available alternatives and employed non-systematic scanning patterns, resulting in poorer decision-making outcomes. In addition, the two experiments from

Cumming and Harris (2001) provided further evidence demonstrating stress induced decision-making impairment. Porcelli and Delgado (2009) also examined the relationship between acute stress and decision-making in students, and found that stress modulates risk taking which in turn leads to an increase in the making of disadvantageous choices (Starcke et al., 2008), especially when the associated reward was considered to be of significant value (Putman et al., 2009).

Preston et al. (2007) examined sex differences in the relationship between stress and decision making using the Iowa Gambling Task and anticipatory stress. Their investigation demonstrated that stress may influence the sexes differently, with men generally making less advantageous decisions and women to make more advantageous decisions; a conclusion that received support by research from Lighthall et al. (2009). However, van den Bos et al. (2009) expanded this sex based dimorphism, and suggested that while the two sexes may be differently sensitive to stress effects, women experience a biphasic effect, where moderate stress leads to the gain of advantageous behaviours and high levels of stress disrupt decision making processes. Finally, a number of reviews (Baumann and Bourbonnais, 1982, LeBlanc, 2009, Starcke and Brand, 2012) largely indicate that stress influences decision making and individuals will often resort to suboptimal decision making processes when faced with stressful situations, which, within the health professions, could be fatal.

Looking specifically at stress and cognitive performance in the health professions, two paramedic-based studies (LeBlanc et al., 2005, LeBlanc et al., 2012) used mobile patient simulators and demonstrated that functioning under high stress impairs clinical performance, regardless of previous experience. In an interview based study, Wetzel et al. (2006) identified that stress often negatively influences the judgement and decision-making ability of surgeons, rendering routine procedures more difficult. However,

another surgeon based study from LeBlanc et al. (2008) indicated the inverse, finding that acute stress may improve an individual's performance. Lastly, a recent nurse based study from Lees and Lal (2017) evaluated perceived stress and its relationship to cognitive performance and found no significant association between the two, introducing further ambiguity and indicating the requirement for more research

Summarily, current literature provides evidence indicating that stress can impair calculation ability, decision-making, working memory and recall, as well as overall performance (Kirschbaum et al., 1996, Lupien et al., 1999, Cumming and Harris, 2001, LeBlanc et al., 2005, Wetzel et al., 2006). However, it also provides evidence that stress may have no effect (Lees and Lal, 2017) or even enhance performance (Lupien et al., 1999, LeBlanc et al., 2008), and thus there is a requirement for additional research to clarify the relationship. Optimal cognitive functioning is vital to health professionals who are required to use higher order cognitive functions in order to diagnose, treat and manage illness and make emergency decisions; and hence without full cognitive functioning, are at risk of decreasing patient care quality and endangering lives. A summary of some of the aforementioned research studies examining stress and the impact it may have on cognitive performance, can be found in Table 1.1.

Table 1.1 – Summary of research studies examining stress, and the impacts it has on the brain and cognitive performance.

	Study	Methodology	Results
Hippocampal atrophy	Sapolsky et al., 1990	Surgically implanted secretory pellets into the brains of monkeys and examined 1 year later.	Hippocampal shrinkage, dark staining and cell damage in the CA2 and 3 regions.
	Starkman et al., 1992	Used MRI to examine hippocampal formation in Cushing's syndrome patients.	Increased cortisol levels were associated with reduced hippocampal formation volume
	Gianaros et al., 2007	Used voxel-based morphology to examine hippocampal grey matter	Higher stress levels predicted decreased grey matter volume in the right hippocampus.
Decision-making capability	Kienan et al., 1987	A computerised multiple choice analogies test, under threat of shock	Increased stress resulted in poorer decision-making outcomes
	Cumming & Harris, 2001	A same-different decision making exercise was administered after a low and high stress task.	Increased anxiety and stress associated with reduced decision making capability.
	leBlanc et al., 2005	Paramedics solved dosage calculations in both a low stress and high stress environment	Acute stress impairs the performance, working memory and memory recall
	Wetzal et a., 2006	Surgeons were interviewed about stress and its impact on their job.	Stress impairs individual judgement and decision making capability.
	leBlanc et al., 2008	Two surgical tasks were completed in a low and high stress situation.	Moderate stress levels are accompanied by improvements in technical performance.
Memory capability	Kirschbaum et al., 1996	Administered a stress task as well as cortisol prior to cognitive testing.	Stress impairs memory recall; memory acquisition & consolidation are unaffected.
	de Quervain et al., 2000	Administered cortisol alongside word recall and recognition tests	Stress impairs memory recall and retrieval.
	Kuhlmann et al., 2005	Memory was assessed after a stress condition and a control condition	Stress impaired memory recall; emotionally arousing material is especially sensitive.

Table 1.1 presents a number of research studies have examined stress and the impacts it can have on both the brain, as well as global and domain specific cognitive performance are shown. Stress has been shown to be associated with decreases in hippocampal volume, as well as impairment of decision-making and memory capability; however, some contrary evidence has suggested performance in these cognitive domains may be improved by stress.

Key: CA = Cornu ammonis; MRI = Magnetic Resonance Imaging

1.3 Anxiety

Anxiety is part of, and associated with the adaptive response, and hence it is often paired with stress. As a concept, Spielberger (1972) famously separated the experience of anxiety into two components; the first is related to the acute experience of apprehension and tension in a given situation, and is referred to as state anxiety. This is accompanied by and interacts with the second component, trait anxiety, or the characteristics of an individual that indicate tendency towards developing state anxiety. In simpler terms, anxiety can be thought of as a state of unpleasant consciously perceived feelings of apprehension and tension, that are associated with the activation of the stress pathways (Spielberger, 1972, Mosby, 2013) and that usually terminates when the causing situation has ended (Bear et al., 2007). However, anxiety can become pathological, and when the response is prolonged or excessive anxiety may become disruptive and interfere with an individual's ability to successfully cope (Steimer, 2002) and subsequently impact their life. Hence, anxiety can be considered an inappropriate and/or excessive expression of fear that most importantly may induce cognitive alterations that could have serious consequences in the health professions

The Australian National Mental health survey reported that approximately 14.4% of Australians aged 16 to 85 years indicated they suffered from an anxiety disorder in the 12 months prior to the survey (Australian Bureau of Statistics, 2007). Research has demonstrated that the health professionals are no exception (Cooper et al., 1989, Sutherland and Cooper, 1992, Rout and Rout, 1994) with up to 55% of all doctors experiencing anxiety (Caplan, 1994, Howie and Porter, 1999). Furthermore, women experience anxiety more commonly than men (Australian Bureau of Statistics, 2007); and again health professionals are no exception, with a number of studies demonstrating

that female health professionals indicate anxiety levels higher than their male counterparts (Cooper et al., 1989, Sutherland and Cooper, 1992, Rout and Rout, 1994), although underreporting by men may also be a factor.

1.3.1 Physiological basis of Anxiety

As mentioned, anxiety is an inappropriate or inordinate activation of the fear response, and often associated with the stressful experiences. As such the physiological basis of anxiety is associated with the autonomic changes governed by the sympathomedullary pathway (e.g. hypertension, tachycardia, and sweating; Figure 1.2) and a similar increase in activation of the neuroendocrine HPA axis (Figure 1.3) and resultant production of stress hormones including cortisol (Andreassi, 2006, Bear et al., 2007). Furthermore, it is important to note the modulatory role of the amygdala on the HPA axis (Figure 1.4), as the amygdala is important in fear and emotion and is involved in the initiation of both the SNS and the HPA axis. Indeed, it has been suggested that hyper-function of the amygdala, as well as diminished activity and/or damage to hippocampal cells, are key components of anxiety disorders and their development (Bear et al., 2007, Shin and Liberzon, 2010).

1.3.2 Anxiety and Cognitive Performance

Following early research that had suggested anxiety impairs the performance of difficult tasks, Eysenck and Calvo (1992) proposed their Processing efficiency theory. In their model, they made a crucial distinction between performance effectiveness (the quality of performance) and processing efficiency (performance effectiveness over time) and suggested that anxiety impairs efficiency more than effectiveness. Moreover, this impairment is dependent upon the availability and utilisation of additional resources for

concurrent tasks, the demands of the task on working memory resources (i.e. storage and processing capacity), the efforts of the individual, and the utilisation of other cognitive processes. More recently, Eysenck et al. (2007) expanded upon the Processing Efficiency theory and proposed the Attentional-Control theory, which is more precise regarding the effects of anxiety on the functioning of the central executive, in particular attentional control. This newer theory suggests that anxiety disrupts the balance of top-down and bottom-up attentional control processes, which in turn impairs processing efficiency (i.e. inhibition and shifting functions), and in turn subsequently impairs cognitive performance. In a two year follow-up, Derakshan and Eysenck (2009) again suggested that anxiety is associated with a wide allocation of attentional resources, which reduces focus and overall performance.

Concerning the demonstrable impact of anxiety, research has associated the experience of anxiety with communicative changes in the brain. Indeed, Bishop (2009) found that individuals with high trait anxiety demonstrated decreased prefrontal activity and slower target identification in response to a competing demand, suggesting that anxiety impaired control mechanisms related to the inhibition of distraction processing. Moreover, Andreescu et al. (2014) examined resting functional state connectivity patterns in the default mode network, and associated anxiety with decreased connectivity in the ventrolateral pre-frontal cortex, and worry severity with ventromedial pre-frontal cortex connectivity. In terms of performance, they suggest that this connectivity reduction would decrease the flexibility of an individual to cognitively modifiy and hence would reduce performance.

Moreover, a number of studies have investigated and compared the cognitive performance of groups with diagnosed anxiety disorders e.g. panic disorder (PD) to that of healthy controls. With respect to PD, it has been demonstrated that visual memory

function (Lucas et al., 1991), verbal learning and memory, as well as short-delay free recall (Asmundson and Stein, 1994) were impaired when compared to healthy controls. Asmundson and Stein (1994) also suggested that the observed diminished performance may be a non-specific correlate of anxiety, and that the disorder type was irrelevant. In contrast, Airaksinen et al. (2005) examined a wider group of anxiety disorders, and found impairments in episodic memory and executive functioning. However, when stratified by disorder, only PD, obsessive compulsive disorder (OCD) and social phobia were implicated, suggesting that the relationship between anxiety and performance may be modified by disorder type. That said, later work from Mantella et al. (2007) implicated generalised anxiety disorder (GAD) and reported impaired short-term and delayed memory functions when compared to healthy controls.

A series of studies from Savage et al., (1996, 1999, 2000) examined OCD and cognitive performance, and came to the consensus that both delayed and free recall are impaired in comparison to healthy controls. Furthermore, Savage et al. (2000) also suggested that strategic organisation is altered in OCD, a result that supported an early result from Veale et al. (1996) and may be associated with frontostriatal dysfunction. Similarly, both Purcell et al. (1998) and Boldrini et al. (2005) reported a number of cognitive deficits in OCD, including impaired spatial working memory and construction, recognition, learning, and motor initiation and execution. Furthermore, Purcell et al. (1998) also indicated that patients with PD differed on some test scores when compared to healthy controls. However, they provided no comment on the significance of these results, unlike Boldrini et al. (2005) who reported impaired spatial memory in PD, and furthered the suggestion of Asmundson and Stein (1994) in that the observed impairments were not condition specific and applied generally to the experience of anxiety. Furthermore, with respect to PD, Lautenbacher et al. (2002) reported an

increased response time on divided attention tasks, suggesting that anxiety leads to the detection and processing of irrelevant information and hence interferes with performance.

Likewise, a number of research studies have investigated the impact of anxiety on cognitive performance by examining purely healthy sample groups. Two early experiments from Darke (1988), investigating the comparative working memory capacities of high and low anxiety subjects, found that highly anxious individuals had reduced working memory storage and processing capacity. Moreover, working memory deficits and impaired cognitive performance have been associated with the experience of math anxiety (Ashcraft, 2002); a relationship that subsequently leads to disruptions in problem solving ability and other executive functions. Kellogg et al. (1999) found that a highly anxious group performed worse, and possessed a higher error rate, on an arithmetic task than their less anxious counterparts. Ashcraft and Kirk (2001) concluded similarly, indicating that anxiety disrupts task-relevant activities associated with working memory, and hence slows and degrades performance.

Moreover, Cumming and Harris (2001) conducted two experiments examining decision-making capability of senior radiography students and the impact of anxiety, finding that higher anxiety was associated with less accurate decision-making on the primary task, but a secondary task was unaffected; a result that aligns with the theory of anxiety facilitating attending to multiple tasks/threats. Similarly, in their examination of college students, Sadeh and Bredemeier (2011) found high anxiety was associated with significant distractor interference, an over processing of irrelevant distractors and predicted task performance, but interestingly was not associated with error rate. Furthermore, two recent studies from Nieuwenhuys et al, (2012, 2015) investigated the effect of anxiety on police officer shooting accuracy and behaviour. In both instances, it

was found that shooting accuracy, as well as response and performance time was significantly reduced under high anxiety. Similarly, Renden et al. (2015) negatively correlated anxiety to performance effectiveness in police officers, and demonstrated that anxiety is associated with inefficient and less effective performance. Most recently, Levita et al. (2016) examined the performance of clinical therapists and anxiety, and found that more anxious clinicians were more likely to focus on the less challenging aspects of delivering cognitive behavioural therapy rather than delivering the full evidence based form, hence delivering inferior care and performing worse.

In contrast, Robinson et al. (2013) demonstrated that induced anxiety and the threat of shock actually reduced participant errors on only no-go tasks, and had no significant impact on go tasks or an individual's overall response time; and attributed this result to promotion of response inhibition. A similar earlier study from Hu et al. (2012) used the threat of bodily harm to examine anxiety and attentional narrowing and suggested that anxiety can lead to a reduction in task irrelevant processing and a subsequent increase in performance, however, high levels of anxiety should generally lead to impairments.

Lastly, it must be mentioned that some research studies have reported that there is no link between the experience of anxiety and either improvement or impairment of cognitive performance. Avila and Parcet (1997) used a Posner paradigm task coupled with a response time measure to investigate negative priming as a function of anxiety and found that task performance did not differ significantly between their anxious and non-anxious group. Shortly thereafter, Gladsjo et al. (1998) conducted a more complex examination using a comprehensive neuropsychological battery, and found that there were no significant differences between patients with panic disorder and health controls in any of the investigated domains including visuospatial functioning, learning, memory attention and psychomotor speed performance. Inside the health professionals, LeBlanc

and Bandiera (2007) examined the impact of subjective anxiety on the performance of emergency medicine residents. Whilst anxiety scores were correlated to the test condition (high vs low stress), they were not significantly correlated with performance scores, further suggesting that other factors including experience may modulate the relationship of anxiety and performance.

Summarily, the majority of literature indicates that anxiety will impair an individual's cognitive performance, in particular, their attention, working memory and executive functions (Ashcraft, 2002, Lautenbacher et al., 2002, Airaksinen et al., 2005). Observations founded on the notion that an individual has a limited pool of working memory resources that are available when completing a task (Baddeley, 1992) and, in anxious individuals, their worries and symptoms of anxiety reduce the available working memory resources, thereby decreasing performance efficiency and efficacy (Eysenck and Calvo, 1992). However, some evidence demonstrating either no effect associated with anxiety (Gladsjo et al., 1998) or actual performance improvements (Hu et al., 2012) has also been reported. A summary of some of the aforementioned research studies that examined the impact anxiety may have on cognitive performance, can be found in Table 1.2

Table 1.2 – Summary of research studies that examined the impact of anxiety on cognitive performance.

Study	Methodology	Results
Gladsjo et al., 1998	Use a comprehensive neuropsychological battery to examine cognitive differences between PD and HC	Found no differences in learning, memory, attention, visuospatial functioning and psychomotor speed.
Savage et al., 2000	Administered the Ray-Osterrieth Complex Figure test and California Verbal learning test to compare memory performance between OCD and HC	Verbal and non-verbal measures of free-recall and organisational strategy were impaired in OCD
Ashcraft & Kirk, 2001	Examine the influence of math anxiety on cognition, using exam tasks of varying difficulty	Math anxiety affects performance by causing a transitory disruption to working memory
Cumming & Harris, 2001	Used a simple dual-task paradigm to examine the diagnostic decision-making capabilities of senior radiology students	Anxiety was associated with reduced performance outcomes on the primary task, but not the secondary task
Lautenbacher et al., 2002	Used selective and divided attention tasks to examine attentional functionality in PD	PD displayed an increase in response time on divided attention tasks only; selective attention was not affected
Boldrini et al., 2005	Used a comprehensive neuropsychological battery to compare cognitive function in OCD and PDA to HC	Impaired fluency, visual-spatial construction, learning and memory were found in OCD; further PD also showed impaired spatial learning
Hu et al., 2012	Used the Stroop colour word test and skin conductance responses to examine the "impairing effects" of anxiety	Experiencing anxiety lead to a reduced distractor effect, and greater task performance
Robinson et al., 2013	Used the Sustained attention to response task, and threat of electrical shock to examine the impact of anxiety	Higher anxiety was associated with a decrease in error rate on no-go tasks; go tasks were not implicated
Nieuwenhuys et al., 2015	Examined the effect of threat and training under treat in a shooting task undertaken by police officers	Anxiety lead to an increase in false positives; a result that persisted following practice
Levita et al., 2016	Used cognitive, behavioural, and physiological measures to examine the impact of anxiety on clinical therapist performance	Increased anxiety was associated with use of less evidence based techniques and a focus on less challenging aspects of CBT

Table 1.2 presents a number of research studies that have examined anxiety and the impacts that it can have on cognitive performance are listed. Anxiety has been shown to be associated with impairment of working memory and executive functioning; however some contrary evidence has suggested that anxiety may improve performance in these cognitive domains.

Key: CBT = Cognitive behavioural therapy; HC = Healthy controls; OCD = Obsessive-compulsive disorder; PD = Panic disorder; PDA = Panic disorder with agoraphobia

1.4 Cognition

Cognition describes the intellectual process by which an individual attends to, perceives and comprehends objects, ideas, situations, and other environment cues (Mosby, 2013). Cognition as a concept is characterised by the utilisation of higher order cognitive functions such as reasoning, visual and language processing, spatial construction, memory and judgement (Müller and Mayes, 2001, Harris et al., 2009, Mosby, 2013).

1.4.1 Cognitive Performance

Research has shown that experiencing stress and/or anxiety can lead to both global and domain specific cognitive impairments (Sections 1.2.6 and 1.3.2, respectively). Cognitive impairment is a broad description for a reduced performance in one or more cognitive domains, e.g. memory and decision making, however, there is currently no finite all-encompassing definition (Scazufca et al., 2009) and, more often than not, is defined as needed. Furthermore, it is worth noting that cognitive impairment can be related to a number of factors including the passage of time on task, aging, fatigue and/or sleepiness and prolonged applied effort (Lal and Craig, 2001, Lal and Craig, 2002, DeLuca, 2005, Trejo et al., 2005), or particular pathologies including stroke, diabetes and dementias (Jin et al., 2006, Porth, 2006). Lastly, aging is associated with a natural cognitive decline, however more severe cognitive impairments are classified as pathological (DeCarli, 2003).

1.4.2 Measurements of Cognitive Performance

Research has utilised a variety of techniques in the assessment of cognitive performance, from observational approaches, to psychometric questionnaires like the Mini-mental State Exam (Folstein et al., 1975) and Cognistat (Mueller et al., 2007).

Furthermore, specifically designed tasks such as the Digit Span task of the Wechsler Adult Intelligence scale (Wechsler, 2008), the Posner Task (Posner, 1980), and various motor tasks have also been extensively utilised. More recently, physiological measurements like electroencephalography (EEG) (Klimesch et al., 1998, Lees and Lal, 2017), event related potentials (ERP) (Hillyard and Anllo-Vento, 1998), and heart rate variability (Hansen et al., 2003, Giblin et al., 2013) have been increasingly utilised due to their inherently quantitative and dynamic nature.

1.4.2.1 Psychometric tools for assessment of cognition

Psychometric tools such as the Mini-Mental State Exam (Folstein et al., 1975), the Montreal Cognistat Assessment (Nasreddine et al., 2005) and the Cognistat (Kiernan et al., 1987) typically quantitatively assess cognitive performance by requiring either a response to an asked question, or a delivered command. Whilst the manner in which cognitive performance is assessed, and the exact components of cognitive performance assessed differs between individual tools, they all generally assess an individual's global and/or domain specific cognitive performance. However, the very design of such tools often limits their utilisation to retrospective examinations only, and as a result, their applicability in the examination of transitory cognitive impairments and the decline into cognitively impaired states is limited.

1.4.2.2 Electroencephalography

Electroencephalography is defined as the detection and recording of the electrical activity of the cortical pyramidal neurons, through electrodes placed upon the scalp (Jasper, 1958, Harris et al., 2009); and as mentioned, its inherently quantitative and

dynamic nature provides the potential to function as a biomarker of cognitive performance.

Traditionally, recorded brain activity is transformed from the time domain into the frequency domain and subsequently separated based on frequency of the wave received (Klimesch, 1999, Niedermeyer and Lopes da Silva, 1999, Schomer and Lopes da Silva, 2010). In frequency analysis, the waveforms found in the lower end of the frequency spectrum (i.e. delta and theta), are typically associated with the various stages of sleep (Harris et al., 2009), and alpha waves are associated with waking relaxation and is considered to be the dominant frequency within humans (Klimesch, 1999). At the upper end of the frequency spectrum, beta waves are associated with alert states and some cognitive processes (Niedermeyer and Lopes da Silva, 1999, Harris et al., 2009). Last of all is the gamma waveform, which is a series of synchronous oscillations that reside in the higher frequencies between 30 and 100 Hz, although the upper frequency limit of these oscillations can be unlimited (Stam et al., 2003, Schomer and Lopes da Silva, 2010). The gamma band has been shown to reflect cognitive processes, primarily memory, however it is also likely fundamental for other cognitive processes including object representation (Mueller et al., 2007). Further, gamma wave abnormalities have been associated with neurological conditions and cognitive impairments (Niedermeyer and Lopes da Silva, 1999, Harris et al., 2009). However, additional research is required to establish the true role of gamma activity in cognitive performance.

As mentioned, conventional EEG analysis largely relies transforming the raw time domain signal prior to analysis. Nevertheless, the time domain can prove insightful; most famously via the three Hjorth Parameters: Activity, Complexity, and Mobility (Hjorth, 1970). The Activity parameter can be referred to as the variance or mean power of an EEG trace; Mobility can be conceived as the mean frequency of an EEG trace, and

finally, Complexity measures the nature of an EEG trace in reference to a sine curve (Hjorth, 1970). These three descriptive parameters are entirely based on time, resistant to the Fourier transform and are used to quantitatively describe the general characteristics of an EEG trace (Hjorth, 1970) and can be utilised to examine or represent behavioural states including cognitive performance (Hjorth, 1973).

1.4.3 Electroencephalographic manifestations of cognitive impairment

As previously discussed, EEG activity has been linked to cognitive processes, e.g. beta and gamma activity for higher order functions, and this association has seen EEG frequently utilised as a research tool in the investigation of cognitive impairment. However, the only current avenue that allows the EEG manifestations of cognitive impairment to be examined is to examine and extrapolate from the results of research that focuses on EEG measurement within diagnosed cognitive impairments, e.g. Dementia, as research in healthy samples and the health professions is limited. Despite this limitation, some literature suggests that power decreases in high frequency waves (alpha, beta and gamma) and increases in low frequency waves (delta and theta) may be associated with cognitive impairment.

Delving into this body of literature, in terms of broad EEG spectral changes, in their longitudinal examination of mild cognitive impairment (MCI), Huang et al. (2000) found that at baseline patients with MCI and patients with Alzheimer's Disease (AD) had relative power increases in the delta and theta frequency bands, as well as a decrease in alpha relative power. Furthermore, patients with AD also possessed decreased beta relative power when compared to controls. Interestingly, a logistic regression model indicated that alpha and theta band changes were best at predicting AD. Moreover, an EEG coherence study from Brunovsky et al. (2003) also related

increased alpha and theta activity to reduced cognitive performance, and additionally, lower beta band activity was similarly implicated. In trialling global field synchronisation in the diagnosis and prediction of cognitive impairment, Koenig et al. (2005) demonstrated that cognitive impairment was reflected by decreases in alpha, beta and gamma power as well as an increase in delta power. Reductions in alpha, beta and gamma power were also found by Pachou et al. (2008) who examined working memory in schizophrenia using spectral EEG, however these findings were for healthy controls, and the relationships were inverted for patients with schizophrenia who presented increases in EEG activity. Pachou et al. (2008) suggested that the observed increase resulted from the greater effort required by patients with schizophrenia to complete the working memory task, and, as such, the increase in activity is more representative of cognitive impairments, than the activity reductions observed in healthy controls. More recently, Aurtenetxe et al. (2013) reported increased frontotemporal and parietal induced theta activity in patients with MCI, and additionally found alpha and gamma power, as well as beta band desynchronisation were reduced. In the following year, Dimpfel (2014) also reported an increase in fronto-temporal theta activity in individuals with MCI when compared to controls, and also similarly implicated delta activity. Most recently, Lees et al. (2016) suggested that reduced global cognitive performance was associated with reduced power in the beta frequency band. Furthermore, it was found that a reduction in delta band oscillation, increases in beta activity and decreases in gamma activity were associated with calculation, memory and judgement performance.

Focusing specifically on the delta frequency, a number of studies from Babiloni and collaborators (Babiloni et al., 2006a, Babiloni et al., 2006b, Babiloni et al., 2008, Babiloni et al., 2010) comparing resting EEG activity of healthy individuals to patients with MCI and patients with AD, have demonstrated that increased delta power

(particularly of the frontal leads) is associated with cognitive impairment. The research from Rossini et al. (2006) and Koenig et al. (2005) also indicate that cognitive impairment is associated with and may be predicted by an increase in delta EEG power. In a review paper from Rossini et al. (2007), they similarly indicate that increased delta power is associated with cognitive impairment. However, Liddell et al. (2007) provides contrary evidence, suggesting that patients with MCI will demonstrate a decrease in delta power but patients with AD will show an increase in delta power. A result that suggests the degree of impairment or the physiological changes in the brain may account for the observed EEG changes, which further obscures the relationship between delta activity and cognitive impairment.

Moving up in the frequency bands, the theta frequency, and any associated changes, are of particular importance, as it has been previous associated with a number of cognitive processes including memory and cognitive control (Klimesch, 1999). Research studies from Trejo et al. (2005) and Koenig et al. (2005) suggest that an increase in theta activity is associated with cognitive impairment and dementia states like AD, a result that was also demonstrated by van der Hiele et al. (2007) who compared EEG measures, during memory activation, between healthy individuals, patients with MCI, and patients with AD. In contrast, a similar comparative study from Cummins et al. (2008) suggested that MCI is associated with decreased theta power, a result similar to that of Missonnier et al. (2006) who found decreased theta event-related synchronisation in progressive MCI. The differing results of these two studies may be associated with the lack of a highly impaired group, however it does indicate that further research examining theta band changes in cognitive performance is required.

In the alpha frequency band, Babiloni et al. (2006b) demonstrated that decreased alpha power was correlated with cognitive performance when patients with MCI or AD were

compared to normal individuals. This result aligns with previously published research (Klimesch, 1999, Tang-Wai et al., 2003, Babiloni et al., 2006b) and has received support from a number of more recent studies that also correlated reduced alpha power with cognitive impairment (Babiloni et al., 2008, Jackson and Snyder, 2008, Zadikoff et al., 2008, Babiloni et al., 2010). An interesting caveat to this result is provided by earlier research from Pijnenburg et al. (2004) who determined that only decreased activity in the upper alpha band was correlated to changes in cognitive performance, and that lower alpha activity was not. Moreover, Babiloni et al. (2006b) also demonstrated this change in EEG power in cognitively impaired patients distinguished them from normal individuals. Furthermore, Luckhaus et al. (2008) localised this reduced alpha activity to the posterior leads, providing further detail to this relationship. In contrast, Trejo et al. (2005) recorded multichannel EEG spectra during a continuous mental arithmetic task and Babiloni et al. (2006b) longitudinally examined quantitative EEG in patients with MCI and patients with AD, and both suggested that alpha power may actually increase in cognitive impairment, providing ambiguity to the research and indicating that further investigation is required.

In the beta frequency band, Stam et al. (2003) examined participants with known memory impairments and suggested that a reduction in beta power, or loss of beta synchronisation was correlated with cognitively impaired states. Similarly, Missonnier et al. (2007) and Pijnenburg et al. (2004) also noted declines in beta synchronisation in patients with AD and patients with progressive MCI, particularly over the parietal leads. More recently, Güntekin et al. (2013) noted decreased beta rhythm variations in MCI when compared to healthy controls, although these were specifically event-related changes and not general declines. A review from Jackson and Snyder (2008) also suggests that a decline in beta frequency power can be associated with cognitive

impairment. In contrast, Lee et al. (2010) positively correlated beta synchronisation to MMSE score in healthy individuals, yet found decreased beta synchronisation in patients with AD, possibly indicating the existence of varied EEG profiles between differing states of cognitive impairment.

Last of all is the gamma frequency band, which, as previously mentioned, has been associated with various cognitive functions (Mueller et al., 2007, Tallon-Baudry, 2009). Early work from Tallon-Baudry et al. (1998) found frontal and occipito-temporal enhancement of gamma band activity whilst completing a memory task and, similar gamma band enhancement was found by both Cho et al. (2006) and Lee et al. (2010). However, with respect to the latter, this result only held for healthy individuals and was inversed for patients with AD. This result directly opposed previous research from van Deursen et al. (2008) who found increase gamma band power (particularly over the parietal, parieto-temporal and occipital leads) in patients with AD when comparing them to both patients with MCI and healthy controls. The work from Moretti et al. (2009) contrasts with these aforementioned studies, whereby examining the EEG recordings and MRI scans of patients with MCI linked declines in gamma power with cognitive impairment. Most recently, Başar et al. (2016) examined event-related EEG oscillations on an oddball paradigm and found that early event-related gamma oscillations were delayed in patients with AD, although the average amplitude of the oscillation itself was greater, suggesting that whilst there is a power enhancement, the timing of the changes may also provide valuable information.

Summarily, cognitive impairments can manifest in a number of ways that are normally detected using various psychometric assessment tools. However, various changes in electroencephalography variables including frequency band power changes and oscillations, event related de/synchronisations, and inter-electrode/trial coherence may

also be representative or indicative of cognitive impairments. Despite this, research examining cognitive impairment and EEG is confined to groups with diagnosed cognitive impairments e.g. Alzheimer's disease, and the examination of healthy and health professional sample groups is limited and is required.

A brief summary of the major findings from some of the aforementioned research that examined the electroencephalographic changes associated with cognitive impairment can be found in Table 1.3.

Table 1.3 – Summary of a number of research studies examining the spectral electroencephalographic manifestations of cognitive impairment.

Study	Sample group information	Delta	Theta	Alpha	Beta	Gamma
Klimesch, 1999	Review Article	↑	-	↓	↓	↓
Stam et al., 2003	20 SMI; 17 MCI; 10 AD	-	-	-	↓	-
Koenig et al., 2005	46 HC; 70 SMI; 92 MCI, 134 AD	↑	-	↓	↓	↓
Trejo et al., 2005	16 HC	-	↑	↑	-	-
Rossini et al., 2006	69 MCI; 24 converted AD	↑	↑	↑	-	-
Van der Hiele et al., 2007	22 HC; 18 MCI; 16 AD	-	↑	↓	-	-
Cummins et al., 2008	12 HC; 12 aMCI	-	↓	-	-	-
Jackson & Snyder, 2008	Review article	↑/↓	-	↓	↓	-
Moretti et al., 2009	79 MCI	-	↑	-	-	↓
Tallon-Baudry., 2009	Review article	-	-	-	-	↑/↓
Babiloni et al., 2010	79 HC; 53 SMI; 51 naMCI, 92 aMCI	↑	↑	-	-	-
Lees et al., 2016	58 N	↑	-	-	↑	↓

Table 1.3 presents some of the aforementioned research studies that have examined the spectral EEG changes that can be attributed to various stages of cognitive impairment are shown. The majority of studies report that cognitive impairment is associated with a decrease in activity in the alpha, beta, and gamma frequency bands, and an increase in the power of the delta and theta frequency bands. However, some contrary evidence does suggest the opposite.

Key: AD = Alzheimer's Disease; aMCI = amnesic Mild cognitive impairment; HC = Healthy Controls; MCI = Mild Cognitive Impairment; N = nurses; naMCI = non-amneisc Mild Cognitive Impairment; SMI = Subject Memory Impairment; ↑ = Increase; ↓ = Decrease; - = No change reported

1.5 Basis for Research

Research examining stress and its effects on humans has indicated that exposure to stress hormones can cause neuronal damage and/or death in the hippocampus and prefrontal cortex (Starkman et al., 1992, Sapolsky, 1996, Sapolsky, 2003, Conrad, 2006). These are important results, as the implicated brain regions are involved in the regulation of the stress response, as well as, memory and a number of other cognitive processes (Lepage et al., 2000, Lupien and Lepage, 2001). Additionally, research has indicated that both acute and chronic stress can have a detrimental effect on an individual's overall performance (Cumming and Harris, 2001, LeBlanc et al., 2005). More specifically, it may affect their visuospatial ability, attention, executive functioning, cognitive flexibility, calculation, decision-making and memory capabilities (Stokes and Raby, 1989, Kivimäki and Lusa, 1994, de Quervain et al., 2000, Wetzel et al., 2006, Mackenzie et al., 2007, Schwabe and Wolf, 2010, Henderson et al., 2012). Finally, research has shown the health professions to be inherently stressful and that this stress can be localised and attributed to multiple different origins (Firth-Cozens, 2003, Thomas, 2004, Sveinsdóttir et al., 2006, Golubic et al., 2009). This inherent stress exposure and associated declines in cognitive performance have been associated to poorer quality patient care (Tarnow-Mordi et al., 2000, Sveinsdóttir et al., 2006, Berland et al., 2008), and in some instances may account for 57% of adverse medical events (Wilson et al., 1999).

Similarly, research has demonstrated that experiencing anxiety is associated with decreased prefrontal activity (Bishop, 2009) and functional connectivity changes in the ventrolateral and ventromedial prefrontal cortex (Andreescu et al., 2014), and such structural changes may manifest as cognitive impairment (Lupien and Lepage, 2001). Moreover, research has associated anxiety with impairments in working memory

(Asmundson and Stein, 1994, Savage et al., 2000, Ashcraft, 2002), attention (Lautenbacher et al., 2002), decision-making (Cumming and Harris, 2001) and various executive functions (Airaksinen et al., 2005, Nieuwenhuys et al., 2015), and largely claimed these impairments are a result of attentional processing deficits (Eysenck et al., 2007). In the nursing profession, these impairments have the real possibility to impact the performance of nurses, their quality of care, and place patients at risk. However, despite research demonstrating that the health professions are inherently stressful, and that both stress and anxiety have the ability to reduce an individual's cognitive performance, which can lead to subsequent declines in quality of patient care, little to no research investigating the impact of stress and anxiety on the cognitive performance of health professionals has been conducted (LeBlanc, 2009). As such, the present study was conducted to explore these empty niches; and to further the understanding of the relationships between stress, anxiety and cognitive performance (Figure 1.5).

Figure 1.5 – Diagrammatic representation of the relationships between concepts that were investigated in the current research.

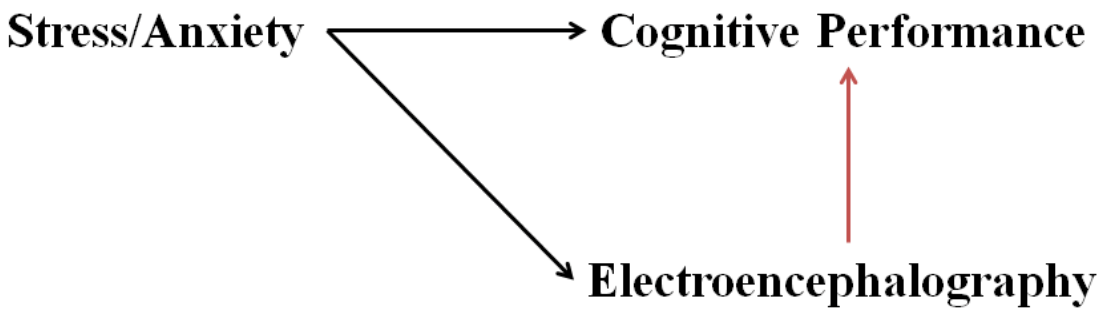

Figure 1.5 presents a diagrammatic representation of the relationships that were investigated by the current research project. The analysis will include: the examination of how stress affects cognitive performance (as measured by psychometric assessment and electroencephalography), as well as how electroencephalography is related to psychometric cognitive performance.

1.6 Aims

The overall aim of this research was to examine the neurocognitive impact of stress, and anxiety on the cognitive performance of nurses and their non-health professional counterparts.

More specifically, the aims of the current study were to investigate the associations between:

1. Stress, and anxiety, and cognitive performance in nurses and non-health professionals.
2. Electroencephalographic variables and global and domain specific cognitive performance in nurses and non-health professionals.

1.7 Hypotheses

The hypotheses for the present study were as follows:

1. Stress and anxiety will be associated with global and/or domain specific cognitive performance in both nurses and non-health professionals.
2. The associations between stress and anxiety, and global and/or domain specific cognitive performance will vary between nurses and non-health professionals.
3. Electroencephalographic variables will be associated with both global and domain specific cognitive performance; with the higher frequency bands being the most likely implicated.

1.8 Significance of Research

The intent of the present project is to provide research based groundwork that may enable further research to inform policy makers and authorities on the effects of stress and anxiety on cognitive performance of nurses. It is possible that by examining the present results and mitigating any cognitive impairment we could improve the performance of our health professionals, as well as understand and manage their stress and hence enhance quality of patient care and, most importantly, reduce the incidence and severity of adverse medical events and patient fatalities.

Chapter 2 – General Methodology

The following chapter provides a broad description of the general methodology that was utilised in the current project. Any experiment specific alterations from this methodology are contained within the shorter methodology sections found in Chapters 3 and 4, respectively.

2.1 Participant Recruitment

Two independent sample groups were utilised in the experiments of this research project. The first sample group was composed of healthy non-health professional individuals, and the second sample group consisted of healthy clinically active nurses.

Participants aged between 18 and 69 years were recruited into the study using a number of advertisements (refer to Appendix 1) placed both physically at various locations within the local community and online. Additionally, both the Faculty of Health of the University of Technology, Sydney and the New South Wales Nurses and Midwives Association further enabled and facilitated recruitment of nurse participants.

Additionally, each participant was asked if they were willing to distribute the experimental advertisements (refer to Appendix 1) to any other individual who may have been interested in participating.

2.2 Ethics approval

This study had UTS Human Research Ethics Committee (HREC) approval (HREC: 2014000110).

An ethical requirement of the current project necessitated all recruited individuals be provided with a detailed explanation regarding the intent of the study, its protocol, and a disclosure stating that individual results would remain confidential and anonymous. Further, during this process the recruited individual was afforded the opportunity to ask any questions they may have had. If the recruited individual agreed to participate in the study, the study consent form (refer to Appendix 2) was read and signed by both participant and researcher, with each retaining a copy.

2.3 Participant Selection Criteria

2.3.1 General requirements

All participants were required to have no ongoing health issues or diseases that would have a known effect upon their cognitive performance. This was determined prior to the experimental session, either via email correspondence and/or immediately before the study. Any participants indicating, they had a chronic illness were excluded from further participation in the study. Furthermore, participants were also required to have consumed less than 16 standard alcoholic drinks, not smoked more than 10 cigarettes or have regularly used any drugs (prescription or illicit) that have known cognitive effects, as these substances may affect the results (Weissenborn and Duka, 2000, Hahn et al., 2009). Participants that did not meet these requirements, as determined by questions 1, 2, 7, 8, and 18 of part 1 of the LAQ (Craig et al., 1996), were excluded from further inclusion in the study.

Additionally, at the commencement of the experimental protocol, three left arm brachial blood pressure measurements were taken for each participant. A requirement of the ethics protocol saw participants that presented with average systolic and/or diastolic blood pressure greater than or equal to 160 mmHg and/or 100 mmHg, respectively,

excluded from further participation and advised to seek medical attention. These blood pressure values are indicative of moderate hypertension which is associated with an increased risk of cardiovascular events (National Heart Foundation of Australia, 2016). Further, participants with average blood pressure values greater than 140/90 mmHg, but less than or equal to 159/99 mmHg, were able to participate, however, they were advised to see a medical professional regarding their blood pressure (per the UTS Human Research Ethics emergency protocol (refer to Appendix 3)). Participants with blood pressure values less than or equal to 139/89 mmHg were included in the study. Table 2.1 displays the inclusion and exclusion criteria for blood pressure relevant to this study.

Table 2.1 – Blood pressure inclusion and exclusion thresholds

BP	BP Classification	Participant
< 120/80 mmHg	Optimal	Included
120-129 and/or 80-84 mmHg	Normal	Included
130-139 and/or 85-89 mmHG	High Normal	
140-159 and/or 90-99 mmHg	Grade 1 (Mild) Hypertension	Included and advised to seek medical care
160-179 and/or 100-109 mmHg	Grade 2 (Moderate) Hypertension	Excluded and offered to be escorted to medical care
≥ 180 and/or 110 mmHg	Grade 3 (Severe) Hypertension	Excluded and offered to be escorted to medical care

Table 2.1 presents the values indicative of various stages of blood pressure including normal and hypertension (National Heart Foundation of Australia, 2016). Participants with BP values above 160/100 mmHg were excluded from the study and offered to be escorted to medical care. Any participant who presented with BP less than or equal to 159/99 mmHg were included in the study, however those above 140/90 mmHg were advised to consult a medical professional.

Key: BP = Blood pressure; mmHg = millimetres mercury; > = Greater than; < = Less than; ≥ = Greater than or equal to; ≤ = Less than or equal to

2.3.2 Requirements for Nurses

Each nurse participant was required to fulfil two additional criteria to be included in the experiment. Firstly, they were required to hold a current nursing qualification (e.g. enrolled nurse (EN), and registered nurse (RN)) and to be able to provide evidence of this. The second criterion was that they be currently clinically active and working in healthcare in some manner. Participants were included regardless of the role in which each individual was employed and were working (e.g. surgical theatre, general ward, or home care) or their employment status (i.e. casual, part-time or full-time).

2.4 Experimental Protocol

Prior to the scheduled experimental session, each participant was asked to refrain from the consumption of caffeine and nicotine for 4 hours, and alcohol for 12 hours. These restrictions were implemented as these substances may influence the physiological data recorded in the study (Chick et al., 1989, Lieberman, 2001, Heishman et al., 2010).

All data was collected in a well-lit, quiet consultation room in order to minimise external stimuli. As previously mentioned, the initial step of the protocol was to brief each participant about the study, and for the participant and the researcher to both sign the consent form, thereby obtaining informed consent from the participant.

2.4.1 Blood Pressure Measurements

After obtaining informed consent, the participant was seated quietly for a 5-minute rest period so as to stabilise any physiological variables that may have been altered by previous activity. Following this rest period, three blood pressure and heart rate measurements (Figure 2.1) were recorded using an automated sphygmomanometer (Omron IA1B/HEM-7200, Omron Healthcare, Japan). A two-minute rest period

between each recording was provided to reduce the chance of occlusion and participant discomfort by allowing the blood vessels of the arm return to normal (National Heart Foundation of Australia, 2016).

Figure 2.1 – Automated method for measuring brachial blood pressure

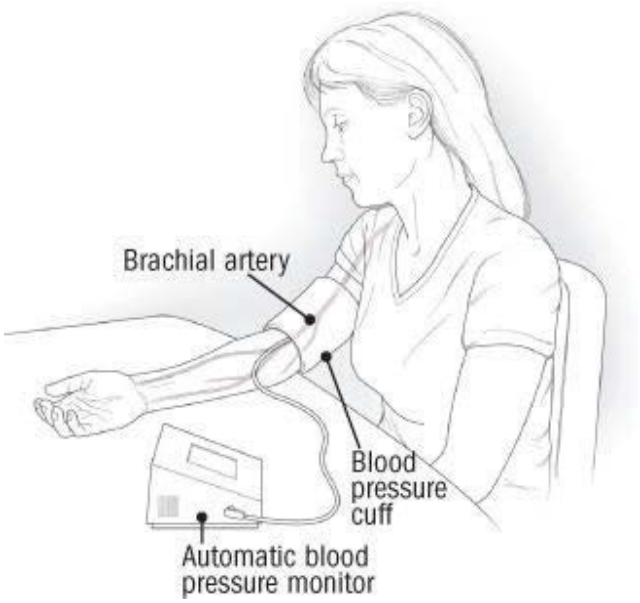

Figure 2.1 demonstrates how participant blood pressure measurements were taken using an automated blood pressure monitor. The correct blood pressure cuff placement on the upper arm, so as to occlude the brachial artery, is shown. Adapted from p1 Harvard Medical School (2009).

As previously mentioned (Section 2.3.1), these blood pressure measurements were used as an exclusion criterion where an average blood pressure greater than or equal to 160/100 mmHg excluded participants from further continuing the experimental protocol.

2.4.2 Pre- Study Questionnaires

After the initial blood pressure measurements, each participant was provided the pre-study questionnaire battery to complete. This battery included the Lifestyle Appraisal Questionnaire (LAQ) (Craig et al., 1996), the Depression, Anxiety, Stress, Scale (DASS) (Lovibond and Lovibond, 1995b) and the Fatigue State Question (Lal and

Craig, 2002). Additionally, the hip and waist circumference of each participant was also obtained, and later used to calculate each individual's waist to hip ratio.

Prior to continuing the study, questions 1a (*"Have you ever regularly smoked cigarettes?"*), 1b (*"Do your presently smoke cigarettes?"*) & 2 (*"How frequently do you smoke?"*), 7 (*"Do you drink alcohol? Indicate your average intake over one week."*), 8 (*"Do you take any drugs or medication other than tea, coffee, alcohol, and nicotine?")* and 18 (*"Do you, at present suffer from any chronic illness such as cancer, heart disease, asthma, diabetes, arthritis, etc.?"*) of part 1 of the LAQ (Craig et al., 1996) were again checked to ensure that the participant was not regularly consuming amounts of caffeine, nicotine and alcohol beyond the respective thresholds (Section 2.3.1), or using any drugs, or suffering from any ongoing illness or chronic condition with cognitive implications.

2.4.2.1 Lifestyle Appraisal Questionnaire

The Lifestyle Appraisal Questionnaire (LAQ) is a reliable and validated questionnaire designed to provide clinically relevant data (Craig et al., 1996), that was used to assess participant lifestyle factors. The LAQ is comprised of two components, part 1 and part 2. Part 1, the first component, contains 22 questions which together provide relevant demographic and lifestyle data concerning alcohol and nicotine consumption, body mass index (BMI), medical history, and also assesses risk factors associated with the development of lifestyle diseases. Obtaining a maximum score of 73 indicates the highest increased risk of an individual developing a chronic illness and experiencing a reduction in their quality of life (Craig et al., 1996). Part 2 of the LAQ (Craig et al., 1996) is comprised of 25 Likert scale (0-3) style questions. Together, these questions allow for an assessment of the participants' perception of life pressures and demands or

life stress that they experience. Within this section, a maximum score of 75 indicates the highest level of perceived life stress experienced (Craig et al., 1996).

Craig et al. (1996) demonstrated the reliability of the LAQ utilising a 3 month test/retest period, where no significant change in test results was found over that period. Further, the internal reliability was shown to be sufficient with Cronbach's Alpha of 0.5 and 0.89 for parts 1 and 2 respectively. Finally, the validity of the LAQ was demonstrated by strong correlations to previously validated questionnaires such as the State Trait Anxiety Inventory (Spielberger et al., 1983). Construct validity was further demonstrated by the lack of associations between the test scores and demographic factors, indicating that each scale accurately represents the components assessed (Craig et al., 1996).

2.4.2.2 Depression, Anxiety, Stress Scale

The Depression, Anxiety, Stress scale (DASS) is a self-reporting questionnaire designed to measure the interrelated negative emotional states of depression, anxiety and stress (Lovibond and Lovibond, 1995b). Its full form is composed of 42 items that are divided into three scales that examine depression, anxiety, and stress respectively. Each scale contains 14 items related to the past week that are scored on a 4-point severity/frequency scale (0-3 Likert scale) and the maximum score possible for each of the major scales is 42. If required, each major scale can be further divided into smaller subscales; specifically, the depression scale can be utilised to assess dysphoria, hopelessness, devaluation of life, self-deprecation, lack of interest/involvement, anhedonia, and inertia. Additionally, the anxiety scale can be used to assess autonomic arousal, skeletal muscle effects, situational anxiety, and subjective experience of

anxious affect. Finally, the stress scale can be used to assess difficulty relaxing, nervous arousal, and being easily upset/agitated, irritable/over-reactive and impatient.

With regards to psychometric properties, both the long and short form of the DASS have been shown to possess high internal consistency (Brown et al., 1997) with Cronbach's Alpha values of 0.97, 0.92, 0.95 and 0.94, 0.87, 0.91 for the 42 and 21 item DASS depression, anxiety and stress scales, respectively (Antony et al., 1998, Crawford and Henry, 2003). Further, the anxiety scale of the DASS correlated strongly with Beck's Anxiety Inventory ($r = 0.81$) and the depression scale with the Beck Depression Inventory ($r = 0.74$) indicating its construct validity (Lovibond and Lovibond, 1995b). Results that have also been further supported by more recent additional research (Brown et al., 1997, Antony et al., 1998, Crawford and Henry, 2003, Ng et al., 2007).

2.4.2.3 Fatigue State Question

The Fatigue state question (Lal and Craig, 2002) is a self-reporting two part question that was designed to measure an individual's fatigue level both prior to and after undertaking an experimental intervention, and was implemented in the present study to examine if the experimental EEG recordings were fatiguing. It asks each individual to answer how fatigued they currently feel on a 4 point Likert scale, where 1 is representative of "Not at all" and 4 is "Markedly".

2.4.3 Electroencephalography

Following the completion of the pre-study questionnaire battery and measurements, participants commenced the electroencephalography (EEG) data collection stage of the experimental protocol (Figure 2.2).

In this stage, data collection was separated into two phases; the first phase was a 5-minute baseline, in which the participants were asked to sit quietly in front of a blank computer screen with their eyes open. The second phase was a 5-minute active phase, in which participants completed a computerised form of the Stroop test (Figure 2.2; Stroop, 1935). The completion of the active phase concluded the EEG data collection.

The specific details regarding EEG data collection including equipment details and electrode positions are provided in Sections 3.2.3 and 4.2.3 respectively.

Figure 2.2 – Laboratory and electroencephalogram set-up of the present research

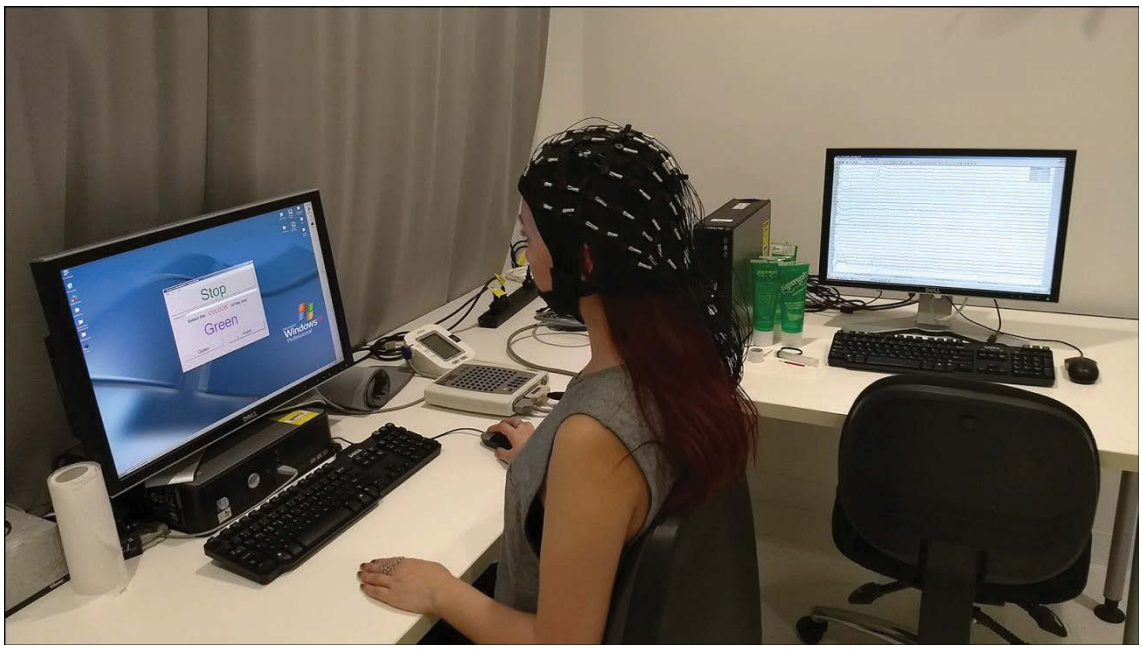

Figure 2.2 depicts a participant undertaking the active phase of testing in one of the laboratory set-ups utilised in the current research, specifically, the set-up of the experiment contained in Chapter 4. The EEG encoder is visible in the centre of the image by the participant's right hand, and the rightmost computer displays the captured EEG data.

Key: EEG = Electroencephalogram

2.4.3.1 Stroop Colour Word Test

The Stroop Colour word test is a reliable and simple test (Lezak et al., 2004) designed early in the 20th century, and widely used in the examination of cognition and cognitive

flexibility (MacLeod, 1991, Djamshidian et al., 2011). Furthermore, the application of the Stroop test extends to the clinical world where it has been previously used in the cognitive assessment of schizophrenia (Barch et al., 2004) and Parkinson's disease (Hsieh et al., 2008).

In this study, the computerised form of the Stroop test (Figure 2.3) presented participants with one of two options both of which required a response to a stimulus. The first option matched the colour named by the word with its ink colour. The second uses an ink colour that is different to the colour named by the word. Participants were required to identify and select the colour of the ink (via mouse button press) as promptly as possible. The duration of the options on the screen was as long as it took the participant to complete that trial.

The generation of the "Stroop interference effect", is the main reason the Stroop Colour Word test is utilised in cognitive assessment as it is considered by literature to be an effective general measure of cognitive flexibility (Uttl and Graf, 1997) or executive functioning (Moering et al., 2004). This effect is generated when an individual undertakes a task in which the suppression of a habitual response in support of an unusual one is required, that is, the naming of the colour ink used to print incongruously named colour words, for example, the word "BLACK" written in red ink (Van der Elst et al., 2006).

Furthermore, the nature of this interference effect has also seen the Stroop colour word test be used as an effective means of inducing stress (Hjemdahl et al., 1984, Tulen et al., 1989, Renaud and Blondin, 1997). However, despite its ability to induce stress, this intervention was used to instigate cognitive functions and was designated as the

cognitively active phase of the experimental recording, which could then subsequently be compared to the baseline phase.

Figure 2.3 – The Stroop Test

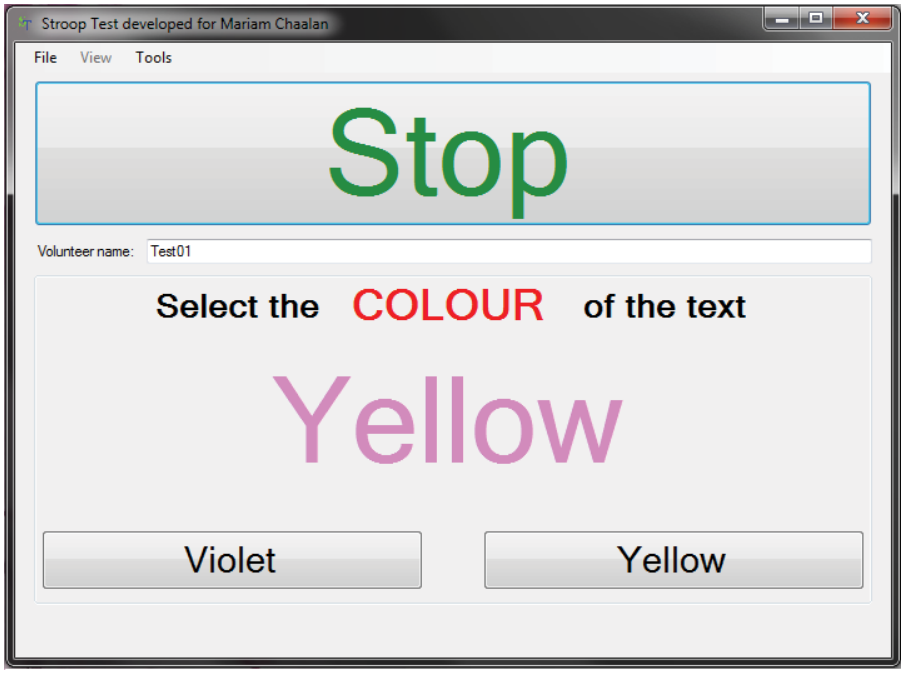

Figure 2.3 is a screenshot of the computerised Stroop test that was used during the present active phase of electrophysiological data collection. The intent of the test is to recognise and select the colour of the text rather than the colour that the word names, as fast as possible. In the figure the correct answer would be violet, as the word yellow is written in the colour violet.

Finally, two output variables were captured from the Stroop test in the present research. The first variable was the number of tests passed, which is defined as the number of trials completed by the participant during the active phase of EEG testing; the second variable was the average response time, which was the participants average response time for all completed trials, both congruent and incongruent.

2.4.4 Cognitive Testing

Once the EEG data collection was completed, cognitive performance was further assessed using the Mini Mental-State Examination (MMSE; Folstein et al., 1975) and

the Cognistat (Kiernan et al., 1987). These tests were administered in a random order (between each participant) so as to negate any potential order effects that may have otherwise developed.

2.2.4.1 Mini-Mental State Examination

The MMSE is a valid and reliable psychometric test (Folstein et al., 1975, Pangman et al., 2000, Marioni et al., 2011) that is comprised of two components that include a total of eleven questions that take approximately 5-10 minutes to complete. Further, it possesses moderate to high test-retest values (ranging from 0.56 to 0.99) (Tombaugh and McIntyre, 1992) and internal consistency (Cronbach's alpha ranging from 0.54 to 0.96) (Tombaugh and McIntyre, 1992) and for these reasons has been, and currently is, used extensively in the assessment of mental function within both research and clinical settings. Collectively, the MMSE examines the domains of orientation, registration, attention and calculation, memory and language (Folstein et al., 1975), but only provides a commentary regarding an individual's global cognitive performance i.e. an individual's overall cognitive performance without reference to any cognitive domain. The maximum score for the MMSE is 30, where a score of less than 23 is considered to be indicative of a potential cognitive impairment (Folstein et al., 1975) and has been quite strongly linked to dementia diagnosis (Lancu and Olmer, 2006).

However, despite its widespread use in cognitive impairment screening (Scazufca et al., 2009), it has been suggested that the standard implementation of the MMSE and the threshold score of 23 may have limited use as a method for the identification of mild cognitive impairment (Yue et al., 1994, McDowell et al., 1997, Tang-Wai et al., 2003, Zadikoff et al., 2008). Indeed, it has been recommended that increasing the threshold score to 26 achieves greater accuracy than the original threshold value of 23 (van Gorp

et al., 1999). Furthermore, the authors of the MMSE recently offered a revised scoring system that indicated that a threshold score of above 27 represents normal cognition, while scores of 21-26 suggest mild cognitive impairment. Scores between 11 and 20 suggest moderate cognitive impairment, and scores of 10 or below are indicative of severe cognitive impairment (Folstein et al., 2001).

Additionally, a number of researchers have suggested that the MMSE is prone to ceiling effects, which may be attributed to the small range of what could be considered easy questions (Lopez et al., 2005, Zadikoff et al., 2008, Hoops et al., 2009). Further, the unequal distribution of sensitivity across the tested domains, the highly verbal nature, the lack of assessment of subcortical functions (Tombaugh and McIntyre, 1992, Byrne et al., 2000) and the lack of standard administration guidelines for delivery, interpretation and scoring, particularly for the more open ended tasks (Molloy et al., 1991) has seen the MMSE draw criticism. Despite this, research (Anthony et al., 1982, Kay et al., 1985, Kafonek et al., 1989, Fillenbaum et al., 1990, Murden et al., 1991) has indicated that the MMSE has moderate to high specificity and sensitivity, with values ranging from 45% to 100% in patients with dementia, and 21% to 76% in general neurology and psychiatric patients (Tombaugh and McIntyre, 1992).

2.2.4.2 Cognistat

The Cognistat (previously known as the Neurobehavioural Cognitive Status Examination) is a robust screening tool that takes approximately 20 minutes to examine cognitive impairment in seven individual domains (orientation, attention, language, construction, memory, calculation and reasoning) (Kiernan et al., 1987, Mueller et al., 2007). It has been shown to possess moderate construct validity (Mueller et al., 2007) and good reliability with a test-retest value of 0.69, as well as moderate inter-rater

reliability of 0.57 (Lamarre and Patten, 1994). Additionally, construct validity was demonstrated by significantly correlating the subtests of the Cognistat to other similar neuropsychological tests (Osato et al., 1993, Marcotte et al., 1997). Furthermore, the internal consistency of the Cognistat is also high with an overall Cronbach's alpha of 0.94 (Kiernan et al., 1987).

The Cognistat makes its assessment by first asking the individual a screening question of average difficulty for a particular domain. Correctly answering and passing the screen question is indicative of a normal level of cognitive function for that domain, and allows the test to progress to the next domain (Deutinger, 2007, Mueller et al., 2007). However, if the screen is failed, the researcher than asks a number of metric questions, which are a series of increasingly more difficult and complex questions associated with the same domain, and provide a more thorough assessment of the level of impairment (Deutinger, 2007, Mueller et al., 2007). Interestingly, Deutinger (2007) demonstrated that an average cognitively healthy person might fail up to 20% of screen questions. Indeed, a number of researchers (Logue et al., 1993, Pichitino and Green, 1999) have suggested abandoning the screen and metric approach, and instead have supported administering both the screen and metric questions to reduce false negatives and obtain maximum accuracy (Drane and Osato, 1997, Oehlert et al., 1997, van Gorp et al., 1999, Drane et al., 2003). Therefore, to reduce false positives in the present study, both the screen and the metric questions were administered.

The Cognistat (Mueller et al., 2007) measures participant cognitive performance using a graded scoring system for each of the individual domains (Table 2.2), where thresholds are provided for varying degrees of impairment i.e. mild, moderate, severe. This scoring method that has been associated with a higher sensitivity than singular global threshold scores (Schwamm et al., 1987, Macaulay et al., 2003).

Table 2.2 – Cognistat cognitive profile and respective impairment threshold scores

			Level of cognitive impairment			
Cognitive domain		**Maximum score**	**Borderline**	**Mild**	**Moderate**	**Severe**
Orientation		12	< 10	8	6	4
Attention		8	< 6	5	3	1
Language	**Comprehension**	6	< 5	4	3	2
	Repetition	12	< 11	9	7	5
	Naming	8	< 7	5	3	2
Construction		6	< 4	3	2	0
Memory		12	< 10	8	6	4
Calculation		4	< 3	2	1	0
Reasoning	**Similarity**	8	< 5	4	3	2
	Judgment	6	< 4	3	2	1
Total score		82	< 65	51	36	21

The respective cognitive impairment thresholds (borderline, mild, moderate and severe) as well as the maximum score achievable in the Cognistat domains are presented. Adapted from Kiernan et al. (1987).

Key: < = Less than

Classifying cognitive impairment using this graded scoring system involves reaching the impairment threshold in one or more of the tested domains and has a specificity of 85%, and sensitivity of 100%. However, if the requirement is shifted to impairment in two or more domains, specificity improves to 100%, but sensitivity is reduced to 94% (Osato et al., 1993). Indeed, in general and in a number of specific contexts, including geriatrics (Fields et al., 1992), psychiatric (Osato et al., 1993), and neuropsychological settings (Schwamm et al., 1987), the Cognistat has demonstrated high sensitivity (Lamarre and Patten, 1994), however, in some instances it has also demonstrated low sensitivity (28 – 56 %) (Osato et al., 1993, Engelhart et al., 1994). Finally, it has been suggested that both the MMSE and Cognistat be administered together to enable

improved sensitivity (Schwamm et al., 1987, Macaulay et al., 2003, Dujardin et al., 2010).

2.4.5 Post study Questionnaires

After the completion of cognitive testing, participants completed a second short post study questionnaire battery that constituted of the Ways of Coping Checklist (Vitaliano et al., 1985), the Fatigue State Question (Lal and Craig, 2002) and, for the nurse participants, the shiftwork and sleep scales of the Standard Shiftwork Index (Barton et al., 1995).

2.4.5.1 Ways of Coping Checklist

The Ways of Coping Checklist (WCCL), as designed by Folkman and Lazarus (1980), is a self-reported binary checklist of 68 items that describe a broad range of behavioural and cognitive coping strategies, derived from literature (Lazarus, 1966, Lazarus and Launier, 1978, Mechanic, 1962, Sidle et al., 1969, Weisman and Worden, 1976), that an individual might utilise when experiencing stress.

The original scales of the WCCL were validated and determined to have a respectable reliability co-efficient ranging from 0.76 to 0.88 (Folkman and Lazarus, 1980). However, Vitaliano et al. (1985) further developed the WCCL, utilising principal component analysis and factor loading to revise and improve its psychometric properties, as it had been indicated that some of the original components may not have been reliable, or were deemed ambiguous against the stress and coping theory of Lazarus and Folkman (1984). This revised WCCL was shortened to 43 items that reside on 5 scales, and again proved to be of respectable internal consistency (values ranging from 0.74 to 0.88) as well as construct and criterion validity (Vitaliano et al., 1985). The

construct validity was further confirmed since demographic variables across three sample groups were not significantly correlated to scale scores (Vitaliano et al., 1985); it was this improved format of the WCCL that was utilised in the present research.

2.4.5.2 Standard Shiftwork Index

The Standard Shiftwork index (SSI) is a battery of self-report questionnaires specifically composed to assess the impact of shift systems upon the individuals (Barton et al., 1995). In this experiment, two components of the SSI were submitted, a number of general questions and the sleep quality and disturbance questionnaire. The general questions were used to provide context regarding an individual's shiftwork habits and are related to both the specific features of the shift system (e.g. the sequencing, timing and duration) and the features of the work context (e.g. degree of subjective workload on different shifts). The sleep quality and disturbance questionnaire consists of two sections made up of 11 items. The first section examines sleep habits, the second component measures sleep quality, and difficulties associated with the shifts worked and the rest days.

As the SSI is a composite of a number of different scales and assessment tools, determining its reliability and sensitivity has proven to be difficult (Pitsopoulos and Greenwood, 2002). However, utilising factor analysis for each individual scale has yielded reliability Cronbach's Alpha scores ranging between 0.60 and 0.94 (Smith et al., 2001, Korompeli et al., 2011). Furthermore, the convergent validity of the SSI scales was confirmed by the strong correlations found between the various scales (Korompeli et al., 2011).

Once the post study questionnaires were completed, three post-study blood pressure measurements were obtained and the experimental session was concluded. Figure 2.3 provides a diagrammatic representation of the entire experimental protocol.

Figure 2.4 – Diagrammatic representation of the experimental protocol

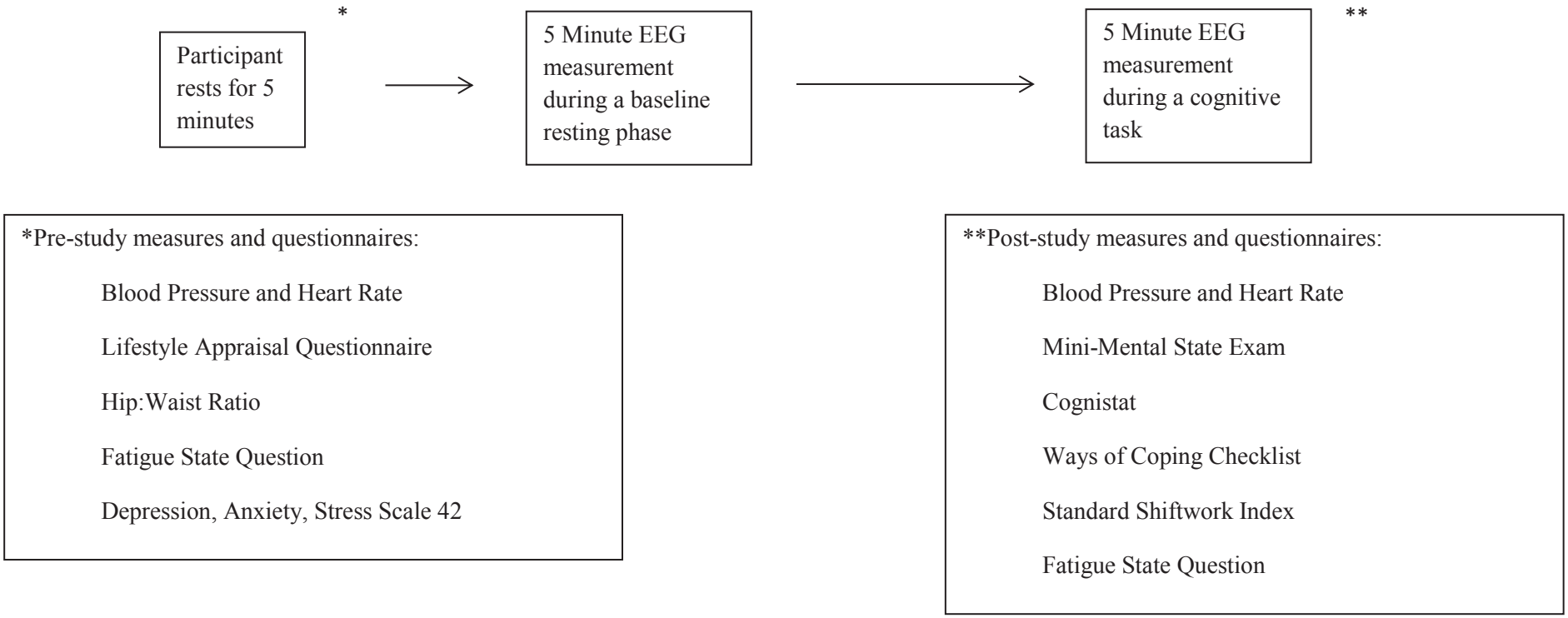

Figure 2.3 is a diagrammatic summary of the experiment protocol. Participants will have an initial 5 minute reset period after which the pre-study measures and questionnaires will be completed. Following this, the baseline and active phase of the EEG data collection will be obtained. Finally the participant will complete the post-study measures and questionnaires which will conclude the experimental session.

2.5 Data Processing

The following data were processed prior to statistical analyses.

2.5.1 Questionnaire & Physiological data

The questionnaire and physiological data collected in the current study, that required processing or scoring prior to being analysed were:

- Blood Pressure and Heart Rate, which was calculated by averaging the three pre- and post- experiment measurements for each participant.
- The Lifestyle Appraisal Questionnaire (Craig et al., 1996); where the scores for part 1 and part 2 were calculated independently for each participant.
- The Depression, Anxiety, Stress, Scale (Lovibond and Lovibond, 1995b); where Stress and Anxiety scores were calculated for each participant.
- The Ways of Coping Checklist (Vitaliano et al., 1985); where the scores for each of the 5 coping strategy subscales was calculated for each participant.
- The Mini-Mental State Exam (Folstein et al., 1975); where the total exam score was totalled for each participant.
- The Cognistat (Kiernan et al., 1987); where both the global and domain specific scores were totalled for each participant.

Further, it is important to note that each questionnaire utilised was scored and totalled as per their specific instructions. For further information on the scoring details of each questionnaire, please refer to their individual references listed above.

2.5.2 Processing of Electroencephalogram data

All collected EEG data was processed prior to statistical analysis. The specific processes applied to each set of electroencephalogram data can be found in detail in Chapter 3, Section 3.2.3 and Chapter 4, Section 4.2.3, respectively.

Briefly, the raw time domain EEG data was filtered using an IIR Butterworth Bandpass filter set at 1.5 Hz and 50 Hz, followed by a Hann Window. The Aligned-artefact average procedure (Croft and Barry, 1998, Croft and Barry, 2000) was then utilised to minimise any ocular artefacts. Following this, the recording was separated into approximately 300 one-second epochs and the EEG activity in 5 frequency bands: delta (0.5–4 Hz), theta (4–8 Hz), alpha (8–13 Hz), beta (13–35 Hz) and gamma (35–50 Hz), was calculated via periodogram power spectral density estimate. The derived power values for each epoch were then averaged together, generating a single power value in each of the delta, theta, alpha, beta and gamma frequency bands for each recording.

2.6 Statistical Analysis

Statistical analysis was conducted using STATISTICA (Version 10, 1999, StatSoft, USA) to explore the associations between stress, anxiety and cognitive performance in a group of nurses, and a group of non-health professionals across two different experiments. Furthermore, the differences between the two groups with respective to their demographic variables, and questionnaire scores were also explored.

Statistical significance was reported at p-values of less than 0.05.

The specific statistical analyses that were utilised across both experiments are explained below. Additionally, any experiment specific analysis methods will be further explained in the following results Chapters 3-5.

2.6.1 Power analysis & Sample size estimation

Cohen (1992) demonstrated that the minimum sample size required for the analysis conducted in this study (t-tests and partial Pearson's correlations), with a power goal of 0.80 and large effect size (e.g. r = 0.5 for Pearson's correlation) is approximately 30. Indeed, sample size is directly related to the statistical significance of Pearson's correlation, where a larger sample size increases the reliability of results and improves the chance that the correlation is reflective of a true relationship (Peacock and Peacock, 2011).

With respect to the present study, all experiments (Chapters 3-5) possess sufficiently adequate sample sizes for the analysis conducted.

2.6.2 Dependent & Independent Sample T test

T-tests are employed to analyse the magnitude and significance of differences in the means of two normally distributed variables from either the same source or two different sources (Peacock and Peacock, 2011).

In this study, dependent sample t-tests were utilised to identify significant differences in heart rate, fatigue and blood pressure data obtained both before and after the experimental session. Furthermore, dependent sample t-tests also assessed the differences in EEG values between the baseline and active phases. Additionally, independent sample t-tests determined if there were any significant differences in demographic variables, LAQ scores, DASS scores, and cognitive performance scores between the nurse and non-health professional groups.

2.6.3 Pearson's Correlation

Pearson's correlation investigates the nature of a linear relationship between a dependent and independent continuous variable, and may also control for covariates if necessary. In this research, Pearson's correlation was used to determine if measures of stress and anxiety were correlated, and if there were associations between measures of stress and anxiety and demographic variables including age, BMI, lifestyle risk factors, and fatigue state. Additionally, partial Pearson's correlations controlling for the covariates of age, BMI (Chapters 3 and 4) and, where possible years of education (Chapter 4), were utilised to assess the associations between stress and anxiety, and cognitive performance (as measured by the MMSE, Cognistat and EEG).

Correlation analysis determines the correlation coefficient (denoted by r), a value which by measure of its magnitude and proximity to 1 or -1 indicates the strength of the association i.e. an r value between 0.10 and 0.30 is considered to be a small effect size, an r value between 0.30 and 0.50 represents a medium effect size, and an r value of 0.50 or greater demonstrates a large effect size (Cohen, 1988, 1992). Further, the correlation coefficient also determines the nature of the investigated relationship (Peacock and Peacock, 2011), where a r value below 0 indicates a negative linear relationship (i.e. when one variable increases the other decreases), and a r value greater than 0 indicates a positive linear relationship (i.e. when one variable increases, the other increases) (Peacock and Peacock, 2011).

2.6.4 Multiple Regression Analysis

Multiple regression analysis examines the nature of the linear relationship between a single dependent variable and multiple independent variables and subsequently identifying the most important independent variable. In completing a regression analysis

a coefficient of determination (often represented as R^2) is generated; this value functions as a measure of effect and reflects the proportion of variability equal to the square of the correlation coefficient between outcomes and their predicted values (Peacock and Peacock, 2010).

This research utilised forward stepwise multiple regression analysis to identify the most significant cognitive predictors of stress and anxiety (Chapter 3). Furthermore, forward stepwise general linear multiple regression analysis was utilised to similarly identify significant cognitive predictors of stress and anxiety (Chapter 4), as well as the most significant EEG predictors of cognitive performance (Chapter 5). In these latter two instances general linear multiple regression analysis was selected because of its greater ability to handle a large number of input variables. Both types of regression analyses were informed via the partial Pearson's correlations, as well as the conducted LASSO analysis (Chapter 5) and, were conducted in the instance of a single dependent variable being significantly correlated to three or more independent variables.

2.6.5 Principal Component Analysis:

Principal component analysis (PCA) is a multivariate statistical procedure that extracts the most salient information from an original dataset, and expresses this information using a new reduced set of values of orthogonal variables called principal components (Jolliffe, 1986, Abdi and Williams, 2010). This new dataset can be then utilised in further analysis, or to evaluate the importance of the variables in the original expanded dataset. Principal component analysis was used in the current study (Chapters 3 and 4) to further identify which EEG activities were important to the present analysis.

2.6.5.1 Principal Component Input

Principal component analysis was performed using MatLab (Version 2015a, Mathworks, USA). An example of the MatLab input that was used is as follows:

1. ProcessedBaseline = mapstd(BaselineEEG', 0, 1)';
2. [Coeff, score, latent, tsquared, explained, mu] = pca(ProcessedBaseline);
3. PCA_ComponentVariance = cumsum(explained);

This analysis was applied to 3 matrices for each sample group; these matrices contained the processed EEG data (both time and frequency domain) from a single testing phase (e.g. baseline), and is represented via the input argument 'BaselineEEG'. Furthermore, the output variable 'PCA_ComponentVariance' provides a cumulative description of the variance represented by each of the newly generated principal components, and was examined to determine the number of principal components that together represented over 95% of the variance of the inputted data.

Following this, the output 'coeff' matrix (which contains the principal component coefficients for each of the original variables) was examined to find the top 5 coefficient values for each of the contributing principal components. This information was combined with the results of the statistical analysis to allow a more detailed and specific analysis of the stress and anxiety related EEG results to be performed.

Chapter 3 – Associations between Stress, Anxiety and Cognitive performance (using a limited EEG montage)

3.1 Introduction

Nurses are frequently faced with challenges that require optimal cognitive performance, including the provision of continuous care, working with vulnerable patients, and time spent on call. These challenges render the nursing profession inherently stressful (Lee and Wang, 2002, Sveinsdóttir et al., 2006, Golubic et al., 2009), a characteristic that can negatively affect nursing and patient care quality (Tarnow-Mordi et al., 2000, Sveinsdóttir et al., 2006, Berland et al., 2008). As such, sub-optimal cognitive functioning could endanger the quality of care provided by nurses (Wilson et al., 1999, Tarnow-Mordi et al., 2000, Sveinsdóttir et al., 2006, Berland et al., 2008), which in turn could place lives at risk; indeed, in some instances stress has accounted for up to 57% of adverse medical events (Wilson et al., 1999).

As a collective, stress literature has demonstrated that consistent and chronic exposure to cortisol can cause neuronal damage and death in the hippocampi and prefrontal cortex (Starkman et al., 1992, Sapolsky, 1996, Sapolsky, 2003, Conrad, 2006). Such results are particularly salient as these neural regions are associated with cognitive processes and stress regulation (Lepage et al., 2000, Lupien and Lepage, 2001). Additionally, the norepinephrine system modulates some cognitive processes including working memory and attention (Ramos and Arnsten, 2007), whereby moderate hormone levels improve cognitive performance and high levels impair cognitive performance (Ramos and Arnsten, 2007). Further, literature has established that global cognitive performance (an individual's overall cognitive performance without reference to any cognitive domain) (LeBlanc et al., 2005, LeBlanc, 2009), and domain specific memory

(Lupien et al., 1999, de Quervain et al., 2000, Kuhlmann et al., 2005) and decision making performance (Cumming and Harris, 2001, Wetzel et al., 2006), can be negatively affected by stress. However, other research has shown stress may have minimal cognitive impact (Lees and Lal, 2017).

Furthermore, anxiety has similarly been associated with functional connectivity changes (Andreescu et al., 2014), decreased prefrontal activity (Bishop, 2009), and specific impairments in working memory (Asmundson and Stein, 1994, Savage et al., 2000, Ashcraft, 2002), attention (Lautenbacher et al., 2002), decision-making (Cumming and Harris, 2001) and various executive functions (Airaksinen et al., 2005, Nieuwenhuys et al., 2015). Yet again, these impairments have the real possibility to affect the performance of nurses, and their quality of care, placing patients at risk.

However, a limited amount of research has investigated the impact of stress and anxiety on the cognitive performance of health professionals (LeBlanc, 2009) and, as such, the aim of this study was to investigate the associations between stress, anxiety and cognitive performance in nurses. All results were of interest as previous research is limited (LeBlanc, 2009), and has indicated the need for additional research in the area (Lees and Lal, 2017). Furthermore, of the cognitive variables investigated, those of most interest were global cognitive performance, as well as memory and judgement performance as these domains are commonly invoked in the occupational tasks of nurses. Thus, it was hypothesized that experiencing higher levels of stress/anxiety will be associated with:

1. Declines in cognitive performance, both in global and specific domains.
2. Alterations in brain activity as measured by EEG; in particular the higher frequency bands (beta and gamma) of the frontal poles.

3.2 Materials and Methods

This experiment utilised the general methodology that was previously described in Chapter 2. The following sections provide a brief summary of the experimental protocol (Section 3.2.2) and describe experimental specific methodologies (Section 3.2.3, and Section 3.2.4).

3.2.1 Study Participants

Data from a total of 58 nurses and 93 non-health professionals was recorded in this experiment. Of these participants, 22 nurses, and 37 non-health professionals were recruited as part of this research project, whilst the remainder of the data was contained within two existing databases.

The first database comprised of data from 36 nurse participants previously collected by the PhD candidate (Lees, 2012); the second database was comprised of data from 55 non-health professionals (Kalatzis, 2014). It is important to note, that the data contained within both of these databases was collected in the Neuroscience Research Unit at the University of Technology Sydney (UTS), using the same experimental protocol and laboratory conditions, as previously described in Chapter 2.

Furthermore, it should be noted that the data from one nurse participant was excluded from the analysis, as this individual was currently using medication that may have altered their cognitive function. This reduced the final sample size of the experiment to 57 nurses and 93 non-health professional individuals.

3.2.2 Experimental Protocol

This experiment utilised the general methodology that was previously detailed in Chapter 2, and is briefly summarised in the following paragraphs.

Initially, participants completed a pre-study questionnaire battery that consisted of the Lifestyle Appraisal Questionnaire (Craig et al., 1996), the Depression, Anxiety, Stress Scale (Lovibond and Lovibond, 1995b), and the Fatigue State Question (Lal and Craig, 2002). Anthropometric measurements, including blood pressure, heart rate, height and weight, as well as waist and hip circumference were recorded. Following the completion of these questionnaires, participants undertook a two-phase electroencephalography recording. The first phase was a baseline recording in which the participants quietly rested with their eyes open, and the second phase was an active phase in which the participants completed a computerised format of the Stroop Test (see Section 2.4.3.1 for further details).

After the electrophysiological recording, participant cognitive performance was assessed using the Mini-Mental State Exam (Folstein et al., 1975), and the Cognistat (Kiernan et al., 1987). Which was followed by a post-study questionnaire battery, that included the Fatigue state Question (Lal and Craig, 2002), the revised Ways of Coping Checklist (Vitaliano et al., 1985), and two scales from the Standard Shiftwork Index (Barton et al., 1995) for nurse participants only. Lastly, participant blood pressure was recorded again, concluding the experimental protocol (Figure 2.3).

3.2.3 Electroencephalogram data collection

A two-channel transverse bipolar electroencephalogram montage with a sampling rate of 2048 Hz was recorded using a Flexcomp Infiniti encoder (Thought Technology Ltd, Canada; Figure 3.1) in conjunction with the BioGraph Infiniti software package (Thought Technology Ltd, Canada).

Figure 3.1 – Flexcomp Infiniti electroencephalogram encoder and electrodes

Figure 3.1 presents the Flexcomp Infiniti encoder and the gold plated electrodes that were utilised in this experiment are shown.

Gold-plated cup electrodes were filled with Signa gel (Parker Laboratories Inc, USA) and set up using the locations: Frontal Polar 1 (Fp_1) and 2 (Fp_2), as well as Central 3 (C_3) and 4 (C_4) of the International 10-20 system. Additionally, non-linked reference ear clip electrodes were placed at positions Auricular 1 (A_1) and 2 (A_2). Refer to Figure 3.2 for electrode locations utilised in the present work.

Figure 3.2 – A top-down view of the relevant electrode placements of the International 10-20 System

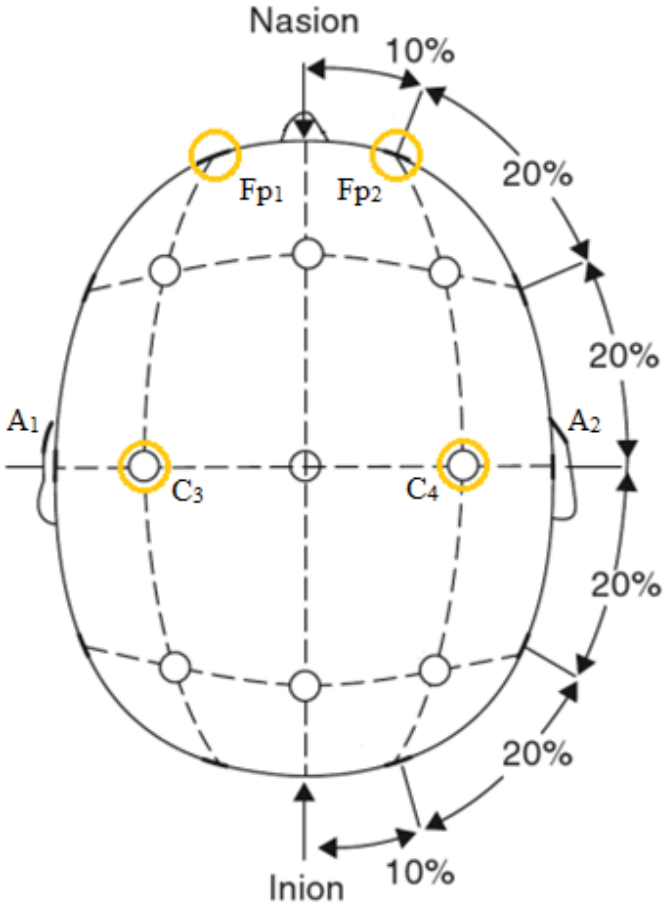

Figure 3.2 illustrates a top-down view of the International 10-20 system of electrode placement as it was original described by Jasper (1958) is shown. Indicated by the yellow circles are the active electrode positions (Fp_1, Fp_2, C_3, and C_4) utilised in this experiment. The reference electrode positions are also indicated by A_1 and A_2.

Key: A = Auricular; C = Central; Fp = Frontal pole

These electrode locations were selected because the brain regions associated with these sites have been previously associated with various cognitive functions including attention, psychomotor execution, and working memory (Beisteiner et al., 1995, Miller and Cohen, 2001, Georgopoulos, 2002, Arnsten, 2009).

Following the placement of the electrodes, an initial short EEG recording was commenced to allow the EEG signal to be examined and troubleshot if necessary. If the signal was found to be of poor quality, adjustments on location or fixation of the electrode were made and the recording checked again until rectified and a reasonable EEG signal (Figure 3.3) was obtained.

Figure 3.3 – A two lead bipolar electroencephalogram recording

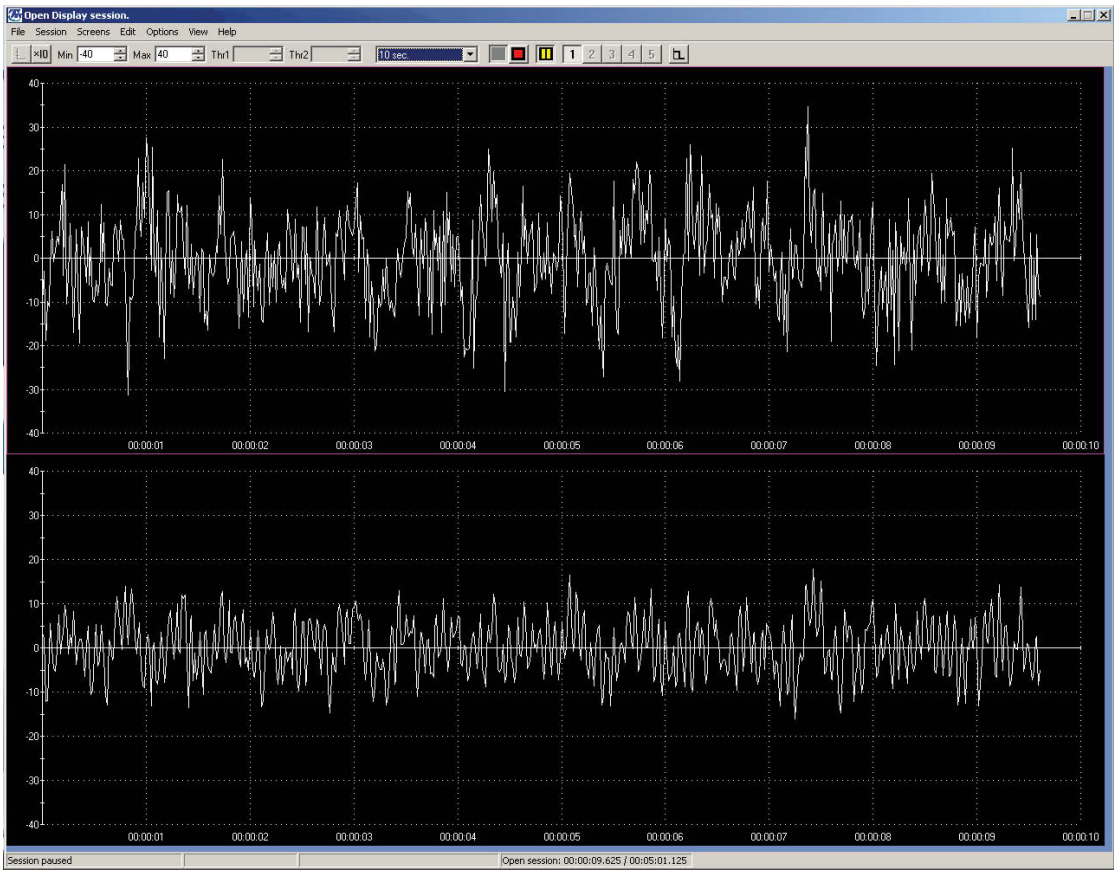

Figure 3.3 displays an acceptable electroencephalogram recorded in the present experiment. The figure also provides a representation of the nature of the bipolar montage utilised, where the top trace was recorded from the electrodes located at Fp_1 and Fp_2; the bottom trace was recorded from the electrodes located at C_3 and C_4. The X axis plots time in seconds, whilst the Y axis plots amplitude in microvolts.

Key: C = Central; Fp = Frontal pole

3.2.4 Electroencephalography Data processing

As noted in Section 2.5.2, prior to statistical analysis, the raw EEG data sets obtained for both the baseline and active phases were processed according to the steps stipulated below.

1. A Butterworth IIR Bandpass filter set at 1.5 and 50 Hz was applied to remove any direct current residue and/or any high frequency artefacts (e.g. movement artefacts). This was followed by the application of a Hann Window.
2. The Aligned-artefact average procedure (Croft and Barry, 1998, Croft and Barry, 2000) was applied to minimise any artefacts linked to eye movement.
3. Both the baseline and active phase recordings (approximately 5 minutes each in length) were sectioned into approximately 300 one-second epochs.
4. Following being separated into epochs, the EEG activity (of each epoch) in the delta (1.5 - 4 Hz), theta (4 - 8 Hz), alpha (8 - 13 Hz), beta (13 - 35 Hz) and gamma (35 - 50 Hz) frequency bands, was calculated via Periodogram power spectral density estimate (Bartlett, 1950).
5. The values for all 300 epochs were then averaged together, deriving a single activity value per recording for each of the five EEG frequency bands (Delta, Theta, Alpha, Beta and Gamma).
6. Additionally, the frequency band activity values for both the frontal and central electrode pairs were also averaged together, providing an overall average value for the EEG activity.
7. Finally, a change in EEG activity or reactivity value was calculated by subtracting the baseline values from their respective active phase values. This value was generated to allow the change in EEG activity between the baseline and active phases to be examined.

3.3 Results

3.3.1 Demographics and Comparisons

One-hundred and fifty individuals participated in this experiment study; 57 were nurses (49 Females, 8 Males) aged between 19 and 59 years (average 33.72 ± 11.14 years), and 93 (48 Females, 45 Males) were non-health professionals aged between 18 and 63 years (average 27.70 ± 11.30 years). Further, it is important to note that age differed significantly between the two groups ($p = 0.001$; Table 3.1). Additionally, regarding the registration status of the nurse participants, at the time of testing, nine were assistants in nursing (AINs), 11 were enrolled nurses (ENs), and the remaining 37 nurses were registered nurses (RNs).

With respect to participant health status, on average, the BMI of both groups (24.09 ± 6.61 kg/m^2 for nurses, and 24.71 ± 4.55 kg/m^2 for non-health professionals) resided within the normal range and did not differ significantly. Interestingly, lifestyle risk factors that increase risk of disease development (LAQ P1) did differ significantly between the sample groups ($p = 0.031$), with nurses reporting a slightly higher score (13.47 ± 6.01) than non-health professionals (11.20 ± 6.29). Similarly, both the pre and post self-report fatigue measures differed significantly between groups ($p = 0.005$, and $p = 0.023$, respectively), with nurses reporting slightly higher values in both instances (2.05 ± 0.67 vs 1.73 ± 0.68, and 2.04 ± 0.80 vs 1.75 ± 0.69, respectively).

Table 3.1 – Demographic data and intergroup comparisons of the two study sample groups

Variable	Group	Value	t	p
Age (years)	Nurses	33.72 ± 11.14	3.18	0.001*
	NHP	27.70 ± 11.30		
BMI (kg/m^2)	Nurses	24.09 ± 6.61	-0.69	0.493
	NHP	24.71 ± 4.55		
WHR	Nurses	0.86 ± 0.08	-1.39	0.166
	NHP	0.88 ± 0.07		
Pre FSQ	Nurses	2.05 ± 0.67	2.84	0.005*
	NHP	1.73 ± 0.68		
Post FSQ	Nurses	2.04 ± 0.80	2.29	0.023*
	NHP	1.75 ± 0.69		
LAQ P1	Nurses	13.47 ± 6.01	2.18	0.031*
	NHP	11.20 ± 6.29		

Table 3.1 displays basic demographic information including age, body mass index, waist hip ratio, pre and post fatigue state, and lifestyle risk factors that increase risk of disease (part 1 of the LAQ) for both sample groups included in this experiment. Additionally, both t and p values are provided for intergroup comparisons for each of these variables.

Key: BMI = Body mass index; FSQ = Fatigue State Questionnaire; LAQ = Lifestyle Appraisal Questionnaire; NHP = Non-health professionals; * = Statistical significance

Finally, dependent sample t-tests revealed that the heart rate of both sample groups reduced significantly over the course of the experimental protocol (Table 3.2), lowering from 75.86 ± 9.80 to 72.87 ± 9.74 bpm ($p < 0.001$), and from 73.85 ± 10.64 to 69.05 ± 8.75 bpm ($p < 0.001$), for nurses and non-nurses respectively. Lastly, blood pressure and heart rate did not differ significantly over the experimental protocol for either sample group.

Table 3.2 – The differences in pre and post study measures of blood pressure and fatigue for both sample groups

	Sample Group	Pre Mean (SD)	Post Mean (SD)	t	p
SBP (mmHg)	Nurses	114.32 ± 13.13	113.45 ± 12.71	0.71	0.480
	NHP	115.85 ± 11.80	115.71 ± 12.71	0.20	0.196
DBP (mmHg)	Nurses	75.94 ± 9.24	74.88 ± 8.33	1.29	0.201
	NHP	75.39 ± 7.77	75.87 ± 8.18	-0.84	-0.838
HR (BPM)	Nurses	75.86 ± 9.80	72.87 ± 9.74	4.07	< 0.001*
	NHP	73.85 ± 10.64	69.05 ± 8.75	7.22	< 0.001*
FSQ	Nurses	2.05 ± 0.67	2.04 ± 0.80	0.24	0.811
	NHP	1.73 ± 0.68	1.75 ± 0.69	-0.32	0.748

Table 3.2 presents the pre and post experimental testing values for systolic and diastolic blood pressure as well as heart rate and fatigue state. Furthermore, t and p values are provided for intragroup comparisons for these variables.

Key: BPM = Beats per minute; DBP = Diastolic Blood Pressure; FSQ = Fatigue State Questionnaire; HR = Heart rate; mmHg = Millimetres mercury; NHP = Non-health professionals; SBP = Systolic Blood Pressure; SD = Standard Deviation; * = Statistical significance

3.3.2 Cognitive Performance

3.3.2.1 Psychometric Assessment

In terms of global cognitive performance, both sample groups scored above the impairment thresholds for the MMSE and the Cognistat (Table 3.3), with the nurse participants scoring 27.61 ± 1.73, and 74.11 ± 5.21 and the non-health professional participants scoring 28.23 ± 1.44, and 75.49 ± 3.62, respectively. Furthermore, regarding the MMSE result, the non-health professionals scored significantly higher than the nurses ($p = 0.021$).

With respect to domain specific cognitive performance, both sample groups had average scores above the respective cognitive impairment thresholds for all assessed domains. Further, some cognitive domain scores differed significantly between the two groups (Table 3.3). The implicated domains were orientation, where nurses scored 11.88 ± 0.38 compared to 11.35 ± 0.62 for the non-health professional group ($p < 0.001$); construction, where the non-health professionals scored 5.32 ± 0.98 versus the 4.67 ± 1.02 of the nurse group ($p < 0.001$); and similarities, where the non-health professionals returned a higher score of 7.27 ± 1.04 compared 6.82 ± 1.35 of the nurse group ($p = 0.025$). All other domain scores did not differ significantly between sample groups.

Table 3.3 – The global and domain specific cognitive performance scores of the two study sample groups

Variable	Group	Value	t	p
MMSE†	Nurses	27.61 ± 1.73	-2.34	0.021*
	NHP	28.23 ± 1.44		
Orientation	Nurses	11.88 ± 0.38	5.73	< 0.001*
	NHP	11.35 ± 0.62		
Attention	Nurses	7.51 ± 0.76	0.79	0.433
	NHP	7.41 ± 0.76		
Comprehension	Nurses	5.54 ± 0.78	-0.53	0.594
	NHP	5.60 ± 0.55		
Repetition	Nurses	11.28 ± 1.39	-1.48	0.140
	NHP	11.56 ± 0.91		
Naming	Nurses	7.65 ± 0.72	-1.09	0.279
	NHP	7.76 ± 0.56		
Construction	Nurses	4.67 ± 1.02	-3.91	< 0.001*
	NHP	5.32 ± 0.98		
Memory	Nurses	10.65 ± 2.35	-1.70	0.090
	NHP	11.17 ± 1.41		
Calculation	Nurses	3.42 ± 1.39	-1.18	0.239
	NHP	3.62 ± 0.71		
Similarities	Nurses	6.82 ± 1.35	-2.26	0.025*
	NHP	7.27 ± 1.04		
Judgement	Nurses	4.68 ± 0.81	1.74	0.084
	NHP	4.42 ± 0.96		
Cognistat Total†	Nurses	74.11 ± 5.21	-1.92	0.056
	NHP	75.49 ± 3.62		

Table 3.3 presents the mean scores for the Mini-Mental State Exam and the Cognistat (including its domains) of both sample groups. Furthermore, t and p values are provided for intergroup comparisons for each of these variables.

Key: n = Sample size; NHP = Non-health professionals; * = Statistical significance; † = Measure of global cognitive performance; < = Less than

As a computerised format of the Stroop test was utilised in the present experiment, it was possible to capture data regarding individual performance, and the two variables captured were the number of tests passed, and average response time (Table 3.4).

Regarding the number of tests passed, the non-health professional sample group performed significantly better ($p < 0.001$) than the nurse sample group, scoring 253.06 ± 46.79 compared to 193.02 ± 51.26. Similarly, the average response time of the non-health professional group was significantly better ($p < 0.001$), returning a value of 1260.99 ± 341.82 milliseconds compared to the 1566.98 ± 426.64 milliseconds associated with the nurse participants.

Table 3.4 – The Stroop test performance outcomes of both of study sample groups

Variable	Group	Value	t	p
Tests Passed	Nurses	193.02 ± 51.26	-7.14	< 0.001*
	NHP	253.06 ± 46.79		
Response Time (ms)	Nurses	1566.98 ± 426.64	4.69	< 0.001*
	NHP	1260.99 ± 341.82		

Table 3.4 displays the mean values recorded for the two Stroop performance variables (response time and number of test passed) for both sample groups. Furthermore, t and p values are provided for intergroup comparisons for each of these variables.

Key: ms = Milliseconds; n = Sample size; NHP = Non-health professionals; * = Statistical significance; < = Less than

Furthermore, in both sample groups, age was significantly correlated to both of the Stroop performance variables (Table 3.5), namely, the average response time ($r = 0.54$; $p < 0.001$ and $r = 0.62$; $p < 0.001$ for nurses and non-health professionals, respectively), and the number of tests passed ($r = -0.50$; $p < 0.001$ and $r = -0.57$; $p < 0.001$ for nurses and non-health professionals, respectively).

Table 3.5 – The associations between age and Stroop test performance in the two study sample groups

Dependent Variable	Independent Variable	Sample Group	n	r	p
Age (Years)	Response Time (ms)	Nurses	55	0.54	< 0.001*
		NHP	85	0.62	< 0.001*
	Tests Passed	Nurses	55	-0.50	< 0.001*
		NHP	85	-0.57	< 0.001*

Table 3.5 presents the correlations between age, and the two Stroop performance variables (response time and number of tests passed) for both sample groups.

Key: ms = Milliseconds; n = Sample size; NHP = Non-health professionals; * = Statistical significance; < = Less than

3.3.2.2 Electroencephalography

Regarding electroencephalography, the present experiment used both time domain and frequency domain variables as physiological measures of individual cognitive performance.

With respect to time domain parameters (Figure 3.4), it was found that complexity at the central location differed significantly between the baseline (0.21 ± 0.05) and active phase (0.22 ± 0.06) for the nurse sample group ($p = 0.023$). Similarly, in the non-health professional sample group, complexity at the central location was also significantly different across the experimental phases (0.20 ± 0.04 vs 0.21 ± 0.05; $p = 0.023$). Additionally, activity at the frontal pole (67.92 ± 66.38 vs 109.33 ± 176.92) and mobility at the central location (0.10 ± 0.03 vs 0.11 ± 0.03) also significantly differed for the non-health professional group ($p = 0.030$, and $p = 0.026$ respectively).

Figure 3.4 – The average time domain electroencephalography values of both sample groups for the two experimental phases

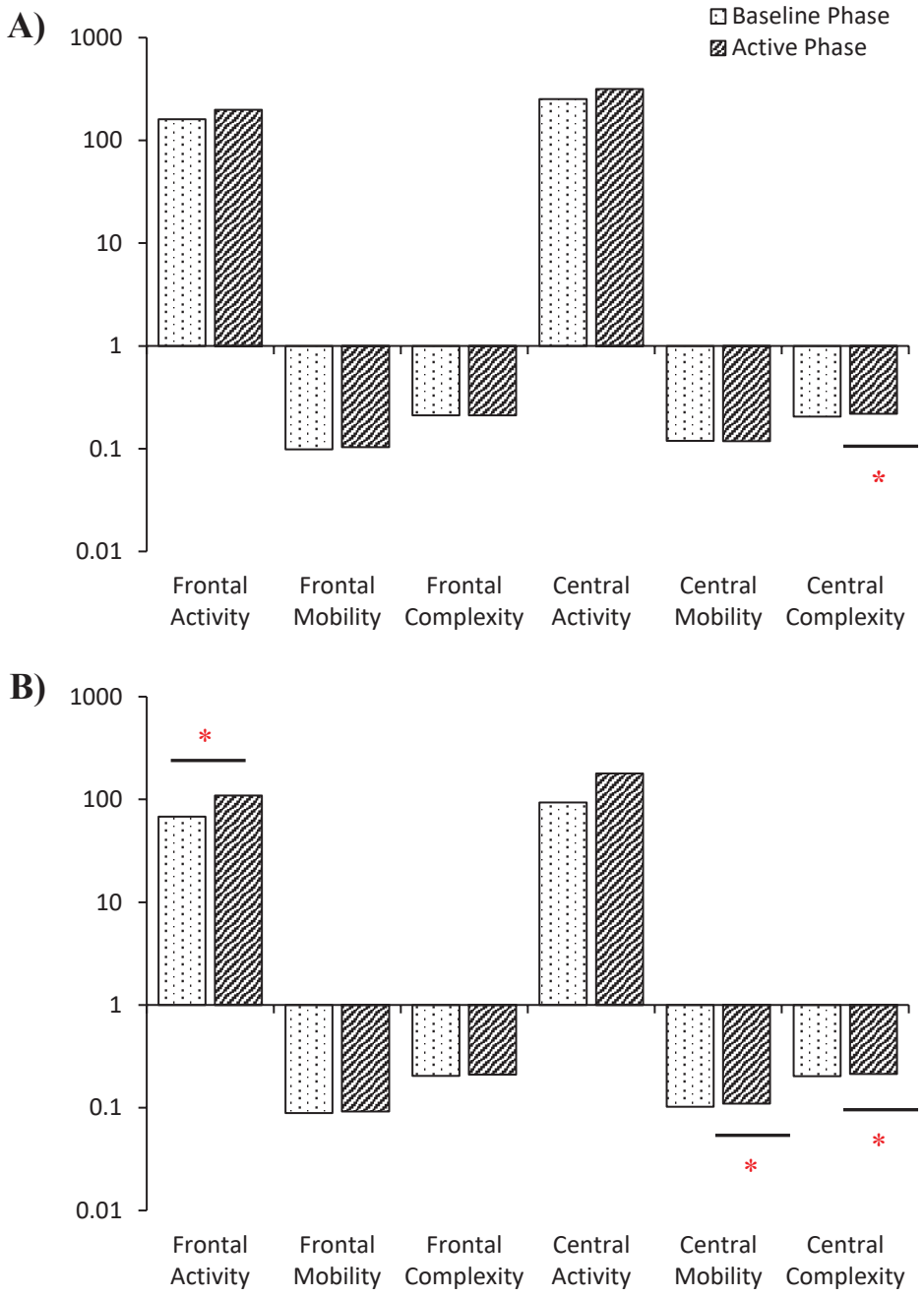

Figure 3.4 presents the mean value of each of the Hjorth parameter variables investigated. Plot A displays the values associated with the nurse participants (n = 57), whilst Plot B displays the values associated with the non-health professional participants (n = 93).

Key: * = $p < 0.05$;

Similarly, a number of frequency domain EEG variables also differed significantly between the two experimental phases (Figure 3.5). In the nurse sample group, frontal pole alpha activity was the only variable that significantly differed ($p < 0.001$), with the active phase ($26.85 \pm 52.10\ \mu V/s^2$) showing a greater value than the baseline phase ($19.10 \pm 27.07\ \mu V/s^2$).

Conversely, in the non-health professional sample group, a number of frequency domain variables were found to differ significantly (Figure 3.5). The active phase data were higher than baseline data for: frontal pole delta activity (21.30 ± 55.14 vs $8.42 \pm 9.70\ \mu V/s^2$; $p = 0.023$), average delta activity (13.04 ± 27.84 vs $6.01 \pm 7.46\ \mu V/s^2$; $p = 0.016$), frontal pole theta activity (8.14 ± 14.52 vs $4.66 \pm 4.81\ \mu V/s^2$; $p = 0.019$), average theta activity (6.15 ± 8.17 vs $3.89 \pm 2.89\ \mu V/s^2$; $p = 0.008$), frontal pole alpha activity (3.20 ± 4.35 vs $1.86 \pm 1.12\ \mu V/s^2$; $p = 0.002$), frontal pole beta activity (10.98 ± 13.61 vs $7.26 \pm 9.22\ \mu V/s^2$; $p = 0.009$), and frontal pole gamma activity (5.69 ± 8.36 vs $4.07 \pm 6.07\ \mu V/s^2$; $p = 0.004$). Finally, it was found that central alpha activity was significantly higher ($p = 0.047$) in the baseline phase ($8.12 \pm 10.10\ \mu V/s^2$) than the active phase ($3.88 \pm 6.68\ \mu V/s^2$).

Figure 3.5 – The average frequency domain electroencephalography power values of both sample groups for the two experimental phases

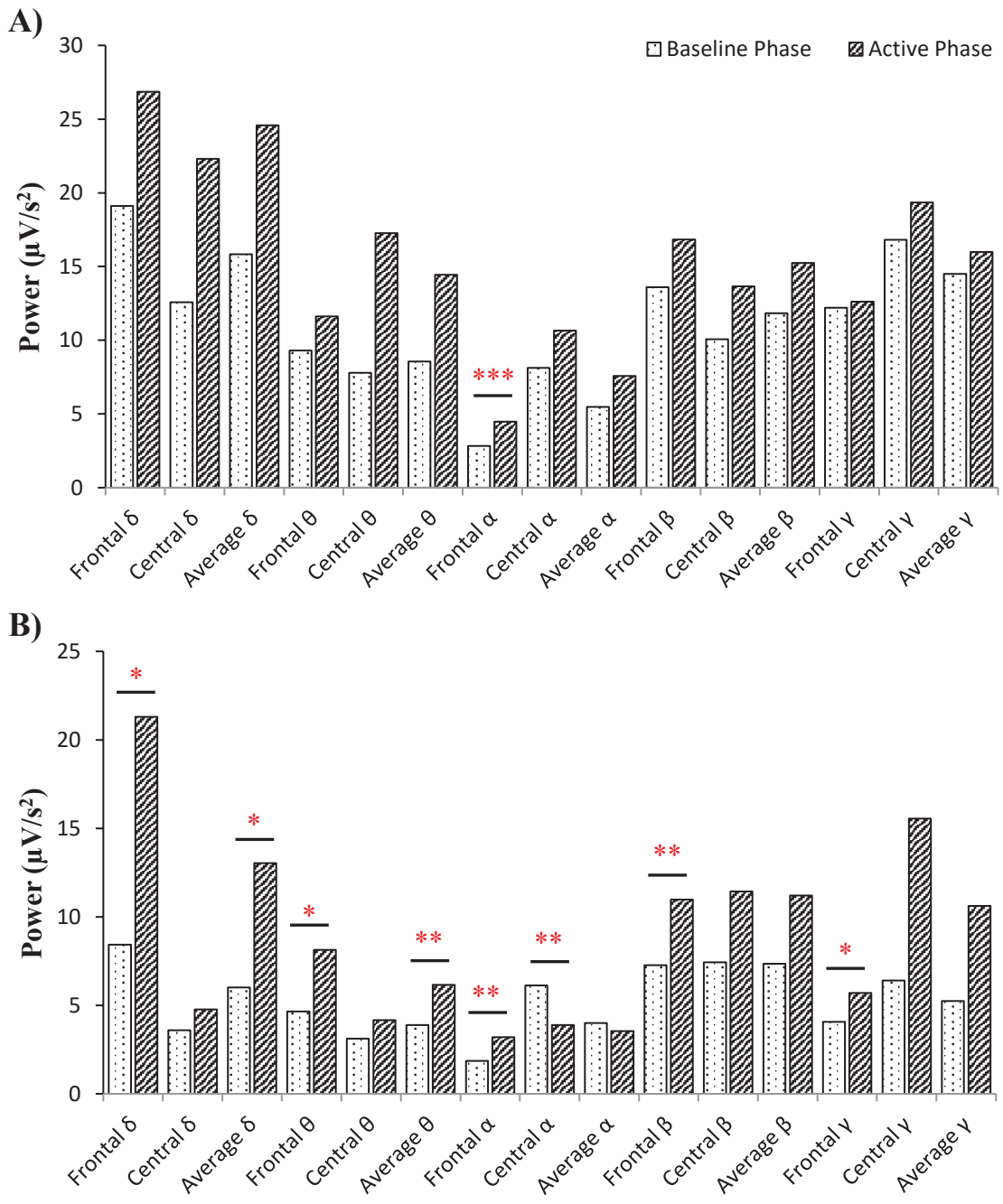

Figure 3.5 presents the mean power value of each of the frequency domain electroencephalography variables investigated are shown. Plot A displays the values associated with the nurse participants (n = 57), whilst Plot B displays the values associated with the non-health professional participants (n = 93).

Key: n = Sample size; α = Alpha; β = Beta; δ = Delta; γ = Gamma; θ = Theta; * = $p < 0.05$; ** = $p < 0.01$; *** = $p < 0.001$

Additionally, principal component analysis was utilised to provide further information regarding both the time domain and frequency domain electroencephalography variables.

Interestingly, in the non-health professional group, the original 21 variables produced 21 principal components, the first 9 of which together represented 95.95% of the variance in the original dataset. The contributions of the original EEG variables to the reduced data set were examined and the five variables that contributed the most to each of the first 9 principal components (as determined by the absolute sum of their eigenvectors; in descending order) are presented in Table 3.6.

Table 3.6 – The top 5 contributing electroencephalography variables per principal component in the non-health professional group

Rank	PC 1	PC 2	PC 3	PC 4	PC 5	PC 6	PC 7	PC 8	PC 9
1	Av-θ	Fp-δ	C-Mob	Fp-γ	Fp-γ	C-Act	C-δ	C-β	Fp-Mob
2	Av-α	Fp-θ	C-Com	Av-β	Fp-β	C-Mob	C-γ	Av-α	C-Com
3	Fp-Act	Av-δ	Fp-Mob	Fp-β	Av-γ	Av-α	Av-γ	C-α	Fp-Com
4	Av-δ	C-α	C-γ	C-β	C-β	C-α	Fp-β	Av-β	C-Mob
5	Fp-α	Fp-Act	Fp-Com	C-δ	C-γ	C-δ	C-Mob	C-δ	Fp-α

Table 3.6 displays the 5 electroencephalography variables that contributed the most to the first 9 principal components, which together represent 95.95% of the variance in the original dataset. The columns represent each principle component, and the rows represent the top 5 variables respectively. These variables were selected according to the absolute sum of their eigenvector which was generated during the computation of the principle components, where the highest values were selected.

Key: Act = Activity; Av = Average; C = Central; Com = Complexity; Fp = Frontal pole; Mob = Mobility; PC = Principal component; α = Alpha; β = Beta; δ = Delta; γ = Gamma; θ = Theta

Likewise, for the nurse group, the original 21 variables produced 21 principal components, the first 7 of which represented 95.07% of the variance in the original data. The contributions of the original EEG variables to the reduced data set were examined and the five variables that contributed the most to each of the first 7 principal components (as determined by the absolute sum of their eigenvectors; in descending order) are presented in Table 3.7.

Table 3.7 – The top 5 contributing electroencephalography variables per principal component in the nurse group

Rank	PC 1	PC 2	PC 3	PC 4	PC 5	PC 6	PC 7
1	Av-β	C-Mob	Fp-δ	C-γ	Fp-γ	Fp-α	Fp-β
2	Av-θ	C-Com	Fp-θ	Av-γ	Fp-β	Fp-Act	Fp-γ
3	C-β	Fp-Com	Fp-Mob	C-Act	Fp-Com	Av-δ	Fp-δ
4	Av-α	Fp-Act	Fp-α	C-α	C-Com	C-β	Av-δ
5	Av-δ	Fp-Mob	Fp-Act	Av-α	Av-β	Av-β	Fp-Act

Table 3.7 presents the 5 electroencephalography variables that contributed the most to the first 9 principal components, which together represent 95.07% of the variance in the original dataset. The columns represent each principle component, and the rows represent the top 5 variables respectively. These variables were selected according to the absolute sum of their eigenvector which was generated during the computation of the principle components,

Key: Act = Activity; Av = Average; C = Central; Com = Complexity; Fp = Frontal pole; Mob = Mobility; PC = Principal component; α = Alpha; β = Beta; δ = Delta; γ = Gamma; θ = Theta

3.3.3 Stress

In terms of self-reported stress scores (Table 3.8), it was found that the nurse group scored 21.19 ± 8.99 and 10.82 ± 8.69 for the LAQ and DASS, respectively. Furthermore, both scores were greater in magnitude than the scores of their non-health professional counterparts (18.48 ± 8.97 and 6.60 ± 5.42, respectively), although only the score of the DASS differed significantly ($p = 0.005$). However, DASS data were only

available for 22 nurse participants, compared to 93 non-health professionals, and this disparity may contribute to the significance found.

Table 3.8 – The self-reported stress scores of the two study sample groups

Variable	Group	n	Value	t	p
LAQ P2	Nurses	57	21.19 ± 8.99	1.79	0.075
	NHP	93	18.48 ± 8.97		
DASS-S	Nurses	22	10.82 ± 8.69	2.88	0.005*
	NHP	93	6.60 ± 5.42		

Table 3.8 presents the mean self-report stress scores of both sample groups for both the LAQ and the DASS. Furthermore, t and p values are provided for intergroup comparisons for each of these variables.

Key: DASS = Depression, Anxiety, Stress Scale; LAQ = Lifestyle Appraisal Questionnaire; NHP = Non-health professional; P2 = Part 2; S = Stress; * = Statistical significance

3.3.3.1 Non-health professionals

Regarding the associations between stress and demographic variables in the non-health professional group (Table 3.9), it was found that stress (as measured by part 2 of the LAQ) was significantly and positively correlated with fatigue state both before ($r = 0.37$; $p < 0.001$) and after ($r = 0.21$; $p = 0.043$) the experimental protocol, as well as lifestyle risk factors ($r = 0.35$; $p = 0.001$). Moreover, it is important to note that stress (as measured by the LAQ) was not significantly associated with age, BMI or Waist to Hip ratio.

Table 3.9 – The associations between stress and demographic variables of the non-health professional group

Dependent Variable	Independent Variable	n	r	p
Stress (LAQ P2)	Age	93	-0.17	0.114
	BMI	93	0.03	0.796
	WHR	93	0.09	0.381
	Pre-FSQ	93	0.37	< 0.001*
	Post-FSQ	93	0.21	0.043*
	LAQ P1	93	0.35	0.001*
Stress (DASS-S)	Age	93	-0.05	0.615
	BMI	93	0.03	0.744
	WHR	93	0.02	0.865
	Pre-FSQ	93	0.38	< 0.001*
	Post-FSQ	93	0.31	0.003*
	LAQ P1	93	0.39	< 0.001*

Table 3.9 displays the correlations between stress, and demographic variables including age, BMI, WHR, fatigue, and lifestyle risk factors (LAQ P1) in the non-health professional group.

Key: BMI = Body Mass Index; FSQ = Fatigue State Question; LAQ = Lifestyle Appraisal Questionnaire; P1 = Part 1; P2 = Part 2; S = Stress; WHR = Waist/Hip Ratio; * = Statistical significance; < = Less than

Moving to the second measure of stress, the stress score of the DASS was positively correlated (Table 3.9) with fatigue state, both before ($r = 0.38$; $p < 0.001$) and after ($r = 0.31$; $p = 0.003$) the experimental protocol, as well as lifestyle risk factors ($r = 0.39$; $p < 0.001$). Further, age, BMI, and Waist to Hip ratio were again not significantly correlated to stress (as measured by the DASS).

Examining the association between stress (as measured by part 2 of the LAQ) and the other negative mental state scores (Table 3.10), a strong positive relationship for both the anxiety score ($r = 0.53$; $p < 0.001$), and stress score of the DASS ($r = 0.65$; $p < 0.001$) was identified. Similarly, stress (as measured by the DASS) demonstrated a strong positive correlation (Table 3.10) to anxiety score ($r = 0.73$; $p < 0.001$).

Table 3.10 – The associations between stress and self-reported negative mental state scores of the non-health professional group

Dependent Variable	Independent Variable	n	r	p
Stress (LAQ P2)	DASS-A	93	0.53	< 0.001*
	DASS-S	93	0.66	< 0.001*
Stress (DASS-S)	DASS-A	93	0.73	< 0.001*

Table 3.10 displays the correlations between stress, and the other self-reported negative mental state scores for the non-health professional group.

Key: A = Anxiety; DASS = Depression, Anxiety, Stress Scale; LAQ = Lifestyle Appraisal Questionnaire; P2 = Part 2; S = Stress; * = Statistical significance; < = Less than

Stress score (as measured by part 2 of the LAQ) of the non-health professional group was significantly and positively correlated with the coping strategy categories of self-blame (r = 0.30; p = 0.003), wishful thinking (r = 0.29; p = 0.005), and avoidance (r = 0.31; p = 0.003; Table 3.11). Further, the stress score from the DASS was also positively correlated to the self-blame (r = 0.27; p = 0.009) and avoidance categories (r = 0.21; p = 0.046).

Table 3.11 – The associations between stress and self-reported coping strategy utilisation of the non-health professional group

Dependent Variable	Independent Variable	n	r	p
Stress (LAQ P2)	Problem Focused	93	0.03	0.746
	Self-support	93	0.06	0.587
	Self-blame	93	0.30	0.003*
	Wishful Thinking	93	0.29	0.005*
	Avoidance	93	0.31	0.003*
Stress (DASS-S)	Problem Focused	93	0.07	0.501
	Self-support	93	0.12	0.253
	Self-blame	93	0.27	0.009*
	Wishful Thinking	93	0.19	0.069
	Avoidance	93	0.21	0.046*

Table 3.11 displays the correlations between stress and the categories of coping strategy examined by the Ways of Coping checklist for the non-health professional group.

Key: DASS = Depression, Anxiety, Stress Scale; LAQ = Lifestyle Appraisal Questionnaire; n = Sample size; P2 = Part 2; S = Stress; * = Statistical significance

In the analysis of cognitive performance outcomes, it was found that stress (as measured by the part 2 of the LAQ) was not associated with either average response time, or the total number of tests passed on the Stroop test (Table 3.12). Moreover, stress (as measured by the DASS) was similarly not significantly associated with either Stroop performance variable.

Table 3.12 – The associations between stress and Stroop test performance outcomes of the non-health professional group

Dependent Variable	Independent Variable	n	r	p
Stress (LAQ P2)	Response Time (ms)	85	-0.08	0.501
	Tests Passed	85	0.09	0.429
Stress (DASS-S)	Response Time (ms)	85	-0.13	0.258
	Tests Passed	85	0.15	0.165

Table 3.12 displays the partial correlations (controlling for age and BMI) between stress, and both of the Stroop performance variables (response time and number of tested passed) in the non-health professional group.

Key: BMI = Body Mass Index; DASS = Depression, Anxiety, Stress Scale; LAQ = Lifestyle Appraisal Questionnaire; ms = Milliseconds; n = Sample size; S = Stress; P2 = Part 2

With respect to the cognitive performance as assessed by the MMSE and Cognistat (Table 3.13), stress (as measured by part 2 of the LAQ) was significantly correlated to only domain specific performance in the language domain of repetition (r = 0.21, p = 0.043). All other global and domain specific variables of the MMSE and Cognistat were not significantly correlated to stress, as measured by part 2 of the LAQ.

Table 3.13 – The associations between stress (as measured by LAQ P2) and cognitive performance variables of the non-health professional group

Dependent Variable	Independent Variable	n	r	p
Stress (LAQ P2)	MMSE	93	0.05	0.665
	Cognistat Total	93	< 0.01	0.985
	Orientation	93	< -0.01	0.951
	Attention	93	-0.06	0.577
	Comprehension	93	-0.07	0.488
	Repetition	93	0.21	0.043*
	Naming	93	0.05	0.614
	Construction	93	-0.10	0.356
	Memory	93	-0.08	0.456
	Calculation	93	0.10	0.334
	Similarities	93	-0.03	0.746
	Judgement	93	0.04	0.737

Table 3.13 displays the partial correlations (controlling for age and BMI) between stress (as scored by the LAQ P2) and the global and domain specific cognitive performance variables of the MMSE and the Cognistat in the non-health professional group.

Key: BMI = Body Mass Index; LAQ = Lifestyle Appraisal Questionnaire; MMSE = Mini-Mental State Exam; P2 = Part 2; * = Statistical significance

Furthermore, stress (as measured by the DASS) was significantly correlated to only memory domain performance (r = -0.22; p = 0.033; Table 3.14). All other global and domain specific variables of the MMSE and Cognistat were not significantly correlated to the DASS stress score.

Table 3.14 – The associations between stress (as measured by DASS-S) and cognitive performance variables of the non-health professional group

Dependent Variable	Independent Variable	n	r	p
Stress (DASS-S)	MMSE	93	-0.10	0.329
	Cognistat Total	93	-0.01	0.227
	Orientation	93	-0.09	0.383
	Attention	93	-0.01	0.346
	Comprehension	93	-0.023	0.818
	Repetition	93	0.03	0.774
	Naming	93	-0.03	0.780
	Construction	93	-0.10	0.331
	Memory	93	-0.22	0.033*
	Calculation	93	0.01	0.912
	Similarities	93	-0.05	0.651
	Judgement	93	0.13	0.214

Table 3.14 displays the partial correlations (controlling for age and BMI) between stress (as scored by the DASS) and the global and domain specific cognitive performance variables of the MMSE and the Cognistat in the non-health professional group.

Key: BMI = Body Mass Index; DASS = Depression, Anxiety, Stress Scale; MMSE = Mini-Mental State Exam; S = Stress * = Statistical significance

In terms of cognitive performance as assessed by EEG, a number of significant positive correlations were found between self-reported stress scores (as measured by part 2 of the LAQ) and electroencephalographic variables (Table 3.15). More specifically, active phase activity of the frontal poles (r = 0.25; p = 0.016), and reactivity activity of the frontal poles (r = 0.24; p = 0.020) were significantly and positively correlated to stress. Additionally, active phase frontal pole delta activity (r = 0.28; p = 0.007), average active phase delta activity (r = 0.27; p = 0.010), active phase frontal pole alpha activity (r = 0.21; p = 0.044); frontal pole delta reactivity (r = 0.29; p = 0.006), average delta reactivity (r = 0.28; p = 0.008), and frontal pole alpha reactivity (r = 0.23; p = 0.032) were significantly associated to stress score.

Table 3.15 – The associations between stress (as measured by LAQ P2) and electroencephalography variables of the non-health professional group

Dependent Variable	Independent Variable	n	r	p
Stress (LAQ P2)	Aδ-Fp	93	0.28	0.007*
	Aδ-Av	93	0.27	0.010*
	Aα-Fp	93	0.21	0.044*
	Rδ-Fp	93	0.29	0.006*
	Rδ-Av	93	0.28	0.008*
	Rα-Fp	93	0.23	0.032*
	AAct-Fp	93	0.25	0.016*
	RAct-Fp	93	0.24	0.020*

Table 3.15 displays the significant partial correlations (controlling for age and BMI) between stress (as measured by the LAQ P2) and time domain and frequency domain electroencephalography variables in the non-health professional group. All other EEG variables were not significantly correlated to stress (as measured by the LAQ P2) and are not presented in this table.

Key: A = Active; Act = Activity; Av = Average; BMI = Body Mass Index; Fp = Frontal pole; LAQ = Lifestyle Appraisal Questionnaire; n = Sample size; P2 = Part 2; R = Reactivity; α = Alpha; δ = Delta; * = Statistical significance

As multiple EEG variables were significantly associated to stress (as measured by part 2 of the LAQ P2), a forward stepwise regression analysis was performed to determined which variable was the strongest predictor of stress. The regression analysis retained two of the eight variables entered (active phase delta activity of the frontal pole, and average active phase delta activity), and had an overall significance of $p < 0.016$ (Table 3.16).

The two variables together explained 8.8% of the variance in perception of stress (as measured by part 2 of the LAQ) ($F = 4.34$; $DF = 2, 90$; $p < 0.016$; $R = 0.297$; $R^2 = 0.088$; $AR^2 = 0.068$). Furthermore, neither of the entered variables presented as independently significant predictors of stress.

Table 3.16 – Regression analysis for stress (LAQ-P2) and significantly correlated electroencephalography variables in the non-health professional group

$R = 0.297$, $R^2 = 0.088$, $AR^2 = 0.068$, $F(2,90) = 4.34$,
$p < 0.016$*, SE of Estimate = 8.66

Variable	β	SE of β	B	SE of B	t(90)	p	n
Intercept			18.35	1.17	15.68	< 0.001*	
Aδ-Fp	1.32	0.88	0.22	0.14	1.51	0.136	93
Aδ-Av	-1.06	0.88	-0.34	0.28	-1.21	0.231	93

Table 3.16 presents a stepwise forward regression analysis between stress (as measured by LAQ P2) and the significantly correlated EEG variables in the non-health professional group. Of the 8 EEG variables originally entered into the model, the analysis retained two: Aδ-F and Aδ-Av.

Key: A = Active; Av = Average; F = Frontal; LAQ = Lifestyle Appraisal Questionnaire; n = Sample size; P2 = Part 2; SE = Standard Error; δ = Delta; * = Statistical significance

Likewise, stress (as measured by the DASS) was positively correlated with seven electroencephalography variables (Table 3.17), including, active phase delta activity of the frontal poles (r = 0.24; p = 0.021); average delta activity (r = 0.24; p = 0.023); active phase alpha activity of the frontal poles (r = 0.22; p = 0.040); frontal pole delta reactivity (r = 0.26; p = 0.013); average delta reactivity (r = 0.28; p = 0.008); frontal pole theta reactivity (r = 0.21; p = 0.048); and frontal pole alpha reactivity (r = 0.25; p = 0.015). Furthermore, a single negative correlation between stress (as measured by the DASS) and reactivity mobility at the frontal poles (r = -0.21; p = 0.046) was also identified.

Table 3.17 – The associations between stress (as measured by DASS-S) and electroencephalography variables of the non-health professional group

Dependent Variable	Independent Variable	n	r	p
Stress (DASS-S)	Aδ-Fp	93	0.24	0.021*
	Aδ-Av	93	0.24	0.023*
	Aα-Fp	93	0.22	0.040*
	Rδ-Fp	93	0.26	0.013*
	Rδ-Av	93	0.28	0.008*
	Rθ-Fp	93	0.21	0.048*
	Rα-Fp	93	0.25	0.015*
	RMob-Fp	93	-0.21	0.046*

Table 3.17 displays the significant partial correlations (controlling for age and BMI) between stress (as measured by the DASS) and time domain and frequency domain electroencephalography variables in the non-health professional group. All other EEG variables were not significantly correlated to stress (as measured by the DASS) and are not presented in this table.

Key: A = Active; Av = Average; BMI = Body Mass Index; DASS = Depression, Anxiety, Stress Scale, Fp = Frontal pole; Mob = Mobility; n = Sample size; R = Reactivity; S = Stress; α = Alpha; δ = Delta; θ = Theta; * = Statistical significance

Additionally, because multiple EEG variables were significantly associated to stress (as measured by the DASS); a forward stepwise regression analysis was performed to determined which variable was the strongest predictor of stress (Table 3.18). The regression analysis retained two of the eight variables entered (average delta reactivity, and reactivity mobility at the frontal poles), and had an overall significance of $p < 0.021$.

The two variables together explained 8.3% of the variance in perception of stress (as measured by the DASS) ($F = 4.06$; $DF = 2, 90$; $p < 0.021$; $R = 0.288$; $R^2 = 0.083$; $AR^2 = 0.062$). Furthermore, average delta reactivity was found to be an independently significant predictor of stress ($p = 0.039$).

Table 3.18 – Regression analysis for stress (DASS-S) and significantly correlated electroencephalography variables in the non-health professional group

$R = 0.288$, $R^2 = 0.083$, $AR^2 = 0.062$, $F(2,90) = 4.06$,
$p < 0.021$*, SE of Estimate = 5.2519

Variable	β	SE of β	B	SE of B	t(90)	p	n
Intercept			6.38	0.52	11.16	< 0.001*	
Rδ-Av	0.22	0.11	0.04	0.021	2.10	0.039*	93
RMob-Fp	-0.13	0.11	-22.94	18.12	-1.27	0.209	93

Table 3.18 presents a stepwise forward regression analysis between stress (as measured by the DASS) and the significantly correlated physiological EEG variables in the non-health professional group. Of the 8 EEG variables originally entered into the model, the analysis retained two: Rδ-Av and RMob-Fp.

Key: Av = Average; DASS = Depression, Anxiety, Stress Scale; Fp = Frontal pole; Mob = Mobility; n = Sample size; R = Reactivity; S = Stress; SE = Standard Error; δ = Delta; * = Statistical significance

3.3.3.2 Nurses

Regarding the nurse group, it was found that stress scores (as measured by part 2 of the LAQ; Table 3.19) were significantly correlated to both age ($r = -0.26$, $p < 0.050$) and lifestyle risk factors ($r = 0.45$; $p < 0.001$). BMI, Waist to hip ratio, and both pre and post experimental protocol fatigue scores were not significantly associated with stress scores. Interestingly, stress (as measured by the DASS) was not significantly correlated to any demographic variable of the nurse sample group including age, BMI, Waist to Hip ratio, both pre and post experimental protocol fatigue scores, and lifestyle risk factors (Table 3.19).

Table 3.19 – The associations between stress (as measured by LAQ P2) and demographic variables of the nurse group

Dependent Variable	Independent Variable	n	r	p
Stress (LAQ P2)	Age	57	-0.26	< 0.050*
	BMI	57	0.10	0.480
	WHR	57	-0.15	0.262
	Pre-FSQ	57	0.09	0.488
	Post-FSQ	57	0.13	0.352
	LAQ P1	57	0.45	< 0.001*
Stress (DASS-S)	Age	21	-0.27	0.246
	BMI	21	-0.18	0.446
	WHR	21	-0.15	0.517
	Pre-FSQ	21	-0.09	0.694
	Post-FSQ	21	-0.23	0.310
	LAQ P1	21	0.13	0.576

Table 3.19 displays the correlations between stress, and demographic variables including age, BMI, WHR, fatigue, and lifestyle risk factors (LAQ P1) in the nurse group.

Key: BMI = Body Mass Index; FSQ = Fatigue State Question; LAQ = Lifestyle Appraisal Questionnaire; n = Sample size; P1 = Part 1; P2 = Part 2; S = Stress; WHR = Waist/Hip Ratio; * = Statistical significance; < = Less than

Unlike their non-health professional counterparts, the stress score (as measured by part 2 of the LAQ) of the nurse group was not significantly correlated to either anxiety scores, or the second measure of stress (Table 3.20). Similarly, the stress score of the DASS was not significantly correlated to anxiety score.

Table 3.20 – The associations between stress (as measured by LAQ P2) and self-reported negative mental state scores of the nurse group

Dependent Variable	Independent Variable	n	r	p
Stress (LAQ P2)	DASS-A	21	-0.13	0.570
	DASS-S	21	0.06	0.796
Stress (DASS-S)	DASS-A	21	0.29	0.198

Table 3.20 displays the correlations between stress, and the other self-reported negative mental state scores (DASS-A, and DASS-S) for the nurse group.

Key: A = Anxiety; BMI = Body Mass Index; DASS = Depression, Anxiety, Stress Scale; LAQ = Lifestyle Appraisal Questionnaire; n = Sample size; P2 = Part 2; S = Stress

It was found that the DASS stress score of the nurse group was positively correlated with the avoidance coping strategy category (r = 0.44; p = 0.049; Table 3.21), while other categories were not significantly correlated with either stress measure.

Table 3.21 – The associations between stress and self-reported coping strategy utilisation of the nurse group

Dependent Variable	Independent Variable	n	r	p
Stress (LAQ P2)	Problem Focused	21	0.18	0.426
	Self-support	21	-0.03	0.905
	Self-blame	21	-0.30	0.185
	Wishful Thinking	21	-0.10	0.677
	Avoidance	21	0.12	0.602
Stress (DASS-S)	Problem Focused	21	-0.12	0.604
	Self-support	21	-0.38	0.087
	Self-blame	21	0.29	0.199
	Wishful Thinking	21	0.21	0.356
	Avoidance	21	0.44	0.049*

Table 3.21 displays the correlations between stress and the categories of coping strategy examined by the Ways of Coping checklist in the nurse group.

Key: DASS = Depression, Anxiety, Stress Scale; LAQ = Lifestyle Appraisal Questionnaire; n = Sample size; P2 = Part 2; S = Stress; * = Statistical significance; < = Less than

In terms of stress (as measured by part 2 of the LAQ) and cognitive performance, it was determined that neither Stroop performance variable (average response time, or number of tests passed) was significantly correlated to stress (Table 3.22). Likewise, Stroop performance variables were also not significantly correlated to stress (as measured by the DASS) in the nurse group (Table 3.22).

Table 3.22 – The associations between stress (as measured by LAQ P2) and Stroop test performance outcomes of the nurse group

Dependent Variable	Independent Variable	n	r	p
Stress (LAQ P2)	Response Time (ms)	55	-0.05	0.746
	Tests Passed	55	0.04	0.788
Stress (DASS-S)	Response Time (ms)	21	-0.13	0.594
	Tests Passed	21	0.36	0.129

Table 3.22 displays the partial correlations (controlling for age and BMI) between stress, and both of the Stroop performance variables (response time and number of tested passed) in the nurse group.

Key: BMI = Body Mass Index; LAQ = Lifestyle Appraisal Questionnaire; ms = Milliseconds; n = Sample size; P2 = Part 2; S = Stress;

However, evaluation of the relationships between the global and domain specific cognitive performance variables of the MMSE and Cognistat showed better promise (Table 3.23). A significant positive relationship was identified between stress (as measured by part 2 of the LAQ) and performance in the memory domain (r = 0.27; p = 0.048). All other variables were not significantly correlated to stress (as measured by part 2 of the LAQ).

Table 3.23 – The associations between stress (as measured by LAQ P2) and cognitive performance variables of the nurse group

Dependent Variable	Independent Variable	n	r	p
Stress (LAQ P2)	MMSE	57	-0.19	0.164
	Cognistat Total	57	< 0.01	0.971
	Orientation	57	-0.09	0.498
	Attention	57	-0.24	0.072
	Comprehension	57	-0.07	0.597
	Repetition	57	0.03	0.855
	Naming	57	< 0.01	0.973
	Construction	57	0.05	0.724
	Memory	57	0.27	0.048*
	Calculation	57	-0.15	0.286
	Similarities	57	-0.02	0.904
	Judgement	57	-0.24	0.076

Table 3.23 displays the partial correlations (controlling for age and BMI) between stress (as scored by the LAQ P2) and the global and domain specific cognitive performance variables of the MMSE and the Cognistat in the nurse group.

Key: BMI = Body Mass Index; LAQ = Lifestyle Appraisal Questionnaire; MMSE = Mini-Mental State Exam; n = Sample size; P2 = Part 2; * = Statistical significance

In the nurse group, stress (as measured by the DASS) was not significantly correlated to any of the global or domain specific cognitive performance variables (Table 3.24).

Table 3.24 – The associations between stress (as measured by DASS-S) and cognitive performance variables of the nurse group

Dependent Variable	Independent Variable	n	r	p
Stress (DASS-S)	MMSE	21	0.31	0.202
	Cognistat Total	21	0.20	0.410
	Orientation	21	-0.14	0.560
	Attention	21	0.20	0.411
	Comprehension	21	0.35	0.139
	Repetition	21	0.08	0.760
	Naming	21	0.11	0.642
	Construction	21	0.28	0.251
	Memory	21	0.11	0.657
	Calculation	21	0.07	0.785
	Similarities	21	-0.02	0.933
	Judgement	21	-0.11	0.641

Table 3.24 displays the partial correlations (controlling for age and BMI) between stress (as scored by the DASS) and the global and domain specific cognitive performance variables of the MMSE and the Cognistat in the nurse group.

Key: BMI = Body Mass Index; DASS = Depression, Anxiety, Stress Scale; MMSE = Mini-Mental State Exam; n = Sample size; S = Stress;

Lastly, for cognitive performance outcomes, correlation analysis revealed only two significant relationships between stress (as measured by part 2 of the LAQ) and EEG variables (Table 3.25), implicating baseline frontal pole beta activity (r = 0.31; p = 0.023), and average baseline beta activity (r = 0.27; p = 0.045). There were no other significant correlations between stress (as measured by part 2 of the LAQ) and any other electroencephalography variable.

Table 3.25 – The associations between stress (as measured by LAQ P2) and electroencephalography variables of the nurse group

Dependent Variable	Independent Variable	n	r	p
Stress (LAQ P2)	Bβ-Fp	57	0.31	0.023*
	Bβ-Av	57	0.27	0.045*

Table 3.25 displays the significant partial correlations (controlling for age and BMI) between stress (as measured by the LAQ P2) and frequency domain electroencephalography variables that were used as physiological markers of cognitive performance in the nurse group. All other EEG variables were not significantly correlated to stress (as measured by the LAQ P2) and are not presented in this table.

Key: Av = Average; B = Baseline; BMI = Body Mass Index; Fp = Frontal pole; LAQ = Lifestyle Appraisal Questionnaire; n = Sample size; P2 = Part 2; β = Beta; * = Statistical significance

There were a number of positive significant correlations between EEG variables and stress (as measured by the DASS) (Table 3.26). These correlations implicated the frequency domain variables of: baseline central delta activity ($r = 0.56$; $p = 0.012$); baseline central theta activity ($r = 0.52$; $p = 0.027$); active phase central delta activity ($r = 0.77$; $p < 0.001$); active phase central theta activity ($r = 0.83$; $p < 0.001$); average active phase theta activity ($r = 0.80$; $p < 0.001$); active phase central alpha activity ($r = 0.83$; $p < 0.001$); average active phase alpha activity ($r = 0.82$; $p < 0.001$); active phase central beta activity ($r = 0.59$; $p = 0.010$); average active phase beta activity ($r = 0.56$; $p = 0.013$); active phase central gamma activity ($r = 0.71$; $p = 0.001$); average central gamma activity ($r = 0.68$; $p = 0.001$); central delta reactivity ($r = 0.55$; $p = 0.019$); central theta reactivity ($r = 0.83$; $p < 0.001$); average theta reactivity ($r = 0.74$; $p < 0.001$); central alpha reactivity ($r = 0.80$; $p < 0.001$); average alpha reactivity ($r = 0.77$; $p < 0.001$); central beta reactivity ($r = 0.51$; $p = 0.030$); average beta reactivity ($r = 0.50$; $p = 0.030$); central gamma reactivity ($r = 0.52$; $p = 0.025$); and lastly, average gamma reactivity ($r = 0.50$;$p = 0.029$). Three time domain variables were also found to be significantly and positively correlated to cognitive performance, including baseline central activity ($r = 0.66$; $p = 0.003$), active phase central activity ($r = 0.78$; $p < 0.001$); and the change in central activity ($r = 0.49$; $p = 0.037$).

Table 3.26 – The associations between stress (as measured by DASS-S) and electroencephalography variables of the nurse group

Dependent Variable	Independent Variable	n	r	p
Stress (DASS-S)	Bδ-C	20	0.56	0.012*
	Bθ-C	20	0.52	0.027*
	Aδ-C	20	0.77	< 0.001*
	Aθ-C	20	0.83	< 0.001*
	Aθ-Av	21	0.81	< 0.001*
	Aα-C	20	0.83	< 0.001*
	Aα-Av	21	0.82	< 0.001*
	Aβ-C	20	0.59	0.010*
	Aβ-Av	21	0.56	0.013*
	Aγ-C	20	0.71	0.001*
	Aγ-Av	21	0.68	0.001*
	Rδ-C	20	0.55	0.019*
	Rθ-C	20	0.83	< 0.001*
	Rθ-Av	21	0.74	< 0.001*
	Rα-C	20	0.80	< 0.001*
	Rα-Av	21	0.77	< 0.001*
	Rβ-C	20	0.51	0.030*
	Rβ-Av	21	0.50	0.030*
	Rγ-C	20	0.52	0.025*
	Rγ-Av	21	0.50	0.029*
	BAct-C	20	0.66	0.003*
	AAct-C	20	0.78	< 0.001*
	RAct-C	20	0.49	0.037*

Table 3.26 displays the significant partial correlations (controlling for age and BMI) between stress (as measured by the DASS) and the electroencephalography variables that were used as physiological markers of cognitive performance in the nurse group. All other EEG variables were not significantly correlated to stress (as measured by the DASS) and are not presented in this table.

Key: A = Active; Act = Activity; Av = Average; B = Baseline; BMI = Body Mass Index; C = Central; DASS = Depression, Anxiety, Stress Scale; n = Sample size; R = Reactivity; S = Stress; α = Alpha; β = Beta; δ = Delta; γ = Gamma; θ = Theta; * = Statistical significance; < = Less than

As there were multiple significant correlations between stress (as scored by the DASS) and EEG variables, a forward stepwise general linear regression analysis was performed. The regression analysis retained 2 of the 23 originally entered variables (central delta reactivity, and central theta reactivity), and had an overall significance of $p < 0.001$ (Table 3.27).

These two variables, together, explained 60.0% of the variance in stress scores ($F = 12.73$; $DF = 2$; $p < 0.001$; $R = 0.774$, $R^2 = 0.600$; $AR^2 = 0.553$). Both of the retained variables (central delta reactivity, and central theta reactivity) also presented as independently significant predictors of stress ($p = 0.036$ and $p < 0.001$, respectively).

Table 3.27 – Regression analysis for stress (DASS-S) and significantly correlated electroencephalography variables in the nurse group

			$R = 0.774$; $R^2 = 0.600$; $AR^2 = 0.553$; SSM = 891.00; dfM= 2; MSM = 445.50 SSR =594.75; dfR = 17; MSR = 34.99 $F = 12.73$; $p < 0.001$*			
Variable	**β**	**SE of β**	**B**	**SE of B**	**t**	**p**
Intercept			9.12	1.40	6.54	< 0.001*
Rδ – C	-0.73	0.32	-0.13	0.06	-2.27	0.036*
Rθ – C	1.33	0.32	0.35	0.09	4.15	< 0.001*

Table 3.27 displays a stepwise forward general linear regression analysis between stress (as measured by the DASS) and the significantly correlated physiological EEG variables in the nurse group. Of the 23 EEG variables originally entered into the model, the analysis retained two: Rδ – C and Rθ – C.

Key: C = Central; dfM = Degrees of freedom Model; dfR = Degrees of freedom Residual; DASS = Depression, Anxiety, Stress Scale; MSM = Mean squares Model; MSR = Mean squares Residual; n = Sample size; R = Reactivity; SE = Standard Error; SSM = Sum of squares Model; SSR = Sum of Squares Residual; δ = Delta; θ = Theta; * = Statistical Significance; < = Less than

3.3.4 Anxiety

With respect to self-reported anxiety scores (Table 3.28), it was found that the nurse group scored significantly higher on the DASS ($p < 0.001$) than their non-health professional counterparts; returning a score of 6.00 ± 5.03 compared to 2.99 ± 3.02. It must be stated that DASS data were only available for 22 nurse participants compared to 93 non-health professionals, and this disparity may contribute to the significance found.

Table 3.28 – The self-reported anxiety scores of the two study sample groups

Variable	Group	n	Value	t	p
Anxiety (DASS-A)	Nurses	22	6.00 ± 5.03	3.65	< 0.001*
	NHP	93	2.99 ± 3.02		

Table 3.28 presents the mean self-report anxiety scores of both sample groups for the DASS. Furthermore, t and p values are provided for intergroup comparisons for each of these variables.

Key: A = Anxiety; DASS = Depression, Anxiety, Stress Scale; n = Sample size; NHP = Non-health professionals; * = Statistical significance; < = Less than

3.3.4.1 Non-health professionals

In the non-health professional group, it was found that anxiety was significantly and positively associated (Table 3.29) with fatigue prior to the experimental protocol ($r = 0.30$; $p = 0.004$) and lifestyle risk factors ($r = 0.37$; $p < 0.001$). However, anxiety was not significantly correlated to age, BMI, or Waist to Hip ratio.

Table 3.29 – The associations between anxiety (as measured by DASS-A) and demographic variables of the non-health professional group

Dependent Variable	Independent Variable	n	r	p
Anxiety (DASS-A)	Age	93	-0.05	0.623
	BMI	93	0.12	0.267
	WHR	93	0.12	0.266
	Pre-FSQ	93	0.30	0.004*
	Post-FSQ	93	0.19	0.064
	LAQ P1	93	0.37	< 0.001*

Table 3.29 displays the correlations between anxiety, and demographic variables (age, BMI, WHR, fatigue, and lifestyle risk factors (LAQ P1)) for the non-health professional group.

Key: A = Anxiety; BMI = Body Mass Index; DASS = Depression, Anxiety, Stress Scale; FSQ = Fatigue State Question; LAQ = Lifestyle Appraisal Questionnaire; n = Sample size; P1 = Part 1; WHR = Waist/Hip Ratio; * = Statistical significance; < = Less than

The correlation analysis identified positive relationships (Table 3.30) between anxiety scores of the non-health professional group, and stress scores, as measured by part 2 the LAQ ($r = 0.53$; $p < 0.001$) and the DASS ($r = 0.73$; $p < 0.001$).

Table 3.30 – The associations between anxiety (as measured by DASS-A) and self-reported stress scores of the non-health professional e group

Dependent Variable	Independent Variable	n	r	p
Anxiety (DASS-A)	LAQ P2	93	0.53	< 0.001*
	DASS-S	93	0.73	< 0.001*

Table 3.30 displays the correlations between anxiety, and self-reported stress scores (LAQ P2 and DASS-S) for the non-health professional group.

Key: A = Anxiety; DASS = Depression, Anxiety, Stress Scale; LAQ = Lifestyle Appraisal Questionnaire; n = Sample size; P2 = Part 2; S = Stress; * = Statistical significance; < = Less than

The present analysis significantly and positively correlated the anxiety score of the non-health professional group with the coping strategy categories of self-blame ($r = 0.29$; $p = 0.004$), wishful thinking ($r = 0.24$; $p = 0.019$) and avoidance ($r = 0.29$; $p = 0.004$; Table 3.31).

Table 3.31 - The associations between anxiety and self-reported coping strategy utilisation of the non-health professional group

Dependent Variable	Independent Variable	n	r	p
Anxiety (DASS-A)	Problem Focused	93	0.10	0.319
	Self-support	93	0.12	0.251
	Self-blame	93	0.29	0.004*
	Wishful Thinking	93	0.24	0.019*
	Avoidance	93	0.29	0.004*

Table 3.31 displays the correlations between anxiety and the categories of coping strategy examined by the Ways of Coping checklist for the non-health professional group.

Key: A = Anxiety; DASS = Depression, Anxiety, Stress Scale; n = Sample size; * = Statistical significance

In terms of Stroop test performance of non-health professionals (Table 3.32), it was found that anxiety score was not significantly correlated to either performance variable.

Table 3.32 – The associations between anxiety (as measured by DASS-A) and Stroop test performance outcomes of the non-health professional group

Dependent Variable	Independent Variable	n	r	p
Anxiety (DASS-A)	Response Time (ms)	85	0.03	0.823
	Tests Passed	85	0.10	0.374

Table 3.32 displays the partial correlations (controlling for age and BMI) between anxiety and the Stroop performance variables (response time and number of tested passed) of the non-health professional group.

Key: A = Anxiety; BMI = Body Mass Index; DASS = Depression, Anxiety, Stress Scale; ms = Milliseconds; n = Sample size; S = Stress

With respect to the psychometric cognitive performance of the non-health professional group (Table 3.33), only domain specific performance in the memory domain was significantly associated with anxiety scores (r = -0.21; p = 0.043). All other global and domain specific cognitive variables of the MMSE and Cognistat were not significantly correlated to anxiety.

Table 3.33 – The associations between anxiety (as measured by DASS-A) and cognitive performance variables of the non-health professional group

Dependent Variable	Independent Variable	n	r	p
Anxiety (DASS-A)	MMSE	93	-0.06	0.557
	Cognistat Total	93	-0.12	0.265
	Orientation	93	-0.13	0.208
	Attention	93	-0.05	0.628
	Comprehension	93	0.04	0.679
	Repetition	93	-0.05	0.642
	Naming	93	-0.07	0.543
	Construction	93	0.01	0.904
	Memory	93	-0.21	0.043*
	Calculation	93	0.03	0.807
	Similarities	93	0.02	0.832
	Judgement	93	-0.01	0.952

Table 3.33 displays the partial correlations (controlling for age and BMI) for the non-health professional group between anxiety and the global and domain specific cognitive performance variables of the MMSE and the Cognistat.

Key: A = Anxiety; BMI = Body Mass Index; DASS = Depression, Anxiety, Stress Scale; MMSE = Mini-Mental State Exam; n = Sample size; * = Statistical significance

Interestingly, anxiety scores for the non-health professional sample group had minimal relation to electroencephalographic variables (Table 3.34), with only a single frequency domain variable being implicated. This result was a negative relationship between anxiety and baseline frontal pole alpha activity (r = -0.21; p = 0.045). All other EEG variables were not significantly correlated to anxiety scores.

Table 3.34 – The associations between anxiety (as measured by DASS-A) and electroencephalography variables of the non-health professional group

Dependent Variable	Independent Variable	n	r	p
Anxiety (DASS-A)	Bα-Fp	93	-0.21	0.045*

Table 3.34 displays the significant partial correlations (controlling for age and BMI) for the non-health professional sample group between anxiety and electroencephalography variables. All other EEG variables were not significantly correlated to anxiety and are not presented in this table.

Key: A = Anxiety; B = Baseline; BMI = Body Mass Index; DASS = Depression, Anxiety, Stress Scale; Fp = Frontal pole; n = Sample size; α = Alpha; * = Statistical significance

3.3.4.2 Nurses

In the nurse group, anxiety was not significantly correlated to any demographic variable including age, BMI, Waist/Hip ratio, pre and post experimental protocol fatigue, and lifestyle risk factors (Table 3.35).

Table 3.35 – The associations between anxiety (as measured by DASS-A) and demographic variables of the nurse group

Dependent Variable	Independent Variable	n	r	p
Anxiety (DASS-A)	Age	21	-0.22	0.350
	BMI	21	-0.05	0.847
	WHR	21	0.15	0.520
	Pre-FSQ	21	-0.17	0.466
	Post-FSQ	21	0.05	0.840
	LAQ P1	21	-0.05	0.840

Table 3.35 displays the correlations between anxiety and demographic variables (age, BMI, WHR, fatigue, and lifestyle risk factors (LAQ P1)) for the nurse group.

Key: A = Anxiety; BMI = Body Mass Index; DASS = Depression, Anxiety, Stress Scale; FSQ = Fatigue State Question; LAQ = Lifestyle Appraisal Questionnaire; n = Sample size; P1 = Part 1; WHR = Waist/Hip Ratio

The anxiety score of the nurse group (Table 3.36) was not significantly correlated to stress scores as measured by part 2 of the LAQ and the DASS, a result that is in direct contrast with the non-health professional group.

Table 3.36 – The associations between anxiety and self-reported stress scores of the nurse group

Dependent Variable	Independent Variable	n	r	p
Anxiety (DASS-A)	LAQ P2	21	-0.13	0.570
	DASS-S	21	0.29	0.198

Table 3.36 displays the correlations between anxiety, and self-reported stress scores (LAQ P2 and DASS-S) for the nurse group.

Key: A = Anxiety; DASS = Depression, Anxiety, Stress Scale; LAQ = Lifestyle Appraisal Questionnaire; n = Sample size; P2 = Part 2; S = Stress;

It was also found that the anxiety score of the nurse group was not significantly correlated with any coping strategy categories from the Ways of Coping Checklist (Table 3.37).

Table 3.37 – The associations between anxiety and self-reported coping strategy utilisation of the nurse group

Dependent Variable	Independent Variable	n	r	p
Anxiety (DASS-A)	Problem Focused	21	0.30	0.183
	Self-support	21	0.09	0.685
	Self-blame	21	0.38	0.090
	Wishful Thinking	21	0.37	0.101
	Avoidance	21	0.35	0.120

Table 3.37 displays the correlations between anxiety and the categories of coping strategy examined by the Ways of Coping checklist for the nurse group.

Key: A = Anxiety; DASS = Depression, Anxiety, Stress Scale; n = Sample size

Correlation analysis examining Stroop performance variables (Table 3.38) indicated that anxiety scores for the nurse group were only significantly associated with the total number of tests passed (r =0.54; p = 0.018).

Table 3.38 – The associations between anxiety and Stroop test performance outcomes of the nurse group

Dependent Variable	Independent Variable	n	r	p
Anxiety (DASS-A)	Response Time (ms)	21	-0.26	0.277
	Tests Passed	21	0.54	0.018*

Table 3.38 displays the partial correlations (controlling for age and BMI) for the nurse group between anxiety, and both of the Stroop performance variables (response time and number of tested passed).

Key: A = Anxiety; BMI = Body Mass Index; DASS = Depression, Anxiety, Stress Scale; ms = Milliseconds; n = Sample size; * = Statistical significance

With respect to psychometric cognitive performance (Table 3.39), anxiety scores of the nurse group were significantly positively correlated to total MMSE score (r = 0.59, p = 0.008). Additionally, it should be noted that the global and domain specific variables of the Cognistat were not significantly correlated to anxiety.

Table 3.39 – The associations between anxiety and cognitive performance variables of the nurse group

Dependent Variable	Independent Variable	n	r	p
Anxiety (DASS-A)	MMSE	21	0.59	0.008*
	Cognistat Total	21	0.28	0.255
	Orientation	21	0.05	0.827
	Attention	21	0.15	0.533
	Comprehension	21	0.13	0.609
	Repetition	21	0.28	0.250
	Naming	21	0.17	0.493
	Construction	21	0.26	0.277
	Memory	21	0.15	0.538
	Calculation	21	-0.02	0.946
	Similarities	21	0.23	0.353
	Judgement	21	-0.20	0.424

Table 3.39 displays the partial correlations (controlling for age and BMI) for the nurse sample group between anxiety and the global and domain specific cognitive performance variables of the MMSE and the Cognistat.

Key: A = Anxiety; BMI = Body Mass Index; DASS = Depression, Anxiety, Stress Scale; MMSE = Mini-Mental State Exam; n = Sample size; * = Statistical significance

Regarding the physiological markers of cognitive performance (Table 3.40), two EEG variables were significantly positively correlated with anxiety score in the nurse group. The implicated variables were average baseline delta activity (r = 0.52, p = 0.023), and baseline central gamma activity (r = 0.54, p = 0.022). Additionally, average delta reactivity was significantly negatively associated with anxiety score (r = -0.53, p = 0.019). No other EEG variable was significantly associated with anxiety.

Table 3.40 – The associations between anxiety and electroencephalography variables of the nurse group

Dependent Variable	Independent Variable	n	r	p
Anxiety (DASS-A)	Bδ-Av	21	0.52	0.023*
	Bγ-C	20	0.54	0.022*
	Rδ-Av	21	-0.53	0.019*

Table 3.40 displays the significant partial correlations (controlling for age and BMI) for the nurse group between anxiety and electroencephalography variables. All other EEG variables were not significantly correlated to anxiety and are not presented in this table.

Key: A = Anxiety; Av = Average; B = Baseline; BMI = Body Mass Index; C = Central; DASS = Depression, Anxiety, Stress Scale; n = Sample size; δ = Delta; γ = Gamma; * = Statistical significance

As there were multiple significant correlations between anxiety and EEG variables, a forward stepwise regression analysis was performed to determine which was the strongest predictor of anxiety The regression analysis retained two of the three originally entered variables (average delta reactivity, and baseline central gamma activity), and had an overall significance of $p < 0.001$ (Table 3.41).

These two variables together explained 47.1% of the variance in anxiety scores ($F = 41.30$; $DF = 2, 17$; $p < 0.001$; $R = 0.686$; $R^2 = 0.471$; $AR^2 = 0.409$). Both variables also presented as independently significant predictors of anxiety ($p = 0.013$, and $p = 0.030$ for average delta reactivity, and baseline central gamma activity respectively).

Table 3.41 – Regression analysis for anxiety and significantly correlated electroencephalography variables in the nurse sample group

n = 20	$R = 0.686$, $R^2 = 0.471$, $AR^2 = 0.409$, $F(2, 17) = 41.30$, $p < 0.001$*, SE of Estimate = 3.27						
Variable	**β**	**SE of β**	**B**	**SE of B**	**t(17)**	**p**	**n**
Intercept	2.00		5.64	0.99	5.70	< 0.001*	
Rδ-Av	-0.49	0.18	-0.06	0.02	-2.78	0.013*	57
Bγ-C	0.42	0.18	0.06	0.02	2.36	0.030*	55

Table 3.41 displays a stepwise forward regression analysis between anxiety in the nurse group and the significantly correlated physiological EEG variables. Of the 3 EEG variables originally entered into the model, the analysis retained two: Rδ-Av and Bγ-C.

Key: A = Anxiety; Av = Average; B = Baseline; C = Central; DASS = Depression, Anxiety, Stress Scale; n = Sample size; R = Reactivity; SE = Standard Error; δ = Delta; γ = Gamma; * = Statistical significance; < = Less than

3.4 Discussion

The present study aimed to investigate the associations between stress, anxiety and cognitive performance in nurses; hypothesising that increased stress and anxiety levels would be associated with impaired global and domain specific cognitive performance, and alterations in frontal pole EEG activity of the beta and gamma frequency bands.

The main findings of the present research significantly associated stress with impaired memory performance and increased delta activity in non-health professionals, as well as increased memory performance, and increased delta, theta and beta activity in nurses. Further, anxiety was associated with impaired memory performance and increased alpha activity in non-health professionals, as well as improved global cognitive performance, and increased delta and gamma activity in nurses.

The following sections will provide a discussion of the present results in relation to the relevant aims, hypotheses, and published literature that has also reported on the cognitive effects of stress and anxiety.

3.4.1 Demographics

The present study captured data for a number of demographic variables, some of which differed significantly between the two sample groups. Age was one such variable with the nurse group reporting a higher average age. Additionally, the age of the nurse sample group was significantly correlated to stress score (as measured by part 2 of the LAQ). This correlation, combined with the known impact of aging on stress and anxiety (Hart and Charles, 2013, Scott et al., 2013), and the association between age and cognitive status (Morris and Price, 2001, Petersen et al., 2001) saw the present analysis control for age where possible.

Similarly, lifestyle risk factors associated with risk of disease development (part 1 of the LAQ) also differed between the sample groups, with the nurse group again reporting a higher score. However, the scores of both groups for part 1 of the LAQ were below the previously published normative score for part 1 of the LAQ (Craig et al., 1996); a score generated from/representing a large general population dataset. The present non-health professional group was relatively young, and in the recent past health literacy has improved and led to better outcomes (Nutbeam, 2000). Further, the nurse group is formally educated in human health and so it follows that both groups would score below normative data published 20 years prior. Additionally, the difference between the two groups could possibly be attributed to the shift-working lifestyle of the nurse sample group, which has been associated with poor lifestyle habits/outcomes (Harrington, 2001, Fido and Ghali, 2008).

3.4.2 Cognitive performance

3.4.2.1 Psychometric

With respect to the present psychometric assessment of cognitive performance, all scores, global and domain specific, for both sample groups were above their respective impairment thresholds (the test score below which potential cognitive impairment or degrees of cognitive impairment is indicated). In addition, some scores significantly differed between the two groups. It was found that the non-health professional group performed better on the MMSE and in the Construction and Similarities domains of the Cognistat, whilst the nurse participants outperformed in the Orientation domain of the Cognistat. These performance differences may be attributed to varying lengths of education, as it has been shown that formal education is associated with cognitive performance (Launer et al., 1999, Le Carret et al., 2003). However, this data was not

available in the present analysis, and should be captured in future work so as to control for this effect. Alternatively, it is possible that the everyday demands placed upon individuals of each sample group primes their performance in certain domains, for example, nurses frequently rely on understanding their immediate spatial and temporal environment, hence, it follows that they would score higher on an assessment of orientation.

3.4.2.2 Electroencephalography

The second cognitive assessment was time-domain EEG, where it was found that in the non-health professional group, both complexity at the central location and mobility at the central location significantly decreased between phases. Complexity at the central location similarly decreased in the nurse sample group as well. Mobility is a measure that can be conceived as the mean frequency of an EEG trace (Hjorth, 1970), and hence would indicate a centrally located shift towards the lower frequencies between the two experimental phases. Whereas, Complexity is a measure of the nature of an EEG trace in reference to a sine curve (Hjorth, 1970). The observed reduction in complexity could be attributed to a decrease in variation between the two phases, which could possibly be conceived as an increase in synchronicity across the central region in both sample groups; however more explicit examinations of the relationship between time-domain EEG parameters and synchronicity is required.

In the frequency domain of EEG, it was found that central alpha activity significantly decreased between the baseline and active phases in the non-health professional group. Additionally, frontal and average delta and theta activity, as well as frontal alpha, beta and gamma activity also significantly increased from the baseline to active phase in this sample group. Frontal alpha activity also showed a similar significant increase in the

nurse group. Furthermore, PCA identified frontal and average delta, theta and alpha activity to be some of the top contributing variables in both sample groups. Therefore, considering the Stroop intervention can be used to initiate cognitive processes (Moering et al., 2004, Uttl and Graf, 1997), it is conceivable that the observed activity increases are representative of processes engaged by the Stroop test, be it executive functions and the interference effect (Stroop, 1935), or the motor functions related to the mouse-click input method.

Indeed, previous research has found associated increases in frontal-central theta activity (Barwick et al., 2012) and changes in theta activity of the fronto-parietal network (Kovacevic et al., 2012) were related to the process of undertaking the Stroop test. Earlier research from Hanslmayr et al. (2008) has similarly reported increases in the theta frequency band, and also associated increased alpha activity with the Stroop interference effect. Research from Tassi et al. (2006), Carp and Compton (2009), and Compton et al. (2011) similarly suggests that increased alpha activity is associated with the Stroop interference effect, and further suggested that the alpha activity increase was associated with correct responses. This may explain the current results, as the present Stroop test was formatted so that the correct response was always eventually selected. Moreover, the beta frequency band has also been associated with the semantic interference effect of the Stroop test (Piai et al., 2012), and is possibly able to discriminate between the various interference effects of the Stroop test (Schack et al., 1999). Conversely, Ergen et al. (2014) indicated that the lower frequency bands were able to discriminate between the congruent and incongruent Stroop effects.

Therefore, it could be suggested that the broad frequency band changes, in particular those of the theta, alpha and beta band may represent the engagement of cognitive processes such as working memory, selective attention, and flexibility, associated with

the Stroop interference effect (Moering et al., 2004). As there was notable change in activity between phases, it can also be suggested that the Stroop intervention was effective in establishing an active phase of experimentation.

3.4.3 Stress

Literature has demonstrated that stress can impact an individual's cognitive performance, leading to both improvements (Beste et al., 2013, Bos et al., 2014) and impairments (Luethi et al., 2008, LeBlanc, 2009), and further, stress can degrade performance and quality of care provided by nurses (Tarnow-Mordi et al., 2000, Berland et al., 2008). In the present study, both stress outcome measures (the LAQ, and the DASS) demonstrated that nurses and non-health professionals experienced stress. With respect to the LAQ, the scores of both groups resided either in line with or below previously published age relevant norms (Craig et al., 1996). Conversely, the non-health professional group DASS score was below the age relevant norm, and the nurse group DASS score was score greater than the age relevant norm (Lovibond and Lovibond, 1995b). However, it is important to consider the limited DASS dataset available for the nurse population, as with more data the stress score may have fallen in line with age relevant norms. Importantly, despite the significant difference between the mean scores of the two sample groups and the variation in sample size, both scores still reside within a normal range (0 – 14; Lovibond and Lovibond (1995b)).

3.4.3.1 Stress and demographics

The relationship of a number of demographic variables to stress was investigated in the present analysis. Stress scores for both sample groups were positively correlated to lifestyle risk factors associated with disease development (part 1 of the LAQ). Further,

the stress score of the non-health professional group was similarly correlated with fatigue state, and the stress score (as determined by part 1 of the LAQ) of the nurse group were negatively correlated to age.

Allostatic overload refers to the burden of stress and any associated changes in personal behaviours/lifestyle factors (McEwen, 2008). Literature has demonstrated that factors such as smoking (Cohen and Lichtenstein, 1990, Steptoe et al., 1996), alcohol consumption (Hemmingsson and Lundberg, 1998, Armeli et al., 2000), obesity (Torres and Nowson, 2007), fatigue/sleep (Åkerstedt et al., 2007), and a number of other lifestyle factors (Maghout Juratli et al., 2011) all increase with greater stress. Moreover, stress can have a negative impact on lifestyle (Hatton et al., 1995), further reconciling the present results associating increased stress with increased lifestyle risk factors. The experience of stress varies over time, and literature suggests that as an individual ages their experience of stress and/or negative affect is tempered by their life experiences/circumstances (Hart and Charles, 2013, Scott et al., 2013), which may help explain the present negative association between stress and age.

The stress scores of the non-health professional group recorded in the present study were positively correlated with self-blame, wishful-thinking and avoidance coping, and the scores of the nurse group were positively correlated with avoidance only, and the limited availability of DASS data for the nurse group must be again considered. Negative coping strategies such as these are often considered ineffectual and possibly detrimental (Koeske et al., 1993, Blalock and Joiner, 2000, Dijkstra and Homan, 2016), whereby long term utilisation can worsen stress (Tattersall et al., 1999, Arnetz, 2001), and, as such, it follows that an increase in stress could be accompanied by an increase in these coping strategies.

Lastly, stress and anxiety are often considered inter-related negative mental states due to shared physiological processes, symptomology and frequent comorbidity (Lovibond and Lovibond, 1995b). In this study, positive correlations were found between both stress measures, as well as to the anxiety measure for the non-health professional group, reflecting this inter-related nature. Interestingly, in the nurse group neither stress measure was correlated to the other nor to the anxiety measure; which could possibly be the result of limited DASS dataset that was available for the nurse sample group.

3.4.3.2 Stress and cognitive performance

With respect to psychometric cognitive performance and stress, the LAQ stress score of the non-health professional group was positively correlated to repetition domain performance. Similar sentence or language repetition tasks are utilised in the examination of specific language impairment (Conti-Ramsden et al., 2001), and more broadly cognition (Nasreddine et al., 2005). It is possible to conceive such a sentence repetition task to be examining language specific working memory, and the role of working memory has been heavily implicated in language performance and disorders (Daneman and Merikle, 1996, Baddeley, 2003, Gathercole and Baddeley, 2014). Furthermore, the DASS stress score of the non-health professional group was negatively correlated to memory score, and the LAQ stress score of the nurse group were positively correlated to memory score; associating both an improvement and impairment in domain performance with rising stress.

Whilst literature previously suggested the relationship between stress and memory to be negatively linear (Kirschbaum et al., 1996, Sandström et al., 2005, Tollenaar et al., 2008, Wolf, 2008, Comijs et al., 2010), the more current and prevailing theory is that of an inverted-U shape function, whereby, low/high levels of stress impair, and

intermediate levels facilitate performance (Sandi, 2013). Indeed, a number of research studies have previously demonstrated enhanced performance across all categorisations of memory including declarative (Henckens et al., 2009, Bos et al., 2014), working (Lupien et al., 1999), and visuospatial memory (Human et al., 2013). Both Buchanan and Lovallo (2001) and Cahill et al. (2003) specified this relationship to memory encoding and long term recall performance of emotional salient stimuli. Lastly, Buchanan and Tranel (2008) demonstrated a biphasic response, associating elevated cortisol with reduced memory retrieval, and stress without cortisol elevation to enhanced retrieval, implicating glucocorticoid physiology in memory processes.

As such, it is possible that the present stress scores of the nurse group facilitated memory performance, and so it could be inferred that this relationship represents the experience of eustress, where stress optimises performance. Whilst the non-health professional group presented a score that impaired memory performance, potentially representing the construct of distress, where stress disrupts performance. However, as both sample groups experienced stress within a normal range (Craig et al., 1996, Lovibond and Lovibond, 1995b) additional confirmatory research, either experimentally manipulating stress or at high stress is required.

3.4.3.3 Stress, cognitive performance and electroencephalography

In the present analysis, the traditional psychometric measures of cognitive performance provided by the MMSE (Folstein et al., 1975) and the Cognistat (Mueller et al., 2007) were complemented by EEG. Additionally, frontal pole and average delta, theta and alpha activity of both sample groups were amongst the top contributing variables identified by PCA.

Stress of the non-health professional group was correlated with a number of EEG variables, primarily delta and alpha band activity, as well as theta band activity and the time domain variables of activity and mobility. Subsequent regression analysis further identified the importance of delta band activity and the time domain variable of mobility. Implicating increases in delta activity aligns well with previous EEG based cognitive research that has associated delta power increases with cognitively impaired states (e.g. MCI and AD) (Huang et al., 2000, Koenig et al., 2005, Babiloni et al., 2006b, Babiloni et al., 2008, Babiloni et al., 2010). Further, it has been suggested that degree of impairment may modulate the relationship of delta activity and cognitive impairment, where mild impairment demonstrates a decrease, and more complete impairments show an increase (Liddell et al., 2007), and as such, qualifying the degree of impairment witnessed may provide a greater understanding of the role of delta activity.

Examining the nurse group found stress to be broadly correlated with a large range of EEG variables, however, the delta, theta and beta frequency bands were demonstrated to be of particular importance via subsequent regression analysis. As previously discussed, increases in the delta frequency band have commonly been associated with cognitively impaired states (Koenig et al., 2005, Babiloni et al., 2006b, Rossini et al., 2007), thus, reconciling the present results of the nurse group. Similarly, the present implication of the theta band also reconciles with the literature, whereby cognitively impaired states have been associated with increases in theta activity (Huang et al., 2000, Brunovsky et al., 2003, Koenig et al., 2005, Trejo et al., 2005). Aurtenetxe et al. (2013) localised this relationship to the fronto-temporal and parietal regions of the brain in patients with MCI. This increase may further be associated with memory process activation in cognitive impairment (van der Hiele et al., 2007).

In the beta frequency band, a reduction in activity is typically associated with cognitive impairment (Stam et al., 2003, Pijnenburg et al., 2004, Missonnier et al., 2007). However, Brunovsky et al. (2003) demonstrated increases in lower beta band activity in cognitive impairment, and Lees et al. (2016) reported negative relationships between beta activity and calculation and memory performance. Further, Lee et al. (2010) positively correlated beta synchronisation to MMSE score in healthy individuals, yet found decreased beta synchronisation in patients with AD, demonstrating varying EEG profiles based on cognitive status. In addition, neuroplastic changes associated with stress related activation of the HPA axis (Cook and Wellman, 2004, Radley et al., 2004) may explain the contrast between the present results and existing literature. Indeed, previous research has suggested that neuronal recruitment in cognitive impairment commonly occurs (Bäckman et al., 1999, Cader et al., 2006) for memory and other cognitive related tasks (Grady et al., 1993). Hence, it is possible that these neuroplastic changes have overcome the typical beta activity reduction observed in cognitive impairment, however further research is required.

3.4.4 Anxiety

Research targeting anxiety has demonstrated that it generally impairs an individual's attention, working memory, and executive functions (Asmundson and Stein, 1994, Ashcraft, 2002, Boldrini et al., 2005), and it is possible for such impairments to impact nursing performance. With respect to the present anxiety scores; it was found that the nurse group reported a score significantly greater than their non-health professional counterparts, and greater than age relevant normative data (Lovibond and Lovibond, 1995b). Additionally, the average score of the non-health professional group resided below the age relevant normative data (Lovibond and Lovibond, 1995b). Furthermore,

it is important to note that despite these differences, the scores of both groups are still considered to be within a normal range (0 – 7; Lovibond and Lovibond (1995b)).

3.4.4.1 Anxiety and demographics

The present analysis investigated a number of demographics variables with the aim of examining their relationship with anxiety. In the non-health professional group, anxiety was associated with lifestyle risk factors (part 1 of the LAQ), and also both stress measures, demonstrating the interconnected nature of stress and anxiety (Lovibond and Lovibond, 1995a). The present positive correlation between anxiety and lifestyle risk factors also follows convention (Mendlowicz and Stein, 2000). Indeed, research has demonstrated that individuals with anxiety have an elevated BMI (Strine et al., 2008), and an increased likelihood of smoking (Lawrence et al., 2009), and comorbid diseases including hypertension (Player and Peterson, 2011), diabetes (Smith et al., 2013), and depression (Fava et al., 2000).

In addition, it was found that anxiety of the non-health professional group was positively associated with the coping strategies of self-blame, wishful thinking, and avoidance. These are a series of results that align with coping literature where negative coping strategies such as these, are considered ineffectual (Tyler and Cushway, 1992, Koeske et al., 1993, Rout and Rout, 1994, Arnetz, 2001) and may actually exacerbate anxiety (Tattersall et al., 1999, Arnetz, 2001). Interestingly, the anxiety score of the nurse group was not associated with any investigated demographic factor, including coping. With respect to non-coping demographic variables, the observed results may be related to the development and utilisation of nursing specific coping mechanisms that mitigate the impact of anxiety rather than eliminating its presence. Another possibility that may explain all results including those related to the coping variables is the lower

number of participants for which anxiety scores had been recorded, which may have limited the analysis.

3.4.4.2 Anxiety and cognitive performance

In terms of psychometric cognitive performance, anxiety of the non-health professional group was negatively associated with memory domain performance. This result is in consensus with the majority of existing literature, where such a relationship is a well-documented cognitive impact of anxiety (Savage et al., 1999, Savage et al., 2000, Ashcraft, 2002). Indeed, literature has further specified and implicated various categories of memory including visual memory (Lucas et al., 1991), verbal memory and short-delay free recall (Asmundson and Stein, 1994), as well as working (Ashcraft and Kirk, 2001), spatial (Boldrini et al., 2005), episodic (Airaksinen et al., 2005) and delayed memory functions (Mantella et al., 2007). Hence, it could be suggested that the observed association between anxiety and memory is most likely related to a short-term free recall impairment, primarily because of the design of the test utilised by the Cognistat (Mueller et al., 2007). Further expanding the assessment of the memory domain to include all the various subcomponents of memory would be prudent for future research.

Interestingly, memory domain performance of the nurse group was not correlated with anxiety; however, anxiety was positively correlated to the number of Stroop tests passed and MMSE performance. Whilst these results do not necessarily align with most literature, a limited amount of research has suggested that anxiety may cause performance improvements, primarily associated with reductions in participant error (Robinson et al., 2013), or a reduction in the processing of task irrelevant information and attentional narrowing (Hu et al., 2012). Moreover, attentional narrowing and a

reduction in processing of task irrelevant information may precede a decrease in participant error, and it is possible that the present results may be explained by such a shift.

The administration of the MMSE relies on question and answer or command based delivery (Folstein et al., 1975) and whilst not necessarily a direct assessment of attention, the MMSE requires adequate attentional capacity. Thus, it could be suggested that an attentional narrowing and focus on the administrator could lead to an improvement in overall performance. Further, the Stroop test generates an interference effect by rotating congruent and incongruent stimuli (Stroop, 1935), and relies on cognitive functions that include working memory as well as selective attention (Moering et al., 2004). Therefore, a reduction in processing of task irrelevant information and attentional narrowing may also produce an increase in Stroop performance. Finally, utilising a specific assessment of attention e.g. the Test of Everyday Attention (Robertson et al., 1996) to confirm this postulation would benefit future research.

3.4.4.3 Anxiety, cognitive performance and electroencephalography

Electroencephalography was used in the present analysis as an additional measure of cognitive performance to complement those of the MMSE (Folstein et al., 1975) and the Cognistat (Mueller et al., 2007). Furthermore, PCA indicated that frontal pole and average delta, theta and alpha activity were amongst the top contributing variables for both sample groups in the present analysis.

In the non-health professional sample group, baseline frontal pole alpha activity was the only EEG variable associated with anxiety, indicating an increase in anxiety was associated with a decrease in alpha activity. Implicating the alpha band aligns well with

previous research that investigated EEG changes in cognitive performance and associated reductions in alpha activity with cognitively impaired states (Klimesch, 1999, Jackson and Snyder, 2008, Zadikoff et al., 2008, Babiloni et al., 2010, Aurtenetxe et al., 2013). Moreover, Pijnenburg et al. (2004) subdivided the alpha band, and associated only the upper portion with cognitive performance, and the future utilisation of such frequency band subdivisions may prove promising. The present results differ from our previously published research (Lees and Lal, 2017), where alpha power was suggested to increase in cognitive impairment. This discrepancy may be attributed to the differing sample group, as the previous result was found for nurses not non-health professionals. It is also possible for the more rigorous present analysis to explain this juxtaposition.

Moving to the nurse group, a different cognitive profile was identified, where anxiety was significantly correlated with baseline average delta activity and central gamma activity, as well as average delta reactivity. These results align well with previous research that has demonstrated delta power increases in cognitively impaired states (Huang et al., 2000, Babiloni et al., 2006b, Babiloni et al., 2008, Babiloni et al., 2010). Importantly, Lees et al. (2016) similarly demonstrated a reduction in delta reactivity was associated with a reduction in cognitive performance. Although the investigation did not examine its relationship with anxiety, when combined with the present results, it nevertheless provides a precedent for the examination of reactivity/oscillatory variables in cognitive EEG research.

With respect to the gamma frequency band, literature has associated frontal, occipito-temporal enhancement of gamma activity with cognition (Tallon-Baudry et al., 1998, Cho et al., 2006). Interestingly, Lee et al. (2010) previously reported this to be true for non-impaired individuals only and the inverse for patients with AD. Additionally, van

Deursen et al. (2008) reported increased gamma band power over the parietal, parieto-temporal and occipital leads in cognitive impairment, thus, expanding the implicated regions to the entirety of the head. Combining the present results with previous literature, it can be suggested that changes in delta and gamma activity may be associated with impaired cognitive capability and potentially the neural changes that result from the experience of anxiety, however, additional research performing direct comparisons to cognitively impaired sample groups would help to confirm this.

A final point to consider with respect to the present EEG analysis is that the majority of past research has been conducted by examining individuals with diagnosed cognitive impairments (i.e. MCI or dementia); however, the sample groups in the present analysis were both demonstrably cognitively able. Thus, further confirmatory research examining non-impaired sample groups, be they occupation specific or otherwise, would be beneficial.

3.5 Limitations, Future Directions & Conclusions

3.5.1 Limitations

The present study effectively examined the relationship between stress, and anxiety and cognitive performance using a cross-sectional design, which provides an initial acute insight into the effects of stress and anxiety, and any examination of the longer-term or chronic effects is forgone. Future research utilising longitudinal designs would allow any temporal variations in the presently identified relationships to be examined, which may be insightful as cognitive performance and indeed stress and anxiety can vary over time. Furthermore, as the present study was an early examination it relied on an observational approach, whereby only associations were determined and more causative statements could not be made. The use of experimental designs utilising varying levels

of stress induced or otherwise, would therefore be worthwhile examinations for the future, particularly as it would also enable direct examination of the constructs of eustress and distress, and possibly even the role of habituation.

Lastly, it should be noted that as the present dataset was a combination of newly collected data and existing databases, the number of observations available for the DASS and WCCL in the nurse sample group was limited to only 21 observations. Whilst this sample size difference could have had impact on the present results, the vast majority of the results associated with the reduced observation count were negative, and so it could be suggested that the most likely impact would be the presence of a false negative as opposed to a false positive. Nonetheless, re-examination of the same parameters with an increased sample size is certainly poignant for future research.

3.5.2 Future Directions

Looking forward, expanding the utilised EEG montage from a 2 lead bipolar montage into a minimum 32 lead unipolar head cap would enable examination of the missing brain regions, as they may also be associated with cognitive performance, or at least function as association areas. Further, it is possible that the existing EEG montage could be hybridised with traditional imaging modalities, to provide both a spatial and temporal image of stress and anxiety. A transition to other functional imaging modalities such as magnetoencephalography or functional MRI may also prove insightful.

Additionally, capturing and including workplace performance metrics, in addition to traditional cognitive performance variables, would benefit future research, as depending on what metrics are captured it is possible for these to function as assessable outcomes. There also exists an argument for future research actively utilising workplace specific tests in their assessment of cognitive performance, as it may provide further insight into

the health professions and the relationship between stress/anxiety and cognitive performance. By doing so, it is plausible that researchers could observe a reduction in generalisability of their results as the role of a nurse and other health professionals can vary significantly depending upon their location inside the industry. Lastly, it is possible that this type of performance-based research could benefit a number of other non-health related professions that also have critically important outcomes (e.g. military, professional driving, and heavy machinery operations).

3.5.3 Conclusions

In conclusion, stress and anxiety are both experienced by non-health professionals and nurses, and may impact the lifestyle, cognitive performance and brain activity of these individuals; however, the nature of these relationships may not be as obvious as expected. The present analysis found that stress was linearly associated with memory performance in both groups; however, literature suggests that an inverted-U shape rather than the observed linear relationship may better reflect the true nature of this association. Further, EEG activity changes related to stress were unique, with delta activity increases being found in both groups, whilst theta and beta increases were found for the nurse group only. Additionally, the associations to anxiety were also distinct for each group; with increases in lifestyle risk factors, impaired memory performance and alpha activity being found in non-health professionals, and increases in Stroop test performance, global cognitive performance and resting delta and gamma activity for nurses.

With respect to the present hypotheses, varied associations, both positive and negative, between stress/anxiety and cognitive parameters were identified; and so the first hypothesis (‘*higher levels of stress/anxiety will be associated with declines in cognitive*

performance, both in global and specific domains') could be rejected or accepted depending upon cognitive parameter and sample group. Furthermore, the second present hypothesis (*'higher levels of stress/anxiety will be associated with alterations in brain activity as measured by EEG; in particular the higher frequency bands (beta and gamma) of the frontal poles.'*) can be partially accepted, as EEG changes were associated with stress/anxiety. However, the implicated frequency bands were sample group dependent e.g. beta and gamma activity were implicated for nurses and not non-health professionals.

Summarily, the present findings develop our understanding of the impact of stress and anxiety, and may enable the development of strategies to ensure nurse performance, quality of patient care, and hence, reduce the incidence of adverse medical events.

Chapter 4 – Associations between Stress, Anxiety and Cognitive performance (using a full head EEG montage)

4.1 Introduction

Research has demonstrated that chronic cortisol exposure can cause neuronal damage and death in the hippocampi and prefrontal cortex (Starkman et al., 1992, Sapolsky, 1996, Sapolsky, 2003, Conrad, 2006). These neural regions have been associated with cognitive processes and stress regulation (Lepage et al., 2000, Lupien and Lepage, 2001). Additionally, the sympathomedullary system modulates a number of cognitive processes including working memory and attention where high system activity is associated with cognitive impairment (Ramos and Arnsten, 2007). Further, it has been well established that global cognition (LeBlanc et al., 2005, LeBlanc, 2009), as well as decision making (Cumming and Harris, 2001, Wetzel et al., 2006) and memory performance (Lupien et al., 1999, de Quervain et al., 2000, Kuhlmann et al., 2005), can be negatively affected by stress. Whilst other research has shown stress can have minimal cognitive impact (Lees and Lal, 2017). However, research specifically examining the impact of stress and anxiety on the cognitive performance of health professionals is currently limited (LeBlanc, 2009).

The experience of anxiety has also been associated with a number of changes both physical and cognitive; decreased prefrontal activity (Bishop, 2009), as well as functional connectivity changes in the ventrolateral and ventromedial prefrontal cortex have both been reported (Andreescu et al., 2014). Impairments in various executive functions (Airaksinen et al., 2005, Nieuwenhuys et al., 2015), decision-making

capability (Cumming and Harris, 2001), attention (Lautenbacher et al., 2002), and working memory (Asmundson and Stein, 1994, Savage et al., 2000, Ashcraft, 2002) have also been reported. As previously stated it is possible for these impairments to impact the performance of nurses and their quality of care, which can in turn place patients at risk.

The nursing profession frequently presents numerous challenges (e.g. working with vulnerable patients, and the provision of continuous care) to its employees, and these challenges require optimal cognitive performance to best manage the health and care of patients. It has been demonstrated that the demands of the nursing profession are inherently stressful (Lee and Wang, 2002, Sveinsdóttir et al., 2006, Golubic et al., 2009), and can impact nurse performance and quality of care (Tarnow-Mordi et al., 2000, Sveinsdóttir et al., 2006, Berland et al., 2008). As such, sub-optimal cognitive functioning associated with stress and/or anxiety has the potential to endanger the quality of care provided by nurses (Wilson et al., 1999, Tarnow-Mordi et al., 2000, Sveinsdóttir et al., 2006, Berland et al., 2008), which in turn would place lives at risk. Indeed, in some instances may account for a number of adverse medical events e.g. medication errors, surgical complication (Wilson et al., 1999).

As such, the aim of this study was to investigate the associations between stress, anxiety, and cognitive performance in nurses, and to expand on the electroencephalographic findings reported in Chapter 3. Global cognitive performance, as well as memory and judgement domain performances were of particular interest, due to their almost daily requirement in the nursing profession. It was hypothesized that experiencing higher levels of stress/anxiety will be associated with:

1. Declines in global and/or domain specific cognitive performance

2. Alterations in brain activity as measured by EEG; in particular the theta, beta and gamma frequency bands.

4.2 Materials and Methods

This experiment utilised the general methodology that was previously described in Chapter 2. The following sections provide a brief summary of the experimental protocol (Section 4.2.1) and also describe experimental specific (Section 4.2.2, and Section 4.2.3).

4.2.1 Study Participants

Data from a total of 64 nurses and 51 non-health professionals were recorded and utilised in this experiment. Of these participants, 15 nurses, and the 51 non-health professionals were recruited into this experiment, whilst the remainder of the data was pre-existing within a database.

This database (Maharaj, 2015) was comprised of data from 49 nurse participants previously collected in the Neuroscience Research Unit at UTS, using the same experimental protocol and laboratory conditions, as previously described in Chapter 2. It is important to note, that data from 24 non-health professionals was collected by another researcher of the Neuroscience Research Unit, UTS.

Finally, data from 3 nurse participants was excluded from analysis, as these individuals failed to meet one or more of the criteria detailed in Section 2.3.1. The data were excluded as one individual smoked more than 10 cigarettes daily, the blood pressure for one individual was too high, and the third individual was taking medicines that may

have affected their cognitive performance at the time of testing. This reduced the final sample size to 61 nurse participants, and 51 non-health professional participants.

4.2.2 Experimental Protocol

This experiment utilised the general methodology previously described in Chapter 2, which can be briefly summarised as follows.

Initially, anthropometric measurements, including blood pressure, heart rate, height and weight, as well as waist and hip circumference were recorded for each participant. A pre-study questionnaire battery containing the Lifestyle Appraisal Questionnaire (Craig et al., 1996), the Depression, Anxiety, Stress Scale (Lovibond and Lovibond, 1995b), and the Fatigue State Question (Lal and Craig, 2002) was also completed. After these questionnaires and measurements, a two phase electroencephalogram recording was completed. The first phase was a quiet seated baseline in which participants rested with their eyes open, and the second was an active phase in which the participants completed a computerised Stroop Test (Stroop, 1935); see Chapter 2, Section 2.4.3.1 for further details.

Following this, the Mini-Mental State Exam (Folstein et al., 1975), and the Cognistat (Kiernan et al., 1987) was used to assess participant cognitive performance. This assessment was then followed by a post-study questionnaire battery made up of the Fatigue state Question (Lal and Craig, 2002), the revised Ways of Coping Checklist (Vitaliano et al., 1985), and for the nurse participants, the sleep and shiftwork scales from the Standard Shiftwork Index (Barton et al., 1995). Lastly, participant blood pressure was recorded again, concluding the experimental protocol.

4.2.3 Electroencephalogram data collection

In this experiment, a 32-lead unipolar electroencephalogram montage with a sampling rate of 1000 Hz was recorded using a SynAmp^2 amplifier (Compumedics, Australia) supported by the Scan software (Version 4.3, Compumedics, Australia). Refer to Figure 4.1 to see the experimental set-up and equipment.

Figure 4.1 – Experimental equipment and set up

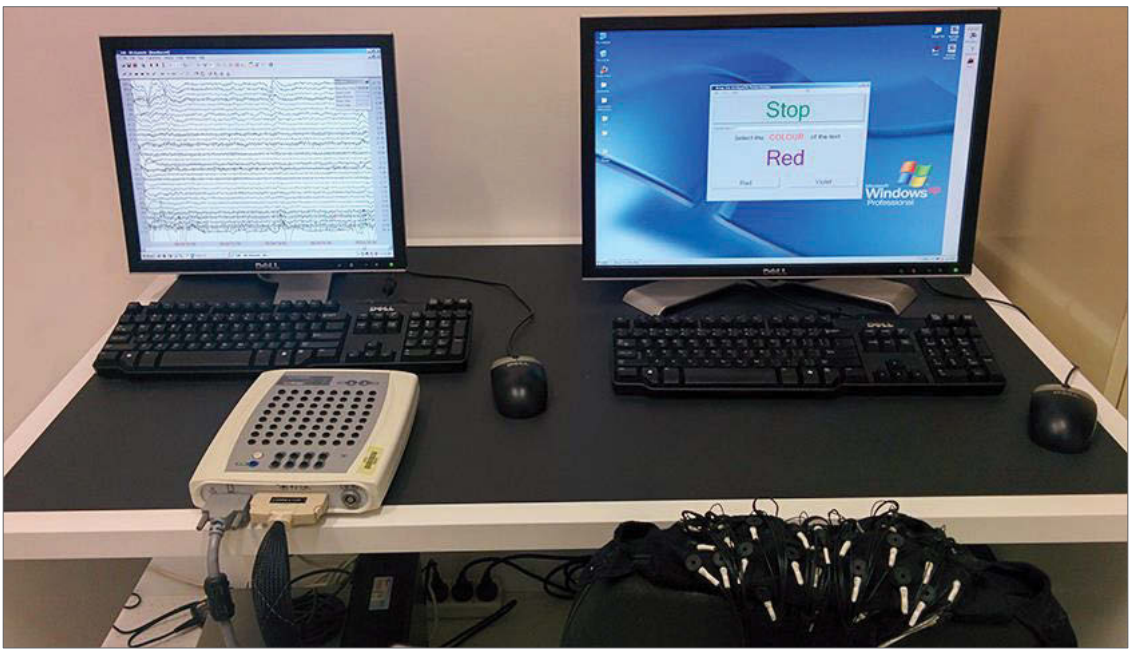

Figure 4.1 displays the currently experimental equipment and set up. This included two computers (one for data acquisition, the other for participant use during the active phase of recording), a SynAmp^2 amplifier, and a 64 channel electroencephalography Quikcap.

Electrodes were placed using a 64 channel electrode QuikCap (Compumedics, Australia); the electrode positions utilised were as follows: Fp_1, Fp_2, F_7, F_3, F_z, F_4, F_8, FT_7, FC_3, FC_z, FC_4, FT_8, T_7, C_3, C_z, C_4, T_8, TP_7, CP_3, CP_z, CP_4, TP_8, P_7, P_3, P_z, P_4, P_8, O_1, O_z, and O_2. Additionally, the reference electrode was placed at the vertex, and the ground electrode at position AF_z. Lastly, a bipolar electrooculogram (EOG) was set up

with one electrode above the left eye, and a second electrode below. Refer to Figure 4.2 for a diagrammatic representation of the electrode locations.

Figure 4.2 – 32 channel electroencephalogram electrode locations

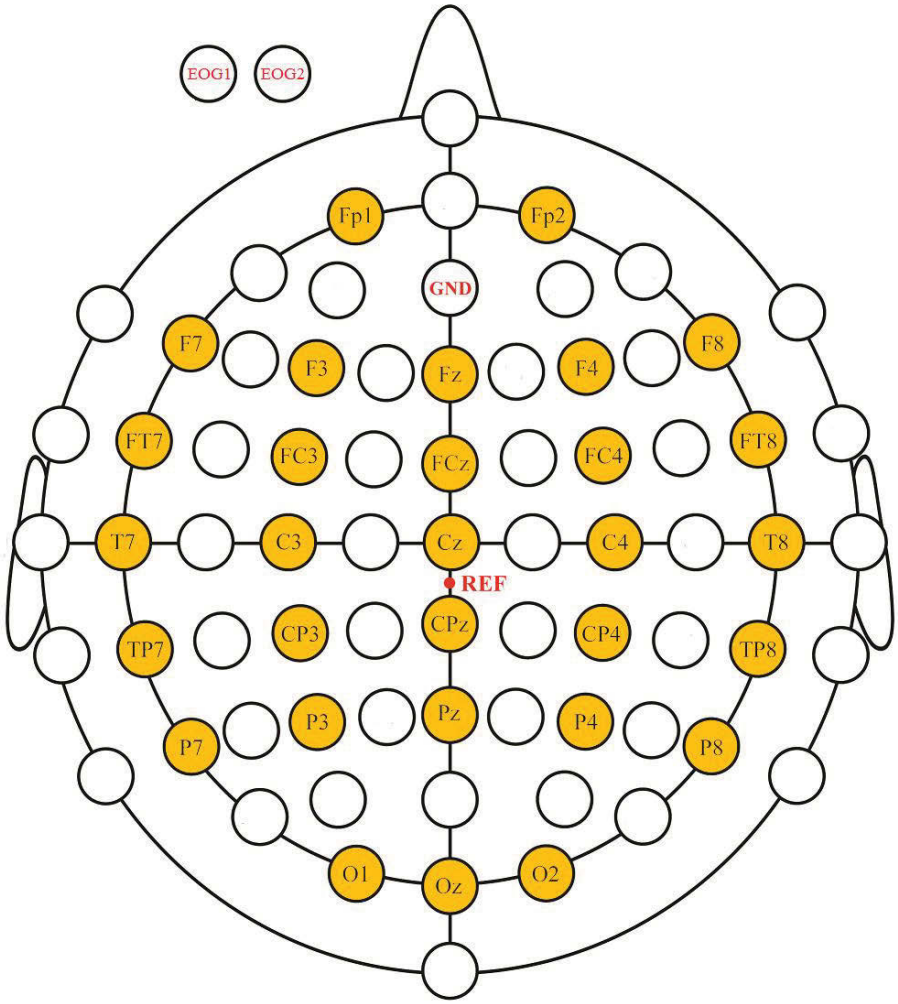

Figure 4.2 illustrates the 32 electrode locations of the International 10-20 system (Jasper, 1958) utilised in the present experiment is shown. Active electrodes were positioned at the following locations: Fp_1, Fp_2, F_7, F_3, F_z, F_4, F_8, FT_7, FC_3, FC_z, FC_4, FT_8, T_7, C_3, C_z, C_4, T_8, TP_7, CP_3, CP_z, CP_4, TP_8, P_7, P_3, P_z, P_4, P_8, O_1, O_z, and O_2. Further, auxiliary electrodes (reference, ground and electrooculogram) are indicated in red.

Key: C = Central; EOG = Electrooculogram; F = Frontal; GND = Ground; O = Occipital; P = Parietal; REF = Reference; T = Temporal; z = Midline

Once the cap was secured and comfortably in place, Signa gel (Parker Laboratories Inc, USA) was injected (using a blunt fill needle and syringe) into each of the relevant electrode cups within the cap. Whilst filling each of the electrode cups, an initial EEG trace was run to allow the signal quality and electrode impedance to be examined. If the quality of the trace was not acceptable, or electrode impedance was too high (above 5 KOhm (Keil et al., 2014) adjustments to EEG electrode fixation and amount of gel applied were made. Refer to Figure 4.3 for an example of an electroencephalogram recorded in the present experiment.

Figure 4.3 – A 32 channel unipolar electroencephalogram recording

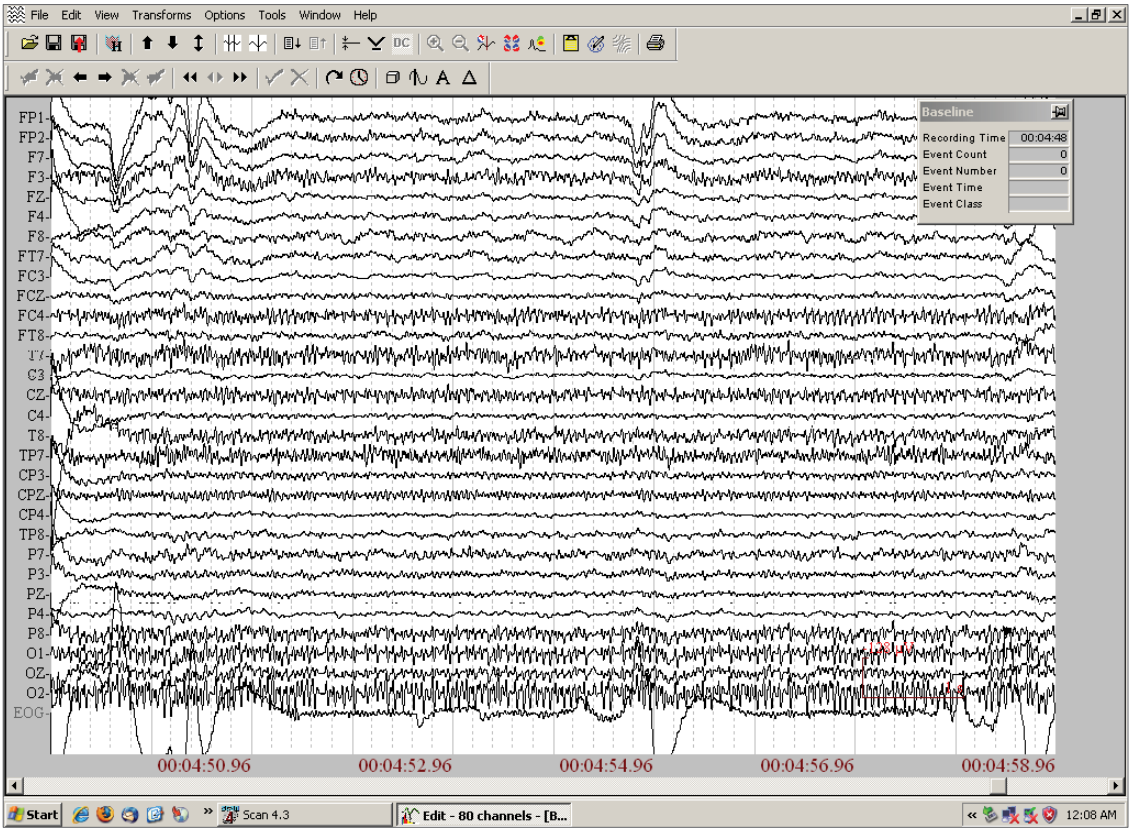

Figure 4.3 displays a 32 channel electroencephalogram recording from the present experiment is shown. Each channel recorded is labelled with its electrode location The X axis plots time in seconds, whilst the Y axis plots amplitude in microvolts.

4.2.4 Electroencephalogram data processing

Prior to statistical analysis, the raw EEG data sets obtained for both the baseline and active phases (Section 2.4.3) were processed according to the steps stipulated below.

1. The raw EEG data was filtered using a Butterworth IIR Bandpass filter set at 1.5 and 50 Hz was applied to remove any direct current residue and/or any high frequency artefacts e.g. movement artefacts. This was followed by the application of a Hann Window.
2. The Aligned-artefact average procedure (Croft and Barry, 1998, 2000) was then applied to minimise any artefacts in the EEG data linked to eye movement.
3. After pre-processing, each set of recordings (baseline and active) was sectioned into approximately 300 one second epochs, after which the EEG activity in the selected frequency bands: delta (1.5 - 4 Hz), theta (4 - 8 Hz), alpha (8 - 13 Hz), beta (13 - 35 Hz) and gamma (35 - 50 Hz), was calculated via Periodogram power spectral density estimate (Bartlett, 1950).
4. The epoch values of each recording were scanned for outliers, which were removed using a modified z-score statistic (Leys et al., 2013) that was calculated using the following equation.

Equation 4.1 – Modified Z-score statistic

$$z = \frac{X - \tilde{x}}{MAD}$$

Where:

X = Epoch value $\tilde{x}$ = Median value MAD = Median Absolute Deviation

Median Absolute deviation was calculated using the following equation:

Equation 4.2 – Median absolute deviation

$$MAD = \tilde{x}_i (|X_i - \tilde{x}_j(X_j)|) \quad (2)$$

Where:

$\tilde{x}$ = Median X = Epoch value

Epoch values were rejected and removed if their modified *z* statistic was greater than or equal to 5.

5. Following the removal of outliers, the activity values for each of the epochs were then averaged to derive a single value for each frequency band at each electrode site. Additionally, the activity values in each frequency band were also averaged, providing an overall average EEG activity value.
6. A reactivity value was generated by subtracting the baseline EEG activity from the active phase for each of the respective frequency bands (beta and gamma). This value was generated to allow the change in EEG activity between the baseline and active phases to be examined.
7. Finally, after all the EEG data had been collated, outliers were removed using the modified Z-score statistic that was previously applied to the epoch values. The threshold at which values were rejected and removed was set at a *z* statistic greater than or equal to 10, a value well beyond the suggested z statistic of 3 (Leys et al., 2013).

4.3 Results

4.3.1 Demographics & Comparisons

A total of 112 individuals participated in this experiment; 61 (50 Females, 11 Males) of which were nurses aged between 19 and 61 years (average 32.75 ± 12.65 years), and 51 (38 Females, 13 Males) of which were non-health professionals aged between 18 and 62 years (average 30.86 ± 11.64 years). Years of education was found to differ significantly ($p = 0.012$) with an average of 16.61 ± 2.97 years in non-health professionals compared to 15.31 ± 2.38 years in nurses (Table 4.1).

Additionally, with respect to the registration status of the 61 nurse participants, at the time of testing 31 were assistants in nursing (AINs), 7 were enrolled nurses (ENs), 2 were currently employed as dental nurses, 1 was a midwife, and the remaining 19 nurses were registered nurses (RNs).

Examining participant demographics, it was found that on average the body mass index (BMI) of non-health professionals resided in the normal range (24.18 ± 4.64 kg/m^2); the nurse participants however resided in the overweight range (25.33 ± 4.91 kg/m^2). Interestingly, these scores were not significantly different. Similarly, both the pre and post fatigue measures did not differ significantly between the two sample groups. In contrast, lifestyle risk factors that increase risk of disease development (part 1 of the LAQ) did differ significantly between the sample groups ($p = 0.020$), with nurses reporting a slightly higher score (13.72 ± 6.90) than non-health professionals (10.88 ± 5.36).

Table 4.1 – Demographic data and intergroup comparison of the two study sample groups

Variable	Group	n	Value	t	p
Age (years)	Nurses	61	32.75 ± 12.65	0.82	0.548
	NHP	51	30.86 ± 11.64		
Years of Education	Nurses	59	15.31 ± 2.38	-2.54	0.012*
	NHP	50	16.61 ± 2.97		
BMI (kg/m^2)	Nurses	61	25.33 ± 4.91	1.27	0.687
	NHP	51	24.18 ± 4.64		
WHR	Nurses	61	0.86 ± 0.07	0.26	0.877
	NHP	51	0.86 ± 0.07		
Pre FSQ	Nurses	15	1.87 ± 0.92	0.47	0.640
	NHP	51	1.76 ± 0.68		
Post FSQ	Nurses	15	1.60 ± 0.74	0.16	0.877
	NHP	51	1.57 ± 0.67		
LAQ P1	Nurses	61	13.72 ± 6.90	2.37	0.020*
	NHP	51	10.88 ± 5.36		

Table 4.1 displays basic demographic information including age, years of education, body mass index, waist hip ratio, fatigue state, and lifestyle risk factors (LAQ Part 1) for both sample groups. Additionally, both t and p values are provided for intergroup comparisons for each of these variables.

Key: BMI = Body mass index; FSQ = Fatigue State Questionnaire; LAQ = Lifestyle Appraisal Questionnaire; n = Sample size; NHP = Non-health professionals; P1= Part one; * = Statistical significance

Finally, it was determined that over the course of the experimental protocol (Table 4.2) the systolic blood pressure of the nurse participants ($p = 0.002$), the fatigue score of non-health professional participants ($p = 0.024$), and the heart rate of both sample groups ($p < 0.001$) significantly declined over the course of the experimental protocol.

Table 4.2 – Differences in pre and post study measures of blood pressure and fatigue for both sample groups

	Sample Group	Pre Mean (SD)	Post Mean (SD)	t	p
SBP (mmHg)	Nurses	113.38 ± 13.40	110.26 ± 13.17	3.26	0.002*
	NHP	110.58 ± 9.80	109.32 ± 10.44	1.23	0.225
DBP (mmHg)	Nurses	76.19 ± 8.63	75.50 ± 8.60	0.85	0.396
	NHP	74.37 ± 7.34	74.24 ± 7.07	0.18	0.858
HR (BPM)	Nurses	78.39 ± 10.20	73.91 ± 9.12	5.07	< 0.001*
	NHP	72.01 ± 9.17	67.67 ± 7.59	4.96	< 0.001*
FSQ	Nurses	1.87 ± 0.92	1.60 ± 0.74	0.59	0.104
	NHP	1.76 ± 0.68	1.57 ± 0.67	2.33	0.024*

Table 4.2 presents the pre and post experimental values for systolic and diastolic blood pressure as well as heart rate and fatigue state. Furthermore, t and p values are provided for intragroup comparisons for these variables.

Key: BPM = Beats per minute; DBP = Diastolic Blood Pressure; FSQ = Fatigue State Questionnaire; HR = Heart rate; mmHg = Millimetres mercury; NHP = Non-health professionals; SBP = Systolic Blood Pressure; SD = Standard Deviation; * = Statistical significance; < = Less than

4.3.2 Cognitive Performance

4.3.2.1 Psychometric Assessment

Both sample groups scored above the global cognitive impairment thresholds for the MMSE and the Cognistat (Table 4.3), with the nurse group scoring 27.39 ± 1.91, and 71.00 ± 6.09 and the non-health professional group scoring 27.86 ± 1.91, and 74.18 ± 4.61 for the MMSE, and Cognistat, respectively. Furthermore, it was found that the global scores of the Cognistat were significantly different between the two groups with the non-health professionals scoring higher than their nurse counterparts ($p = 0.003$).

In terms of domain specific cognitive performance (Table 4.3), it was found that the nurse sample group was below the borderline impairment score (11) in the repetition domain, and that both sample groups fell below the threshold score (4) for the judgement domain; whilst, performance in the remaining domains were all above respective thresholds for both sample groups. Further, a number of cognitive domain scores differed significantly between the two groups (Table 4.3); the domains were: orientation, where nurses scored 11.96 ± 0.18 compared to the 11.67 ± 0.59 of the non-health professionals ($p < 0.001$); attention, where the non-health professionals scored 7.43 ± 0.88 versus the 7.00 ± 1.02 of the nurses ($p < 0.001$); comprehension, where the non-health professionals scored 5.76 ± 0.47 to the 5.33 ± 0.77 of their nurse counterparts ($p < 0.001$); and, similarities, where the non-health professionals scored higher (6.86 ± 1.39) compared to the nurse group (5.90 ± 1.48; $p < 0.001$). The remaining domain scores did not differ significantly between sample groups.

Table 4.3 – The global and domain specific cognitive performance scores of the two study sample groups

Variable	Group	n	Value	t	p
MMSE†	Nurses	61	27.39 ± 1.91	-1.30	0.198
	NHP	51	27.86 ± 1.91		
Orientation	Nurses	61	11.96 ± 0.18	3.78	< 0.001*
	NHP	51	11.67 ± 0.59		
Attention	Nurses	61	7.00 ± 1.02	-2.38	0.019*
	NHP	51	7.43 ± 0.88		
Comprehension	Nurses	61	5.33 ± 0.77	-3.54	< 0.001*
	NHP	51	5.76 ± 0.47		
Repetition	Nurses	61	10.59 ± 1.81	-1.70	0.091
	NHP	51	11.14 ± 1.54		
Naming	Nurses	61	7.11 ± 1.08	-1.70	0.091
	NHP	51	7.45 ± 0.99		
Construction	Nurses	61	5.13 ± 1.12	-0.33	0.745
	NHP	51	5.20 ± 0.96		
Memory	Nurses	61	10.75 ± 1.83	-1.01	0.315
	NHP	51	11.08 ± 1.51		
Calculation	Nurses	61	3.56 ± 0.67	-0.73	0.470
	NHP	51	3.65 ± 0.63		
Similarities	Nurses	61	5.90 ± 1.48	-3.52	< 0.001*
	NHP	51	6.86 ± 1.39		
Judgement	Nurses	61	3.66 ± 1.40	-1.24	0.219
	NHP	51	3.94 ± 0.95		
Cognistat Total†	Nurses	61	71.00 ± 6.09	-3.06	0.003*
	NHP	51	74.18 ± 4.61		

Table 4.3 presents the mean scores for the Mini-Mental State Exam and the Cognistat (including its domains) of both sample groups. Furthermore, t and p values are provided for intergroup comparisons for each of these variables.

Key: MMSE = Mini-mental State Exam; n = Sample size; NHP = Non-health professionals; * = Statistical significance; † = Measure of global cognitive performance < = Less than

In this experiment, two performance variables (average response time, and number of tests passed) related to the Stroop test were captured and examined in the two groups (Table 4.4). Regarding the number of tests passed, the performance of the two sample groups did not differ significantly with the non-health professionals scoring 260.08 ± 61.34 compared to the nurses' score of 240.34 ± 64.35. However, it was found that the average response time of the nurse group was significantly worse ($p = 0.031$), returning a value of 1511.85 ± 539.16 milliseconds compared to the 1324.60 ± 288.22 milliseconds associated with the non-health professional group.

Table 4.4 – The Stroop test performance variables of both of study sample groups

Variable	Group	n	Value	t	p
Tests Passed	Nurses	61	240.34 ± 64.35	-1.61	0.110
	NHP	51	260.08 ± 61.34		
Response Time (ms)	Nurses	61	1511.85 ± 539.16	2.19	0.031*
	NHP	51	1324.60 ± 288.22		

Table 4.4 displays the mean values recorded for the two Stroop performance variables (response time and number of test passed) for both sample groups. Furthermore, t and p values are provided for intergroup comparisons for each of these variables.

Key: ms = Milliseconds; n = Sample size; NHP = Non-health professionals; * = Statistical significance

Furthermore, the number of tests passed was significantly negatively correlated to the age of both the nurses ($r = -0.36$; $p = 0.007$), and non-health professionals ($r = -0.37$; $p = 0.008$). Additionally, a positive correlation between average response time and age ($r = 0.54$, $p < 0.001$) was also found for the non-health professional group.

Table 4.5 – The associations between age and Stroop test performance in both sample groups

Dependent Variable	Independent Variable	Sample Group	n	r	p
Age (Years)	Response Time (ms)	Nurses	56	0.23	0.087
		NHP	50	0.54	< 0.001*
	Tests Passed	Nurses	56	-0.36	0.007*
		NHP	50	-0.37	0.008*

Table 4.5 presents the correlations between age, and the two Stroop performance variables (response time and number of tests passed) for both sample groups.

Key: ms = Milliseconds; n = Sample size; NHP = Non-health professionals; * = Statistical significance

4.3.2.2 Electroencephalography

Regarding electroencephalography, the present experiment used frequency domain variables across the entire head as physiological measures of individual cognitive performance.

With respect to the delta frequency band, the delta activity of the nurse group (Figure 4.4A) significantly varied at: FT_7, increasing from 127.20 ± 198.09 to 146.93 ± 217.24 $\mu V/s^2$ ($p = 0.002$); CP_4 which also increased from 91.09 ± 134.20 to 106.96 ± 163.84 $\mu V/s^2$ ($p = 0.029$); P_z which increased to 186.83 ± 262.56 from 153.20 ± 220.13 $\mu V/s^2$ ($p = 0.046$); and finally, O_2 where activity increased from 216.19 ± 262.15 to 303.86 ± 419.66 $\mu V/s^2$ ($p = 0.049$). However, delta activity significantly varied for only a single electrode location (Fp_2) in non-health professionals (Figure 4.4B), with activity reducing from 75.67 ± 73.16 to 59.21 ± 50.77 $\mu V/s^2$ ($p = 0.037$).

In the theta frequency band, the nurse sample group (Figure 4.5A) saw activity at: Fp_1 increase from 11.81 ± 7.41 to 15.41 ± 8.33 $\mu V/s^2$ ($p < 0.001$); Fp_2 increase to 18.02 ± 15.67 from 13.54 ± 9.85 $\mu V/s^2$ ($p = 0.002$); F_7 move from 14.17 ± 10.09 to 17.76 ±

10.99 $\mu V/s^2$ ($p = 0.002$); F_8 change to 22.21 ± 25.27 from 19.66 ± 22.78 $\mu V/s^2$ ($p = 0.023$); and FT_7 increase from 11.89 ± 9.09 to 14.67 ± 11.11 μV ($p = 0.038$). Similarly, the non-health professional group (Figure 4.5B) saw activity at: Fp_1 increase from 14.99 ± 12.59 to 17.47 ± 14.12 $\mu V/s^2$ ($p = 0.035$); F_7 move from 13.26 ± 10.66 to 16.26 ± 10.90 $\mu V/s^2$ ($p = 0.005$); F_3 shift from 9.27 ± 8.71 to 11.10 ± 10.32 $\mu V/s^2$ ($p = 0.031$); FT_7 increase from 12.27 ± 11.75 to 15.22 ± 12.82 $\mu V/s^2$ ($p = 0.001$); and, C_4 change from 3.01 ± 2.08 to 4.01 ± 4.25 $\mu V/s^2$ ($p = 0.045$).

Of the investigated frequency bands, the alpha frequency saw the greatest number of significant changes between the experimental phases (Figure 4.6); interestingly, activity at all locations across both sample groups only decreased. In the nurse group (Figure 4.6A), activity significantly decreased at: Fp_1, which moved from 10.58 ± 8.50 to 8.00 ± 4.24 $\mu V/s^2$ ($p = 0.027$); F_7, which decreased from 12.94 ± 13.46 to 10.34 ± 10.36 $\mu V/s^2$ ($p = 0.024$); F_z, which changed from 7.06 ± 6.83 to 4.18 ± 3.17 $\mu V/s^2$ ($p < 0.001$); FC_3, which decreased to 3.85 ± 3.47 from 6.30 ± 6.19 $\mu V/s^2$ ($p = 0.002$); FC_z, which decreased from 3.94 ± 4.44 to 2.88 ± 3.66 $\mu V/s^2$ ($p = 0.045$); FC_4, which moved from 7.57 ± 8.78 to 4.62 ± 4.60 $\mu V/s^2$ ($p = 0.010$); FT_8, which shifted to 8.77 ± 6.39 from 12.57 ± 14.61 $\mu V/s^2$ ($p = 0.016$); C_3, which diminished to 3.73 ± 4.93 from 5.33 ± 7.11 $\mu V/s^2$ ($p = 0.009$); C_4, which reduced to 3.68 ± 4.22 from 5.03 ± 5.27 $\mu V/s^2$ ($p = 0.005$); CP_3, which became 3.23 ± 3.28 from 5.31 ± 6.66 $\mu V/s^2$ ($p = 0.011$); CP_4, which decreased from 5.84 ± 6.05 to 3.06 ± 2.58 $\mu V/s^2$ ($p = 0.001$); P_7, which shifted from 15.71 ± 18.31 to 10.47 ± 10.81 $\mu V/s^2$ ($p = 0.012$); P_3, which changed from 12.26 ± 14.36 to 6.81 ± 6.86 $\mu V/s^2$ ($p = 0.015$); P_4, which reduced to 15.31 ± 20.39 from 11.22 ± 15.85 $\mu V/s^2$ ($p = 0.022$); and P_8, which decreased to 12.71 ± 17.36 from 17.97 ± 22.54 $\mu V/s^2$ ($p = 0.048$).

Figure 4.4 – The average values of delta electroencephalographic variables across the two experimental phases for both sample groups

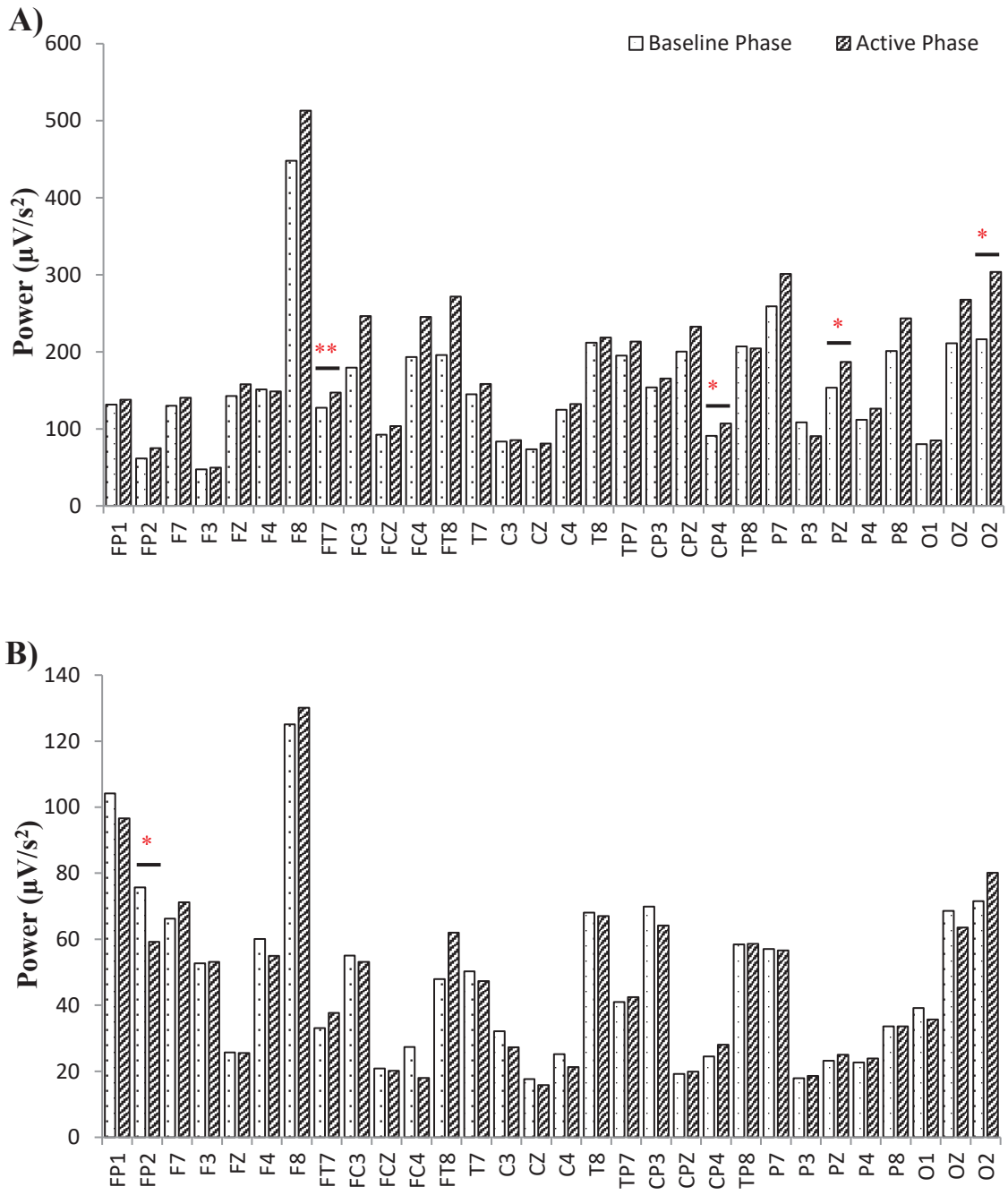

Figure 4.4 presents the mean value of each of the delta electroencephalography variables investigated is shown. Plot A displays the values associated with nurse group, whilst Plot B displays the values associated with the non-health professional participants.

Key: C = Central; F = Frontal; n = Sample size; P = Parietal, T = Temporal; z = Midline * = $p < 0.05$; ** = $p < 0.01$; *** = $p < 0.001$

Figure 4.5 – The average values of theta electroencephalographic variables across the two experimental phases for both sample groups

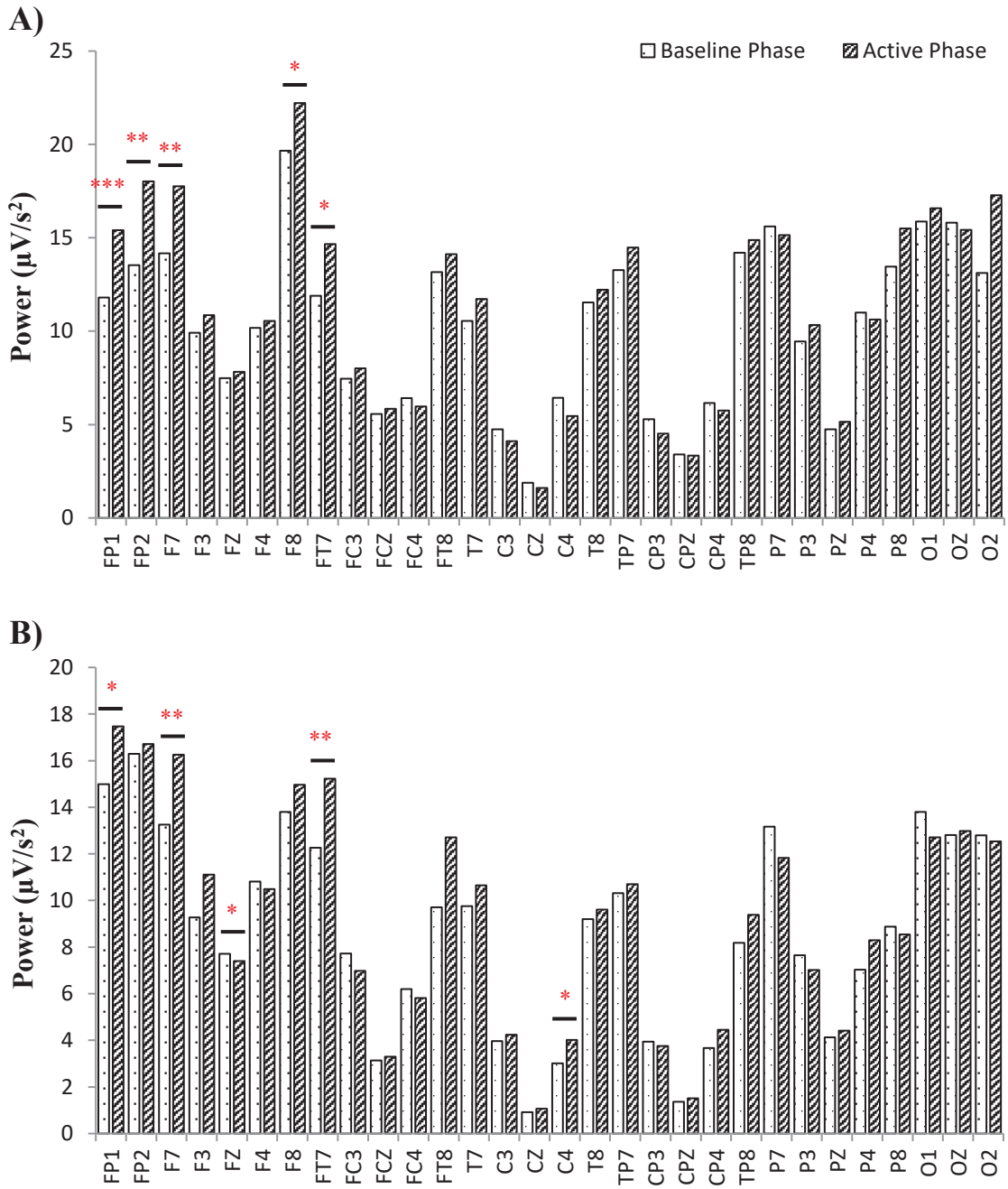

Figure 4.5 displays the mean power value of each of the theta electroencephalography variables investigated. Plot A displays the values associated with nurse group, whilst Plot B displays the values associated with the non-health professional participants.

Key: C = Central; F = Frontal; n = Sample size; P = Parietal, T = Temporal; z = Midline * = $p < 0.05$; ** = $p < 0.01$; *** = $p < 0.001$

Similarly, alpha decreases were also found for the non-health professional group (Figure 4.6B), where activity at: Fp_1 decreased from 13.07 ± 13.45 to 9.14 ± 7.13 $\mu V/s^2$ ($p = 0.015$); F_7 shifted to 9.81 ± 7.82 from 12.43 ± 12.45 $\mu V/s^2$ ($p = 0.047$); F_z diminished from 7.14 ± 6.10 to 4.84 ± 3.95 $\mu V/s^2$ ($p < 0.001$); F_4 changed from 10.09 ± 11.57 to 6.54 ± 7.08 $\mu V/s^2$ ($p = 0.039$); FT_7 shifted from 8.90 ± 5.54 to 6.79 ± 2.91 $\mu V/s^2$ ($p = 0.003$); FC_z decreased to 2.09 ± 1.55 from 3.14 ± 2.58 $\mu V/s^2$ ($p = 0.001$); FC_4 reduced from 7.82 ± 9.28 to 4.07 ± 4.00 $\mu V/s^2$ ($p = 0.006$); T_7 moved from 11.05 ± 10.90 to 8.24 ± 8.31 $\mu V/s^2$ ($p = 0.027$); C_3 decreased from 5.11 ± 5.79 to 2.72 ± 1.89 μV ($p = 0.004$); C_4 became 3.02 ± 2.81 from 4.82 ± 4.66 $\mu V/s^2$ ($p = 0.003$); T_8 shifted to 7.47 ± 7.08 from 11.54 ± 12.87 $\mu V/s^2$ ($p = 0.041$); TP_7 diminished to 7.58 ± 6.00 from 12.67 ± 13.22 $\mu V/s^2$ ($p = 0.001$); CP_3 changed to 2.99 ± 2.50 from 4.85 ± 4.51 $\mu V/s^2$ ($p < 0.001$); CP_z moved from 1.01 ± 1.53 to 0.65 ± 0.77 $\mu V/s^2$ ($p = 0.024$); CP_4 shifted from 5.95 ± 5.92 to 3.61 ± 3.65 $\mu V/s^2$ ($p = 0.004$); TP8 moved from 16.97 ± 21.83 to 9.59 ± 9.73 $\mu V/s^2$ ($p = 0.029$); P_7 decreased from 13.44 ± 13.13 to 8.95 ± 8.03 $\mu V/s^2$ ($p = 0.015$); P_3 diminished from 15.02 ± 19.18 to 8.35 ± 8.53 $\mu V/s^2$ ($p = 0.015$); P_z, changed to 3.39 ± 2.69 from 6.07 ± 5.99 to $\mu V/s^2$ ($p < 0.001$); P_4 became 5.95 ± 5.04 from 12.75 ± 15.26 $\mu V/s^2$ ($p = 0.004$); P_8 reduced to 8.26 ± 8.20 from 18.04 ± 23.30 $\mu V/s^2$ ($p = 0.004$); and O_1 which moved from 14.76 ± 15.10 to 9.12 ± 8.00 $\mu V/s^2$ ($p = 0.028$).

Moreover, a number of beta activity variables also significantly differed between experimental phases for both groups (Figure 4.7). Interestingly, beta activity at FC_4 significantly decreased for non-health professionals (6.48 ± 6.89 vs 5.24 ± 5.82 $\mu V/s^2$; $p = 0.035$) and nurses (9.95 ± 12.27 vs 7.10 ± 8.39 $\mu V/s^2$; $p = 0.009$). Additionally, in nurses (Figure 4.7A), activity at electrode locations: F_7 reduced from 15.20 ± 16.87 to 11.75 ± 9.92 $\mu V/s^2$; F_z reduced from 7.21 ± 8.40 to 5.14 ± 4.78 $\mu V/s^2$; and P_z reduced

from 7.43 ± 10.94 to 5.41 ± 7.53 $\mu V/s^2$. Furthermore, in non-health professionals (Figure 4.7B), C_3 ($p = 0.049$), and CP_z ($p = 0.039$) beta activity also significantly reduced, from 5.62 ± 6.90 and 1.27 ± 1.95 to 3.88 ± 4.40 and 0.97 ± 1.23 $\mu V/s^2$, respectively.

Lastly, in both sample groups gamma activity at a number of electrode locations significantly increased from the baseline to active phases (Figure 4.8). In the nurse group (Figure 4.8A), activity at: Fp_1 increased from 0.86 ± 0.95 to 1.27 ± 1.39 $\mu V/s^2$ ($p = 0.049$); Fp_8 shifted from 1.22 ± 1.50 to 2.35 ± 3.52 $\mu V/s^2$ ($p = 0.020$); T_8 moved from 1.73 ± 2.21 to 2.74 ± 3.87 $\mu V/s^2$ ($p = 0.019$); P_7 increased to 1.00 ± 1.22 from 0.65 ± 0.80 $\mu V/s^2$ ($p = 0.005$); P_4 changed from 0.44 ± 0.63 to 0.72 ± 1.10 $\mu V/s^2$ ($p = 0.028$); P_8 increased to 1.36 ± 1.99 from 1.07 ± 1.57 $\mu V/s^2$ ($p = 0.024$); and lastly, O_2 activity increased from 1.24 ± 2.07 to 3.34 ± 5.36 $\mu V/s^2$ ($p = 0.005$). Similarly, in the non-health professional group (Figure 4.8B), activity at: FT_8 increased from 0.74 ± 0.79 to 1.12 ± 1.41 $\mu V/s^2$ ($p = 0.035$); TP_7 moved from 0.96 ± 0.98 to 1.38 ± 1.11 $\mu V/s^2$ ($p = 0.005$); TP_8 shifted from 0.54 ± 0.58 to 0.95 ± 1.21 $\mu V/s^2$ ($p = 0.028$); P_4 increased from 0.61 ± 0.73 to 0.90 ± 1.24 $\mu V/s^2$ ($p = 0.038$); O_z changed from 1.12 ± 1.53 to 1.97 ± 3.25 $\mu V/s^2$ ($p = 0.018$); and lastly, O_2 activity changed from 0.97 ± 1.46 to 1.85 ± 2.91 $\mu V/s^2$ ($p = 0.008$).

Figure 4.6 – The average values of alpha electroencephalographic variables across the two experimental phases for both sample groups

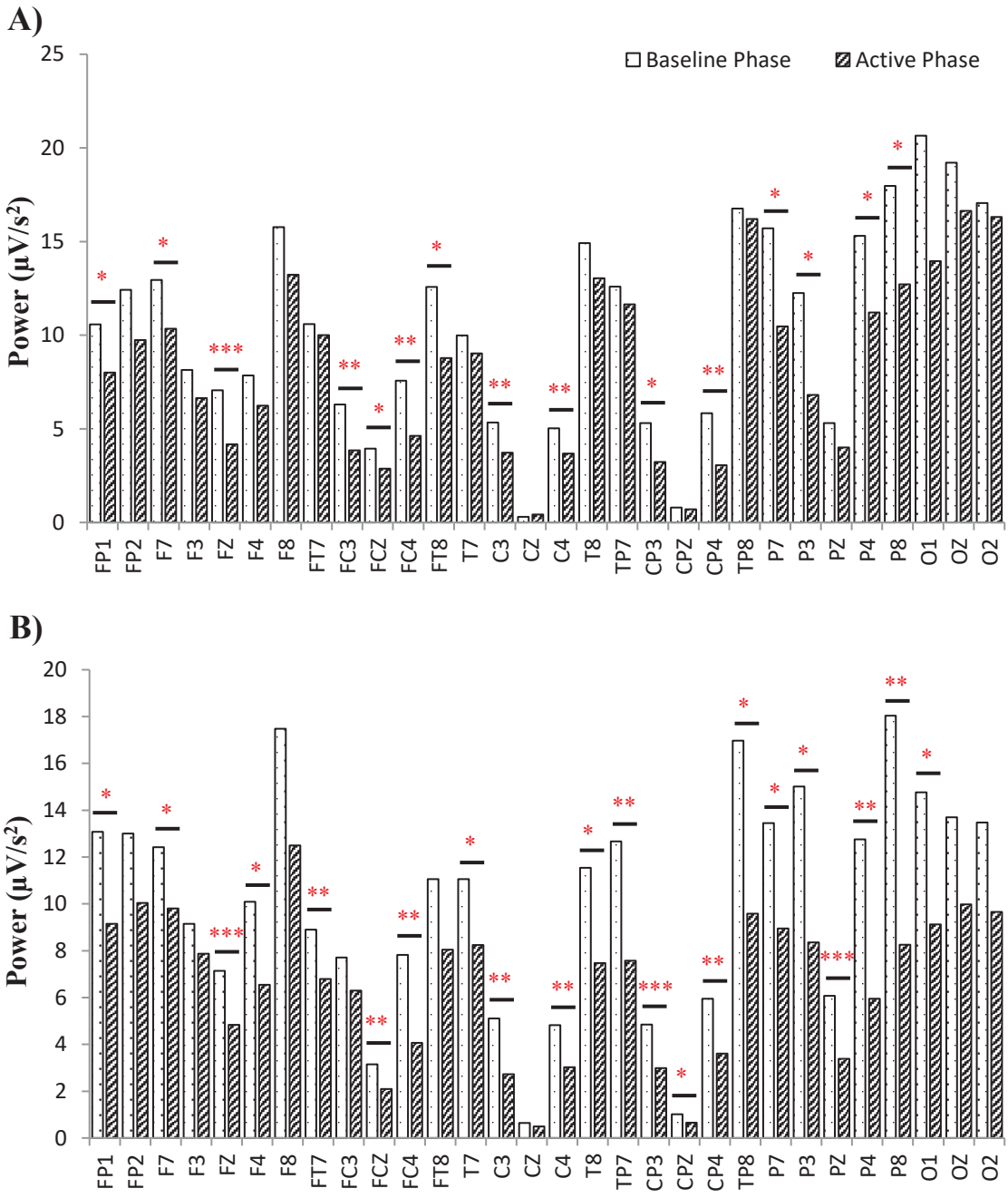

Figure 4.6 displays the mean power value of each of the alpha electroencephalography variables investigated. Plot A displays the values associated with nurse group, whilst Plot B displays the values associated with the non-health professional participants.

Key: C = Central; F = Frontal; n = Sample size; P = Parietal, T = Temporal; z = Midline * = $p < 0.05$; ** = $p < 0.01$; *** = $p < 0.001$

Figure 4.7 – The average values of beta electroencephalographic variables across the two experimental phases for both sample groups

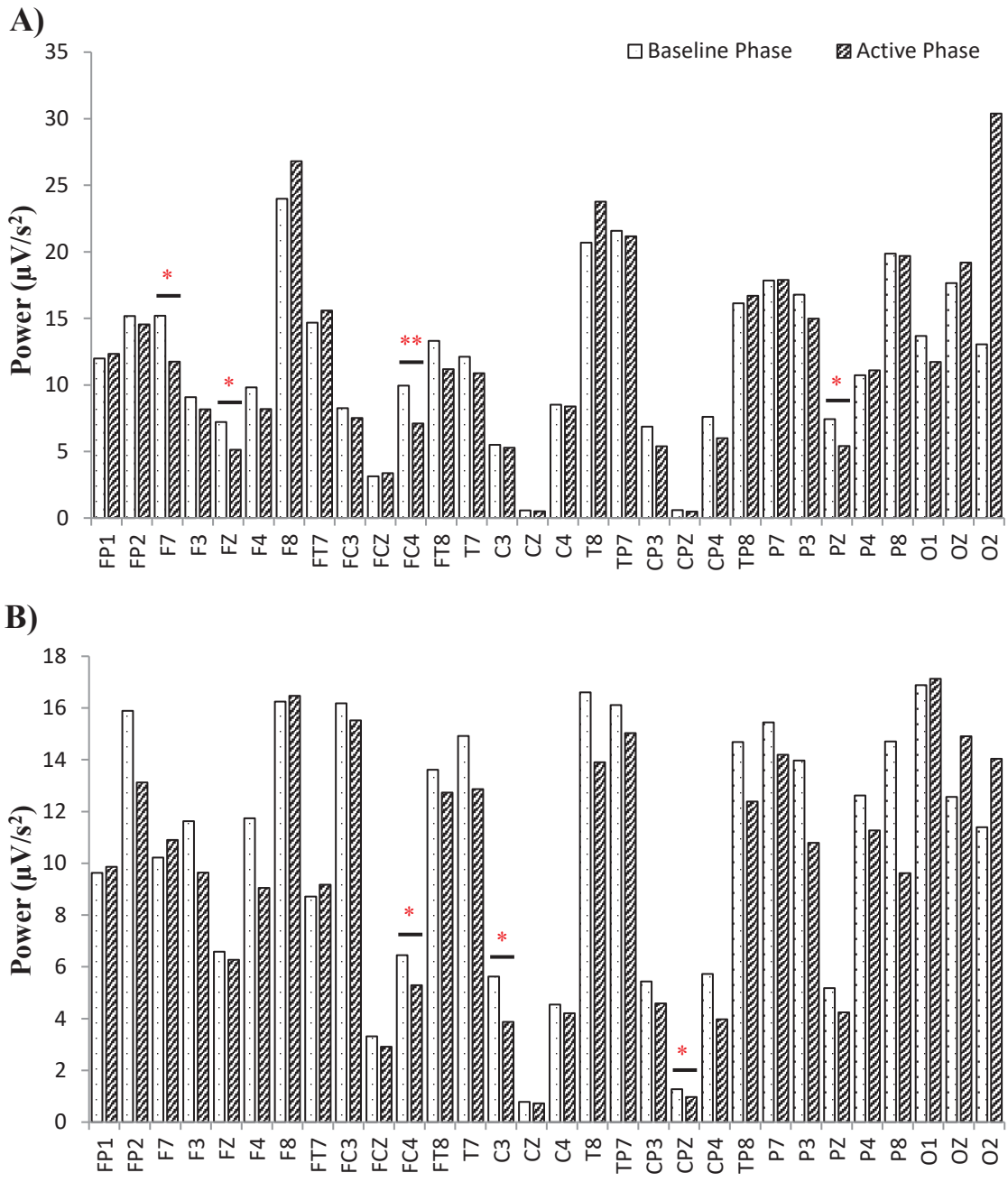

Figure 4.7 displays the mean power value of each of the beta electroencephalography variables investigated. Plot A displays the values associated with nurse group, whilst Plot B displays the values associated with the non-health professional participants.

Key: C = Central; F = Frontal; n = Sample size; P = Parietal, T = Temporal; z = Midline * = $p < 0.05$; ** = $p < 0.01$; *** = $p < 0.001$

Figure 4.8 – The average values of gamma electroencephalographic variables across the two experimental phases for both sample groups

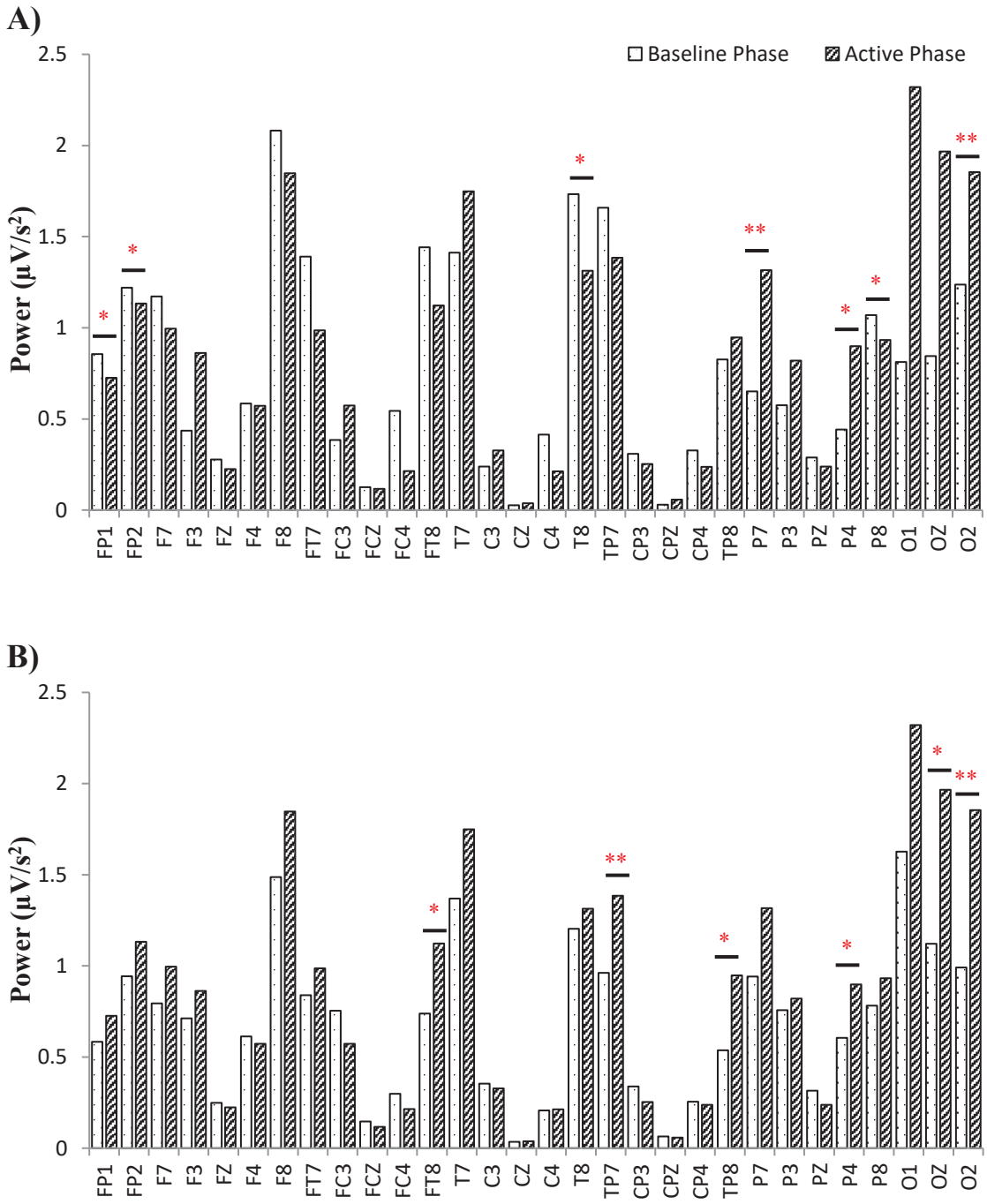

Figure 4.8 displays the mean power value of each of the theta electroencephalography variables investigated. Plot A displays the values associated with nurse group, whilst Plot B displays the values associated with the non-health professional participants.

Key: C = Central; F = Frontal; n = Sample size; P = Parietal, T = Temporal; z = Midline * = $p < 0.05$; ** = $p < 0.01$; *** = $p < 0.001$

Furthermore, principal component analysis was utilised to provide further information regarding the electroencephalography variables. In the non-health professional group, the original 150 EEG variables (delta, theta, alpha, beta, and gamma activity at 30 electrode sites) were reduced to 32 principal components, the first 14 of which together represented 96.02% of the variance in the original dataset. The contributions of the original EEG variables to the reduced data set were examined and the five variables that contributed the most to each of the first 14 principal components (as determined by the absolute sum of their eigenvectors; in descending order) are found in Table 4.6.

In the nurse group, the original 150 EEG variables (delta, theta, alpha, beta, and gamma activity at 30 electrode sites) were reduced to 31 principal components, the first 12 of which together represented 96.02% of the variance in the original dataset. The contributions of the original EEG variables to the reduced data set were examined and the five variables that contributed the most to each of the first 12 principal components (as determined by the absolute sum of their eigenvectors; in descending order) are found in Table 4.7.

Table 4.6 – The top 5 contributing electroencephalography variables per principal component in the non-health professional group

Rank	PC1	PC2	PC3	PC4	PC5	PC6	PC7	PC8	PC9	PC10	PC11	PC12	PC13	PC14
1	FT_7-β	C_4-δ	FC_3-γ	FC_3-θ	TP_8-γ	F_8-δ	C_z-θ	FC_z-δ	FC_3-γ	F_z-δ	C_4-δ	C_4-γ	F_8-δ	Oz-θ
2	F_3-γ	FC_3-θ	F_7-γ	F_8-δ	P_3-β	P_3-β	F_8- δ	FC_3-θ	O_2-γ	C_z-θ	O_2-γ	TP_8-γ	F_4-δ	F_7-δ
3	F_3-α	C_z-δ	CP_3-δ	C_z-δ	TP_7-γ	F_z-δ	F_3-θ	FC_3-γ	FT_8-γ	TP_7-γ	C_z-δ	CP_3-δ	TP_8-δ	CP_3-δ
4	CP_4-θ	T_8-δ	FC_3-θ	FC_3-α	FT_7-β	O_2-β	C_z-α	F_3-γ	C_4-δ	O_z- δ	O_2- β	FT_8-δ	FT_8-θ	C_4-γ
5	F_7-γ	F_2-δ	F_7-δ	F_4-δ	C_4-δ	O_2- γ	C_z-β	FT_8-δ	O_2-β	C_z-γ	F_8-δ	F_3-δ	FT_8-δ	C_z-δ

Table 4.6 presents the 5 electroencephalography variables that contributed the most to the first 14 principal components, which together represent 96.02% of the variance in the original dataset. The columns represent each principle component, and the rows represent the top 5 variables respectively. These variables were selected according to the absolute sum of their eigenvector which was generated during the computation of the principle components,

Key: C = Central; F = Frontal; n = sample size; O = Occipital; P = Parietal; PC = Principal component; T = Temporal; z = Midline; α = Alpha; β = Beta; δ = Delta; γ = Gamma; θ = Theta

Table 4.7 – The top 5 contributing electroencephalography variables per principal component in the nurse group

Rank	PC1	PC2	PC3	PC4	PC5	PC6	PC7	PC8	PC9	PC10	PC11	PC12
1	T_7-δ	F_7-γ	F_8-δ	F_8-δ	Fp_1-δ	P_3-δ	Fp_1-θ	O_1-δ	FC_4-γ	C_4-γ	Fp_1-γ	P_8-γ
2	F_z-δ	C_z-γ	P_3-δ	P_3-δ	FC_4-γ	F_8-δ	F_3-θ	O_1-α	FC_z-δ	F_4-γ	O_1-δ	F_4-γ
3	FC_z-δ	FC_z-γ	Fp_1-δ	C_z-γ	F_7-γ	Fp_1-δ	F_8-θ	P_3-δ	F_z-δ	P_4-δ	Fp_2-γ	P_8-θ
4	C_z-γ	FC_4-γ	C_4-δ	C_4-δ	F_3-θ	O_1-δ	O_z-θ	O_z-θ	Fp_2-δ	P_8-γ	Fp_1-β	Fp2-γ
5	F_7-γ	P_3-δ	F_4-γ	FC_z-θ	T_7-δ	Fp_1-θ	O_1-δ	C_4-γ	C_3-δ	P_3-α	T_7-γ	CP_4-δ

Table 4.7 presents the 5 electroencephalography variables that contributed the most to the first 12 principal components, which together represent 96.02% of the variance in the original dataset. The columns represent each principle component, and the rows represent the top 5 variables respectively. These variables were selected according to the absolute sum of their eigenvector which was generated during the computation of the principle components,

Key: C = Central; F = Frontal; Fp = Frontal pole; n = sample size; O = Occipital; P = Parietal; PC = Principal component; T = Temporal; z = Midline; α = Alpha; β = Beta; δ = Delta; γ = Gamma; θ = Theta

4.3.3 Stress

With respect to self-reported stress scores (Table 4.8), the nurse group reported higher values than their non-health professional counterparts for part 2 of the LAQ, scoring 24.37 ± 11.99 versus 21.45 ± 12.19, although this was found to be a non-significant difference. Similarly, for the DASS measure of stress, the nurse group scored higher than the non-health professional group, reporting values of 14.41 ± 10.09 versus 10.80 ± 10.37; again this was found to be a non-significant difference.

Table 4.8 – The self-reported stress scores of the two study sample groups

Variable	Group	n	Value	t	p
Stress (LAQ P2)	Nurses	61	24.37 ± 11.99	1.25	0.213
	NHP	51	21.45 ± 12.19		
Stress (DASS-S)	Nurses	61	14.41 ± 10.09	1.86	0.066
	NHP	51	10.80 ± 10.37		

Table 4.8 presents the mean self-report stress scores of both sample groups for both the LAQ and the DASS. Furthermore, t and p values are provided for intergroup comparisons for each of these variables.

Key: DASS = Depression, Anxiety, Stress Scale; LAQ = Lifestyle Appraisal Questionnaire; n = Sample size; NHP = Non-health professionals; P2 = Part 2; S = Stress

4.3.3.1 Non-health professionals

In the non-health professional group, a number of demographic variables were significantly correlated to stress (Table 4.9). Stress (as measured by part 2 of the LAQ) was significantly and positively correlated with fatigue state both before ($r = 0.43$; $p = 0.002$) and after ($r = 0.33$; $p = 0.019$) the experimental protocol, as well as lifestyle risk factors ($r = 0.50$; $p < 0.001$). Whereas, age, BMI, waist to hip ratio and years of education were not significantly associated with stress (as measured by part 2 of the LAQ). When measured by the DASS, stress was also positively correlated to fatigue

score before ($r = 0.48$; $p < 0.001$) and after ($r = 0.33$; $p = 0.019$) the experimental protocol, and lifestyle risk factors ($r = 0.50$; $p < 0.001$). In concordance with the results associated to the LAQ, stress scores (as measured by the DASS) were again not significantly correlated with age, BMI, Waist to Hip ratio and years of education.

Table 4.9 – The associations between stress (as measured by LAQ P2) and demographic variables of the non-health professional group

Dependent Variable	Independent Variable	n	r	p
Stress (LAQ P2)	Age	51	-0.22	0.122
	Yrs. Education	50	-0.10	0.484
	BMI	51	-0.12	0.403
	WHR	51	0.10	0.498
	Pre-FSQ	51	0.43	0.002*
	Post-FSQ	51	0.33	0.019*
	LAQ P1	51	0.50	< 0.001*
Stress (DASS-S)	Age	51	-0.17	0.232
	Yrs. Education	50	-0.01	0.492
	BMI	51	-0.07	0.611
	WHR	51	0.08	0.594
	Pre-FSQ	51	0.48	< 0.001*
	Post-FSQ	51	0.33	0.019*
	LAQ P1	51	0.50	< 0.001*

Table 4.9 displays the correlations between stress (as measured by LAQ P2, and DASS), and demographic variables including age, years of education, BMI, WHR, fatigue, and lifestyle risk factors (LAQ P1) in the non-health professional group.

Key: BMI = Body Mass Index; FSQ = Fatigue State Question; LAQ = Lifestyle Appraisal Questionnaire; n = Sample size; P1 = Part 1; P2 = Part 2; S = Stress; WHR = Waist/Hip Ratio; Yrs = Years; * = Statistical significance; < = Less than

Furthermore, a strong positive association between stress (as measured by part 2 of the LAQ; Table 4.10) and both the anxiety score ($r = 0.76$; $p < 0.001$), and stress score of the DASS ($r = 0.89$; $p < 0.001$) was identified. Similarly, a strong positive correlation between stress (as measured by the DASS) and anxiety score was also found ($r = 0.84$; $p < 0.001$).

Table 4.10 – The associations between stress and self-reported negative mental state scores of the non-health professional group

Dependent Variable	Independent Variable	n	r	p
Stress (LAQ P2)	DASS-A	51	0.76	< 0.001*
	DASS-S	51	0.89	< 0.001*
Stress (DASS-S)	DASS-A	51	0.84	< 0.001*

Table 4.10 displays the correlations between stress and the other self-reported negative mental state scores for the non-health professional group.

Key: A = Anxiety; DASS = Depression, Anxiety, Stress Scale; LAQ = Lifestyle Appraisal Questionnaire; n = Sample size; P2 = Part 2; S = Stress; * = Statistical significance; < = Less than

Additionally, it was found that both stress scores (LAQ P2 and DASS) of the non-health professional group were significantly and positively correlated to reported coping categories (Table 4.11) of self-blame ($r = 0.40$; $p = 0.004$ and $r = 0.43$; $p = 0.002$), wishful thinking ($r = 0.50$; $p < 0.001$ and $r = 0.51$; $p < 0.001$) and avoidance ($r = 0.47$; $p = 0.001$ and $r = 0.43$; $p = 0.002$).

Table 4.11 – The associations between stress and self-reported coping strategy utilisation of the non-health professional group

Dependent Variable	Independent Variable	n	r	p
Stress (LAQ P2)	Problem Focused	51	0.23	0.110
	Self-support	51	0.17	0.234
	Self-blame	51	0.40	0.004*
	Wishful Thinking	51	0.50	< 0.001*
	Avoidance	51	0.47	0.001*
Stress (DASS-S)	Problem Focused	51	0.25	0.077
	Self-support	51	0.18	0.198
	Self-blame	51	0.43	0.002*
	Wishful Thinking	51	0.51	< 0.001*
	Avoidance	51	0.43	0.002*

Table 4.11 displays the correlations between anxiety and the categories of coping strategy examined by the Ways of Coping checklist for the non-health professional group.

Key: DASS = Depression, Anxiety, Stress Scale; LAQ = Lifestyle Appraisal Questionnaire; n = Sample size; P2 = Part 2; S = Stress; * = Statistical significance; < = Less than

With respect to cognitive performance outcomes, it was found that stress (as measured by part 2 the LAQ) in the non-health professional group was associated with average response time (r = 0.33; p = 0.024), but not the total number of tests passed on the Stroop test (Table 4.12). Interestingly, stress (as measured by the DASS) was significantly associated with both average response time (r = 0.41, p = 0.005) and number of tests passed (r = -0.31; p = 0.037).

Table 4.12 – The associations between stress and Stroop test performance outcomes of the non-health professional group

Dependent Variable	Independent Variable	n	r	p
Stress (LAQ P2)	Response Time (ms)	50	0.33	0.024*
	Tests Passed	50	-0.23	0.127
Stress (DASS-S)	Response Time (ms)	50	0.41	0.005*
	Tests Passed	50	-0.31	0.037*

Table 4.12 displays the partial correlations (controlling for age, BMI, and years of education) between stress (as scored by the LAQ P2 and DASS), and both of the Stroop performance variables (response time and number of tested passed) in the non-health professional group

Key: BMI = Body Mass Index; DASS = Depression, Anxiety, Stress Scale; LAQ = Lifestyle Appraisal Questionnaire; ms = Milliseconds; n = Sample size; P2 = Part 2; S = Stress; * = Statistical significance

Additionally, stress (as measured by part 2 of the LAQ; Table 4.13) in the non-health professional group was significantly correlated to judgement domain performance ($r = 0.37$, $p = 0.011$). All other global and domain specific variables of the MMSE and Cognistat were not significantly correlated to stress as measured by part 2 of the LAQ in the non-health professional group.

Table 4.13 – The associations between stress (as measured by LAQ P2) and cognitive performance variables of the non-health professional group

Dependent Variable	Independent Variable	n	r	p
Stress (LAQ P2)	MMSE	51	-0.17	0.253
	Cognistat Total	51	-0.09	0.564
	Orientation	51	0.01	0.951
	Attention	51	-0.17	0.267
	Comprehension	51	0.10	0.511
	Repetition	51	-0.03	0.801
	Naming	51	0.04	0.768
	Construction	51	-0.23	0.116
	Memory	51	-0.12	0.439
	Calculation	51	-0.18	0.226
	Similarities	51	-0.10	0.504
	Judgement	51	0.37	0.011*

Table 4.13 displays the partial correlations (controlling for age, BMI, and years of education) between stress (as scored by the LAQ P2) and the global and domain specific cognitive performance variables of the MMSE and the Cognistat in the non-health professional group.

Key: BMI = Body Mass Index; LAQ = Lifestyle Appraisal Questionnaire; MMSE = Mini-Mental State Exam; n = Sample size; P2 = Part 2; * = Statistical significance

Stress (as measured by the DASS) in the non-health professional group was not significantly correlated to any global and domain specific variables of the MMSE and Cognistat (Table 4.14).

Table 4.14 – The associations between stress (as measured by DASS-S) and cognitive performance variables of the non-health professional group

Dependent Variable	Independent Variable	n	r	p
Stress (DASS-S)	MMSE	51	-0.21	0.154
	Cognistat Total	51	-0.21	0.161
	Orientation	51	-0.04	0.789
	Attention	51	-0.16	0.269
	Comprehension	51	0.10	0.486
	Repetition	51	-0.08	0.591
	Naming	51	-0.03	0.865
	Construction	51	-0.15	0.311
	Memory	51	-0.22	0.147
	Calculation	51	-0.21	0.165
	Similarities	51	-0.19	0.197
	Judgement	51	0.17	0.244

Table 4.14 displays the partial correlations (controlling for age, BMI, and years of education) between stress (as scored by the DASS) and the global and domain specific cognitive performance variables of the MMSE and the Cognistat in the non-health professional group.

Key: BMI = Body Mass Index; DASS = Depression, Anxiety, Stress Scale; MMSE = Mini-Mental State Exam; n = Sample size; S = Stress

In terms of the investigated EEG variables; stress (as measured by the LAQ P2; Table 4.15) in the non-health professional group was significantly and positive correlated with 9 variables, which were: baseline T_8 delta activity ($r = 0.41$; $p = 0.008$), baseline T_8 theta activity ($r = 0.41$; $p = 0.012$), F_3 alpha reactivity ($r = 0.34$; $p = 0.042$), FC_4 beta reactivity ($r = 0.32$; $p = 0.044$), baseline T_8 gamma activity ($r = 0.31$; $p = 0.044$), baseline P_z gamma activity ($r = 0.36$; $p = 0.028$), active phase F_3 gamma activity ($r = 0.34$; $p < 0.050$), active phase T_7 gamma activity ($r = 0.446$; $p = 0.004$), and F_3 gamma reactivity ($r = 0.36$; $p = 0.029$).

Additionally, a further 21 negative correlations were also found between stress (as measured by the LAQ P2) and: active phase O_1 delta activity ($r = -0.366$; $p = 0.03$), active phase O_z delta activity ($r = -0.39$; $p = 0.021$), F_3 delta reactivity ($r = -0.31$; $p = 0.046$), FC_z delta reactivity ($r = -0.32$; $p = 0.038$), FT_8 delta reactivity ($r = -0.38$; $p = 0.012$), T_7 delta reactivity ($r = -0.31$; $p = 0.047$), P_4 delta reactivity ($r = -0.32$; $p = 0.039$), O_1 delta reactivity ($r = -0.33$; $p = 0.045$), C_z theta reactivity ($r = -0.38$; $p = 0.017$), T_8 theta reactivity ($r = -0.38$; $p = 0.016$), P_3 theta reactivity ($r = -0.33$; $p = 0.040$), Fp_2 alpha reactivity ($r = -0.34$; $p = 0.025$), baseline FC_4 beta activity ($r = -0.35$; $p = 0.032$), baseline C_z beta activity ($r = -0.36$; $p = 0.031$), baseline O_2 beta activity ($r = -0.35$; $p = 0.048$), active phase F_4 beta activity ($r = -0.34$; $p = 0.046$), T_8 beta reactivity ($r = -0.32$; $p = 0.049$), CP_z beta reactivity ($r = -0.40$; $p = 0.013$), active phase T_7 gamma activity ($r = -0.45$; $p = 0.004$), CP_z gamma reactivity ($r = -0.34$; $p = 0.038$), P_7 gamma reactivity ($r = -0.39$; $p = 0.013$), and P_z gamma reactivity ($r = -0.40$; $p = 0.013$).

All other EEG variables were not significantly correlated to stress (as measured by part 2 of the LAQ) in the non-health professional group.

Table 4.15 – The associations between stress (as measured by LAQ P2) and electroencephalography variables of the non-health professional group

Dependent Variable	Independent Variable	n	r	p
Stress (LAQ P2)	Bδ-T_8	47	0.41	0.008*
	Aδ-O_1	40	-0.37	0.030*
	Aδ-O_z	40	-0.39	0.021*
	Rδ-F_3	45	-0.31	0.046*
	Rδ-FC_z	45	-0.32	0.038*
	Rδ-FT_8	46	-0.38	0.012*
	Rδ-T_7	47	-0.31	0.047*
	Rδ-P_4	44	-0.32	0.039*
	Rδ-O_1	41	-0.33	0.045*
	Bθ-T_8	41	0.41	0.012*
	Rθ-C_z	42	-0.38	0.017*
	Rθ-T_8	43	-0.38	0.016*
	Rθ-P_3	44	-0.33	0.040*
	Rα-Fp_2	47	-0.34	0.025*
	Rα-F3	40	0.34	0.042*
	Bβ-FC_4	42	-0.35	0.032*
	Bβ-C_z	40	-0.36	0.031*
	Bβ-O_2	36	-0.35	0.048*
	Aβ-F_4	40	-0.34	0.046*
	Rβ-FC_4	44	0.32	0.044*
	Rβ-T_8	43	-0.32	0.049*
	Rβ-CP_z	42	-0.40	0.013*
	Bγ-T_8	46	0.31	0.044*
	Bγ-P_z	41	0.36	0.028*
	Aγ-F_3	38	0.35	< 0.050*
	Aγ-T_7	44	0.45	0.004*
	Rγ-F_3	40	0.36	0.029*
	Rγ-CP_z	42	-0.34	0.038*
	Rγ-P_7	45	-0.39	0.013*
	Rγ-P_z	41	-0.40	0.013*

Table 4.15 presents the significant partial correlations (controlling for age, BMI, and years of education) between stress (as measured by the LAQ P2) and electroencephalography variables in the non-health professional group. All other EEG variables were not significantly correlated to stress and are not presented in this table.

Key: A = Active phase; B = Baseline; BMI = Body Mass Index; C = Central; F = Frontal; Fp = Frontal pole; LAQ = Lifestyle Appraisal Questionnaire; n = Sample size; O = Occipital; P = Parietal; P2 = Part 2; R = Reactivity; T = Temporal; z = Midline α = Alpha; β = Beta; δ = Delta; γ = Gamma; θ = Theta; * = Statistical significance; < = Less than

As multiple EEG variables were significantly correlated with stress (as scored by part 2 of the LAQ) in the non-health professional group, a forward stepwise general linear regression analysis was performed.

The regression analysis retained 15 of the 30 originally entered variables (baseline T_8 delta activity, active phase O_1 delta activity, active phase O_z delta activity, FC_z delta reactivity, FT_8 delta reactivity, T_7 delta reactivity, baseline T_8 theta activity, C_z theta reactivity, T_8 theta reactivity, CP_z beta reactivity, baseline P_z gamma activity, active phase F_3 gamma activity, active phase T_7 gamma activity, F_3 gamma reactivity, and CP_z gamma reactivity), and had an overall significance of $p = 0.003$ (Table 4.16).

Together these 15 variables explained 99.9% of the variance in stress scores ($F = 8.95 \times 10^4$; $DF = 15$; $p = 0.003$; $R = 1.000$, $R^2 = 0.999$; $AR^2 = 0.999$). Furthermore, all of the retained variables presented as independently significant predictors of anxiety ($p \leq 0.030$), with FT_8 delta reactivity emerging as the strongest predictor ($p = 0.001$)

Table 4.16 – Regression analysis for stress (LAQ P2) and significantly correlated electroencephalography variables in the non-health professional group

$R = 1.00$; $R^2 = 0.999$; $AR^2 = 0.999$;
SSM = 2149.53; dfM = 15; MSM = 143.30;
SSR = 0.002; dfR = 1; MSR = 0.002
$F = 8.95 \times 10^4$; $p = 0.003$*

Variable	β	SE of β	B	SE of B	t	p
Intercept			47.46	0.069	687.57	< 0.001*
Bδ-T_8	0.38	< 0.01	0.07	0.001	73.71	0.009*
Aδ-O_1	-1.15	< 0.01	-0.50	0.002	-205.56	0.003*
Aδ-O_z	0.91	< 0.01	0.13	0.001	226.83	0.003*
Rδ-FC_z	0.23	0.01	0.59	0.028	21.26	0.030*
Rδ-FT_8	-0.92	< 0.01	-0.51	0.001	-459.26	0.001*
Rδ-T_7	-0.37	< 0.01	-0.57	0.004	-142.90	0.004*
Bθ-T_8	-0.34	< 0.01	-1.01	0.012	-82.79	0.008*
Rθ-C_z	-0.43	< 0.01	-12.44	0.074	-168.87	0.004*
Rθ-T_8	-0.52	< 0.01	-2.86	0.026	-111.71	0.006*
Rβ-CP_z	0.56	< 0.01	21.16	0.255	82.94	0.008*
Bγ-P_z	0.30	0.01	25.46	0.823	30.94	0.021*
Aγ-F_3	-0.41	< 0.01	-8.14	0.107	-75.90	0.008*
Aγ-T_7	-0.08	< 0.01	-0.56	0.011	-50.31	0.013*
Rγ-F_3	-0.48	< 0.01	-19.82	0.252	-78.76	0.008*
Rγ-CP_z	-0.27	< 0.01	-209.64	6.018	-34.83	0.018*

Table 4.16 displays a stepwise forward general linear regression analysis between stress (as measured by the LAQ P2) and the significantly correlated physiological EEG variables in the non-health professional group. Of the 30 EEG variables originally entered into the model, the analysis retained 15: Bδ-T8, Aδ-O1, Aδ-Oz, Rδ-FCz, Rδ-FT8, Rδ-T7, Bθ-T8, Rθ-Cz, Rθ-T8, Rβ-CPz, Bγ-Pz, Aγ-F3, Aγ-T7, Rγ-F3, and Rγ-CPz.

Key: A = Active phase; dfM = Degrees of freedom model; dfR = Degrees of freedom residual; F = Frontal; MSM = Mean square model; MSR = Mean square residual; O = Occipital; P = Parietal; R = Reactivity; SE = Standard Error; SSM = Sum of Squares Model; SSR = Sum, of squares residual; z = midline; β = Beta; δ = Delta; γ = Gamma; θ = Theta; * = Statistical significance; < = Less than

Likewise, stress (as measured by the DASS) in the non-health profession group was positively correlated with 10 electroencephalography variables (Table 4.17): baseline CP_4 delta activity ($r = 0.36$; $p = 0.036$), active phase F_3 alpha activity ($r = 0.39$; $p = 0.022$), F_3 alpha reactivity ($r = 0.39$; $p = 0.019$), O_2 beta reactivity ($r = 0.36$; $p = 0.041$), active phase F_3 gamma activity ($r = 0.349$; $p = 0.043$), active phase T_7 gamma activity ($r = 0.45$; $p = 0.004$), active phase C_4 gamma activity ($r = 0.35$; $p = 0.039$), active phase P_4 gamma activity ($r = 0.36$; $p = 0.023$), F_3 gamma reactivity ($r = 0.39$; $p = 0.017$), and T_7 gamma reactivity ($r = 0.34$; $p = 0.027$).

Additionally, a further 9 significant negative correlations were found between stress (as measured by the DASS) and: FC_z delta reactivity ($r = -0.34$; $p = 0.029$), FT_8 delta reactivity ($r = -0.39$; $p = 0.010$), O_1 delta reactivity ($r = -0.33$; $p = 0.047$); baseline T_8 theta activity ($r = -0.42$; $p = 0.010$), active phase FC_4 theta activity ($r = -0.37$; $p = 0.024$), C_z theta reactivity ($r = -0.35$; $p = 0.028$), T_8 theta reactivity ($r = -0.34$; $p = 0.033$), T_8 alpha reactivity ($r = -0.34$; $p = 0.034$), and P_7 gamma reactivity ($r = -0.31$; $p = 0.046$).

All other EEG variables were not significantly correlated to stress (as measured by the DASS) in the non-health professional group.

Table 4.17 – The associations between stress (as measured by DASS-S) and electroencephalography variables of the non-health professional group

Dependent Variable	Independent Variable	n	r	p
Stress (DASS-S)	Bδ-CP_4	42	0.36	0.036*
	Rδ-FC_z	45	-0.34	0.029*
	Rδ-FT_8	46	-0.39	0.010*
	Rδ-O_1	41	-0.33	0.047*
	Bθ-T_8	41	-0.42	0.010*
	Aθ-FC_4	41	-0.37	0.024*
	Rθ-C_z	42	-0.35	0.028*
	Rθ-T_8	43	-0.34	0.033*
	Aα-F_3	38	0.39	0.022*
	Rα-F_3	40	0.39	0.019*
	Rα-T_8	43	-0.34	0.034*
	Rβ-O_2	36	0.36	0.041*
	Aγ-F_3	38	0.35	0.043*
	Aγ-T_7	44	0.45	0.004*
	Aγ-C_4	40	0.35	0.039*
	Aγ-P_4	43	0.36	0.023*
	Rγ-F_3	40	0.39	0.017*
	Rγ-T_7	46	0.34	0.027*
	Rγ-P_7	45	-0.31	0.046*

Table 4.17 displays the significant partial correlations (controlling for age, BMI, and years of education) between stress (as measured by the DASS) and electroencephalography variables that were used as physiological markers of cognitive performance in the non-health professional group. All other EEG variables were not significantly correlated to stress and are not presented in this table.

Key: A = Active phase; B = Baseline; BMI = Body Mass Index; C = Central; DASS = Depression, Anxiety, Stress Scale, F = Frontal; n = Sample size; O = Occipital; P = Parietal; R = Reactivity; S = Stress; T = Temporal; z = Midline; α = Alpha; β = Beta; δ = Delta; γ = Gamma; θ = Theta; * = Statistical significance; < = Less than

Yet again, as multiple EEG variables were significantly correlated with stress (as measured by the DASS) in the non-health professional group, a forward stepwise general linear regression analysis was performed. The regression analysis retained 2 of the 19 originally entered variables (FT_8 delta reactivity, and active phase P_4 gamma activity), and had an overall significance of $p < 0.001$ (Table 4.18).

These 2 variables together explained 55.8% of the variance in stress scores ($F = 11.998$; $DF = 2$; $p < 0.001$; $R = 0.747$, $R^2 = 0.558$; $AR^2 = 0.512$). Furthermore, both retained variables presented as independently significant predictors of stress ($p \leq 0.015$).

Table 4.18 – Regression analysis for stress (DASS-S) and significantly correlated electroencephalography variables in the non-health professional group

$R = 0.747$; $R^2 = 0.558$; $AR^2 = 0.512$;
SSM = 1334.60; dfM= 2; MSM = 667.30;
SSR = 1056.72; dfR = 19; MSR = 55.62
$F = 11.99$; $p < 0.001$*

Variable	β	SE of β	B	SE of B	t	p
Intercept			8.43	1.93	4.37	< 0.001*
Rδ-FT_8	-0.41	0.15	-0.11	0.04	-2.67	0.015*
Aγ-P_4	0.57	0.15	6.67	1.79	3.73	0.001*

Table 4.18 displays a stepwise forward general linear regression analysis between stress (as measured by the DASS) and the significantly correlated physiological EEG variables in the non-health professional group. Of the 19 EEG variables originally entered into the model, the analysis retained 2: Rδ-FT8, and Aγ-P4.

Key: A = Active phase; dfM = Degrees of freedom model; dfR = Degrees of freedom residual; F = Frontal; MSM = Mean square Model; MSR = Mean square Residual; O = Occipital; P = Parietal; R = Reactivity; SE = Standard Error; SSM = Sum of Squares Model; SSR = Sum, of squares residual; δ = Delta; γ = Gamma; * = Statistical significance; < = Less than

4.3.3.2 Nurses

The stress scores (as measured by part 2 of the LAQ; Table 4.19) of the nurse group were significantly correlated to lifestyle risk factors ($r = 0.45$; $p < 0.001$), but not age, BMI, waist to hip ratio, years of education, and both pre and post experimental protocol fatigue scores. Interestingly, stress (as measured by the DASS) was not significantly correlated to lifestyle risk factors, but instead post experimental protocol fatigue score in the nurse group ($r = 0.63$; $p = 0.012$). Further, no other demographic factor was significantly correlated to stress (as measured by the DASS) in the nurse group.

Table 4.19 – The associations between stress and demographic variables in the nurse group

Dependent Variable	Independent Variable	n	r	p
Stress (LAQ P2)	Age	57	-0.21	0.113
	Yrs. Education	55	-0.16	0.258
	BMI	57	-0.11	0.409
	WHR	15	-0.07	0.799
	Pre-FSQ	15	0.44	0.098
	Post-FSQ	15	0.36	0.183
	LAQ P1	57	0.41	0.002*
Stress (DASS-S)	Age	61	-0.02	0.867
	Yrs. Education	61	-0.18	0.181
	BMI	61	0.10	0.455
	WHR	15	0.07	0.805
	Pre-FSQ	15	0.46	0.088
	Post-FSQ	15	0.63	0.012*
	LAQ P1	57	0.21	0.126

Table 4.19 displays the correlations between stress (as measured by part 2 of the LAQ), and demographic variables including age, BMI, WHR, fatigue, and lifestyle risk factors (LAQ P1) in the nurse group.

Key: BMI = Body Mass Index; DASS = Depression, Anxiety, Stress Scale; FSQ = Fatigue State Question; LAQ = Lifestyle Appraisal Questionnaire; n = Sample size; P1 = Part 1; P2 = Part 2; S = Stress; WHR = Waist/Hip Ratio; Yrs = Years; * = Statistical significance

Additionally, the stress score of the nurse group (as measured by part 2 of the LAQ) was significantly correlated to both the anxiety score ($r = 0.62$; $p < 0.001$), and the DASS stress score of the nurse group ($r = 0.63$; $p < 0.001$; Table 4.20). Similarly, the stress score of the DASS was also significantly correlated to anxiety score ($r = 0.82$; $p < 0.001$).

Table 4.20 – The associations between stress and self-reported negative mental state scores in the nurse group

Dependent Variable	Independent Variable	n	r	p
Stress (LAQ P2)	DASS-A	57	0.62	< 0.001*
	DASS-S	61	0.63	< 0.001*
Stress (DASS-S)	DASS-A	61	0.82	< 0.001*

Table 4.20 displays the correlations between stress and the other self-reported negative mental state scores (DASS-A, and DASS-S) for the nurse group.

Key: A = Anxiety; BMI = Body Mass Index; DASS = Depression, Anxiety, Stress Scale; LAQ = Lifestyle Appraisal Questionnaire; n = Sample size; P2 = Part 2; S = Stress; * = Statistical significance

It was found that the stress scores of the nurse group for both measures were not significantly associated to any of the coping categories captured by the Ways of Coping Checklist (Table 4.21); although the low observation count may go towards explaining this.

Table 4.21 – The associations between stress and self-reported coping strategy utilisation of the nurse group

Dependent Variable	Independent Variable	n	R	p
Stress (LAQ P2)	Problem Focused	15	-0.27	0.328
	Self-support	15	0.47	0.079
	Self-blame	15	0.44	0.100
	Wishful Thinking	15	0.39	0.153
	Avoidance	15	0.18	0.512
Stress (DASS-S)	Problem Focused	15	-0.24	0.384
	Self-support	15	0.10	0.722
	Self-blame	15	0.21	0.456
	Wishful Thinking	15	0.28	0.315
	Avoidance	15	< -0.01	0.993

Table 4.21 displays the correlations between stress and the categories of coping strategy examined by the Ways of Coping checklist for the nurse group.

Key: DASS = Depression, Anxiety, Stress Scale; LAQ = Lifestyle Appraisal Questionnaire; n = Sample size; P2 = Part 2; S = Stress

With respect to Stroop performance variables, stress (as measured by part 2 of the LAQ) of the nurse group was significantly correlated to average response time (r = -0.31, p = 0.027), but not total number of tests passed (Table 4.22). Neither Stroop performance variable was significantly correlated to stress (as measured by the DASS) in the nurse group (Table 4.22).

Table 4.22 – The associations between stress (as measured by LAQ P2) and Stroop test performance outcomes of the nurse group

Dependent Variable	Independent Variable	n	r	p
Stress (LAQ P2)	Response Time (ms)	55	-0.31	0.027*
	Tests Passed	55	0.25	0.075
Stress (DASS-S)	Response Time (ms)	56	-0.26	0.068
	Tests Passed	56	0.15	0.299

Table 4.22 displays the partial correlations (controlling for age, BMI, and years of education) between stress, and both of the Stroop performance variables (response time and number of tested passed) in the nurse group.

Key: BMI = Body Mass Index; DASS = Depression, Anxiety, Stress Scale; LAQ = Lifestyle Appraisal Questionnaire; ms = Milliseconds; n = Sample size; P2 = Part 2; S = Stress; * = Statistical significance

Regarding psychometric assessment of cognitive performance, correlation analysis determined that stress (as measured by part 2 of the LAQ) in the nurse group was significantly correlated with domain specific performance in the memory domain (r = -0.28; p = 0.047; Table 4.23), whilst all other variables were not significantly correlated to stress (as measured by part 2 of the LAQ).

Table 4.23 – The associations between stress (as measured by LAQ P2) and cognitive performance variables of the nurse group

Dependent Variable	Independent Variable	n	r	p
LAQ P2	MMSE	57	0.08	0.595
	Cognistat Total	57	-0.15	0.286
	Orientation	57	< -0.01	0.972
	Attention	57	-0.19	0.170
	Comprehension	57	-0.12	0.397
	Repetition	57	0.05	0.702
	Naming	57	-0.08	0.580
	Construction	57	-0.14	0.320
	Memory	57	-0.28	0.047*
	Calculation	57	0.05	0.739
	Similarities	57	0.13	0.357
	Judgement	57	-0.15	0.284

Table 4.23 displays the partial correlations (controlling for age, BMI, and years of education) between stress (as scored by the LAQ P2) and the global and domain specific cognitive performance variables of the MMSE and the Cognistat in the nurse group.

Key: BMI = Body Mass Index; LAQ = Lifestyle Appraisal Questionnaire; MMSE = Mini-Mental State Exam; n = Sample size; P2 = Part 2; * = Statistical significance

Furthermore, stress (as measured by the DASS) of the nurse group was similarly significantly correlated to domain specific performance in the memory domain (r = -0.29; p = 0.030; Table 4.24); all other psychometric cognitive performance variables were not significantly correlated with stress (as measured by the DASS).

Table 4.24 – The associations between stress (as measured by DASS-S) and cognitive performance variables of the nurse group

Dependent Variable	Independent Variable	n	r	p
DASS-S	MMSE	61	0.02	0.913
	Cognistat Total	61	-0.13	0.328
	Orientation	61	0.09	0.532
	Attention	61	-0.17	0.212
	Comprehension	61	-0.11	0.428
	Repetition	61	0.05	0.722
	Naming	61	0.03	0.834
	Construction	61	-0.21	0.128
	Memory	61	-0.29	0.030*
	Calculation	61	0.04	0.779
	Similarities	61	0.08	0.538
	Judgement	61	-0.05	0.712

Table 4.24 displays the partial correlations (controlling for age, BMI, and years of education) between stress (as scored by the DASS) and the global and domain specific cognitive performance variables of the MMSE and the Cognistat in the nurse group.

Key: BMI = Body Mass Index; DASS = Depression, Anxiety, Stress Scale; MMSE = Mini-Mental State Exam; n = Sample size; S = Stress; * = Statistical significance

With respect to physiological cognitive performance outcomes, correlation analysis determined 8 significant relationships between stress (as measured by part 2 of the LAQ) and EEG variables in the nurse group (Table 4.25). Positive correlations were found between stress and: T_8 delta reactivity (r = 0.34; p = 0.027), CP_3 delta reactivity (r = 0.30; p = 0.040), CP_4 delta reactivity (r = 0.41; p = 0.008), baseline F_3 beta activity (r = 0.32; p = 0.037), baseline FC_z gamma activity (r = 0.41; p = 0.011), and active phase F_3 gamma activity (r = 0.37; p = 0.021). Additionally, two significant negative associations were found between stress (as measured by the LAQ P2) and: F_4 beta reactivity (r = -0.32; p = 0.042), and F_4 gamma reactivity (r = -0.34; p = 0.034). Furthermore, there were no other EEG variable was significantly correlated to stress (as measured by part 2 of the LAQ).

Table 4.25 – The associations between stress (as measured by LAQ P2) and electroencephalography variables in the nurse group

Dependent Variable	Independent Variable	n	r	p
Stress (LAQ P2)	Rδ-T_8	46	0.34	0.027*
	Rδ-CP_3	49	0.34	0.040*
	Rδ-CP_4	44	0.41	0.008*
	Bβ-F_3	45	0.32	0.037*
	Rβ-F_4	44	-0.32	0.042*
	Bγ-FC_z	41	0.41	0.011*
	Aγ-F_3	42	0.37	0.021*
	Rγ-F_4	42	-0.34	0.034*

Table 4.25 displays the significant partial correlations (controlling for age, BMI and years of education) between stress (as measured by the LAQ P2) and frequency domain electroencephalography variables that were used as physiological markers of cognitive performance in the nurse group. All other EEG variables were not significantly correlated to stress and are not presented in this table.

Key: A = Active phase; B = Baseline; BMI = Body Mass Index; C = Central; F = Frontal; LAQ = Lifestyle Appraisal Questionnaire; n = Sample size; P2 = Part 2; T = Temporal; z = Midline; β = Beta; δ = Delta; γ = Gamma; * = Statistical significance

As there were multiple significant correlations between stress (as scored by part 2 of the LAQ) and EEG variables in the nurse group, a forward stepwise general linear regression analysis was performed. The regression analysis retained only one of the 8 originally entered variables (F_4 gamma reactivity), and had an overall significance of $p = 0.013$ (Table 4.26); this variable explained 21.6% of the variance in stress scores ($F = 7.17$; $DF = 1$; $p = 0.013$; $R = 0.465$, $R^2 = 0.216$; $AR^2 = 0.186$).

Table 4.26 – Regression analysis for stress (LAQ P2) and significantly correlated electroencephalography variables in the nurse group

			$R = 0.465$; $R^2 = 0.216$; $AR^2 = 0.186$; SSM = 847.94; dfM= 1; MSM = 847.94; SSR = 3076.74; dfR = 26 ; MSR = 118.34 F = 7.17; p = 0.013*			
Variable	**β**	**SE of β**	**B**	**SE of B**	**t**	**p**
Intercept			24.93	2.11	11.79	< 0.001
Rγ-F_4	-0.47	0.17	-13.26	4.95	-2.68	0.013*

Table 4.26 presents displays a stepwise forward general linear regression analysis between stress (as measured by the LAQ P2) and the significantly correlated physiological EEG variables in the nurse group. Of the 8 EEG variables originally entered into the model, the analysis retained Rγ-F4.

Key: A = Active phase; C = Central; dfM = Degrees of freedom Model; dfR = Degrees of freedom Residual; F = Frontal; LAQ = Lifestyle Appraisal Questionnaire; MSM = Mean squiares Model; MSR = Mean squares Residual; n = Sample size; P = Parietal; P2 = Part 2; SE = Standard Error; SSM = Sum of squares Model; SSR = Sum of squares Residual; δ = Delta; γ = Gamma; * = Statistical significance

In total, 54 physiological EEG variables were significantly correlated to stress (as measured by the DASS; Table 4.27) in the nurse group. Forty-eight of these were found to be positive correlations between stress (as measured by the DASS) and: baseline F_7 delta activity ($r = 0.32$; $p = 0.034$), baseline F_3 delta activity ($r = 0.33$; $p = 0.041$), baseline FC_4 delta activity ($r = 0.31$; $p = 0.035$), baseline C_z delta activity ($r = 0.40$;

p = 0.007), baseline T_8 delta activity (r = 0.39; p = 0.008), baseline CP_4 delta activity (r = 0.45; p = 0.002), baseline P_3 delta activity (r = 0.44; p = 0.003), active phase C_z delta activity (r = 0.46; p = 0.002), active phase O_2 delta activity (r = 0.30; p = 0.036), FC_z delta reactivity (r = 0.34; p = 0.031), CP_3 delta reactivity (r = 0.33; p = 0.024), baseline TP_7 theta activity (r = 0.28; p = 0.044), baseline CP_3 theta activity (r = 0.35; p = 0.022), baseline O_2 theta activity (r = 0.52; p = 0.001), active phase CP_z theta activity (r = 0.415; p = 0.005), active phase O_z theta activity (r = 0.32; p = 0.042), active phase O_2 theta activity (r = 0.34; p = 0.030), CP_z theta reactivity (r = 0.50; p < 0.001), active phase C_3 alpha activity (r = 0.41; p = 0.010), baseline Fp_2 beta activity (r= 0.46; p = 0.003), baseline F_7 beta activity (r = 0.389; p = 0.011), baseline F_4 beta activity (r = 0.34; p = 0.024), baseline FT_7 beta activity (r = 0.39; p = 0.009), baseline FC_z beta activity (r = 0.37; p = 0.019), baseline T_7 beta activity (r = 0.50; p = 0.001), baseline C_3 beta activity (r = 0.37; p = 0.021), baseline P_3 beta activity (r = 0.34; p = 0.025), baseline O_2 beta activity (r = 0.37; p = 0.034), active phase FC_z beta activity (r = 0.35; p = 0.024), active phase TP_8 beta activity (r = 0.32; p = 0.047), active phase O_2 beta activity (r = 0.35; p = 0.037), baseline Fp_2 gamma activity (r = 0.41; p = 0.007), baseline F_3 gamma activity (r = 0.34; p = 0.027), baseline F_4 gamma activity (r = 0.41; p = 0.006), baseline FT_7 gamma activity (r = 0.40; p = 0.006), baseline FC_z gamma activity (r = 0.40; p = 0.011), baseline T_7 gamma activity (r = 0.39; p = 0.009), baseline T_8 gamma activity (r = 0.31; p = 0.044), baseline TP_7 gamma activity (r = 0.31; p = 0.034), baseline P_3 gamma activity (r = 0.52; p = 0.001), baseline P_z gamma activity (r = 0.35; p = 0.031), baseline P_4 gamma activity (r = 0.35; p = 0.032), baseline O_z gamma activity (r = 0.35; p = 0.027), active phase FC_4 gamma activity (r = 0.33; p = 0.043), active phase P_3 gamma activity (r = 0.51; p = 0.001), and active phase P_z gamma activity (r = 0.34; p = 0.039). Additionally, the remaining 8 associations were

found to be significant negative relationships between stress (as measured by the DASS) and: T_8 delta reactivity ($r = -0.38$; $p = 0.012$); CP_4 delta reactivity ($r = -0.52$; $p < 0.001$), P_8 delta reactivity ($r = -0.29$; $p = 0.034$); F_4 theta reactivity ($r = -0.29$; $p = 0.045$), FT_7 beta reactivity ($r = -0.31$; $p = 0.040$), CP_4 beta reactivity ($r = -0.34$; $p = 0.026$), F_4 gamma reactivity ($r = -0.31$; $p = 0.045$), and FT_8 gamma reactivity ($r = -0.30$; $p = 0.045$); all other EEG variables were not significantly associated with the DASS stress score of the nurse group.

Table 4.27 – The associations between stress (as measured by DASS-S) and electroencephalography variables in the nurse group

DV	IV	n	r	p	IV	n	r	p	IV	n	r	p
Stress (DASS)	Bδ-F_7	49	0.32	0.034*	Aθ-O_z	46	0.32	0.042*	Rβ-CP_4	47	-0.34	0.026*
	Bδ-F_3	43	0.33	0.0418	Aθ-O_2	44	0.34	0.030*	Bγ-Fp_2	46	0.41	0.007*
	Bδ-FC_4	50	0.31	0.035*	Rθ-F_4	51	-0.29	0.0458	Bγ-F_3	47	0.34	0.027*
	Bδ-C_z	49	0.40	0.007*	Rθ-CP_z	53	0.50	< 0.001*	Bγ-F_4	47	0.41	0.006*
	Bδ-T_8	50	0.39	0.008*	Aα-C_3	43	0.41	0.010*	Bγ-FT_7	50	0.40	0.006*
	Bδ-CP_4	50	0.45	0.002*	Bβ-Fp_2	46	0.46	0.003*	Bγ-FC_z	44	0.40	0.011*
	Bδ-P_3	48	0.43	0.003*	Bβ-F_7	47	0.39	0.011*	Bγ-T_7	48	0.39	0.009*
	Aδ-C_z	49	0.46	0.002*	Bβ-F_4	47	0.34	0.024*	Bγ-T_8	47	0.31	0.044*
	Aδ-O_2	52	0.30	0.036*	Bβ-FT_7	49	0.39	0.009*	Bγ-TP_7	51	0.31	0.034*
	Rδ-FC_z	46	0.34	0.031*	Bβ-FC_z	46	0.37	0.019*	Bγ-P_3	44	0.52	0.001*
	Rδ-T_8	48	-0.38	0.012*	Bβ-T_7	46	0.50	0.001*	Bγ-P_z	43	0.35	0.031*
	Rδ-CP_3	53	0.33	0.024*	Bβ-C_3	43	0.37	0.021*	Bγ-P_4	42	0.35	0.032*
	Rδ-CP_4	48	-0.52	< 0.001*	Bβ-P_3	48	0.34	0.025*	Bγ-O_z	44	0.35	0.027*
	Rδ-P_8	45	-0.29	0.034*	Bβ-O_2	37	0.37	0.034*	Aγ-FC_4	43	0.33	0.043*
	Bθ-TP_7	56	0.28	0.044*	Aβ-FC_z	46	0.35	0.024*	Aγ-P_3	43	0.51	0.001*
	Bθ-CP_3	49	0.35	0.022*	Aβ-TP_8	43	0.32	0.047*	Aγ-P_z	43	0.34	0.039*
	Bθ-O_2	42	0.52	0.001*	Aβ-O_2	40	0.35	0.037*	Rγ-F_4	46	-0.31	0.045*
	Aθ-CP_z	51	0.41	0.005*	Rβ-FT_7	49	-0.31	0.040*	Rγ-FT_8	50	-0.30	0.045*

Table 4.27 displays the significant partial correlations (controlling for age, BMI, and years of education) between stress (as measured by the DASS) and time domain and frequency domain electroencephalography variables that were used as physiological markers of cognitive performance in the nurse group. All other EEG variables were not significantly correlated to stress (as measured by the DASS) and are not presented in this table.

Key: A = Active phase; B = Baseline; BMI = Body Mass Index; C = Central; DASS = Depression, Anxiety, Stress Scale; DV = Dependent Variable; F = Frontal; Fp = Frontal pole; IV = Independent variable; n = Sample size; O = Occipital; P = Parietal; R = Reactivity; T = Temporal; z = Midline; α = Alpha; β = Beta; δ = Delta; γ = Gamma; θ = Theta; * = Statistical significance; < = Less than

As there were multiple significant correlations between stress (as scored by the DASS) and EEG variables, a forward stepwise general linear regression analysis was performed.

The regression analysis retained 14 of the 54 originally entered variables (baseline F_7 delta activity, baseline FC_4 delta activity, baseline C_z delta activity, baseline CP_4 delta activity, baseline P_3 delta activity, active phase O_2 delta activity, FC_z delta reactivity, active phase O_2 theta activity, F_4 theta reactivity, baseline FC_z beta activity, baseline T_7 beta activity, CP_4 beta reactivity, baseline TP_7 gamma activity, and baseline P_4 gamma activity), and had an overall significance of $p < 0.001$ (Table 4.28).

These 14 variables together explained 100.0% of the variance in stress scores ($F = 1.79 \times 10^7$; $DF = 14$; $p < 0.001$; $R = 1.000$, $R^2 = 1.000$; $AR^2 = 1.000$). Furthermore, all of the retained variables except for F_4 theta reactivity, presented as independently significant predictors of stress ($p \leq 0.014$).

Table 4.28 – Regression analysis for stress (DASS-S) and significantly correlated electroencephalography variables in the nurse group

$R = 1.000$; $R^2 = 1.000$; $AR^2 = 1.000$;
SSM =934.00; dfM= 14; MSM = 66.71
SSR < 0.001; dfR = 1; MSR < 0.001
$F = 1.79 \times 10^7$; $p < 0.001$*

Variable	β	SE of β	B	SE of B	t	p
Intercept			4.03	0.005	847.71	< 0.001*
Bδ-F_7	1.23	< 0.001	0.16	< 0.001	9120.47	< 0.001*
Bδ-FC_4	-0.97	< 0.001	-0.07	< 0.001	-4740.52	< 0.001*
Bδ-C_Z	-0.44	< 0 .001	-0.08	< 0.001	-2669.71	< 0.001*
Bδ-CP_4	1.16	< 0.001	0.11	< 0.001	4475.22	< 0.001*
Bδ-P_3	-0.35	< 0.001	-0.02	< 0.001	-2134.55	< 0.001*
Aδ-O_2	0.14	< 0.001	0.01	< 0.001	560.91	0.001*
Rδ-FC_z	-0.09	< 0.001	-0.04	< 0.001	-693.02	< 0.001*
Aθ-O_2	-0.16	< 0.001	-0.19	< 0.001	-327.74	0.002*
Rθ-F_4	< -0.01	< 0.001	< -0.01	< 0.001	-10.21	0.062
Bβ-FC_z	0.40	< 0.001	1.11	< 0.001	1814.14	< 0.001*
Bβ-T_7	0.07	< 0.001	0.05	< 0.001	166.29	0.004*
Rβ-CP_4	-0.01	< 0.001	-0.04	< 0.001	-45.80	0.014*
Bγ-TP_7	-0.10	< 0.001	-0.65	< 0.01	-288.29	0.002*
Bγ-P_4	0.15	< 0.001	2.63	< 0.01	1408.08	< 0.001*

Table 4.28 presents displays a stepwise forward general linear regression analysis between stress (as measured by the DASS) and the significantly correlated physiological EEG variables. Of the 54 EEG variables originally entered into the model, the analysis retained 14: Bδ-F7, Bδ-FC4, Bδ-CZ, Bδ-CP4, Bδ-P3, Aδ-O2, Rδ-FCz, Aθ-O2, Rθ-F4, Bβ-FCz, Bβ-T7, Rβ-CP4, Bγ-TP7, and Bγ-P4.

Key: A = Active phase; dfM = Degrees of freedom model; dfR = Degrees of freedom residual; F = Frontal; MSM = Mean square model; MSR = Mean square residual; O = Occipital; P = Parietal; R = Reactivity; SE = Standard Error; SSM = Sum of Squares Model; SSR = Sum, of squares residual; β = Beta; δ = Delta; γ = Gamma; θ = Theta; * = Statistical significance; < = Less than

4.3.4 Anxiety

Regarding participant self-reported anxiety scores (Table 4.29), it was found that nurse participants scored higher on the DASS than their non-health professional counterparts; returning a score of 9.16 ± 8.74 compared to 6.14 ± 7.44, however this difference did not reach statistical significance.

Table 4.29 – The self-reported anxiety scores of the two study sample groups

Variable	Group	n	Value	t	p
Anxiety (DASS)	Nurses	61	9.16 ± 8.74	1.95	0.054
	NHP	51	6.14 ± 7.44		

Table 4.29 presents the mean self-report anxiety scores of both sample groups for the DASS. Furthermore, t and p values are provided for intergroup comparisons for each of these variables.

Key: DASS = Depression, Anxiety, Stress Scale; n = Sample size; NHP = Non-health professionals

4.3.4.1 Non-health professionals

In the non-health professional group (Table 4.30), it was found that anxiety was significantly and positively associated fatigue prior to the experimental protocol ($r = 0.34$; $p = 0.014$) and lifestyle risk factors ($r = 0.41$; $p = 0.003$). Anxiety was not significantly correlated to age, BMI, Waist to Hip Ratio, and years of education.

Table 4.30 – The associations between anxiety and demographic variables of the non-health professional group

Dependent Variable	Independent Variable	n	r	p
Anxiety (DASS)	Age	51	-0.21	0.132
	Yrs. Education	50	-0.08	0.563
	BMI	51	0.06	0.679
	WHR	51	0.02	0.883
	Pre-FSQ	51	0.34	0.014*
	Post-FSQ	51	0.25	0.084
	LAQ P1	51	0.41	0.003*

Table 4.30 displays the correlations between anxiety, and demographic variables (age, BMI, WHR, fatigue, and lifestyle risk factors (LAQ P1)) for the non-health professional group.

Key: BMI = Body Mass Index; DASS = Depression, Anxiety, Stress Scale; FSQ = Fatigue State Question; LAQ = Lifestyle Appraisal Questionnaire; n = Sample size; P1 = Part 1; WHR = Waist/Hip Ratio; Yrs = Years; * = Statistical significance

The present analysis, identified strong positive relationships (Table 4.31) between anxiety scores, and stress scores as measured by part 2 of the LAQ ($r = 0.76$; $p < 0.001$) and the DASS ($r = 0.84$; $p < 0.001$).

Table 4.31 – The associations between anxiety (as measured by DASS-A) and self-reported stress scores of the non-health professional group

Dependent Variable	Independent Variable	n	r	p
Anxiety (DASS)	LAQ P2	51	0.76	< 0.001*
	DASS-S	51	0.84	< 0.001*

Table 4.31 displays the correlations between anxiety, and self-reported stress scores (LAQ P2 and DASS-S) for the non-health professional group.

Key: DASS = Depression, Anxiety, Stress Scale; LAQ = Lifestyle Appraisal Questionnaire; n = Sample size; P2 = Part 2; S = Stress; * = Statistical significance; < = Less than

Anxiety scores of the non-health professional group were found to be significantly and positively correlated (Table 4.32) with wishful thinking (r = 0.40; p = 0.004) and avoidance based coping strategies (r = 0.35; p = 0.013).

Table 4.32 – The associations between stress and self-reported coping strategy utilisation of the non-health professional group

Dependent Variable	Independent Variable	n	r	p
Anxiety (DASS)	Problem Focused	51	0.21	0.146
	Self-support	51	0.19	0.184
	Self-blame	51	0.26	0.061
	Wishful Thinking	51	0.40	0.004*
	Avoidance	51	0.35	0.013*

Table 4.32 displays the correlations between anxiety and the categories of coping strategy examined by the Ways of Coping checklist for the non-health professional group.

Key: DASS = Depression, Anxiety, Stress Scale; n = Sample size; * = Statistical significance

In terms of Stroop test performance (Table 4.33), the anxiety score of non-health professionals was significantly correlated to average response time (r = 0.31; p = 0.037), but not the total number of tests passed.

Table 4.33 – The associations between anxiety and Stroop test performance outcomes of the non-health professional group

Dependent Variable	Independent Variable	n	r	p
Anxiety (DASS)	Response Time (ms)	50	0.31	0.037*
	Tests Passed	50	-0.23	0.130

Table 4.33 displays the partial correlations (controlling for age, BMI, and years of education) between anxiety and both of the Stroop performance variables (response time and number of tested passed) for the non-health professional group.

Key: BMI = Body Mass Index; DASS = Depression, Anxiety, Stress Scale; ms = Milliseconds; n = Sample size; * = Statistical significance

With respect to the psychometric cognitive performance (Table 4.34), anxiety scores of the non-health professional group were negatively and significantly correlated to global cognitive performance as assessed by the MMSE (r = -0.35; p = 0.015) and the Cognistat (r = -0.31; p = 0.036). Furthermore, domain specific performance in the attention domain was also significantly and negatively associated with anxiety scores (r = -0.31 p = 0.036). All other domain specific cognitive variables of the MMSE and Cognistat were not significantly correlated to anxiety.

Table 4.34 – The associations between anxiety and cognitive performance variables of the non-health professional group

Dependent Variable	Independent Variable	n	r	p
Anxiety (DASS)	MMSE	51	-0.35	0.015*
	Cognistat Total	51	-0.31	0.036*
	Orientation	51	-0.09	0.547
	Attention	51	-0.31	0.036*
	Comprehension	51	0.15	0.327
	Repetition	51	-0.23	0.114
	Naming	51	-0.13	0.383
	Construction	51	-0.18	0.233
	Memory	51	-0.22	0.136
	Calculation	51	-0.13	0.386
	Similarities	51	-0.21	0.155
	Judgement	51	0.20	0.179

Table 4.34 displays the partial correlations (controlling for age, BMI, and years of education) for the non-health professional sample group between anxiety and the global and domain specific cognitive performance variables of the MMSE and the Cognistat in the non-health professional group.

Key: BMI = Body Mass Index; DASS = Depression, Anxiety, Stress Scale; MMSE = Mini-Mental State Exam; n = Sample size; * = Statistical significance

In the non-health professional group (Table 4.35), anxiety was significantly and positively associated with: baseline T_8 theta activity (r = 0.34; p = 0.038); active phase F_3 alpha activity (r = 0.40; p = 0.020); F_3 alpha reactivity (r = 0.34; p = 0.046); baseline P_7 gamma activity (r = 0.36; p = 0.026); active phase CP_3 gamma activity (r = 0.36; p = 0.034); active phase P_4 gamma activity (r = 0.38; p = 0.018); active phase O_2 activity (r = 0.42; p = 0.016); F_3 gamma reactivity (r = 0.36; p = 0.030); and P_4 gamma reactivity (r = 0.38; p = 0.014). Furthermore, anxiety was also significantly negatively correlated to P_7 gamma reactivity (r = -0.38; p = 0.015). All other EEG variables were not significantly correlated to anxiety scores of the non-health professional group.

Table 4.35 – The associations between anxiety and electroencephalography variables of the non-health professional group

Dependent Variable	Independent Variable	n	r	p
Anxiety (DASS)	Bθ-T_8	41	0.34	0.038*
	Aα-F_3	38	0.40	0.020*
	Rα-F_3	40	0.34	0.046*
	Bγ-P_7	42	0.36	0.026*
	Aγ-CP_3	40	0.36	0.034*
	Aγ-P_4	43	0.38	0.018*
	Aγ-O_2	37	0.42	0.016*
	Rγ-F_3	40	0.36	0.030*
	Rγ-P_7	45	-0.38	0.015*
	Rγ-P_4	45	0.38	0.014*

Table 4.35 displays the significant partial correlations (controlling for age, BMI and years of education) for the non-health professional sample group between anxiety and electroencephalography variables that were used as physiological markers of cognitive performance. All other EEG variables were not significantly correlated to anxiety and are not presented in this table.

Key: A = Active Phase; B = Baseline; BMI = Body Mass Index; C = Central; DASS = Depression, Anxiety, Stress Scale; F = Frontal; n = Sample size; O = Occipital; P = Parietal; R = Reactivity; T = Temporal; α = Alpha; γ = Gamma; θ = Theta; * = Statistical significance

As there were multiple significant correlations between anxiety and EEG variables, a forward stepwise general linear regression analysis was performed. The regression analysis retained 4 of the 10 originally entered variables (F_3 alpha reactivity, active phase P_4 gamma activity, active phase O_2 gamma activity, and F_3 gamma reactivity), and had an overall significance of $p < 0.001$ (Table 4.36). Together these four variables explained 71.7% of the variance in anxiety scores ($F = 15.17$; $DF = 4$; $p < 0.001$; $R = 0.846$, $R^2 = 0.717$; $AR^2 = 0.669$). All of the retained variables presented as independently significant predictors of anxiety ($p \leq 0.004$).

Table 4.36 – Regression analysis for anxiety and significantly correlated electroencephalography variables in the non-health professional group

			$R = 0.846$; $R^2 = 0.717$; $AR^2 = 0.669$; SSM = 1372.04; dfM= 4; MSM = 343.01; SSR = 542.79; dfR = 24; MSR = 22.62 $F = 15.17$; $p < 0.001$			
Variable	**β**	**SE of β**	**B**	**SE of B**	**t**	**p**
Intercept			3.83	1.21	3.16	0.004*
Rα-F_3	0.68	0.14	0.90	0.18	4.95	< 0.001*
Aγ-P_4	0.71	0.16	4.16	0.96	4.33	< 0.001*
Aγ-O_2	0.39	0.12	1.09	0.34	3.24	0.004*
Rγ-F_3	-0.62	0.17	-11.43	3.23	-3.54	0.002*

Table 4.36 presents displays a stepwise forward general linear regression analysis between anxiety (as measured by the DASS) and the significantly correlated physiological EEG variables in the non-health professional. Of the 10 EEG variables originally entered into the model, the analysis retained 4: Rα-F3, Aγ-P4, Aγ-O2, and Rγ-F3.

Key: A = Active; dfM = Degrees of freedom model; dfR = Degrees of freedom residual; F = Frontal; MSM = Mean square model; MSR = Mean square residual; O = Occipital; P = Parietal; R = Reactivity; SE = Standard Error; SSM = Sum of Squares Model; SSR = Sum, of squares residual; α = Alpha; γ = Gamma; * = Statistical significance; < = Less than

4.3.4.2 Nurses

In the nurse group (Table 4.37), anxiety was significantly correlated with fatigue scores before (r = 0.66; p = 0.007) and after (r = 0.59; p = 0.020) the experimental protocol. Additionally, anxiety was not significantly correlated to age, BMI, Waist to Hip ratio, years of education, and lifestyle risk factors (part 1 of the LAQ).

Table 4.37 – The associations between anxiety and demographic variables of the nurse group

Dependent Variable	Independent Variable	n	r	p
Anxiety (DASS)	Age	61	-0.01	0.920
	Yrs. Education	59	-0.25	0.060
	BMI	61	< -0.01	0.991
	WHR	15	0.10	0.729
	Pre-FSQ	15	0.66	0.007*
	Post-FSQ	15	0.59	0.020*
	LAQ P1	57	0.16	0.237

Table 4.37 displays the correlations between anxiety (as measured by the DASS), and demographic variables (age, BMI, WHR, fatigue, and lifestyle risk factors (LAQ P1)) for the nurse group.

Key: BMI = Body Mass Index; DASS = Depression, Anxiety, Stress Scale; FSQ = Fatigue State Question; LAQ = Lifestyle Appraisal Questionnaire; n = Sample size; P1 = Part 1; WHR = Waist/Hip Ratio; Yrs = Years; * = Statistical significance

Similar to their non-health professional counter parts, the anxiety score of the nurse group (Table 4.38) was significantly and positively correlated to stress scores as measured by part 2 of the LAQ (r = 0.615; p < 0.001) and the DASS (r = 0.823; p < 0.001).

Table 4.38 – The associations between anxiety and self-reported stress scores of the nurse group

Dependent Variable	Independent Variable	n	r	p
Anxiety (DASS)	LAQ P2	57	0.62	< 0.001*
	DASS-S	61	0.82	< 0.001*

Table 4.38 displays the correlations between anxiety, and self-reported stress scores (LAQ P2 and DASS-S) for the nurse group.

Key: DASS = Depression, Anxiety, Stress Scale; LAQ = Lifestyle Appraisal Questionnaire; n = Sample size; P2 = Part 2; S = Stress; * = Statistical significance; < = Less than

Unlike their non-health professional counterparts, the anxiety score of the nurse sample group was not significantly associated with any of the coping strategy categories examined (Table 4.39), although the low sample count may explain this.

Table 4.39 - The associations between anxiety and self-reported coping strategy utilisation of the nurse group

Dependent Variable	Independent Variable	n	r	p
Anxiety (DASS)	Problem Focused	15	-0.22	0.429
	Self-support	15	0.48	0.068
	Self-blame	15	0.27	0.339
	Wishful Thinking	15	0.13	0.638
	Avoidance	15	-0.21	0.439

Table 4.39 displays the correlations between anxiety and the categories of coping strategy examined by the Ways of Coping checklist for the nurse group.

Key: DASS = Depression, Anxiety, Stress Scale; n = Sample size;

In addition, the anxiety score for the nurse group was not significantly correlated to either Stroop performance variable (Table 4.40).

Table 4.40 – The associations between anxiety and Stroop test performance outcomes of the nurse sample group

Dependent Variable	Independent Variable	n	r	p
Anxiety (DASS)	Response Time (ms)	56	-0.21	0.134
	Tests Passed	56	0.10	0.474

Table 4.40 displays the partial correlations (controlling for age, BMI and years of education) for the nurse sample group between anxiety, and both of the Stroop performance variables (response time and number of tested passed) in the nurse group.

Key: BMI = Body Mass Index; DASS = Depression, Anxiety, Stress Scale; n = Sample size; ms = Milliseconds

Further, regarding the psychometric cognitive performance of the nurse group (Table 4.41), anxiety score was significantly positively correlated to domain specific performance in the memory domain ($r = -0.39$, $p = 0.003$); all other psychometric cognitive variables were not significantly correlated to anxiety.

Table 4.41 – The associations between anxiety and cognitive performance variables of the nurse group

Dependent Variable	Independent Variable	n	r	p
Anxiety (DASS)	MMSE	61	-0.03	0.842
	Cognistat Total	61	-0.20	0.140
	Orientation	61	0.11	0.441
	Attention	61	-0.23	0.092
	Comprehension	61	-0.18	0.187
	Repetition	61	-0.02	0.880
	Naming	61	-0.10	0.455
	Construction	61	-0.16	0.240
	Memory	61	-0.39	0.003*
	Calculation	61	0.12	0.371
	Similarities	61	0.16	0.244
	Judgement	61	-0.11	0.442

Table 4.41 displays the partial correlations (controlling for age, BMI and years of education) for the nurse sample group between anxiety and the global and domain specific cognitive performance variables of the MMSE and the Cognistat in the nurse group.

Key: BMI = Body Mass Index; DASS = Depression, Anxiety, Stress Scale; MMSE = Mini-Mental State Exam; n = Sample size; * = Statistical significance

The present correlation analysis found that anxiety score in the nurse group was significantly correlated with 52 EEG variables (Table 4.42). Positive correlations were found between anxiety and: baseline F_3 delta activity ($r = 0.45$; $p = 0.004$), baseline F_4 delta activity ($r = 0.35$; $p = 0.021$), baseline FT_7 delta activity ($r = 0.42$; $p = 0.006$), baseline FC_4 delta activity ($r = 0.37$; $p = 0.014$), baseline C_z delta activity ($r = 0.49$; $p = 0.001$), baseline CP_4 delta activity ($r= 0.36$; $p = 0.015$), baseline P_3 delta activity ($r = 0.40$; $p = 0.009$), active phase F_7 delta activity ($r = 0.30$; $p = 0.049$), active phase F_3 delta activity ($r = 0.33$, $p = 0.035$), active phase F_4 delta activity ($r = 0.37$, $p = 0.017$), active phase C_z delta activity ($r = 0.51$, $p < 0.001$), FC_z delta reactivity ($r = 0.34$; $p = 0.029$), CP_3 delta reactivity ($r = 0.31$; $p = 0.030$), P_8 delta reactivity ($r = 0.30$; $p = 0.043$), baseline F_7 theta activity ($r = 0.31$; $p = 0.043$), baseline FC_z theta activity ($r = 0.40$; $p = 0.005$), baseline C_3 theta activity ($r = 0.32$; $p = 0.034$), baseline CP_3 theta activity ($r = 0.44$; $p = 0.003$), baseline P_3 theta activity ($r = 0.31$; $p = 0.042$), baseline O_2 theta activity ($r = 0.50$; $p = 0.001$), active phase C_3 theta activity ($r = 0.44$; $p = 0.005$), active phase T_8 theta activity ($r = 0.37$; $p = 0.013$), active phase TP_7 theta activity ($r = 0.36$; $p = 0.011$), active phase CP_z theta activity ($r = 0.45$; $p = 0.002$), CP_z theta reactivity ($r = 0.41$; $p = 0.004$), active phase CP_z alpha activity ($r = 0.41$; $p = 0.010$), F_8 alpha reactivity ($r = 0.34$; $p = 0.024$), baseline Fp_2 beta activity ($r = 0.45$; $p = 0.024$), baseline F_7 beta activity ($r = 0.43$; $p = 0.005$), baseline F_4 beta activity ($r = 0.36$; $p = 0.018$), baseline T_7 beta activity ($r = 0.39$; $p = 0.013$), baseline C_3 beta activity ($r = 0.43$; $p = 0.008$), baseline C_4 beta activity ($r = 0.34$; $p = 0.028$), baseline CP_z beta activity ($r = 0.37$; $p = 0.021$), baseline CP_4 beta activity ($r = 0.34$; $p = 0.032$), baseline P_z beta activity ($r = 0.32$; $p = 0.049$), active phase F_7 beta activity ($r = 0.35$; $p = 0.029$), active phase FC_z beta activity ($r = 0.38$; $p = 0.014$), baseline Fp_2 gamma activity ($r = 0.46$; $p = 0.003$), baseline F_3 gamma activity ($r = 0.43$; $p = 0.005$), baseline

F_4 gamma activity (r = 0.34; p = 0.024), baseline FC_3 gamma activity (r = 0.44; p = 0.005), baseline FC_z gamma activity (r = 0.40; p = 0.011), baseline C_3 gamma activity (r = 0.44; p = 0.006), baseline C_4 gamma activity (r = 0.41; p = 0.007), baseline P_3 gamma activity (r = 0.46; p = 0.003), baseline P_z gamma activity (r = 0.45; p = 0.004), active phase P_3 gamma activity (r = 0.48; p = 0.002), and active phase P_z gamma activity (r = 0.36; p = 0.028). Additionally, negative correlations were found between anxiety and: CP_4 delta reactivity (r = -0.43; p = 0.004), CP_4 beta reactivity (r = -0.32; p = 0.041), and FT_8 gamma reactivity (r = -0.31; p = 0.039); no other EEG variable was significantly associated with anxiety.

As multiple EEG variables were significantly correlated to anxiety of the nurse group, a forward stepwise general linear regression analysis was performed. The regression analysis retained 12 of the 52 originally entered variables (baseline F_3 delta activity, baseline FT_7 delta activity, baseline C_z delta activity, baseline P_3 delta activity, active phase F_3 delta activity, P_8 delta reactivity, active phase C_3 theta activity, baseline FC_3 gamma activity, baseline FC_z gamma activity, baseline P_3 gamma activity, baseline P_z gamma activity, and active phase P_3 gamma activity), and had an overall significance of p = 0.003 (Table 4.42).

These 12 variables explained 99.9% of the variance in anxiety scores ($F = 7.48 \times 10^2$ DF = 12; p = 0.003; R = 0.999, R^2 = 0.999; AR^2 = 0.999). Furthermore, all of the retained variables also presented as independently significant predictors of anxiety ($p \leq 0.036$).

Table 4.42 – The associations between anxiety and electroencephalography variables in the nurse group

DV	IV	n	r	p	IV	n	r	p	IV	n	r	p
Anxiety (DASS)	Bδ-F3	43	0.45	0.004*	Bθ-CP_3	49	0.44	0.003*	Bβ-P_z	44	0.32	0.049*
	Bδ-F4	48	0.35	0.021*	Bθ-P_3	48	0.31	0.042*	Aβ-F_7	44	0.35	0.029*
	Bδ-FT7	46	0.42	0.006*	Bθ-O_2	42	0.50	0.001*	Aβ-FC_z	46	0.38	0.014*
	Bδ-FC4	50	0.37	0.014*	Aθ-C_3	44	0.44	0.005*	Rβ-CP_4	47	-0.32	0.041*
	Bδ-Cz	49	0.49	0.001*	Aθ-T_8	47	0.37	0.0138*	Bγ-Fp_2	46	0.46	0.003*
	Bδ-CP4	50	0.36	0.015*	Aθ-TP_7	52	0.36	0.011*	Bγ-F_3	47	0.43	0.005*
	Bδ-P3	48	0.40	0.009*	Aθ-CP_z	51	0.45	0.002*	Bγ-F_4	47	0.34	0.024*
	Aδ-F7	50	0.30	0.049*	Rθ-CP_z	53	0.41	0.004*	Bγ-FC_3	44	0.44	0.005*
	Aδ-F3	47	0.33	0.035*	Aα-CP_z	43	0.41	0.010*	Bγ-FC_z	44	0.40	0.011*
	Aδ-F4	46	0.37	0.017*	Rα-F_8	47	0.34	0.024*	Bγ-C_3	42	0.44	0.006*
	Aδ-Cz	49	0.51	< 0.001*	Bβ-Fp_2	46	0.45	0.003*	Bγ-C_4	46	0.41	0.007*
	Rδ-FCz	46	0.34	0.029*	Bβ-F_7	47	0.43	0.0058	Bγ-P_3	44	0.46	0.003*
	Rδ-CP3	53	0.31	0.030*	Bβ-F_4	47	0.36	0.018*	Bγ-P_z	43	0.45	0.004*
	Rδ-CP4	48	-0.43	0.004*	Bβ-T_7	46	0.39	0.013*	Aγ-P_3	43	0.48	0.002*
	Rδ-P8	52	0.30	0.043*	Bβ-C_3	43	0.43	0.008*	Aγ-P_z	43	0.36	0.028*
	Bθ-F7	50	0.31	0.039*	Bβ-C_4	47	0.34	0.028*	Rγ-FT_8	50	-0.31	0.039*
	Bθ-FCz	52	0.40	0.005*	Bβ-CP_z	44	0.37	0.021*				
	Bθ-C3	47	0.32	0.034*	Bβ-CP_4	46	0.34	0.032*				

Table 4.42 displays the significant partial correlations (controlling for age, BMI, and years of education) between anxiety and electroencephalography variables that were used as physiological markers of cognitive performance for the nurse group. All other EEG variables were not significantly correlated to anxiety and are not presented in this table.

Key: A = Active phase; B = Baseline; BMI = Body Mass Index; C = Central; DASS = Depression, Anxiety, Stress Scale; DV; Dependent Variable; F = Frontal; Fp = Frontal pole; IV = Independent variable; n = Sample size; O = Occipital; P = Parietal; R = Reactivity; T = Temporal; z = Midline; α = Alpha; β = Beta; δ = Delta; γ = Gamma; θ = Theta; * = Statistical significance; < = Less than

Table 4.43 – Regression analysis for anxiety and significantly correlated electroencephalography variables in the nurse group

			$R = 0.999$; $R^2 = 0.999$; $AR^2 = 0.999$; SSM = 880.93; dfM= 12; MSM = 73.41; SSR < 0.001; dfR = 1; MSR < 0.001 $F = 7.48 \times 10^2$; p = 0.003*			
Variable	**β**	**SE of β**	**B**	**SE of B**	**t**	**p**
Intercept			6.59	0.07	99.80	0.006*
Bδ-F_3	-2.13	0.01	-0.58	< 0.01	-194.01	0.003*
Bδ-FT_7	1.25	< 0.01	0.24	< 0.01	375.78	0.002*
Bδ-C_z	0.50	< 0.01	0.10	< 0.001	220.63	0.003*
Bδ-P_3	-0.26	< 0.01	-0.01	< 0.001	-78.87	0.008*
Aδ-F_3	0.63	< 0.01	0.14	< 0.01	82.99	0.008*
Rδ-P_8	-0.09	< 0.01	-0.02	< 0.01	-17.46	0.036*
Aθ-C_3	-0.18	< 0.01	-1.25	0.03	-49.61	0.013*
Bγ-FC_3	2.43	0.01	129.84	0.51	257.07	0.002*
Bγ-FC_z	-0.42	< 0.01	-27.11	0.13	-202.53	0.003*
Bγ-P_3	-0.97	0.01	-44.83	0.29	-155.09	0.004*
Bγ-P_z	-0.61	< 0.01	-47.66	0.31	-155.05	0.004*
Aγ-P_3	-0.22	< 0.01	-8.10	0.10	-79.40	0.008*

Table 4.43 presents displays a stepwise forward general linear regression analysis between anxiety (as measured by the DASS) and the significantly correlated physiological EEG variables in the nurse group. Of the 52 EEG variables originally entered into the model, the analysis retained 12: Bδ-F3, Bδ-FT7, Bδ-Cz, Bδ-P3, Aδ-F3, Rδ-P8, Aθ-C3, Bγ-FC3, Bγ-FCz, Bγ-P3, and Bγ-Pz.

Key: A = Active phase; B = Baseline; C = Central; dfM = Degrees of freedom model; dfR = Degrees of freedom residual; F = Frontal; MSM = Mean square model; MSR = Mean square residual; P = Parietal; SE = Standard error; SSM = Sum of squares model; SSR = Sum of squares residual; T = Temporal; z = Midline; δ = Delta; γ = Gamma; θ = Theta; * = Statistical significance; < = Less than

4.4 Discussion

The present study aimed to investigate the associations between stress, anxiety, and cognitive performance in nurses, and expand on the electroencephalographic findings reported in Chapter 3; hypothesising that increased stress and anxiety levels would be associated with cognitive impairment, and alterations in EEG activity particularly of the theta, beta and gamma frequency bands.

Presently, stress was associated with improved judgement and decreased fronto-temporal delta activity in non-health professionals, as well as impaired memory, increased fronto-central delta activity, and increased fronto-central and front-temporal beta activity in nurses. Additionally, increases in fronto-temporal and fronto-parietal gamma activity were positively associated with stress in both sample groups. Anxiety was associated with decreased global cognitive performance and attention domain performance in non-health professionals, and decreased memory performance in nurses. Further, increases in fronto-parietal delta and gamma activity were positively correlated with anxiety in both sample groups.

In the following sections, these results will be discussed with respect to these aims and hypotheses, as well as previous literature that has also reported on the impact of stress and anxiety on cognitive function, and the results previously presented in Chapter 3.

4.4.1 Demographics

The present study captured a number of important demographic variables for the analysis, some of which differed significantly between the non-health professional and nurse sample groups. Lifestyle risk factors associated with an increased risk of disease development (part 1 of the LAQ) was one such variable with the nurse group reporting a

higher score. This difference may be related to the shift-working lifestyle of the nurse group, which has been previously associated with poor lifestyle outcomes (Harrington, 2001, Munakata et al., 2001, Berger and Hobbs, 2006). Furthermore, despite this intergroup difference, when compared to published normative data (the representative questionnaire score for a large general population; Craig et al. (1996)), the scores for part 1 of the LAQ of both groups were below the published value. A result that may be attributed to improved health literacy (Nutbeam, 2000), and/or in the instance of the nurse group their formal health education.

Furthermore, it was found that the length of formal education differed between the two groups, with the non-health professional group reporting a lengthier education than their nurse counterparts; possibly due to the nature of nursing where some registrations (e.g. enrolled nurses) require short training periods (1 -2 years). Research has suggested that formal education may modulate cognitive performance (Launer et al., 1999, Le Carret et al., 2003) and for that reason the present analysis controlled for years of education where relevant.

4.4.2 Cognitive performance

4.4.2.1 Psychometric

With respect to the present psychometric assessment of cognitive performance, it was found that global cognitive performance scores of both the non-health professional and nurse groups were above the relevant impairment thresholds (the test score/s below which potential cognitive impairment or degrees of cognitive impairment is indicated). However, both groups were below the borderline threshold score for the judgement domain (Mueller et al., 2007). Additionally, the nurse group were also below the borderline threshold score for the repetition domain (Mueller et al., 2007), potentially

demonstrating the importance of these domains. The scores of both groups on all remaining domain specific measures were above their respective thresholds.

When comparing the cognitive performance of the two groups, a number of domains were identified to differ between the two sample groups. It was found that the nurse group outperformed their non-health professional counterparts in the orientation domain, whilst the non-health professionals outperformed in global performance of the Cognistat, as well as the attention, similarities, and comprehension domains. It could be suggested that these differences are a result of the everyday demands, which may prime the various cognitive processes of an individual. Since task repetition, i.e. training, is associated with improved performance (Edwards et al., 2005, Anguera et al., 2013) it would follow that individuals who rely more frequently certain processes e.g. nurses and orientation, would outperform individuals who are not primed by this process. Furthermore, it is possible that the aforementioned variance in years of education and the effect this can have on cognitive performance could have also contributed to these reported differences, although the present analysis controlled for education where possible.

4.4.2.2 Electroencephalography

Frequency domain EEG provided a second and physiological measure of cognitive performance in the present analysis; and when comparing the two experimental phases a number of significant changes were identified. Additionally, PCA identified the importance of the delta, theta and gamma frequency bands for both sample groups.

With respect to EEG activity changes, it was found that Fp_2 delta activity decreased significantly in the non-health professional group between the experimental phases (from baseline to active), whilst FT_7, CP_4, P_z and O_z delta activity increased

significantly in the nurse group. Moreover, in the theta frequency band, activity at Fp_1, Fp_2, F_7, F_8 and FT_7 in the nurse group, and at Fp_1, F_7, FT_7 and C_4 in the non-health professional group increased significantly; further, F_z theta activity decreased significantly in the non-health professional group.

Alpha activity was found to be the most variable of the investigated frequency bands between the experimental phases with significant decreases at locations Fp_1, F_7, F_z, F_4, FT_7, FC_z, FC_4, T_7, C_3, C_4, T_8, TP_7, CP_3, CP_z, CP_4, TP_8, P_7, P_3, P_z, P_4, P_8, and O_1, and Fp_1, F_7, F_z, FC_3, FC_7, FC_4, FT_8, C_3, C_4, CP_3, CP_4, P_7, P_3, P_4, and P_8 found for the non-health professional and nurse groups respectively. Likewise, beta activity was also found to have decreased between the phases in both groups, with locations FC_4, C_3, CP_z and at F_7, F_z, FC_4, P_z being implicated for the non-health professional and nurse groups respectively. Lastly, gamma activity at FT_8, TP_7, TP_8, P_4, O_z, in the non-health professional and at O_2 and P_7, P_4, and O_2 in the nurse group was found to have increased significantly. Further, significant decreases in gamma activity at Fp_1, Fp_2, T_8, and P_8 were also recorded for the nurse group.

When examining the spectral EEG correlates of the Stroop test, it is commonly reported that frontal theta activity is associated with the cognitive processes associated with the Stroop test (Hanslmayr et al., 2008, Kovacevic et al., 2012, Jiang et al., 2015). Similarly increased alpha (West and Bell, 1997, Tassi et al., 2006, Hanslmayr et al., 2008, Compton et al., 2011) and beta activity (Schack et al., 1999, Ergen et al., 2014) have also been associated with the Stroop interference effect. The aforementioned activity changes observed in the present analysis align with literature in the theta frequency band, but are in contrast with respect to the alpha and beta bands.

It is possible for these broad activity changes to be representative of the cognitive processes associated with the Stroop test and its interference effect (Moering et al., 2004). Indeed, spectral EEG data have been previously associated with cognitive processes including working memory (Gevins et al., 1997, Onton et al., 2005, Basar-Eroglu et al., 2007), and attention (Harmony et al., 1996, Sauseng et al., 2005), which are engaged during the Stroop test. Alternatively, it could be suggested that repeatedly performing the same motor action and completing the same challenge, as in the Stroop test, could be considered a monotonous task. Thusly, an enhancement of slow wave EEG activity and suppression of fast wave EEG activity similar to the present results would be observed (Torsvall and Åkerstedt, 1988, Papadelis et al., 2006, Jap et al., 2009, Jap et al., 2011). Finally, a third possible explanation for the present results may be the processes associated with preparation and execution of movement which have been previously associated with decreased beta activity (Engel and Fries, 2010) and increased gamma activity (Schoffelen et al., 2005, Donner et al., 2009). Hence, it could be suggested that the observed beta and gamma activity changes are attributable to the hand/arm movements associated with the mouse-click required to complete the present computerised Stroop test, rather than any cognitive interference or monotony effect. Future examination of EEG activity changes related to Stroop response methods (e.g. mouse button press, and verbal report) could be an interesting research avenue, particularly to separate the neural changes associated with cognitive interface effects from the neural changes of the response method.

4.4.3 Stress

Stress research has demonstrated an individual's cognitive performance may be either facilitated (Buchanan and Lovallo, 2001, Beste et al., 2013) or impaired (LeBlanc,

2009, Henderson et al., 2012) by stress, and most importantly, this impairing effect extends to the quality of care provided by nurses (Tarnow-Mordi et al., 2000, Berland et al., 2008). With respect to self-reported stress from the LAQ, the non-health professional group reported a score in line with the previously published age relevant normative data for the LAQ, whilst the nurse group reported scores greater than the normative data (Craig et al., 1996). However, the DASS stress scores of both sample groups were greater than the previously published age relevant normative data (Lovibond and Lovibond, 1995b). Furthermore, the scores of both sample groups were found to be within the normal range (0 – 14); with the nurse sample group bordering the mild range (15- 19) (Lovibond and Lovibond, 1995b).

4.4.3.1 Stress and demographics

The burden of stress placed on an individual, and any changes in behaviour/lifestyle factors that occur as a result has been termed allostatic overload (McEwen, 2008). Stress literature has demonstrated that greater magnitude of experienced stress is associated with increased alcohol consumption (Hemmingsson and Lundberg, 1998, Armeli et al., 2000), fatigue and poor sleep (Åkerstedt et al., 2007), incidence of obesity (Torres and Nowson, 2007) and smoking (Cohen and Lichtenstein, 1990, Steptoe et al., 1996), as well as comorbid diseases including hypertension (Sparrenberger et al., 2008), diabetes (Heraclides et al., 2009), and immunosuppression (Kemeny and Schedlowski, 2007). Presently, both the nurse and non-health professional groups reported a stress score that was correlated with lifestyle risk factors associated with an increased risk of disease development, and fatigue state; in line with literature which indicates stress can have a negative impact on lifestyle (Hatton et al., 1995), further supporting the present results. Additionally, the present analysis identified positive associations between the

two stress scores, and between stress and anxiety scores in both sample groups. These correlations reflect the notion that stress and anxiety are considered to be inter-related negative mental states due to shared physiological processes, and frequent comorbidity (Lovibond and Lovibond, 1995a).

Lastly, the stress scores of the nurse sample group were not correlated with any coping variable. However, both stress scores of the non-health professional group were positively correlated with self-blame, wishful thinking and avoidance coping; strategies that are often considered detrimental (Koeske et al., 1993, Blalock and Joiner, 2000, Dijkstra and Homan, 2016) and have been shown to worsen stress over time (Tattersall et al., 1999, Arnetz, 2001). Hence it is conceivable that an increase in these coping strategies accompanies increased self-reported stress.

4.4.3.2. Stress and cognitive performance

With respect to psychometric cognitive performance, the present analysis found that in the non-health professional group, the perception of stress score obtained from part 2 of the LAQ was positively correlated to judgement domain score (which reflects decision-making performance), and it could be inferred that this relationship reflects the experience of eustress, where performance is optimised by stress. Most previous research does not align with the present result, as it generally indicates that stress influences individuals to use suboptimal decision making processes therefore degrading performance (Baumann and Bourbonnais, 1982, LeBlanc, 2009, Starcke and Brand, 2012). These suboptimal processes may be related to the use of non-systematic scanning strategies and not fully examining all alternatives (Keinan et al., 1987), modulating risk taking behaviour (Porcelli and Delgado, 2009), or an increase in selection of disadvantageous choices (Starcke et al., 2008, Putman et al., 2009). However, stress

may influence the sexes differently, causing men to generally make less advantageous decisions and women to make more advantageous decisions (Preston et al., 2007, Lighthall et al., 2009). Additionally, it is possible that women experience a biphasic effect, where moderate stress improves decision-making and high levels of stress impair it (van den Bos et al., 2009). Such a dimorphism may explain why the present analysis (where stress was categorised as normal in non-health professionals and mild in nurses) associated decision-making capability to stress in the non-health professional group and not in the nurse group, despite both groups being primarily female. However, a more comprehensive sex based inter and intragroup comparison of stress would be required to confirm this.

With reference to the nurse group, it was found that both of the recorded stress scores were negatively correlated to memory domain performance, potentially representing the experience of distress, where stress impairs performance. This is yet another result that is supported by current literature, which largely suggests that memory domain performance is adversely impacted by stress and stress hormones (Sandström et al., 2005, Tollenaar et al., 2008, Wolf, 2008, Comijs et al., 2010). This impairment may be related to stress challenging various components of memory including declarative memory (Kirschbaum et al., 1996, Buchanan and Tranel, 2008), free-recall (de Quervain et al., 2000, Domes et al., 2004, Kuhlmann et al., 2005), and/or working memory (Lupien et al., 1999, Luethi et al., 2008). That said, more recent research has suggested that an inverted U relationship better explains the relationship between stress and memory performance (Human et al., 2013, Sandi, 2013), where low/high levels of stress impair memory and intermediate levels facilitate memory performance. As such, the experimental manipulation of stress between low and high levels to examine this proposed inverted U hypothesis warrants future attention.

4.4.3.3 Stress, cognitive performance and electroencephalography

Frequency domain electroencephalography was used to supplement traditional psychometric measures of cognitive performance. In the present results, the stress score obtained using part 2 of the LAQ for both groups were correlated with delta, beta and gamma band variables. Additionally, theta and alpha band variables were also correlated to the stress score obtained using part 2 of the LAQ of the non-health professional group. Moreover, the DASS stress score of both sample groups was significantly correlated with EEG variables from all five investigated frequency bands (delta, theta, alpha, beta, and gamma), with subsequent regression analysis indicating the importance of fronto-central increases and fronto-temporal decreases in delta activity, fronto-temporal and parietal increases in gamma activity of both groups, and fronto-central and temporal beta activity increases for the nurse group.

Research concerning the delta frequency band and cognitive performance is almost universal in its reporting of delta activity increases being associated with cognitive impairment (Huang et al., 2000, Babiloni et al., 2006b, Babiloni et al., 2008, Babiloni et al., 2010, Dimpfel, 2014). That said, some research has demonstrated delta activity decreases (Lees et al., 2016), and even suggested that early impaired states will demonstrate delta activity decreases, whilst later more complete stages will demonstrate increases (Liddell et al., 2007). In the present analysis, fronto-temporal decreases were observed in the non-health professional group and fronto-central increases in delta activity were observed in the nurse sample group. It could be suggested that these differing changes in delta activity could be related to degree of impairment, and indeed may explain the differing effect of stress on the psychometric performance of the two sample groups.

Moving higher in the frequency bands, the present analysis reported fronto-central and temporal increases in beta activity; a result that is in juxtaposition to most literature which has associated a reduction in beta activity with cognitively impaired states (Huang et al., 2000, Stam et al., 2003, Koenig et al., 2005, Güntekin et al., 2013, Lees et al., 2016). However, it has been suggested that cognitive status may modulate this relationship, with beta activity increases associated to performance in healthy individuals and decreases in cognitively impaired individuals (Lee et al., 2010), and indeed some research has demonstrated beta activity increases in cognitive impairment (Brunovsky et al., 2003, Pachou et al., 2008). Further, with respect to the gamma frequency band the present analysis found increases in fronto-temporal and parietal gamma activity were associated with increasing stress. In its current state, cognitive EEG literature is divided, in that both decreases (Koenig et al., 2005, Aurtenetxe et al., 2013, Lees et al., 2016) and increases in gamma activity (Tallon-Baudry et al., 1998, van Deursen et al., 2008, Lee et al., 2010) have been previously associated with cognitive impairment. Pachou et al. (2008) importantly reported reductions in gamma power in healthy controls, and increases patients with schizophrenia which further suggests that this increase translated to the greater cognitive effort required in the impaired state.

Stress and glucocorticoid exposure has demonstrably led to neuronal cell death and communicative alterations particularly in the frontal lobes, which can manifest as cognitive impairment (Lupien and Lepage, 2001, Cook and Wellman, 2004, Gianaros et al., 2007). Furthermore, in cognitively impaired states it is possible for neighbouring or associated neural regions to be recruited as a compensatory response to maintain performance (Dickerson et al., 2005, Clément and Belleville, 2010). As such, it could be suggested that the observed increases in both beta and gamma activity are resultant of

an adaptive response to overcome the neuronal changes that occur as a result of the experience of stress; however, confirmatory imaging analysis is required.

4.4.4 Anxiety

Research has demonstrated that anxiety can impair performance (Renden et al., 2015), and more specifically impact cognitive functions including memory and attention (Savage et al., 1999, Lautenbacher et al., 2002, Airaksinen et al., 2005), and hence may impact quality of care and nurse performance. With respect to the present analysis, the anxiety score of the two groups were found to not differ significantly. However, it was found that the score of both the non-health professional and nurse groups were greater than age relevant normative data (Lovibond and Lovibond, 1995b) suggesting that the present cohort experienced greater amounts of anxiety. Interestingly, the score of the non-health professional group resided within a normal range (0 – 7; Lovibond and Lovibond (1995b)), whilst the score of the nurse sample group was found to be in the upper end of the mild range (8 – 9; (Lovibond and Lovibond, 1995b)).

4.4.4.1 Anxiety and demographics

Demographics variables were captured in the present analysis, so as to examine their relationship with anxiety; the anxiety score of both sample groups were correlated to both of the stress measures utilised (LAQ, and the DASS), demonstrating the established interconnected nature of stress and anxiety (Lovibond and Lovibond, 1995b).

Furthermore, in non-health professional group anxiety was positively correlated with lifestyle risk factors that are associated with an increased risk of disease development (LAQ P1) and fatigue state; similarly the anxiety score of the nurse group was also

correlated with individual fatigue state. As such the present results reconcile with previous research where individuals with anxiety have been demonstrated to have an increased fatigue level (Jiang et al., 2003), BMI (Strine et al., 2008) and smoking incidence (Lawrence et al., 2009). Moreover, comorbid diseases including hypertension (Player and Peterson, 2011), diabetes (Smith et al., 2013), and depression (Fava et al., 2000) also have increased incidence in the presence of anxiety.

Additionally, previous coping research has demonstrated that negative coping strategies, e.g., avoidance, are ineffectual (Tyler and Cushway, 1992, Koeske et al., 1993, Rout and Rout, 1994, Arnetz, 2001) and may exacerbate anxiety (Tattersall et al., 1999, Arnetz, 2001), lending credence to present results where anxiety of the non-health professional group was positively associated with wishful thinking and avoidance coping. Interestingly, the anxiety score of the nurse sample group was not correlated with any of the recorded coping strategies; a result that is presumably attributable to the limited availability of coping data.

4.4.4.2 Anxiety and cognitive performance

With respect to the psychometric measures of cognitive performance, the anxiety score of the non-health professional group was negatively correlated with both measures of global cognitive performance (MMSE and Cognistat), as well as attention domain performance. These results align well with previous research, which has demonstrated that the experience of anxiety can impact performance (Renden et al., 2015, Nieuwenhuys et al., 2015) and suggested that anxiety may lead to the delivery of inferior care (Levita et al., 2016). Moreover, anxiety has also been associated with broad impairments with cognitive processes including executive function (Airaksinen et al., 2005), memory (Asmundson and Stein, 1994, Savage et al., 2000), learning

(Boldrini et al., 2005), problem solving and mathematical ability (Kellogg et al., 1999, Ashcraft, 2002), as well as decision-making capability (Cumming and Harris, 2001). Furthermore, it has been suggested that the experience of anxiety is associated with a widening of attentional resources (Derakshan and Eysenck, 2009), and the subsequent detection and processing of irrelevant information (Lautenbacher et al., 2002), thereby decreasing performance.

Within the nurse group, it was found that memory domain performance was negatively associated with experienced anxiety, but not global cognitive performance. Memory impairment is a well-documented effect of anxiety including short and delayed memory functions (Savage et al., 1999, Savage et al., 2000), as well as a number of memory subtypes including working (Darke, 1988, Ashcraft, 2002), visual (Lucas et al., 1991) verbal (Asmundson and Stein, 1994), episodic (Airaksinen et al., 2005), and spatial memory (Boldrini et al., 2005). Furthermore, as the design of the memory test of the Cognistat largely assesses short-term free-recall (Mueller et al., 2007) and relies on efficient attentional control, it could be suggested the wide allocation of attentional resources known to occur in anxiety (Derakshan and Eysenck, 2009) may be responsible for the decline in performance. As such, a more expansive attentional assessment, such as the test of everyday attention (Robertson et al., 1996), may benefit future research, as the present assessment utilises a digit span task which largely assesses selective attention.

4.4.4.3 Anxiety, cognitive performance and electroencephalography

Presently, power spectral analysis of the recorded EEG was used to provide an additional measure of cognitive performance to support the data obtained using the MMSE (Folstein et al., 1975) and the Cognistat (Mueller et al., 2007). The present

analysis found correlations between frontal and parietal EEG activity in the theta, alpha and gamma frequency bands with the anxiety score of the non-health professional group and mostly frontal, central and parietal EEG variables of all five frequency bands to anxiety in the nurse group. The ensuing regression analysis identified primarily fronto-parietal delta and gamma frequency band activity to be of chief importance. Furthermore, PCA also identified the delta and gamma frequency bands to be highly important variables in the present study.

With respect to the delta frequency band, the present results found increases in delta activity to be associated with increases in anxiety score. A result in line with the findings of the majority of previous literature which have demonstrated that delta activity increases in cognitive impaired states particularly at frontal sites (Babiloni et al., 2010, Dimpfel, 2014), and that it may be able to functionally predict and/or represent cognitive impairment (Koenig et al., 2005, Babiloni et al., 2006b). Furthermore, it has been suggested that the physiological changes related to cognitively impaired states, such as atrophy, neurofibrillary tangles, and beta amyloid accumulation, may be responsible for these witnessed changes (Liddell et al., 2007). Correlation and/or hybridisation of EEG data with traditional spatial imaging data would enable a better determination of this.

The gamma frequency band is particularly interesting as it has been previously associated with a number of cognitive functions (Mueller et al., 2007, Tallon-Baudry, 2009). However, it's correlation with cognitive impairment is less clear with both increases, particularly at parietal locations (Tallon-Baudry et al., 1998, van Deursen et al., 2008, Başar et al., 2016) and decreases having been reported (Aurtenetxe et al., 2013, Lees et al., 2016). In the present analysis gamma activity variables of both groups were positively correlated to anxiety scores. Recruitment of additional neuronal

resources has been demonstrated in cognitive impairment (Bäckman et al., 1999, Cader et al., 2006), and it has been suggested that the interaction between gamma band synchronisations and desynchronisations and subsequent activity changes may represent enhanced or impaired local neural communication (Gallinat et al., 2004, Lachaux et al., 2005, Lachaux et al., 2008). As such it could be suggested that the observed increase in gamma activity may be related to an increased neural communication and association area recruitment in an attempt to overcome the impact of anxiety on an individual; however, this requires confirmation.

Overall, fronto-parietal delta and gamma activity are associated with self-reported anxiety, and may be impacted by the neurophysiological changes associated with anxiety, or in the instance of gamma activity perhaps neurophysiological recruitment intended to overcome the impact of anxiety. However, it should be noted that the majority of EEG based cognitive research is conducted in cognitive impaired groups comprised of patients with MCI, AD, etc., either by direct observation or in some instances, comparison to healthy controls, and therefore extrapolation and interpretation to non-impaired groups requires some caution. Further research in non-impaired groups would be beneficial.

4.5 Limitations, Future Directions & Conclusions

4.5.1 Limitations

The present study effectively examined the relationship/s between stress and anxiety, and cognitive performance; this observational approach allows the determination of associations to be made, however more causative statements cannot be made. Future research utilising these experimental designs to examine the differing impacts of perceived stress, and actively induced stress of varying levels (low, medium, high)

could further illuminate the relationships identified in the present analysis, particularly in relation to the constructs of eustress and distress, and their experience. Furthermore, this analysis relied on a cross-sectional design and only provides an initial acute examination of stress and anxiety, and forgoes commenting on any longer term or chronic effects. Studies making use of a longitudinal or follow-up design across 1, 3, and 6 months to 1 year tests, would allow researchers to account for natural variance in cognitive performance, stress, and anxiety over time and examine the temporal nature of the relationships found in the present analysis.

Finally, it is important to note that in the present analysis the available Ways of Coping checklist data was limited in the nurse sample group. The limited nature of this data was a result of compiling a number of similar but non-identical databases (as described in Section 4.2.1), and should be kept in mind when interpreting the present coping results of the nurse sample group, where neither stress or anxiety were significantly correlated to coping parameters.

4.5.2 Future Directions

Firstly, it is important to mention that this type of performance based research would be beneficial for not only other health professionals e.g., physicians, but also a number of other industries that have critically important performance needs, e.g., aviation, military, etc., or those where performance impairments are a condition to mitigate, e.g., legal work, management, etc. Furthermore, future research should look to combine and/or hybridise the existing EEG montage with more traditional imaging modalities such as MRI, to not only provide a temporal image of stress and anxiety, but also a spatial image. Moreover, combining/hybridising EEG with other functional imaging modalities

such as magnetoencephalography or blood oxygen level dependent MRI may also be promising avenues for future research.

Further, the biochemical measurement of stress via cortisol or alpha amylase could provide a strong supplementary measure of stress, and should be used in conjunction with self-reported stress measures in future research. Finally, the inclusion of any workplace performance metrics would be beneficial supplements to traditional cognitive variables, as it is possible such variables could also function as assessable performance outcomes, and provide a more realistic examination. Similarly, the utilisation of workplace specific tests, such as patient simulators, in the assessment of cognitive performance may provide further insight into the relationship of stress/anxiety and cognitive performance in the health professions. That said, it is possible that there would be an observable reduction in generalisability of results as the role of a nurse and indeed other health professionals can vary significantly depending upon their location inside the industry, and their workplace.

4.5.3 Conclusions

Overall, it is apparent that both non-health professionals and nurses experience both stress and anxiety, and that these phenomena may impact lifestyle, cognitive performance and brain activity, with a unique profile developing for each sample group. Stress was associated with facilitated decision-making or judgment domain performance in non-health professionals, and impaired memory domain performance of nurses. The electroencephalographic analysis associated fronto-temporal delta activity decreases in non-health professionals, and fronto-central delta increases in nurses to stress. Further, fronto-temporal and parietal gamma activity increased in both groups with increasing stress, while fronto-central and temporal beta activity also increased in the nurse group.

Furthermore, anxiety was associated with worsened global cognitive and attention domain performance in non-health professionals, and impaired memory domain performance of nurses. Interestingly, with respect to EEG activity, the results for both groups were not unique and demonstrated increases in both fronto-parietal delta and gamma activity to be associated with anxiety.

With respect to the present hypotheses, both positive and negative associations between stress/anxiety and cognitive parameters were demonstrated, and so the first hypothesis (‘*higher levels of stress/anxiety will be associated with declines in global and/or domain specific cognitive performance’)* could be rejected or accepted depending upon the cognitive parameter. Moving forward it is important for stress/anxiety research to consider each cognitive parameters independently. Further, the second present hypothesis (*‘higher levels of stress/anxiety will be associated alterations in brain activity as measured by EEG; in particular the theta, beta and gamma frequency bands.’*) can be partially accepted when considering the present results, which demonstrated alterations in beta and gamma activity to be associated with stress/anxiety, however theta activity was not implicated.

Overall, these findings may enable the development and implementation of strategies that could be utilised to preserve nurse performance, and reduce the incidence of adverse medical events.

Chapter 5 – Electroencephalography and its prediction of cognitive performance

5.1 Introduction

As a concept, cognition is a broad descriptor of the intellectual processes that an individual utilises in their perception and comprehension of the world. Typically, these processes are characterised by the use functions including reasoning and memory (Müller and Mayes, 2001, Harris et al., 2009). It follows that a reduction in performance of these functions and hence an individual's cognitive performance has been loosely termed cognitive impairment. It is important to note that a natural decline in cognitive function is associated with age, where these functions deteriorate and subsequently an individual's faculties diminish. However, more severe cognitive deficits beyond this normal decline are defined to be pathological (DeCarli, 2003), although, there is no readily accepted definition of cognitive impairment (Scazufca et al., 2009).

Neuroscientists and psychologists alike have utilised a variety of techniques in their investigations of cognitive performance, most common of which are psychometric questionnaires like the Mini-mental State Exam (Folstein et al., 1975), the Montreal Cognitive Assessment (Nasreddine et al., 2005), and Cognistat (Mueller et al., 2007). These quantitatively assess cognitive performance using question and answer or command based tasks. However, this design limits the function of such tasks mostly to retrospective examinations, which in turn limits the applicability of these tasks in examining the decline into mild cognitive impairment (MCI), and other transitory cognitive impairments.

More recently, physiological measurements like electroencephalography (EEG) (Klimesch et al., 1998, Lees et al., 2016) have been increasingly utilised to measure cognitive performance and detect cognitive decline due to their inherently quantitative and dynamic nature. Indeed, research has suggested that increases in delta and theta frequency activity may represent cognitive impairment (Rossini et al., 2006, Babiloni et al., 2010). However, contradictory findings have suggested that delta (Jackson and Snyder, 2008) and theta (Cummins et al., 2008) activities will decrease in cognitive impairment. Additionally, changes in higher frequency EEG activity including decreased upper alpha band activity (Pijnenburg et al., 2004) and alpha reactivity (van der Hiele et al., 2007) have also been correlated to changes in cognitive performance. Furthermore, beta (Jackson and Snyder, 2008, Lees et al., 2016) and gamma (Moretti et al., 2009, Tallon-Baudry, 2009) activity changes have also been implicated. While in contrast, others suggest that alpha and gamma activity will increase (Tallon-Baudry, 2009). As such, the current state of literature regarding EEG and cognitive performance is ambiguous and requires further research.

As it stands, most research examining cognitive performance and EEG activity is confined to groups with MCI, dementia, or some other diagnosed cognitive impairment, while research utilising non-impaired groups is limited. Because of this, the examination of the cognitive performance of non-impaired individuals should be conducted prior to the development of predictive tools that intend to identify transitory and early cognitive impairment. Therefore, the aims of this study were to further investigate the relationship between EEG activity and the global and domain specific cognitive performance of healthy non-impaired individuals and a similar group of nurses, and to determine the significance and predictive capabilities of these relationships. With respect to hypotheses; it was hypothesised that:

1. Cognitive performance will be associated with decreases in slow wave (delta, and theta) activity, and increases in fast wave (alpha, beta and gamma) activity.
2. EEG variables will be able to predict both global and domain specific cognitive performance.

Finally, it is important to note this experiment was conducted as a follow-up to research previously published by the author of this thesis (Lees et al., 2016).

5.2 Materials and Methods

The data analysed in this Chapter was collected for the experiment previously reported in Chapter 4; the relevant experimental protocol is described in Section 4.2. The following section describes the analysis specific to this Chapter.

5.2.1 Analysis

STATISTICA (Version 10, 1999, StatSoft, USA) was used to conduct the statistical analysis. Partial Pearson's correlation analysis was used to examine the associations between the collected electroencephalography data and global cognitive performance, as well as performance in the Attention, Memory, and Judgement domains. These domains were selected as they were previously significantly correlated to cognitive function in Chapter 4.

In this chapter, the Least absolute shrinkage and selection operator (LASSO) (Tibshirani, 1996) analysis provided a further linear examination of the relationship between EEG and cognitive performance, supplementing the correlation analysis. LASSO was computed using Matlab (Version 2016a, Mathworks, USA), as per the following equation:

Equation 5.1 – Least absolute shrinkage and selection operator (LASSO) equation

$$\min_{\beta_0 \beta} \left(\frac{1}{2N} \sum_{i=1}^{N} \left(y_i - \beta_0 - x_i^T \beta \right)^2 + \lambda \sum_{j=1}^{p} |\beta_j| \right)$$

Where:

- N is the number of observations.
- y_i is the response at observation i.
- x_i is data, a vector of p values at observation i.
- λ is a nonnegative regularization parameter corresponding to one value of Lambda.
- The parameters β_0 and β are scalar and p-vector respectively.
- As λ increases, the number of nonzero components of β decreases.
- The lasso problem involves the L^1 norm of β, as contrasted with the elastic net algorithm.

The LASSO is a method for shrinkage and selection for regression and generalised regression problems (Tibshirani, 1996). It defines a continuous shrinking operation that provides coefficients or weights (that can equal exactly zero) that evaluate the importance of inputted variables for subsequent analysis (Tibshirani, 1996). In the present analysis, a cut-off normalised weight of an absolute value of 0.75 was utilised to identify only the most important EEG variables.

Additionally, it must be noted that prior to LASSO analysis, an imputation to replace all previously removed EEG values was performed as is common (Keil et al., 2014). The imputation in the present analysis was performed by calculating the average of the previous and next non-missing value for the variable containing the missing value.

Following the correlation and LASSO analysis, if significant relationships to three or more dependent variables were identified, general linear multiple regression analysis was utilised to determine the predictive capability of EEG, and the strongest individual predictor of a single independent variable.

5.3 Results

With respect to global cognitive performance, both the nurse and non-health professional groups scored above the borderline threshold of 23 of the MMSE, scoring 27.39 ± 1.91 and 27.86 ± 1.91, respectively. Similar results were found for the Cognistat total score, with both the nurses and non-health professionals scoring above the threshold score of 65, returning scores of 71.00 ± 6.09 and 74.18 ± 4.61, respectively.

Further information regarding domain specific performance in the Attention, Memory and Judgement domains, and intergroup comparisons, can be found in Chapter 4, Section 4.3.2.

5.3.1. Global Cognitive Performance

5.3.1.1 Non-health professionals

In the non-health professional group, global cognitive performance (as assessed by the MMSE) was significantly correlated to 42 EEG variables (Table 5.1). This analysis revealed negative correlations between MMSE score and: baseline CP_3 theta activity ($r = -0.38$; $p = 0.020$), baseline C_3 alpha activity ($r = -0.34$; $p = 0.036$), baseline T_8 alpha activity ($r = -0.32$; $p = 0.049$), active phase CP_3 alpha activity ($r = -0.42$; $p = 0.009$), baseline F_7 beta activity ($r = -0.36$; $p = 0.029$); baseline F_z beta activity ($r = -0.35$; $p = 0.039$); baseline C_3 beta activity ($r = -0.43$; $p = 0.007$), baseline CP_3 beta activity ($r = -0.49$; $p = 0.002$), baseline P_z beta activity ($r = -0.36$; $p = 0.027$), baseline O_z beta activity ($r = -0.49$; $p = 0.004$), baseline O_2 beta activity ($r = -0.36$; $p = 0.043$), active phase F_7 beta activity ($r = -0.32$; $p = 0.048$); active phase F_z beta activity ($r = -0.36$; $p = 0.027$), active phase CP_3 beta activity ($r = -0.44$; $p = 0.007$), active phase CP_4 beta activity ($r = -0.52$; $p = 0.001$), active phase P_7 beta activity ($r = -0.33$; $p = 0.040$), baseline C_3 gamma activity ($r = -0.33$; $p = 0.045$), baseline CP_3 gamma

activity ($r = -0.44$; $p = 0.008$), baseline TP_8 gamma activity ($r = -0.37$; $p = 0.028$), baseline P_7 gamma activity ($r = -0.38$; $p = 0.020$), baseline P_3 gamma activity ($r = 0.40$; $p = 0.012$), baseline P_8 gamma activity ($r = -0.44$; $p = 0.004$), active phase Fp_1 gamma activity ($r = -0.35$; $p = 0.037$), active phase F_z gamma activity ($r = -0.43$; $p = 0.010$); active phase CP_3 gamma activity ($r = -0.54$; $p = 0.001$), active phase P_z gamma activity ($r = 0.38$; $p = 0.025$), active phase P_4 gamma activity ($r = -0.46$; $p = 0.004$), F_3 gamma reactivity ($r = -0.38$; $p = 0.021$), and P_4 gamma reactivity ($r = -0.38$, $p = 0.014$).

Additionally, positive correlations between MMSE score were found for: F_3 theta reactivity ($r = 0.41$; $p = 0.012$), O_2 theta reactivity ($r = 0.34$; $p = 0.041$); F_7 alpha reactivity ($r = 0.40$; $p = 0.010$), T_7 alpha reactivity ($r = 0.32$; $p = 0.045$), C_3 alpha reactivity ($r = 0.33$; $p = 0.042$), T_8 alpha reactivity ($r = 0.33$; $p = 0.042$), FT_8 beta reactivity ($r = 0.32$; $p = 0.046$), C_3 beta reactivity ($r = 0.49$; $p = 0.002$), P_8 beta reactivity ($r = 0.35$; $p = 0.032$), O_2 beta reactivity ($r = 0.43$; $p = 0.014$), T_8 gamma reactivity ($r = 0.31$; $p = 0.045$); TP_8 gamma reactivity ($r = 0.41$; $p = 0.012$), and P_8 gamma reactivity ($r = 0.52$; $p < 0.001$). Lastly, all other EEG variables were not significantly correlated to MMSE score.

Table 5.1 – The associations between global cognitive performance (as measured by MMSE) and electroencephalography variables of the non-health professional group

DV	IV	n	r	p	IV	n	r	p
MMSE	Bθ-CP_3	41	-0.38	0.020*	Aβ-P_7	42	-0.33	0.040*
	Rθ-F_3	42	0.41	0.012*	Rβ-FT_8	43	0.32	0.046*
	Rθ-O_2	40	0.34	0.041*	Rβ-C_3	42	0.49	0.002*
	Bα-C_3	42	-0.34	0.036*	Rβ-P_8	42	0.35	0.032*
	Bα-T_8	42	-0.32	0.049*	Rβ-O_2	36	0.43	0.014*
	Aα-CP_3	41	-0.42	0.009*	Bγ-C_3	41	-0.33	0.045*
	Rα-F_7	45	0.40	0.010*	Bγ-CP_3	40	-0.44	0.008*
	Rα-T_7	44	0.32	0.045*	Bγ-TP_8	39	-0.37	0.028*
	Rα-C_3	43	0.33	0.042*	Bγ-P_7	42	-0.38	0.020*
	Rα-T_8	43	0.33	0.042*	Bγ-P_3	43	-0.40	0.012*
	Bβ-F_7	41	-0.36	0.029*	Bγ-P_8	44	-0.44	0.004*
	Bβ-F_z	40	-0.35	0.039*	Aγ-Fp_1	40	-0.35	0.037*
	Bβ-C_3	41	-0.43	0.007*	Aγ-F_z	39	-0.43	0.010*
	Bβ-CP_3	40	-0.49	0.002*	Aγ-CP_3	40	-0.54	0.001*
	Bβ-P_z	41	-0.36	0.027*	Aγ-P_z	39	-0.38	0.025*
	Bβ-O_z	37	-0.49	0.004*	Aγ-P_4	43	-0.46	0.004*
	Bβ-O_2	36	-0.36	0.043*	Rγ-F_3	40	-0.38	0.021*
	Aβ-F_7	42	-0.32	0.048*	Rγ-T_8	47	0.31	0.045*
	Aβ-F_z	42	-0.36	0.027*	Rγ-TP_8	41	0.41	0.012*
	Aβ-CP_3	40	-0.44	0.007*	Rγ-P_4	45	-0.38	0.014*
	Aβ-CP_4	41	-0.52	0.001*	Rγ-P_8	45	0.52	< 0.001*

Table 5.1 displays the significant partial correlations (controlling for age, BMI and years of education) between global cognitive performance score (as assessed by the MMSE) of the non-health professional group and electroencephalography variables. All other EEG variables were not significantly correlated to MMSE score and are not presented in this table.

Key: A = Active phase; B = Baseline; C = Central; DV = Dependent variable; F = Frontal; IV = Independent variable; n = Sample size; O = Occipital; P = Parietal; R = Reactivity; T = Temporal; z = Midline; α = Alpha; β = Beta; γ = Gamma; θ = Theta; * = Statistical significance; < = Less than

Furthermore, the LASSO analysis for the MMSE in the non-health professionals reduced the weights of all investigated EEG variables to zero.

As multiple EEG variables were significantly correlated to the MMSE score of the non-health professional group, a forward stepwise general linear regression was performed. The regression analysis retained 3 of the 42 originally entered variables (F_3 delta reactivity, active phase CP_3 alpha activity, and FT_8 beta reactivity), and had an overall significance of p = 0.013 (Table 5.2). Together these 3 variables explained 44% of the variance in MMSE score (F = 4.72 DF = 3; p = 0.013; R = 0.664, R^2 = 0.440; AR^2 = 0.347). Furthermore, active phase CP_3 alpha activity also presented as an independently significant predictor of the non-health professional MMSE score (p = 0.011).

Table 5.2 – Regression analysis for global cognitive performance (as measured by the MMSE) and significantly correlated electroencephalography variables in the non-health professional group

			R = 0.664; R^2 = 0.440; AR^2 = 0.347; SSM = 26.40; dfM= 3; MSM = 8.80; SSR = 33.60; dfR = 18; MSR = 1.87 F = 4.715; p = 0.013*			
Variable	**β**	**SE of β**	**B**	**SE of B**	**t**	**p**
Intercept			29.32	0.56	52.28	< 0.001*
Rθ-F_3	0.24	0.20	0.08	0.07	1.22	0.239
Aα-CP_3	-0.55	0.20	-0.78	0.28	-2.82	0.011*
Rβ-FT_8	0.32	0.20	0.02	0.01	1.61	0.125

Table 5.2 displays a stepwise forward general linear regression between global cognitive performance of the non-health professional group (as measured by the MMSE) and the significantly correlated physiological EEG variables. Of the 42 EEG variables originally entered into the model, the analysis retained 3: Rθ-F_3, Aα-CP_3, and Rβ-FT_8.

Key: A = Active phase; C = Central; dfM = Degrees of Freedom Model; dfR = Degrees of Freedom Residual; F = Frontal; MSM = Mean square Model; MSR = Mean square Residual; P = Parietal; R = Reactivity; SE = Standard error; SSM = Sum of squares Model; SSR= Sum of squares Residual; T = Temporal; α = Alpha; β = Beta; θ = Theta; * = Statistical significance; < = Less than

Similarly, the total Cognistat score of the non-health professional group was also significantly correlated to a number of EEG variables (Table 5.3). Significant and positive correlations were found for: baseline FC_3 theta activity ($r = 0.36$; $p = 0.035$), Fp_2 alpha reactivity ($r = 0.31$; $p = 0.040$), O_2 alpha reactivity ($r = 0.35$; $p = 0.038$), C_3 beta reactivity ($r = 0.41$; $p = 0.011$), P_z beta reactivity ($r = 0.38$; $p = 0.016$), O_2 beta reactivity ($r = 0.36$; $p = 0.044$), FC_z gamma reactivity ($r = 0.37$; $p = 0.020$), and C_3 gamma reactivity ($r = 0.35$; $p = 0.029$).

Additionally, significant negative correlations were found for: baseline C_3 beta activity ($r = -0.44$; $p = 0.006$), baseline CP_3 beta activity ($r = -0.44$; $p = 0.007$), baseline P_z beta activity ($r = -0.45$; $p = 0.005$), active phase F_3 beta activity ($r = -0.37$; $p = 0.035$), active phase CP_3 beta activity ($r = -0.37$; $p = 0.029$), baseline FC_z gamma activity ($r = -0.33$; $p = 0.047$), baseline C_3 gamma activity ($r = -0.39$; $p = 0.017$), baseline CP_3 gamma activity ($r = -0.34$; $p = 0.044$), baseline CP_z gamma activity ($r = -0.37$; $p = 0.031$), baseline P_7 gamma activity ($r = -0.35$; $p = 0.031$), baseline P_3 gamma activity ($r = -0.33$; $p = 0.041$), active phase Fp_1 gamma activity ($r = -0.34$; $p = 0.041$), active phase CP_3 gamma activity ($r = -0.53$; $p = 0.001$), active phase P_z gamma activity ($r = -0.45$; $p = 0.007$), active phase P_4 gamma activity ($r = -0.42$; $p = 0.007$), and P_4 gamma reactivity ($r = -0.40$; $p = 0.009$).

In addition, utilising a cut-off normalised weight of an absolute value of 0.75, the LASSO analysis demonstrated the importance of baseline P_z beta activity (Figure 5.1) for predicting the total Cognistat score of the non-health professional group.

Table 5.3 – The associations between global cognitive performance (as measured by the Cognistat) and electroencephalography variables of the non-health professional group

Dependent Variable	Independent Variable	n	r	p
Cognistat (Total)	Bθ-FC_3	39	0.36	0.035*
	Rα-Fp_2	47	0.31	0.040*
	Rα-O_2	40	0.35	0.038*
	Bβ-C_3	41	-0.44	0.006*
	Bβ-CP_3	40	-0.44	0.007*
	Bβ-P_z	41	-0.45	0.005*
	Aβ-F_3	37	-0.37	0.035*
	Aβ-CP_3	40	-0.37	0.029*
	Rβ-C_3	42	0.41	0.011*
	Rβ-P_z	43	0.38	0.016*
	Rβ-O_2	36	0.36	0.044*
	Bγ-FC_z	41	-0.33	0.047*
	Bγ-C_3	41	-0.39	0.017*
	Bγ-CP_3	40	-0.34	0.044*
	Bγ-CP_z	39	-0.37	0.031*
	Bγ-P_7	42	-0.35	0.031*
	Bγ-P_3	43	-0.33	0.041*
	Aγ-Fp_1	40	-0.34	0.041*
	Aγ-CP_3	40	-0.53	0.001*
	Aγ-P_z	39	-0.45	0.007*
	Aγ-P_4	43	-0.42	0.007*
	Rγ-FC_z	44	0.37	0.020*
	Rγ-C_3	43	0.35	0.029*
	Rγ-P_4	45	-0.40	0.009*

Table 5.3 displays the significant partial correlations (controlling for age, BMI and years of education) between global cognitive performance score (as assessed by the Cognistat) of non-health professional group and the electroencephalography variables. All other EEG variables were not significantly correlated to Cognistat Total score and are not presented in this table.

Key: A = Active phase; B = Baseline; BMI = Body Mass Index; C = Central; F = Frontal; Fp = Frontal pole; n = Sample size; O = Occipital; P = Parietal; R = Reactivity; T = Temporal; z = Midline; α = Alpha; β = Beta; γ = Gamma; θ = Theta; * = Statistical significance

Figure 5.1 - Normalised LASSO weights for the global cognitive performance (as measured by the Cognistat) of the non-health professional group

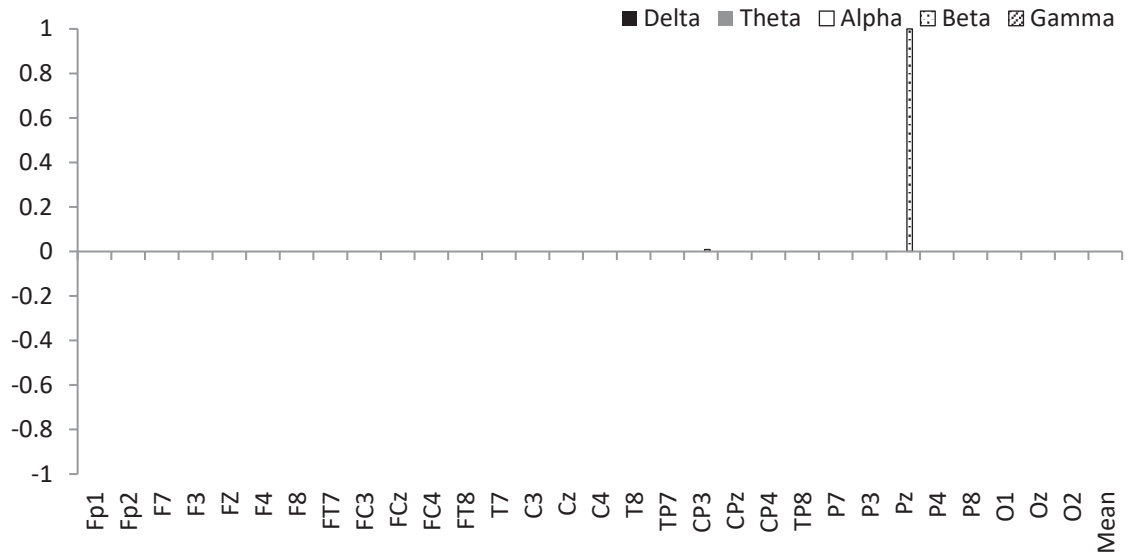

Figure 5.1 presents the normalised LASSO weights for baseline EEG variables and global cognitive performance (as measured by the Cognistat) of the non-health professional group. Additionally, the weights for all active phase and reactivity variables were zeroed and hence are not presented. Lastly, some frequency bands (delta, theta, alpha and gamma) are not visible as all variable weights were reduced to zero.

Key: C = Central; F = Frontal; Fp = Frontal pole; O = Occipital; P = Parietal; T = Temporal; z = Midline

Since the total Cognistat score of the non-health professional group was significantly correlated with multiple EEG variables a forward stepwise general linear regression was performed to determine predictive capability; the LASSO analysis results also informed this regression.

The regression analysis retained 7 of the 24 originally input variables (baseline FC_3 theta activity, baseline P_z beta activity, active phase F_3 and CP_3 beta activity, as well as active phase Fp_1 and CP_3 gamma activity, and C_3 gamma reactivity), and had an overall significance of $p < 0.001$ (Table 5.4). Together these 7 variables explained a substantial 77.8% of the variance in total Cognistat score ($F = 8.02$, $df = 7$; $p < 0.001$; $R = 0.882$, $R^2 = 0.778$; $AR^2 = 0.681$).

Furthermore, baseline P_z beta activity, active phase F_3 and CP_3 beta activity, as well as active phase Fp_1 and CP_3 gamma activity all presented as independently significant predictors of total Cognistat score in the non-health professional group ($p \leq 0.043$).

Table 5.4 – Regression analysis for global cognitive performance (as measured by the Cognistat) and significantly correlated electroencephalography variables in the non-health professional group

			$R = 0.882$; $R^2 = 0.778$; $AR^2 = 0.681$ SSM = 393.76; dfM= 7; MSM = 56.25; SSR = 112.20; dfR = 16; MSR = 7.01 F = 8.02; $p < 0.001$*			
Variable	**β**	**SE of β**	**B**	**SE of B**	**t**	**P**
Intercept			75.38	1.6	55.63	< 0.001*
Bθ-FC_3	0.25	0.13	0.12	0.06	2.00	0.063
Bβ-P_z	0.85	0.39	0.83	0.38	2.20	0.043*
Aβ-F_3	0.62	0.25	0.38	0.16	2.51	0.023*
Aβ-CP_3	-1.50	0.49	-2.52	0.83	-3.04	0.008*
Aγ-Fp_1	0.33	0.15	4.33	1.96	2.21	0.042*
Aγ-CP_3	-0.53	0.22	-17.89	7.48	-2.39	0.029*
Rγ-C_3	0.24	0.18	3.88	3.01	1.29	0.216

Table 5.4 displays a stepwise forward general linear regression analysis between global cognitive performance of the non-health professional group (as measured by the Cognistat) and the significantly correlated EEG variables. Of the 24 EEG variables originally entered into the model, the analysis retained 7: Bθ-FC_3, Bβ-P_z, Aβ-F_3, Aβ-CP_3, Aγ-Fp_1, Aγ-CP_3, and Rγ-C_3.

Key: A = Active phase; B = Baseline; C = Central; dfM = Degrees of Freedom Model; dfR = Degrees of Freedom Residual; F = Frontal; Fp = Frontal pole; MSM = Mean square Model; MSR = Mean square Residual; P = Parietal; R = Reactivity; SE = Standard error; SSM = Sum of squares Model; SSR= Sum of squares Residual; z = Midline; β = Beta; γ = Gamma; θ = Theta; * = Statistical significance; < = Less than

5.3.1.2 Nurses

The global cognitive performance (as measured by the MMSE) for the nurse group was found to be significantly correlated to 22 EEG variables in the present analysis (Table 5.5). A number of these correlations were positive relationships between MMSE score and: FC_3 theta reactivity ($r = 0.40$, $p = 0.006$), P_z theta reactivity ($r = 0.34$, $p = 0.021$), baseline Fp_1 alpha activity ($r = 0.34$; $p = 0.025$), baseline F_z alpha activity ($r = 0.37$; $p = 0.016$), baseline F_4 alpha activity ($r = 0.31$ $p = 0.046$), baseline FT_8 alpha activity ($r = 0.34$; $p = 0.021$), and FC_3 alpha reactivity ($r = 0.33$; $p = 0.036$).

Furthermore, the present analysis also identified a number of negative correlations for: C_4 delta reactivity ($r = -0.32$; $p = 0.037$), TP_7 delta reactivity ($r = -0.34$; $p = 0.024$), baseline FC_3 theta reactivity ($r = -0.49$; $p = 0.001$), baseline C_3 theta activity ($r = -0.33$; $p = 0.031$), TP_8 theta reactivity ($r = -0.34$; $p = 0.016$), active phase TP_8 alpha activity ($r = -0.42$; $p =0.004$), F_z alpha reactivity ($r = -0.36$; $p = 0.015$), F_8 alpha reactivity ($r = -0.44$; $p = 0.003$, TP_8 alpha reactivity ($r = -0.43$; $p = 0.004$), O_1 alpha reactivity ($r = -0.33$; $p = 0.029$), baseline FC_3 beta activity ($r = -0.37$; $p = 0.029$), baseline C_4 beta activity ($r = -0.31$; $p = 0.049$), active phase Fp_2 beta activity ($r = -0.33$; $p = 0.046$), active phase FC_3 beta activity ($r = -0.45$; $p = 0.004$), and active phase CP_3 beta activity ($r = -0.36$; $p = 0.028$).

Moreover, when utilising a cut-off normalised weight of an absolute value of 0.75 the LASSO regression (Figure 5.2) indicated the importance of baseline FC_3 and TP_8 theta activity as well as baseline F_3 alpha activity for the prediction of MMSE score of the nurse group.

Table 5.5 – The associations between global cognitive performance (as measured by MMSE) and electroencephalography variables of the nurse group

Dependent Variable	Independent Variable	n	r	p
MMSE	Rδ-C_4	47	-0.32	0.037*
	Rδ-TP_7	50	-0.34	0.024*
	Bθ-FC_3	49	-0.49	0.001*
	Bθ-C_3	47	-0.33	0.031*
	Rθ-FC_3	50	0.40	0.006*
	Rθ-TP_8	53	-0.34	0.016*
	Rθ-P_z	50	0.34	0.021*
	Bα-Fp_1	48	0.34	0.025*
	Bα-F_z	48	0.37	0.016*
	Bα-F_4	47	0.31	0.046*
	Bα-FT_8	49	0.34	0.021*
	Aα-TP_8	48	-0.42	0.004*
	Rα-F_z	49	-0.36	0.015*
	Rα-F_8	47	-0.44	0.003*
	Rα-FC_3	46	0.33	0.036*
	Rα-TP_8	49	-0.43	0.004*
	Rα-O_1	47	-0.33	0.029*
	Bβ-FC_3	45	-0.37	0.018*
	Bβ-C_4	47	-0.31	0.049*
	Aβ-Fp_2	43	-0.33	0.046*
	Aβ-FC_3	44	-0.45	0.004*
	Aβ-CP_3	41	-0.36	0.028*

Table 5.5 displays the significant partial correlations (controlling for age, BMI and years of education) between global cognitive performance score (as assessed by the MMSE) of the nurse group and electroencephalography variables. All other EEG variables were not significantly correlated to MMSE score and are not presented in this table.

Key: A = Active phase; B = Baseline; BMI = Body Mass Index; C = Central; F = Frontal; Fp = Frontal pole; n = Sample size; O = Occipital; P = Parietal; R = Reactivity; T = Temporal; z = Midline; α = Alpha; β = Beta; δ = Delta; θ = Theta; * = Statistical significance

Figure 5.2 – Normalised LASSO weights for the global cognitive performance (as measured by the MMSE) of the nurse group

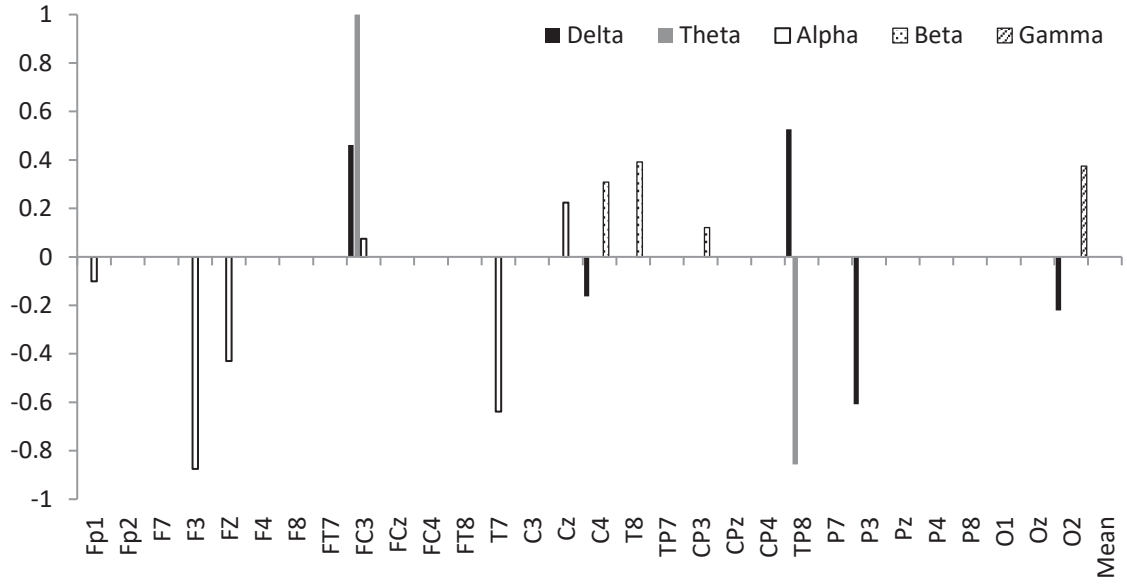

Figure 5.3 presents the normalised LASSO weights for baseline EEG variables and global cognitive performance (as measured by the MMSE) of the nurse group. Additionally, the weights for all active phase and reactivity variables were zeroed and hence are not presented.

Key: C = Central; F = Frontal; Fp = Frontal pole; O = Occipital; P = Parietal; T = Temporal; z = Midline

Subsequently, a forward stepwise general linear regression analysis was performed as the MMSE score for the nurse group was significantly correlated with multiple EEG variables. Additionally, the LASSO results also informed this regression.

The regression analysis retained 7 of the 24 originally entered variables (C_4 delta reactivity, P_z theta reactivity, baseline F_3 alpha activity, TP_8 and O_1 alpha reactivity, as well as active phase CP_3 beta activity), and had an overall significance of $p < 0.001$ (Table 5.6). Together these 7 variables explained a considerable 85.6% of the variance in MMSE score ($F = 12.75$, $DF = 7$; $p < 0.001$; $R = 0.925$, $R^2 = 0.856$; $AR^2 = 0.789$).

P_z theta reactivity, baseline F_3 alpha activity, TP_8 and O_1 alpha reactivity, as well as active phase CP_3 beta activity all presented as independently significant predictors of MMSE score in the nurse group ($p \leq 0.049$).

Table 5.6 – Regression analysis for global cognitive performance (as measured by the MMSE) and significantly correlated electroencephalography variables in the nurse group

			$R = 0.925$; $R^2 = 0.856$; $AR^2 = 0.789$; SSM = 46.61; dfM = 7; MSM = 6.66; SSR = 7.83; dfR = 15; MSR = 0.52; F = 12.75; $p < 0.001$*			
Variable	**β**	**SE of β**	**B**	**SE of B**	**t**	**P**
Intercept			26.24	0.28	94.60	< 0.001*
Rδ-C_4	-0.40	0.10	-0.01	< 0.01	-4.06	0.001*
Rθ-P_z	0.23	0.17	0.27	0.20	1.35	0.196
Bα-F_3	1.53	0.22	0.34	0.05	7.00	< 0.001*
Rα-FC_3	0.55	0.12	0.12	0.03	4.60	< 0.001*
Rα-TP_8	0.76	0.23	0.14	0.04	3.38	0.004*
Rα-O_1	-0.28	0.11	-0.03	0.01	-2.55	0.022*
Aβ-CP_3	-0.35	0.1	-0.15	0.07	-2.14	0.049*

Table 5.6 displays a stepwise forward general linear regression analysis between global cognitive performance of the nurse group (as measured by the MMSE) and the significantly correlated EEG variables. Of the 24 EEG variables originally entered into the model, the analysis retained 7: Rδ-C_4, Rθ-P_z, Bα-F_3, Rα-FC_3, Rα-TP_8, Rα-O_1, and Aβ-CP_3.

Key: A = Active phase; B = Baseline; C = Central; dfM = Degrees of Freedom Model; dfR = Degrees of Freedom Residual; F = Frontal; MSM = Mean square Model; MSR = Mean square Residual; O = Occipital; P = Parietal; R = Reactivity; SE = Standard error; SSM = Sum of squares Model; SSR= Sum of squares Residual; T = Temporal; z = Midline; α = Alpha; β = Beta; δ = Delta; θ = Theta; * = Statistical significance; < = Less than

Similarly, the Cognistat measure of global cognitive performance was also significantly correlated to a number of EEG variables in the nurse sample group (Table 5.7). A number of positive correlations were found for: baseline Fp_2 alpha activity ($r = 0.34$; $p = 0.025$), baseline F_z alpha activity ($r = 0.31$; $p = 0.046$), baseline C_4 alpha activity ($r = 0.32$; $p = 0.035$), and baseline CP_4 alpha activity ($r = 0.43$; $p = 0.046$.

In addition, the following were significantly negatively correlated to total Cognistat score: baseline FT_7 delta activity ($r = -0.34$; $p = 0.029$), baseline P_3 delta activity ($r = -0.37$; $p = 0.015$), active phase FT_7 delta activity ($r = -0.33$; $p = 0.033$), baseline F_8 theta activity ($r = -0.36$; $p = 0.015$), active phase F_8 theta activity ($r = -0.30$; $p = 0.045$), active phase T_8 theta activity ($r = -0.37$; $p = 0.014$), active phase O_1 theta activity ($r = -0.36$; $p = 0.016$), FT_8 theta reactivity ($r = -0.34$; $p = 0.017$), O_z theta reactivity, active phase F_8 alpha activity ($r = -0.43$; $p = 0.005$), Fp_2 alpha reactivity ($r = -0.30$; $p = 0.042$), F_8 alpha reactivity ($r = -0.32$; $p = 0.036$), CP_4 alpha reactivity ($r = -0.36$; $p = 0.019$), baseline F_7 gamma activity ($r = -0.36$; $p = 0.022$), baseline FT_8 gamma activity ($r = -0.42$; $p = 0.005$), and active phase F_4 gamma activity ($r = -0.39$; $p = 0.013$).

Table 5.7 – The associations between global cognitive performance (as measured by the Cognistat) and electroencephalography variables of the nurse sample group

Dependent Variable	Independent Variable	n	r	p
Cognistat (Total)	Bδ-FT_7	46	-0.34	0.029*
	Bδ-P_3	48	-0.37	0.015*
	Aδ-FT_7	48	-0.33	0.033*
	Bθ-F_8	49	-0.36	0.015*
	Aθ-F_8	50	-0.30	0.045*
	Aθ-T_8	47	-0.37	0.014*
	Aθ-O_1	47	-0.36	0.016*
	Rθ-FT_8	52	-0.34	0.017*
	Rθ-O_z	49	-0.36	0.015*
	Bα-Fp_2	49	0.34	0.025*
	Bα-F_z	48	0.31	0.046*
	Bα-C_4	48	0.32	0.035*
	Bα-CP_4	45	0.43	0.006*
	Aα-F_8	45	-0.43	0.005*
	Rα-Fp_2	50	-0.30	0.042*
	Rα-F_8	47	-0.32	0.036*
	Rα-CP_4	46	-0.36	0.019*
	Bγ-F_7	46	-0.35	0.022*
	Bγ-FT_8	49	-0.42	0.005*
	Aγ-F_4	45	-0.39	0.013*

Table 5.7 displays the significant partial correlations (controlling for age, BMI and years of education) between global cognitive performance score (as assessed by the Cognistat) of the nurse group and electroencephalography variables. All other EEG variables were not significantly correlated to Cognistat Total score and are not presented in this table.

Key: A = Active phase; B = Baseline; BMI = Body Mass Index; C = Central; F = Frontal; Fp = Frontal pole; n = Sample size; O = Occipital; P = Parietal; R = Reactivity; T = Temporal; z = Midline; α = Alpha; δ = Delta; γ = Gamma; θ = Theta; * = Statistical significance

Further, the relationship between global cognitive performance (as measured by the Cognistat) and EEG variables in the nurse group was further explored using LASSO regression (Figure 5.4) which indicated the importance of baseline FT_8 gamma activity, as well as FC_z and O_z theta reactivity when utilising a cut-off normalised weight of an absolute value of 0.75.

As the Cognistat total score of the nurse group was significantly correlated with multiple EEG variables, a forward stepwise general linear regression analysis was performed. Additionally, it should be noted that the results of the LASSO also informed this regression analysis.

The regression analysis retained 4 of the 20 originally entered variables (FC_z and O_z theta reactivity, as well as baseline C_4 alpha and F_7 gamma activity), and had an overall significance of $p < 0.001$ (Table 5.8). Together these 4 variables a generous explained 81.0% of the variance in total Cognistat score ($F = 21.31$, $df = 4$; $p < 0.001$; $R = 0.900$, $R^2 = 0.810$; $AR^2 = 0.772$). Furthermore, O_z theta reactivity, as well as baseline C_4 alpha and F_7 gamma activity also presented as independently significant predictors of total Cognistat score in the nurse group ($p \leq 0.012$).

Figure 5.3 – Normalised LASSO weights for the global cognitive performance (as measured by the Cognistat) of the nurse group

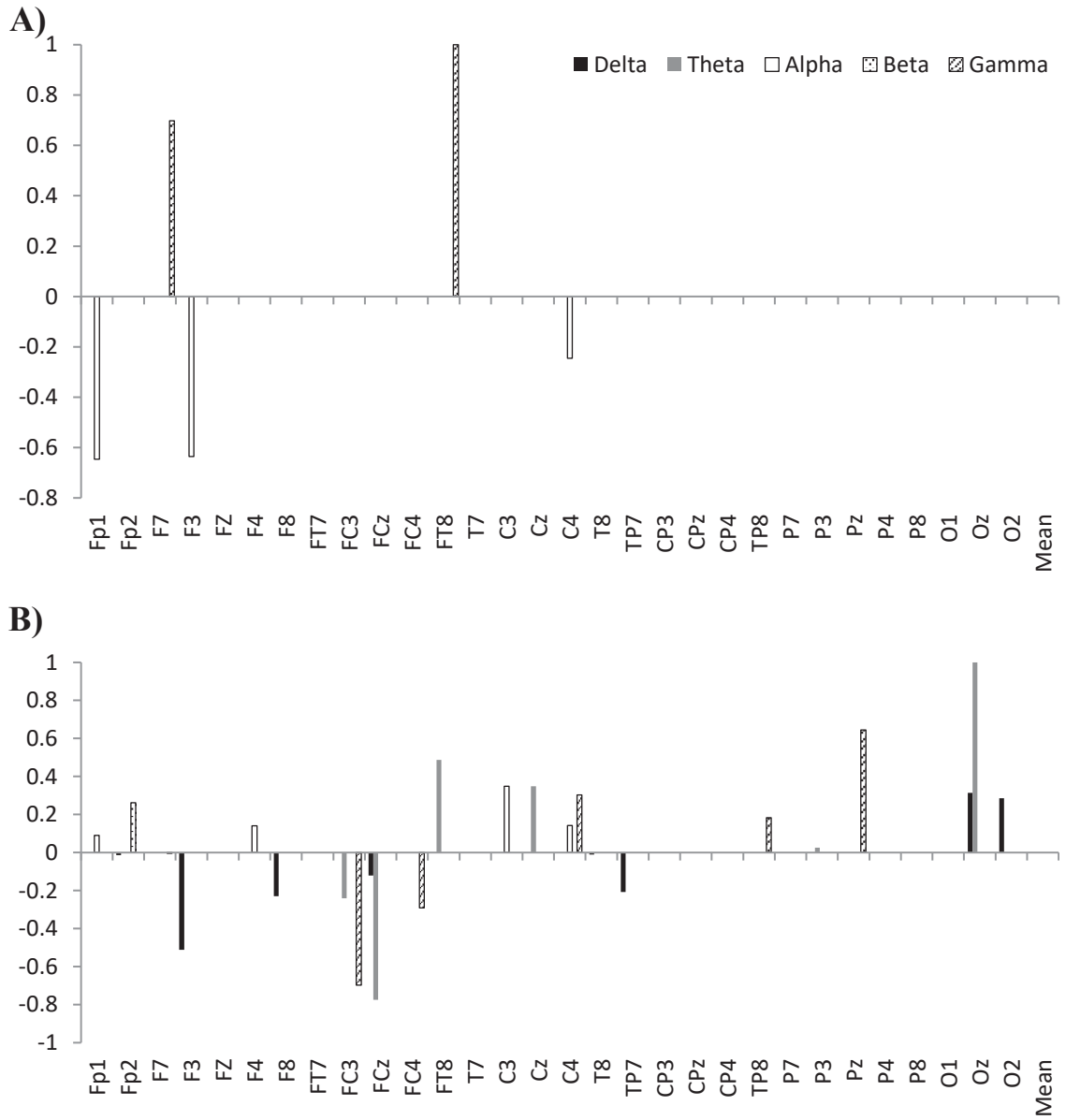

Figure 5.3 presents the normalised LASSO weights for electroencephalography variables for global cognitive performance (as measured by the Cognistat) of the nurse group. Plot A displays the baseline variables; Plot B displays the reactivity variables. Additionally, the weights for all active phase variables were zeroed and hence are not presented. Lastly, the beta frequency band is not visible as all variable weights were reduced to zero.

Key: C = Central; F = Frontal; Fp = Frontal pole; O = Occipital; P = Parietal; T = Temporal; z = Midline

Table 5.8 – Regression analysis for global cognitive performance (as measured by the Cognistat) and significantly correlated electroencephalography variables in the nurse group

			$R = 0.900$; $R^2 = 0.810$; $AR^2 = 0.772$; SSM = 949.12; dfM = 4; MSM =237.28; SSR = 222.72; dfR = 20; MSR = 11.14 F = 21.31; $p < 0.001$*			
Variable	**β**	**SE of β**	**B**	**SE of B**	**t**	**p**
Intercept			73.00	1.04	70.18	< 0.001*
Rθ-FC_z	0.15	0.10	0.57	0.38	1.48	0.154
Rθ-O_z	-0.45	0.10	-0.23	0.05	-4.47	< 0.001*
Bα-C_4	0.28	0.10	0.32	0.12	2.78	0.012*
Bγ-F_7	-0.57	0.10	-3.29	0.58	-5.63	< 0.001*

Table 5.8 displays a stepwise forward general linear regression analysis between global cognitive performance of the nurse group (as measured by the Cognistat) and the significantly correlated EEG variables. Of the 20 EEG variables originally entered into the model, the analysis retained 4: Rθ-FC_z, Rθ-O_z, Bα-C_4, and Bγ-F_7.

Key: B = Baseline; C = Central; dfM = Degrees of Freedom Model; dfR = Degrees of Freedom Residual; F = Frontal; Fp = Frontal pole; MSM = Mean square Model; MSR = Mean square Residual; O = Occipital; R = Reactivity; SSM = Sum of squares Model; SSR= Sum of squares Residual; α = Alpha; γ = Gamma; θ = Theta; * = Statistical significance; < = Less than

5.3.2 Attention

5.3.2.1 Non-health professionals

Performance in the Attention domain of the non-health professional group was significantly correlated to a number of EEG variables (Table 5.9); three of these correlations were positive relationships with: FC_z delta reactivity ($r = 0.34$; $p = 0.027$), O_2 beta reactivity ($r = 0.36$; $p = 0.046$), and T_7 gamma reactivity ($r = 0.36$; $p = 0.020$). While negative relationships were found with: baseline P_7 delta activity ($r = -0.32$; $p = 0.036$), active phase Fp_1 delta activity ($r = -0.43$; $p = 0.005$), CP_3 delta reactivity ($r = -0.32$; $p = 0.031$), active phase P_7 theta activity ($r = -0.32$; $p = 0.043$), active phase P_7 beta activity ($r = -0.35$; $p = 0.029$), baseline CP_3 gamma activity ($r = -0.48$; $p = 0.003$), baseline P_7 gamma activity ($r = -0.39$; $p = 0.016$), and active phase CP_3 gamma activity ($r = -0.60$; $p < 0.001$).

Table 5.9 – The associations between Attention domain performance and electroencephalography variables of the non-health professional group

Dependent Variable	Independent Variable	n	r	p
Attention (Cognistat)	Bδ-P_7	46	-0.32	0.036*
	Aδ-Fp_1	45	-0.43	0.005*
	Rδ-FC_z	45	0.34	0.027*
	Rδ-CP_3	50	-0.32	0.031*
	Aθ-P_7	44	-0.32	0.043*
	Aβ-P_7	42	-0.35	0.029*
	Rβ-O_2	36	0.36	0.046*
	Bγ-CP_3	40	-0.48	0.003*
	Bγ-P_7	42	-0.39	0.016*
	Aγ-CP_3	40	-0.60	< 0.001*
	Rγ-T_7	46	0.36	0.020*

Table 5.9 displays the significant partial correlations (controlling for age, BMI and years of education) between Attention domain performance score of the non-health professional group and electroencephalography variables. All other EEG variables were not significantly correlated to Attention domain performance score and are not presented in this table.

Key: A = Active phase; B = Baseline; BMI = Body Mass Index; C = Central; F = Frontal; Fp = Frontal pole; n = Sample size; O = Occipital; P = Parietal; R = Reactivity; T = Temporal; z = Midline; β = Beta; δ = Delta; γ = Gamma; θ = Theta; * = Statistical significance

The relationship between EEG variables and Attention domain performance of the non-health professional group was further examined using LASSO analysis. Interestingly, this analysis reduced the weights of all investigated EEG variables to zero.

Moreover, a forward stepwise general linear regression analysis was performed as multiple EEG variables were significantly correlated to the Attention score of the non-health professional group.

The regression analysis retained 1 of the 11 originally entered variables (baseline CP_3 gamma activity), and had an overall significance of $p < 0.03$ (Table 5.10). This single variable explained 20.4% of the variance in Attention domain score ($F = 6.93$; $df = 1$; $p <= 0.014$; $R = 0.452$, $R^2 = 0.204$; $AR^2 = 0.175$).

Table 5.10 – Regression analysis for Attention domain performance and significantly correlated electroencephalography variables in the non-health professional group

			R = 0.452; R^2 = 0.204; AR^2 = 0.175; SSM = 4.33; dfM = 1; MSM = 4.33; SSR = 16.85; dfR = 27; MSR = 0.62 F = 6.93; p = 0.014*			
Variable	**β**	**SE of β**	**B**	**SE of B**	**t**	**p**
Intercept			7.67	0.17	45.20	< 0.001*
Bγ-CP_3	-0.45	0.17	-1.25	0.47	-2.63	0.014*

Table 5.10 displays a stepwise forward general linear regression analysis between Attention domain performance score of the non-health professional group and the significantly correlated EEG variables. Of the 11 EEG variables originally entered into the model, the analysis retained 1: Bγ-CP_3.

Key: B = Baseline; C = Central; dfM = Degrees of Freedom Model; dfR = Degrees of Freedom Residual; MSM = Mean square Model; MSR = Mean square Residual; P = Parietal; SSM = Sum of squares Model; SSR= Sum of squares Residual; γ = Gamma; * = Statistical significance; < = Less than

5.3.2.2 Nurses

In the nurse group (Table 5.11) C_3 delta reactivity ($r = 0.44$; $p = 0.005$), active phase Fp_2 theta activity ($r = 0.29$; $p < 0.050$), F_8 theta reactivity ($r = 0.42$; $p = 0.004$), FC_z theta reactivity ($r = 0.29$; $p = 0.047$), baseline FC_4 alpha activity ($r = 0.29$; $p < 0$ .050), baseline CP_4 alpha activity ($r = 0.42$; $p = 0.007$), baseline CP_z gamma activity ($r = 0.34$; $p = 0.042$), and CP_4 gamma reactivity ($r = 0.34$; $p = 0.032$) were significantly and positively correlated with Attention domain score. Furthermore, negative correlations were found for baseline FT_7 delta activity ($r = -0.31$; $p = 0.048$), baseline P_3 delta activity ($r = -0.32$; $p = 0.035$), active phase FT_7 delta activity ($r = -0.30$; $p = 0.048$), O_1theta reactivity ($r = -0.31$; $p = 0.037$), CP_4 alpha reactivity ($r = -0.33$; $p = 0.037$), active phase O_2 beta activity ($r = -0.36$; $p = 0.033$), average active phase beta activity ($r = -0.34$; $p = 0.014$), baseline FT_7 gamma activity ($r = -0.31$; $p = 0.040$), baseline O_2 gamma activity ($r = -0.37$; $p = 0.029$), active phase Fp_1 gamma activity ($r = -0.44$; $p = 0.003$), active phase FT_8 gamma activity ($r = -0.36$; $p = 0.019$), active phase T_7 gamma activity ($r = -0.34$; $p = 0.028$), and active phase TP_8 gamma activity ($r = -0.38$; $p = 0.019$) were also found.

A further examination of the relationship between attention domain performance for the nurse group and electroencephalography was conducted using LASSO (Figure 5.4), and a cut-off normalised weight of an absolute value of 0.75. This analysis indicated the importance of baseline CP_4 alpha activity and baseline F_8 gamma activity.

Table 5.11 – The associations between Attention domain performance and electroencephalography variables of the nurse group

Dependent Variable	Independent Variable	n	r	p
Attention (Cognistat)	Bδ-FT_7	46	-0.31	0.048*
	Bδ-P_3	48	-0.32	0.035*
	Aδ-FT_7	48	-0.30	0.048*
	Rδ-C_3	45	0.44	0.005*
	Aθ-Fp_2	50	0.29	< 0.050*
	Rθ-F_8	51	0.42	0.004*
	Rθ-FC_z	52	0.29	0.047*
	Rθ-O_1	50	-0.31	0.037*
	Bα-FC_4	50	0.29	< 0.050*
	Bα-CP_4	45	0.42	0.007*
	Rα-CP_4	46	-0.33	0.037*
	Aβ-O_2	40	-0.36	0.033*
	Aβ-Mean	56	-0.34	0.014*
	Bγ-FT_7	50	-0.31	0.040*
	Bγ-CP_z	42	0.34	0.042*
	Bγ-O_2	38	-0.37	0.029*
	Aγ-Fp_1	48	-0.44	0.003*
	Aγ-FT_8	47	-0.36	0.019*
	Aγ-T_7	46	-0.34	0.028*
	Aγ-TP_8	41	-0.38	0.019*
	Rγ-CP_4	46	0.34	0.032*

Table 5.11 displays the significant partial correlations (controlling for age, BMI and years of education) between Attention domain performance score of the nurse group and electroencephalography variables. All other EEG variables were not significantly correlated to Attention domain performance score and are not presented in this table.

Key: A = Active phase; B = Baseline; BMI = Body Mass Index; C = Central; F = Frontal; Fp = Frontal pole; n = Sample size; O = Occipital; P = Parietal; R = Reactivity; T = Temporal; z = Midline; α = Alpha; β = Beta; δ = Delta; γ = Gamma; θ = Theta; * = Statistical significance; < = Less than

Figure 5.4 – Normalised LASSO weights for the Attention domain performance of nurse group

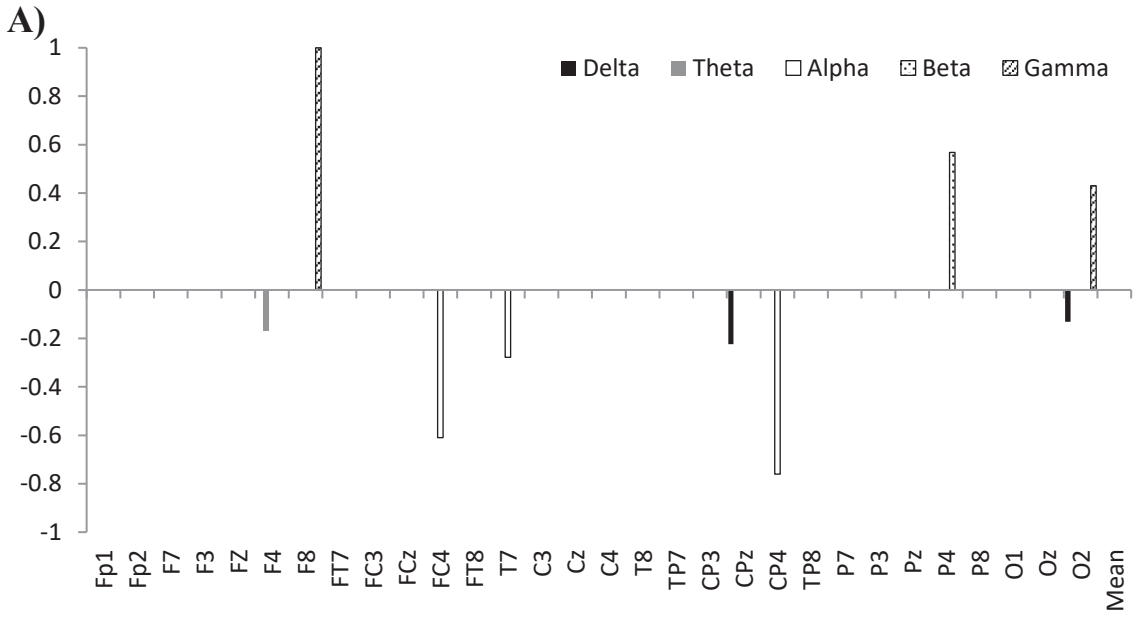

Figure 5.6 presents the normalised LASSO weights for baseline electroencephalography variables and attention domain performance of the nurse group. Additionally, the weights for all active phase and reactivity variables were zeroed and hence are not presented.

Key: C = Central; F = Frontal; Fp = Frontal pole; O = Occipital; P = Parietal; T = Temporal; z = Midline

Further, a forward stepwise general linear regression analysis was performed as Attention domain performance was significantly correlated with multiple EEG variables. Additionally, the EEG variables indicated to be important using the LASSO analysis were also entered into the regression analysis.

The regression analysis retained 6 of the 22 originally entered variables (baseline FT_7 and P_3 delta activity, active phase FT_7 delta activity, active phase Fp_2 theta activity, baseline FT_7 gamma activity, and active phase T_7 gamma activity), and had an overall significance of $p < 0.001$ (Table 5.12). Together these 7 variables explained 76.7% of the variance in Attention domain score ($F = 7.68$, $DF = 6$; $p < 0.001$; $R = 0.876$, $R^2 = 0.767$; $AR^2 = 0.667$).

Furthermore, baseline FT_7 and P_3 delta activity, active phase FT_7 delta activity, baseline FT_7 gamma activity, and active phase T_7 gamma activity, all presented as independently significant predictors of the attention score of the nurse group ($p \leq 0.022$).

Table 5.12 – Regression analysis for Attention domain performance and significantly correlated electroencephalography variables in the nurse group

			$R = 0.876$; $R^2 = 0.767$; $AR^2 = 0.667$; SSM = 14.22; dfM= 6 ; MSM = 2.34; SSR = 4.33; dfR = 14 ; MSR = 0.31 F = 7.68; $p < 0.001$*			
Variable	**β**	**SE of β**	**B**	**SE of B**	**t**	**p**
Intercept			7.34	0.40	18.49	< 0.001*
Bδ-FT_7	-0.92	0.36	-0.02	< 0.01	-2.58	0.022*
Bδ-P_3	-0.72	0.15	< -0.01	< 0.01	-4.91	< 0.001*
Aδ-FT_7	1.16	0.36	0.019	< 0.01	3.24	0.006*
Aθ-Fp_2	-0.27	0.19	-0.04	0.03	-1.42	0.176
Bγ-FT_7	-0.53	0.16	-0.74	0.22	-3.29	0.005*
Aγ-T_7	0.66	0.18	0.63	0.17	3.64	0.003*

Table 5.12 displays a stepwise forward general linear regression analysis between Attention domain performance score of the nurse group and the significantly correlated EEG variables. Of the 22 EEG variables originally entered into the model, the analysis retained 6: Bδ-FT_7, Bδ-P_3, Aδ-FT_7, Aθ-Fp_2, Bγ-FT_7, and Aγ-T_7.

Key: A = Active phase; B = Baseline; dfM = Degrees of Freedom Model; dfR = Degrees of Freedom Residual; F = Frontal; Fp = Frontal pole; MSM = Mean square Model; MSR = Mean square Residual; P = Parietal; SSM = Sum of squares Model; SSR= Sum of squares Residual; T = Temporal; δ = Delta; γ = Gamma; θ = Theta; * = Statistical significance; < = Less than

5.3.3 Memory

5.3.3.1 Non-health professionals

In the non-health professional group (Table 5.13), a number of EEG reactivity variables including: FC_z delta reactivity ($r = 0.33$; $p = 0.036$), P_7 delta reactivity ($r = 0.34$; $p = 0.021$), O_z delta reactivity ($r = 0.36$; $p = 0.026$), P_7 alpha reactivity ($r = 0.38$; $p = 0.015$), O_z beta reactivity ($r = 0.46$; $p = 0.008$), Fp_2 gamma reactivity ($r = 0.35$; $p = 0.027$), and FT_7 gamma reactivity ($r = 0.36$; $p = 0.024$) were positively correlated to Memory domain score. Additionally, negative correlations were found for baseline FC_z delta activity, ($r = -0.34$; $p = 0.034$), baseline F_3 theta activity ($r = -0.33$; $p = 0.044$), active phase Fp_2 theta activity ($r = -0.31$; $p = 0.045$), active phase F_8 gamma activity ($r = -0.33$; $p = 0.045$), and Fp_1gamma reactivity ($r = -0.34$; $p = 0.033$).

Moreover, the relationship between EEG and Memory domain performance of the non-health professional group was further examined using LASSO analysis. Utilising a cut-off normalised weight of an absolute value of 0.75, the LASSO regression (Figure 5.5) indicated that P_7 and O_z delta reactivity were of particular importance.

Table 5.13 – The associations between Memory domain performance and electroencephalography variables of the non-health professional group

Dependent Variable	Independent Variable	n	r	p
Memory (Cognistat)	Bδ-FC_z	43	-0.34	0.034*
	Rδ-FC_z	45	0.33	0.036*
	Rδ-P_7	49	0.34	0.021*
	Rδ-O_z	43	0.36	0.026*
	Bθ-F_3	42	-0.33	0.044*
	Aθ-Fp_2	46	-0.31	0.045*
	Rα-P_7	45	0.38	0.015*
	Rβ-O_z	37	0.46	0.008*
	Aγ-F_8	42	-0.33	0.045*
	Rγ-Fp_1	43	-0.34	0.033*
	Rγ-Fp_2	45	0.35	0.027*
	Rγ-FT_7	44	0.36	0.024*

Table 5.13 displays the significant partial correlations (controlling for age, BMI and years of education) between Memory domain performance of the non-health professional group and electroencephalography variables. All other EEG variables were not significantly correlated to Memory domain performance score and are not presented in this table.

Key: A = Active phase; B = Baseline; BMI = Body Mass Index; C = Central; F = Frontal; Fp = Frontal pole; n = Sample size; O = Occipital; P = Parietal; R = Reactivity; T = Temporal; z = Midline; α = Alpha; β = Beta; δ = Delta; γ = Gamma; θ = Theta; * = Statistical significance

Figure 5.5 – Normalised LASSO weights for the Memory domain performance of the non-health professional group

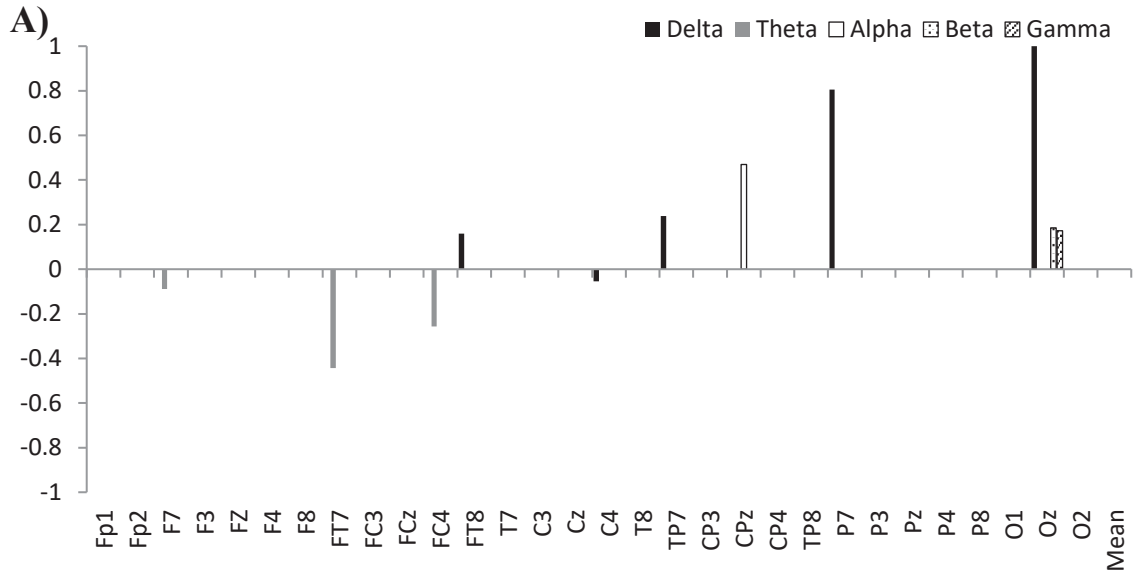

Figure 5.5 presents the normalised LASSO weights for electroencephalography reactivity variables and Memory domain performance of the non-health professional group. Additionally, the weights for all baseline and active phase variables were zeroed and hence are not presented

Key: C = Central; F = Frontal; Fp = Frontal pole; O = Occipital; P = Parietal; T = Temporal; z = Midline

A forward stepwise general linear regression analysis informed by significant correlations to non-health professional Memory score, and the LASSO analysis was subsequently performed.

The regression analysis retained 3 of the 14 originally entered variables (P_7 delta reactivity, active phase Fp_2 theta activity, and Fp_2 gamma reactivity), and had an overall significance of $p < 0.010$ (Table 5.14). Together these 3 variables explained 44.3% of the variance in Memory domain score ($F = 5.04$; $df = 3$; $p < 0.010$; $R = 0.666$, $R^2 = 0.443$; $AR^2 = 0.355$).

Furthermore, P_7 delta reactivity also presented as an independently significant predictor of Memory domain performance in the non-health professional group ($p = 0.013$).

Table 5.14 – Regression analysis for Memory domain performance and significantly correlated EEG variables in the non-health professional group

			$R = 0.666$; $R^2 = 0.443$; $AR^2 = 0.355$; SSM = 22.59; dfM = 3; MSM = 7.53; SSR = 28.37; dfR = 19; MSR = 1.49 $F = 5.04$; $p < 0.010$*			
Variable	**β**	**SE of β**	**B**	**SE of B**	**t**	**p**
Intercept			11.97	0.56	21.57	< 0.0018
Rδ-P_7	0.49	0.18	< 0.01	< 0.01	2.75	0.013*
Aθ-Fp_2	-0.35	0.17	-0.06	0.03	-2.03	0.057
Rγ-Fp_2	0.19	0.18	1.04	0.98	1.06	0.304

Table 5.14 displays a stepwise forward general linear regression analysis between Memory domain performance of the non-health professional group and the significantly correlated EEG variables. Of the 14 EEG variables originally entered into the model, the analysis retained 3: Rδ-P_7, Aθ-Fp_2, and Rγ-Fp_2.

Key: A = Active phase; dfM = Degrees of Freedom Model; dfR = Degrees of Freedom Residual; Fp = Frontal pole; MSM = Mean square Model; MSR = Mean square Residual; P = Parietal; R = Reactivity; SSM = Sum of squares Model; SSR= Sum of squares Residual; z = Midline; δ = Delta; γ = Gamma; θ = Theta; * = Statistical significance

5.3.3.2 Nurses

Interestingly, the Memory domain score of the nurse group was positively correlated with only three EEG variables (Table 5.15); FT_8 delta reactivity (r = 0.30; p = 0.039), T_8 delta reactivity (r = 0.34; p = 0.026), CP_4 delta reactivity (r = -0.33; p = 0.030).

Significant negative correlations were found with baseline F_z delta activity (r = -0.39; p = 0.007), baseline F_4 delta activity (r = -0.36; p = 0.016), baseline FT_7 delta activity (r = -0.47; p = 0.002), baseline FC_z delta activity (r = -0.38; p = 0.013), baseline FT_8 delta activity (r = -0.30; p = 0.038), baseline T_7 delta activity (r = -0.38; p = 0.010), baseline C_z delta activity (r = -0.32; p = 0.034), baseline C_4 delta activity (r = -0.38; p = 0.013), baseline T_8 delta activity (r = -0.34; p = 0.024), baseline TP_7 delta activity (r = -0.32; p = 0.028), baseline CP_z delta activity (r = -0.33; p = 0.019), baseline P_7 delta activity (r = -0.30; p =0.038), baseline P_3 delta activity (r = -0.44; p = 0.003), baseline P_z delta activity (r = -0.29; p = 0.045), baseline O_z delta activity (r = -0.43; p = 0.003), active phase F_7 delta activity (r = -0.32; p = 0.032), active phase F_z delta activity (r = -0.31; p = 0.042), active phase F_4 delta activity (r = -0.40; p = 0.010), active phase FT_7 delta activity (r = -0.50; p = 0.001), active phase FC_z delta activity (r = -0.33; p = 0.029), active phase T_7 delta activity (r = -0.41; p = 0.005), active phase C_z delta activity (r = -0.44; p = 0.003), active phase C_4 delta activity (r = -0.41; p = 0.007), active phase P_3 delta activity (r = -0.44; p = 0.004), C_z delta reactivity (r = -0.35; p = 0.020), baseline F_7 theta activity (r = -0.31; p = 0.036), baseline F_8 theta activity (r = -0.38; p = 0.009), baseline T_8 theta activity (r = -0.34; p = 0.022), baseline CP_z theta activity (r = -0.31; p = 0.034), baseline O_1 theta activity (r = -0.32; p = 0.032), average baseline theta activity (r = -0.31; p = 0.025), active phase F_7 theta activity (r = -0.35; p = 0.015), active phase F_8 theta activity (r = -0.36; p = 0.014), active phase T_8 theta activity (r = -0.45; p = 0.003), active phase CP_z theta activity (r = 0.30; p = 0.047),

average active phase theta activity (r = -0.32; p = 0.023), active phase F_8 alpha activity (r = -0.45; p = 0.003), Fp_1 alpha reactivity (r = 0.32; p = 0.036), F_z alpha reactivity (r = -0.32; p = 0.037), F_8 alpha reactivity (r = -0.31; p = 0.044), active phase Fp_2 beta activity (r = -0.34; p = 0.037), active phase F_7 beta activity (r = -0.39; p = 0.013), Fp_2 beta reactivity (r = -0.31; p = 0.046), baseline F_7 gamma activity (r = -0.38; p = 0.014), baseline F_4 gamma activity (r = -0.34; p = 0.025), baseline FT_8 gamma activity (r = -0.47; p = 0.001), active phase F_7 gamma activity (r = -0.37; p = 0.018), and active phase F_4 gamma activity (r = -0.49; p = 0.001).

In addition, LASSO analysis was used to further explore the relationship between EEG variables and Memory domain performance of the nurse group (Figure 5.6). A cut-off normalised weight of an absolute value of 0.75 indicated the importance of baseline F_z delta activity, baseline FT_8 gamma activity, active phase F_z delta activity, active phase F_4 gamma activity, and C_z delta reactivity.

Table 5.15 – The associations between Memory domain performance and electroencephalography variables of the nurse group

DV	IV	n	r	p	IV	n	r	p	IV	n	r	p
Memory	Bδ-F_z	51	-0.39	0.007*	Aδ-FT_7	48	-0.50	0.001*	Aθ-T_8	47	-0.45	0.003*
	Bδ-F_4	48	-0.36	0.016*	Aδ-FC_z	50	-0.33	0.029*	Aθ-CP_z	51	-0.30	0.047*
	Bδ-FT_7	46	-0.47	0.002*	Aδ-T_7	50	-0.41	0.005*	Aθ-Mean	56	-0.32	0.023*
	Bδ-FC_z	47	-0.38	0.013*	Aδ-C_z	49	-0.44	0.003*	Aα-F_8	45	-0.45	0.003*
	Bδ-FT_8	52	-0.30	0.038*	Aδ-C_4	47	-0.41	0.007*	Rα-Fp_1	48	-0.32	0.036*
	Bδ-T_7	49	-0.38	0.010*	Aδ-P_3	45	-0.44	0.004*	Rα-F_z	49	-0.32	0.037*
	Bδ-C_z	49	-0.32	0.034*	Rδ-FT_8	52	0.30	0.039*	Rα-F_8	47	-0.31	0.044*
	Bδ-C_4	48	-0.38	0.013*	Rδ-C_z	49	-0.35	0.020*	Aβ-Fp_2	43	-0.34	0.037*
	Bδ-T_8	50	-0.34	0.024*	Rδ-T_8	48	0.34	0.026*	Aβ-F_7	44	-0.39	0.013*
	Bδ-TP_7	51	-0.32	0.028*	Rδ-CP_4	48	0.33	0.030*	Rβ-Fp_2	47	-0.31	0.046*
	Bδ-CP_z	54	-0.33	0.019*	Bθ-F_7	50	-0.31	0.036*	Bγ-F_7	46	-0.38	0.014*
	Bδ-P_7	52	-0.30	0.038*	Bθ-F_8	49	-0.38	0.009*	Bγ-F_4	47	-0.34	0.025*
	Bδ-P_3	48	-0.44	0.003*	Bθ-T_8	49	-0.34	0.022*	Bγ-FT_8	49	-0.47	0.001*
	Bδ-P_z	53	-0.29	0.045*	Bθ-CP_z	51	-0.31	0.034*	Aγ-F_7	46	-0.37	0.018*
	Bδ-O_z	50	-0.43	0.003*	Bθ-O_1	48	-0.32	0.032*	Aγ-F_4	45	-0.49	0.001*
	Aδ-F_7	50	-0.32	0.032*	Bθ-Mean	57	-0.31	0.025*				
	Aδ-F_z	50	-0.31	0.042*	Aθ-F_7	51	-0.35	0.015*				
	Aδ-F_4	46	-0.40	0.010*	Aθ-F_8	50	-0.36	0.014*				

Table 5.15 displays the significant partial correlations (controlling for age, BMI and years of education) between memory domain performance of the nurse group and electroencephalography variables. All other EEG variables were not significantly correlated to Memory domain performance score and are not presented in this table.

Key: A = Active phase; B = Baseline; BMI = Body Mass Index; C = Central; DV = Dependent Variable; F = Frontal; Fp = Frontal pole; IV = Independent Variable; n = Sample size; O = Occipital; P = Parietal; R = Reactivity; T = Temporal; z = Midline; α = Alpha; β = Beta; δ = Delta; γ = Gamma; θ = Theta; * = Statistical significance; < = Less than

Figure 5.6 – Normalised LASSO weights for the Memory domain performance of the nurse group

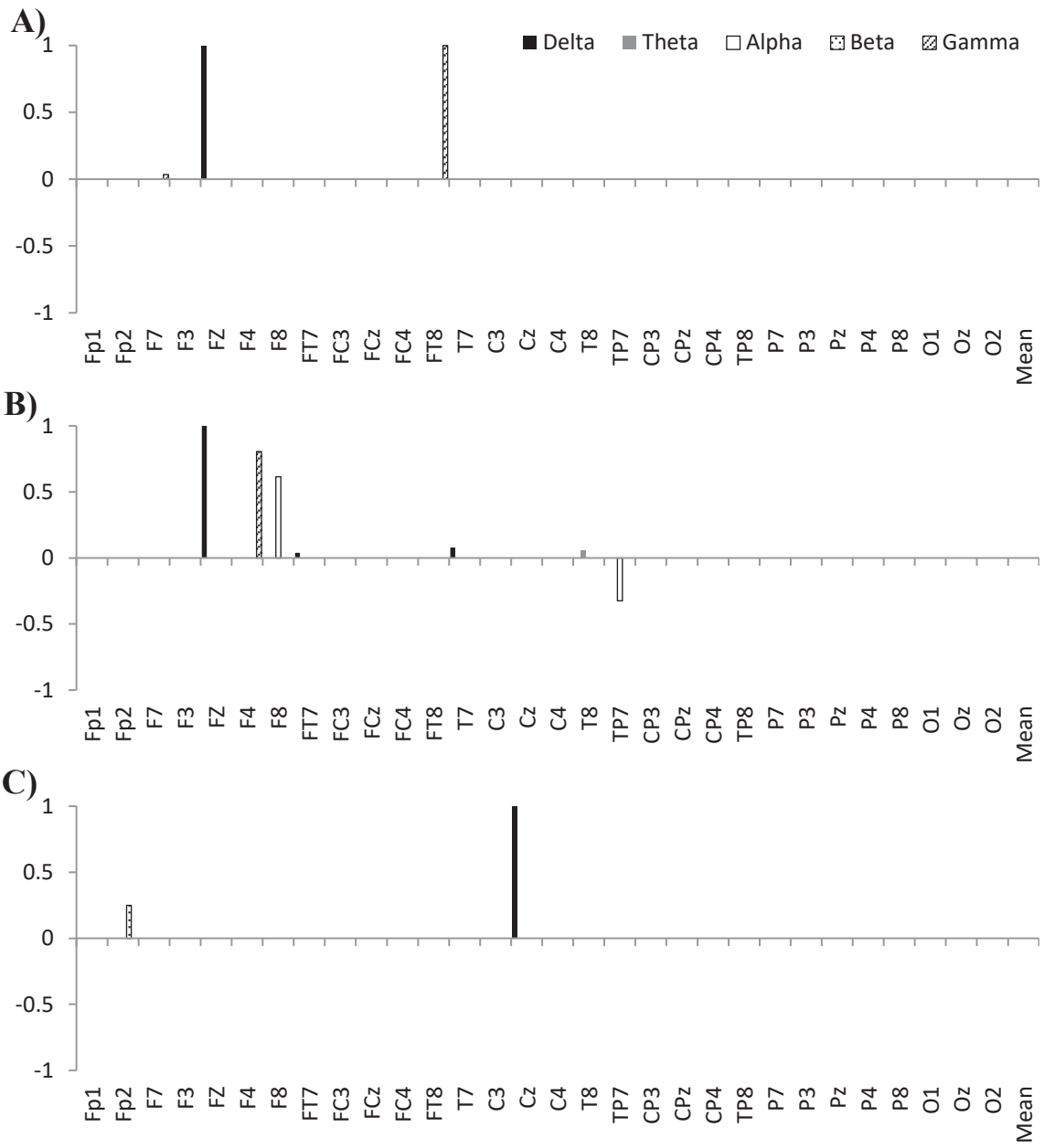

Figure 5.6 presents the normalised LASSO weights for electroencephalography variables and Memory domain performance of the nurse group. Plot A displays baseline variables; Plot B displays active phase variables; and Plot C displays reactivity variables.

Key: C = Central; F = Frontal; Fp = Frontal pole; O = Occipital; P = Parietal; T = Temporal; z = Midline

Following this analysis, a forward stepwise general linear regression was performed informed by the significant correlations to Memory score of the nurse group, as well as the LASSO analysis.

The regression analysis retained 13 of the 51 originally entered variables (baseline F_z, T_7, TP_z, CP_z and P_7 delta activity, as well as active phase T_7 delta activity, FT_8 and T_8 delta reactivity, active phase CP_z and F_8 theta activity, Fp_2 beta reactivity, and baseline and active phase F_7 gamma activity), and had an overall significance of $p < 0.001$ (Table 5.16). Together these 13 variables explained a large 99.8% of the variance in Memory score ($F = 229.87$, $df = 13$; $p < 0.001$; $R = 0.999$, $R^2 = 0.998$; $AR^2 = 0.994$).

All retained variables, except for active phase FT_8 and T_8 delta reactivity, presented as independently significant predictors of Memory domain performance in the nurse sample group ($p \leq 0.047$).

Table 5.16 – Regression analysis for Memory domain and significantly correlated electroencephalography variables in the nurse group

			$R = 0.999; R^2 = 0.998; AR^2 = 0.994;$ $SSM = 63.68; dfM = 13; MSM = 4.90;$ $SSR = 0.11; dfR = 5; MSR = 0.02$ $F = 229.87$ $p < 0.001$*			
Variable	**β**	**SE of β**	**B**	**SE of B**	**t**	**p**
Intercept			12.14	0.09	129.34	< 0.001*
Bδ-F_z	-0.34	0.06	< -0.01	< 0.01	-5.70	0.002*
Bδ-T_7	1.23	0.30	0.01	< 0.01	4.07	0.010*
Bδ-TP_z	-0.13	0.05	< -0.01	< 0.01	-2.62	0.047*
Bδ-CP_z	0.21	0.05	< 0.01	< 0.01	4.56	0.006*
Bδ-P_7	-0.21	0.03	< -0.01	< 0.001	-7.08	0.001*
Aδ-T_7	-0.98	0.35	-0.01	< 0.01	-2.77	0.039*
Rδ-FT_8	0.12	0.09	< 0.01	< 0.01	1.44	0.210
Rδ-T_8	0.27	0.11	0.03	0.01	2.51	0.054
Aθ-CP_z	0.18	0.04	0.09	0.02	4.97	0.004*
Aθ-F_8	-0.32	0.03	-0.12	0.01	-9.68	< 0.001*
Rβ-Fp_2	-0.12	0.03	-0.05	0.01	-3.76	0.013*
Bγ-F_7	-1.86	0.12	-2.57	0.16	-15.99	< 0.001*
Aγ-F_7	1.07	0.09	2.22	0.18	12.27	< 0.001*

Table 5.16 displays a stepwise forward general linear regression analysis between Memory domain performance of the nurse group and the significantly correlated physiological EEG variables. Of the 51 EEG variables originally entered into the model, the analysis retained 13: Bδ-F_z, Bδ-T_7, Bδ-TP_z, Bδ-CP_z, Bδ-P_7, Aδ-T_7, Rδ-FT_8, Rδ-T_8, Aθ-CP_z, Aθ-F_8, Rβ-Fp_2, Bγ-F_7, and Aγ-F_7.

Key: A = Active phase; B = Baseline; C = Central; dfM = Degrees of Freedom Model; dfR = Degrees of Freedom Residual; F = Frontal; Fp = Frontal pole; MSM = Mean square Model; MSR = Mean square Residual; P = Parietal; R = Reactivity; SSM = Sum of squares Model; SSR= Sum of squares Residual; T = Temporal; z = Midline; β = Beta; δ = Delta; γ = Gamma; θ = Theta; * = Statistical significance; < = Less than

5.3.4 Judgement

5.3.4.1 Non-health professionals

Positive correlations were found between the Judgement domain score of the non-health professional group (Table 5.17) and baseline C_z delta activity ($r = 0.38$; $p = 0.018$), and baseline T_8 delta activity ($r = 0.31$; $p = 0.044$). Furthermore, it was found that Judgement domain score was negatively and significantly correlated with active phase FC_4 delta activity ($r = -0.41$; $p = 0.021$), F_8 delta reactivity ($r = -0.34$; $p = 0.025$), FC_3 delta reactivity ($r = -0.35$; $p = 0.019$), T_8 delta reactivity ($r = -0.32$; $p = 0.033$), C_4 theta reactivity ($r = -0.44$; $p = 0.005$), baseline FT_7 beta activity ($r = -0.38$; $p = 0.028$), active phase CP_3 beta activity ($r = -0.33$; $p = 0.046$), active phase CP_z beta activity ($r = -0.35$; $p = 0.040$), active phase CP_z gamma activity ($r = 0.42$; $p = 0.012$), active phase TP_8 ($r = -0.40$; $p = 0.020$), active phase P_7 gamma activity ($r = -0.37$; $p = 0.019$), active phase P_z gamma activity ($r = -0.55$; $p = 0.001$), TP_7 gamma reactivity ($r = -0.42$; $p = 0.005$) and P_7 gamma reactivity ($r = -0.33$; $p = 0.037$).

Table 5.17 – The associations between Judgment domain performance and electroencephalography variables of the non-health professional group

Dependent Variable	Independent Variable	n	r	p
Judgement (Coginstat)	Bδ-C_z	42	0.38	0.018*
	Bδ-T_8	47	0.31	0.044*
	Aδ-FC_4	35	-0.41	0.021*
	Rδ-F_8	47	-0.34	0.025*
	Rδ-FC_3	49	-0.35	0.019*
	Rδ-T_8	49	-0.32	0.033*
	Rθ-C_4	42	-0.44	0.005*
	Bβ-FT_7	37	-0.38	0.028*
	Aβ-CP_3	40	-0.33	0.046*
	Aβ-CP_z	39	-0.35	0.040*
	Aγ-CP_z	39	-0.42	0.012*
	Aγ-TP_8	38	-0.40	0.020*
	Aγ-P_7	44	-0.37	0.019*
	Aγ-P_z	39	-0.55	0.001*
	Rγ-TP_7	47	-0.42	0.005*
	Rγ-P_7	45	-0.33	0.037*

Table 5.17 displays the significant partial correlations (controlling for age, BMI and years of education) between judgement domain performance score of the non-health professional group and electroencephalography variables. All other EEG variables were not significantly correlated to Judgement domain performance score and are not presented in this table.

Key: A = Active Phase; B = Baseline; BMI = Body Mass Index; C = Central; F = Frontal; n = Sample size; P = Parietal; T = Temporal; z = Midline; β = Beta; δ = Delta; γ = Gamma; θ = Theta; * = Statistical significance

A further examination of the relationship between Judgment domain performance of the non-health professional group and EEG variables was conducted using the LASSO (Figure 5.7). A cut-off normalised weight of an absolute value of 0.75 implicated baseline T_8 delta, F_4 alpha, and FT_7 beta activity, as well asl active phase C_z delta, and F_z alpha activity.

As a number of EEG variables were significantly correlated to the Judgement score in the nurse sample group, and provided a non-zero weight by the LASSO analysis, a forward stepwise general linear regression was performed.

The regression analysis retained 9 of the 19 originally entered variables (baseline T_8 delta activity, active phase C_z delta activity, T_8 delta reactivity, C_4 theta reactivity, baseline F_4 alpha activity, active phase F_z alpha activity, baseline FT_7 beta activity, active phase TP_8 and P_7 gamma activity), and had an overall significance of $p < 0.001$ (Table 5.18). Together these variables explained a substantial 89.6% of the variance in the Judgement score ($F = 13.35$ $df = 9$; $p < 0.001$; $R = 0.946$, $R^2 = 0.896$; $AR^2 = 0.828$).

Furthermore, baseline T_8 delta activity, T_8 delta reactivity, baseline F_4 alpha activity, and active phase F_z alpha activity, as well as active phase TP_8 and P_7 gamma activity, also presented as independently significant predictors of Judgement domain performance in the non-health professional group ($p \leq 0.010$).

Figure 5.7 – Normalised LASSO weights for the Judgement domain performance of the non-health professional group

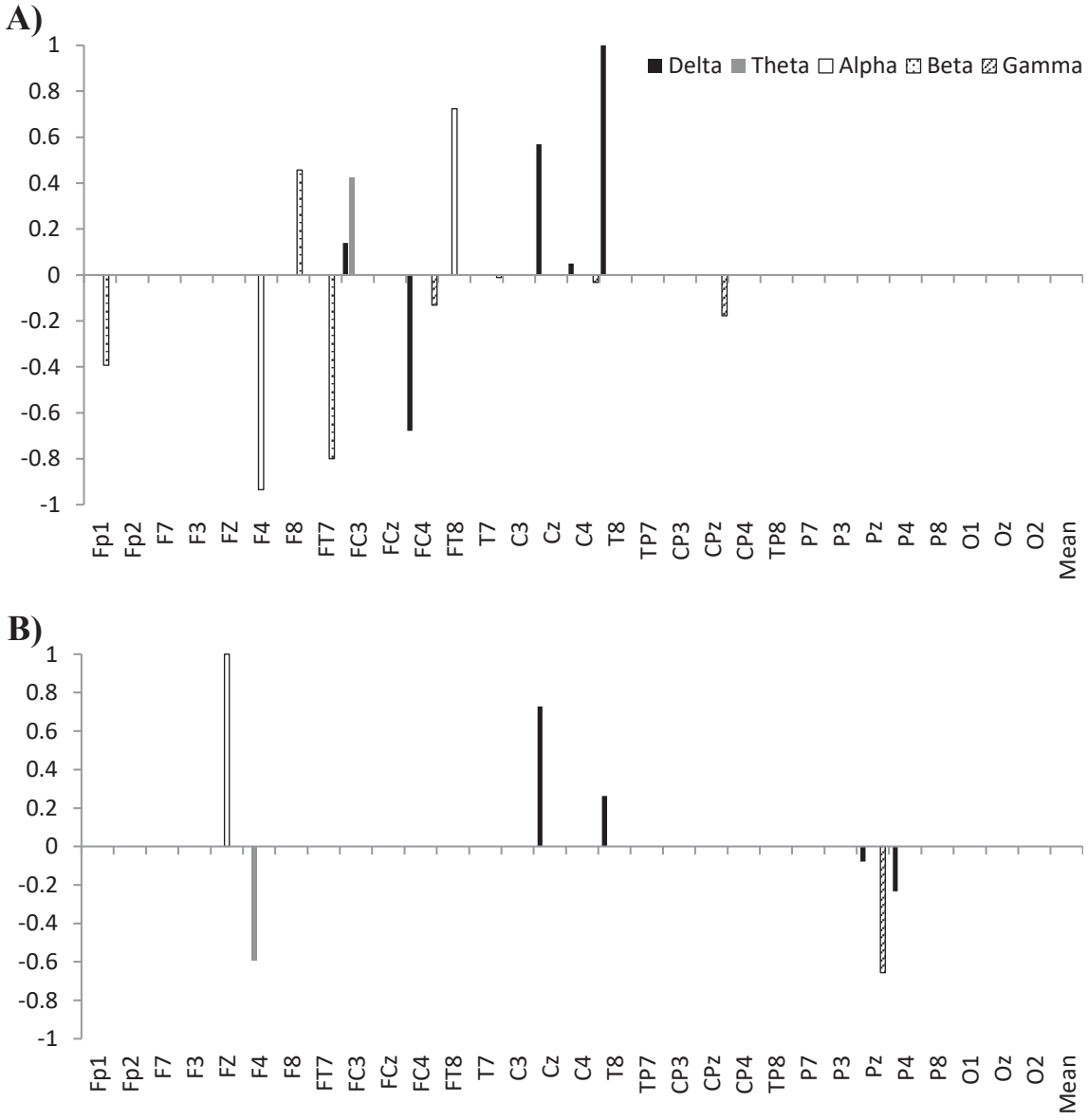

Figure 5.7 presents the normalised LASSO weights for electroencephalography variables and Judgement domain performance of the non-health professional group. Plot A displays baseline variables; and Plot B displays active phase variables. Additionally, the weights for all reactivity variables were zeroed and hence are not presented

Key: C = Central; F = Frontal; Fp = Frontal pole; O = Occipital; P = Parietal; T = Temporal; z = Midline

Table 5.18 – Regression analysis for Judgement domain performance and significantly correlated electroencephalography variables in the non-health professional group

			$R = 0.946$; $R^2 = 0.896$; $AR^2 = 0.828$; SSM = 19.55; dfM= 9; MSM = 2.17; SSR = 2.28; dfR = 14; MSR = 0.16 $F = 13.35$; $p < 0.001$*			
Variable	**β**	**SE of β**	**B**	**SE of B**	**t**	**p**
Intercept			3.39	0.25	13.38	< 0.001*
Bδ-T_8	0.85	0.16	0.02	< 0.01	5.34	< 0.001*
Aδ-C_z	0.17	0.10	0.01	< 0.01	1.69	0.113
Rδ-T_8	0.58	0.17	0.03	< 0.01	3.35	0.005*
Rθ-C_4	-0.16	0.07	-0.14	0.08	-1.77	0.099
Bα-F_4	-0.23	0.10	-0.02	< 0.01	-2.37	0.033*
Aα-F_z	0.58	0.10	0.15	0.03	5.98	< 0.001*
Bβ-FT_7	0.23	0.16	0.05	0.03	1.40	0.182
Aγ-TP_8	0.34	0.13	0.29	0.11	2.66	0.019*
Aγ-P_7	-1.42	0.25	-1.32	0.23	-5.72	< 0.001*

Table 5.18 displays a stepwise forward general linear regression analysis between judgement domain performance of the non-health professional group and the significantly correlated EEG variables. Of the 19 EEG variables originally entered into the model, the analysis retained 9: Bδ-T_8, Aδ-C_z, Rδ-T_8, Rθ-C_4, Bα-F_4, Aα-F_z, Bβ-FT_7, Aγ-TP_8, and Aγ-P_7.

Key: A = Active Phase; B = Baseline; C = Central; dfM = Degrees of freedom Model; dfR = Degrees of freedom Residual; F = Frontal; MSM = Mean square Model; MSR = Mean square Residual; P = Parietal; R = Reactivity; SE = Standard error; SSM = Sum of squares Model; SSR = Sums of squares Residual; T = Temporal; α = Alpha; β = Beta; δ = Delta; γ = Gamma; θ = Theta * = Statistical significance

5.3.4.2 Nurses

In the nurse group (Table 5.19), Judgement domain score was found to be significantly and positively correlated to both F_7 theta reactivity (r = 0.28; p = 0.048) and active phase T_7 beta activity (r = 0.34; p = 0.032). Additionally, a number of significant negative correlations were also found: baseline Fp_2 delta activity (r = -0.37; p = 0.020), baseline F_4 delta activity (r = -0.39, p = 0.009) baseline O_1 delta activity (r = -0.36; p = 0.021), active phase F_4 delta activity (r = -0.36; p = 0.021), and FC_4 beta reactivity (r = -0.30; p = 0.049).

Table 5.19 – The associations between Judgement domain performance and electroencephalography variables of the nurse group

Dependent Variable	Independent Variable	n	r	p
Judgement (Cognistat)	Bδ-Fp_2	44	-0.37	0.020*
	Bδ-F_4	48	-0.39	0.009*
	Bδ-O_1	44	-0.36	0.021*
	Aδ-F_4	46	-0.36	0.021*
	Rθ-F_7	53	0.28	0.048*
	Aβ-T_7	44	0.34	0.032*
	Rβ-FC_4	48	-0.30	0.049*

Table 5.19 displays the significant partial correlations (controlling for age, BMI and years of education) between judgement domain performance score of the nurse group and electroencephalography variables. All other EEG variables were not significantly correlated to Judgement domain performance score and are not presented in this table.

Key: A = Active Phase; B = Baseline; BMI = Body Mass Index F = Frontal; Fp = Frontal pole; O = Occipital; n = Sample size; δ = Delta; * = Statistical significance

Lastly, a further examination of the nature of the relationship between EEG and Judgement domain performance of the nurse group was performed using LASSO analysis (Figure 5.10). Utilising a cut-off normalised weight of an absolute value of 0.75, the LASSO analysis indicated the importance of baseline F_4 delta activity.

Figure 5.8 – Normalised LASSO weights for the Judgement domain performance of the nurse group

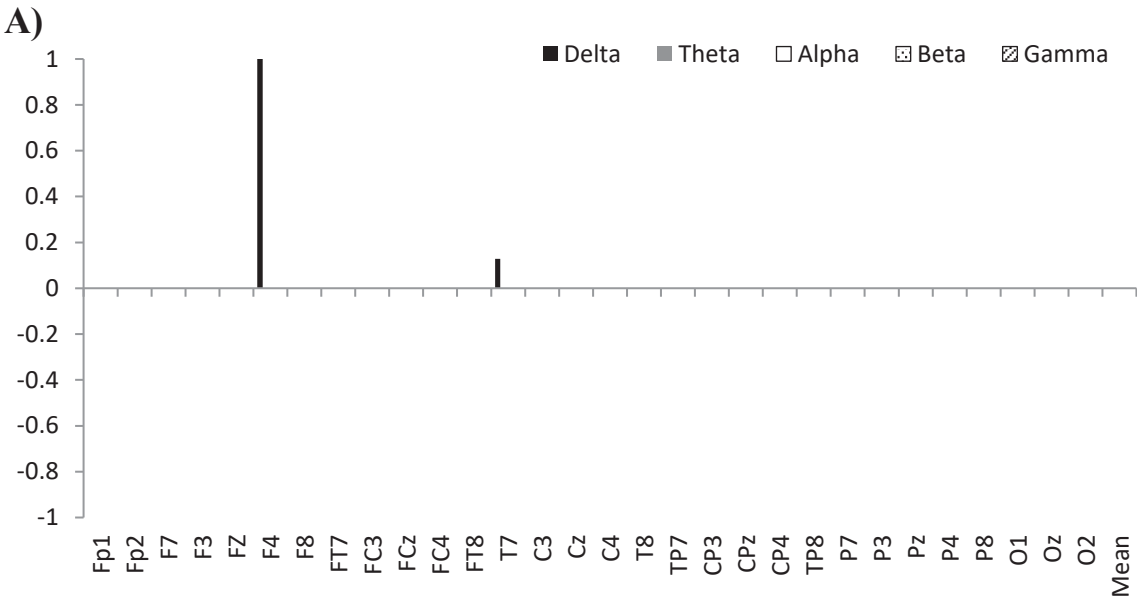

Figure 5.8 presents the normalised LASSO weights for baseline electroencephalography variables and Judgement domain performance of the nurse group. Additionally, the weights for all active phase and reactivity variables were zeroed and hence are not presented. Lastly, some frequency bands (theta, alpha, beta, and gamma) are not visible as all variable weights were reduced to zero.

Key: C = Central; F = Frontal; Fp = Frontal pole; O = Occipital; P = Parietal; T = Temporal; z = Midline

Furthermore, a forward stepwise general linear regression analysis for Judgement domain score, informed by significant correlations and the LASSO analysis was performed.

The regression analysis retained 2 of the 7 originally entered variables (baseline Fp_2 delta activity, and FC_4 beta reactivity), and had an overall significance of p = 0.001 (Table 5.20). These 2 variables together explained 42.1% of the variance in Judgement score (F = 9.44; DF = 2; p = 0.001; R = 0.649, R^2 = 0.421; AR^2 = 0.376). Furthermore, baseline Fp_2 delta activity presented as an independently significant predictor of the Judgement domain performance in the nurse group (p = 0.002).

Table 5.20 – Regression analysis for Judgement domain performance and significantly correlated electroencephalography variables in the nurse group

			R = 0.649; R^2 = 0.421; AR^2 = 0.376; SSM = 26.49; dfM= 2; MSM = 13.24; SSR = 36.48; dfR = 26; MSR = 1.40 F = 9.44; p = 0.001*			
Variable	**β**	**SE of β**	**B**	**SE of B**	**t**	**p**
Intercept			4.26	0.28	15.19	< 0.001*
Bδ-Fp_2	-0.52	0.15	< -0.01	< 0.01	-3.42	0.002*
Rβ-FC_4	-0.31	0.15	-0.04	0.02	-2.03	0.052

Table 5.20 presents a stepwise forward general linear regression analysis between judgement domain performance of the nurse group and the significantly correlated EEG variables. Of the 7 EEG variables originally entered into the model, the analysis retained 2: Bδ-Fp_2 and Rβ-FC_4.

Key: B = Baseline; C = Central; dfM = Degrees of freedom Model; dfR = Degrees of freedom Residual; F = Frontal; Fp = Frontal pole; MSM = Mean square Model; MSR = Mean square Residual; R = Reactivity; SE = Standard Error; SSM = Sum of squares Model; SSR = Sums of squares Residual; β = Beta; δ = Delta; * = Statistical significance

5.4 Discussion

The present study aimed to investigate associations between electroencephalography and cognitive performance in healthy nurses and non-health professionals; hypothesising that decreases in slow wave (delta, and theta) activity, and increased in fast wave (alpha, beta, and gamma) activity would be associated with cognitive performance. Furthermore, the secondary aim was to comment on the predictive capability of these associations with the goal to lay groundwork for future research into EEG biomarkers of early cognitive impairment.

Examination of the present correlative analysis leads to the direct suggest that the first hypothesis of the present research *('Cognitive performance will be associated with decreases in slow wave (delta, and theta) activity, and increases in fast wave (alpha, beta and gamma) activity.')* must be rejected. This analysis indicates that the nature (positive vs negative) of the relationship between EEG activity and cognitive performance depends three factors. The first is the cognitive domain examined, the second is the frequency band utilised, and the third is the localisation of that activity; and so, broad suggestions regarding entire frequency bands (like those made in the first hypothesis) are unable to be confirmed by an EEG examination of cognitive performance.

With respect to the main predictive findings of the present research were that: global cognitive performance was best predicted by EEG variables of the alpha, beta and theta frequency bands. Whilst in the specific domains, combined alpha, beta and gamma EEG variables best predicted attention domain performance; a combination of delta, gamma and theta activities predicted memory domain performance; and judgement domain performance was predicted by a combination of delta, beta and gamma variables.

5.4.1 Prediction of Global cognitive performance

Presently, global cognitive performance was assessed using both the MMSE (Folstein et al., 1975) and the Cognistat (Mueller et al., 2007). With respect to the MMSE, the recorded score of both groups were above the impairment threshold value of 24; similarly, both groups also scored above the total impairment threshold score (65) of the Cognistat. Additionally, electroencephalographic variables from the theta, alpha, beta and gamma frequency bands were all significantly correlated to global cognitive performance measures in both sample groups; further, delta band variables were also implicated for the nurse group.

In the non-health professional group, regression analysis predicted 44% of the variance in MMSE score, and 77.8% of the variance in Cognistat score, and highlighted the importance of frontal, fronto-temporal, centro-parietal, and parietal beta activity, frontal pole, central, and centro-parietal gamma activity, as well as frontal and fronto-central theta activity. Regression analysis in the nurse group, predicted 85.6% of the variance in MMSE score, and 81.0% variance in the Cognistat score. Interestingly, the theta frequency band was again demonstrated to be of particular importance, although the locations varied slight with fronto-central, parietal, and occipital sites being implicated. Furthermore, alpha activity at frontal, fronto-central, central, temporo-parietal and occipital locations was also demonstrated to be important. Together, these two sets of results indicate that global cognitive performance is primarily associated with alpha and beta activity, as well as theta variables.

Cognitive EEG research has largely concluded that reduced cognitive performance is associated with reduced alpha activity (Klimesch, 1999, Tang-Wai et al., 2003, Babiloni et al., 2006b, Rossini et al., 2006, Zadikoff et al., 2008), particularly at the parietal locations (Luckhaus et al., 2008); although some contrary results do exist (Trejo et al.,

2005, Babiloni et al., 2006b). Interestingly, some state that subdivision of the alpha band demonstrates that only lower alpha activity is correlated to changes in cognitive performance (Pijnenburg et al., 2004). Similar results have been found for the beta frequency band, where decreased beta power has been correlated with cognitive impairment (Brunovsky et al., 2003, Koenig et al., 2005, Lees et al., 2016) and cognitively impaired states (Stam et al., 2003, Pijnenburg et al., 2004, Missonnier et al., 2007, Jackson and Snyder, 2008, Lee et al., 2010, Basar et al., 2013). Finally, cognitive research has largely associated increases in theta activity with reduced cognitive performance (Brunovsky et al., 2003, Trejo et al., 2005, van der Hiele et al., 2007). Furthermore, increases in resting theta, particularly at fronto-temporal (Aurtenetxe et al., 2013, Dimpfel, 2014) and parietal locations (Aurtenetxe et al., 2013) have been demonstrated in cognitively impaired states including MCI and AD (Huang et al., 2000).

Hence, in the present study, it appears that frontal, fronto-temporal, centro-parietal, and parietal beta activity, in combination with frontal, fronto-central, parietal and occipital theta activity, and alpha activity across the whole head characterise global cognitive performance changes, and may prove useful in its prediction.

5.4.2 Prediction of Attention domain performance

With respect to attention domain performance, it was again found that both groups reported scores greater than the impairment threshold (Mueller et al., 2007). In addition, the attention domain score of both the non-health professional and nurse groups were significantly correlated with electroencephalographic variables of the delta, theta, beta and gamma frequency bands. Further, alpha band variables were also correlated to the attention score of the nurse group. In the non-health professional group, regression

analysis demonstrated the importance of centro-parietal gamma activity, and predicted 20.4% of the variance in attention domain score. Similar analysis in the nurse group implicated fronto-temporal, and parietal delta activity, frontal pole theta activity, as well as fronto-temporal and temporal gamma activity; and predicted a generous 76.7% of the variance in attention domain score.

Electroencephalography research has associated both gamma activity increases (Cho et al., 2006, van Deursen et al., 2008, Lee et al., 2010, Başar et al., 2016) and gamma activity decreases (Koenig et al., 2005, Pachou et al., 2008, Moretti et al., 2009, Aurtenetxe et al., 2013) with cognitive impairment. Research regarding slow wave activity is a little more definite, and has largely suggested that reductions in cognitive performance (Brunovsky et al., 2003) and/or cognitive impaired states are associated with increases in theta activity (Huang et al., 2000, Trejo et al., 2005, van der Hiele et al., 2007), particularly at fronto-temporal and parietal electrode locations (Aurtenetxe et al., 2013). However, it should be noted that a lesser quantity of research has demonstrated decreased theta power in MCI (Missonnier et al., 2006, Cummins et al., 2008). Furthermore, delta band research largely replicates findings reported within the theta band, whereby delta activity increases, particularly of the frontal leads, have been associated with cognitive impairment (Koenig et al., 2005, Babiloni et al., 2006b, Rossini et al., 2007, Babiloni et al., 2010, Dimpfel, 2014). Thus, literature and the present results indicate that fronto-temporal, centro-parietal and temporal gamma activity, as well as frontal pole theta activity, and fronto-temporal and parietal delta activity may represent an individual's attentional performance, and can with a moderate degree of success predict this performance.

5.4.3 Prediction of Memory domain performance

In the present analysis, it was found that the memory domain score of both the nurse and non-health professional groups were greater than the reported impairment threshold (Mueller et al., 2007). Moreover, the present analysis significantly correlated electroencephalographic variables from all of the investigated EEG frequency bands to the memory domain score of both sample groups. In the non-health professional group, 44.3% of the variance in memory domain score was predicted by the present regression analysis, which further indicated the importance of frontal pole theta and gamma activity, as well as parietal delta activity in the prediction of memory domain performance. Furthermore, the regression analysis conducted for the memory score of the nurse group predicted an extensive 99.8% of the variance, and demonstrated the importance of frontal, fronto-temporal, centro-parietal, temporal, temporo-parietal and parietal delta activity, frontal and centro-parietal theta activity, as well as frontal gamma activity.

In previous research, memory processes have been traditionally associated with the alpha and theta EEG frequency bands (Onton et al., 2005); indeed, the present analysis found associations between frontal, and centro-parietal theta activity and memory domain performance. In consensus, broader cognitive research has associated increases in theta activity with reduced cognitive performance (Brunovsky et al., 2003, Trejo et al., 2005, van der Hiele et al., 2007). Further, patients with MCI and AD have both been shown to possess increases in resting theta activity (Huang et al., 2000), particularly at fronto-temporal (Aurtenetxe et al., 2013, Dimpfel, 2014) and parietal locations (Aurtenetxe et al., 2013). Interestingly, the present analysis demonstrated that the delta and gamma bands were of greater importance for prediction of memory performance than the theta band. Previous EEG research investigating the delta frequency bands has

indicated that cognitive impairment is largely associated with increases in delta band activity, particularly at frontal electrode locations (Koenig et al., 2005, Babiloni et al., 2006b, Babiloni et al., 2008, Babiloni et al., 2010, Dimpfel, 2014). Although, it has been suggested that the degree of impairment could cause a biphasic response in delta activity (Liddell et al., 2007). Research examining the gamma frequency band has found it more difficult to reach a consensus, with both increases (Tallon-Baudry et al., 1998, Cho et al., 2006, van Deursen et al., 2008, Lee et al., 2010) and decreases in gamma activity (Pachou et al., 2008, Moretti et al., 2009, Aurtenetxe et al., 2013) being associated with cognitive impairment. As such, it could be suggested that gamma activity changes at frontal pole, and frontal locations, as well as shifts in frontal, fronto-temporal, centro-parietal, temporal, temporo-parietal and parietal delta as well as frontal and centro-parietal theta activity may represent and be predictive of varying memory capacities which could prove useful for the diagnosis of conditions like MCI and AD.

5.4.4 Prediction of Judgment domain performance

In contrast to the other results of this research, it was found that judgement domain performance for both groups was below the borderline impairment threshold score (Mueller et al., 2007). Furthermore, judgement domain score of both sample groups was significantly associated with a number of electroencephalographic variables from the delta, theta, beta, and gamma frequency bands. In the non-health professional group, the present regression analysis predicted a large 89.6% of the variance in judgement domain scores. Additionally, the analysis indicated the importance of central and temporal delta activity, temporal beta activity, as well as temporo-parietal and parietal gamma activity for the prediction of judgement domain performance. Similar analysis in the nurse group was less successful, representing only 42.1% of the variance in judgement score;

however, it did implicate the predictive importance of frontal pole delta activity, and fronto-central beta reactivity.

Previous EEG research has typically investigated judgement domain performance utilising event-related potentials (Van Rullen and Thorpe, 2001, Philiastides et al., 2006, Esposito et al., 2009, Polezzi et al., 2010) as opposed to spectral analysis; although, some spectral research has previously implicated gamma activity (Lees et al., 2016). However, a broader examination of cognitive EEG research demonstrates that each of the aforementioned EEG activities have been previously associated with cognitive performance and/or cognitive impairment.

With respect to the delta frequency band, this body of research largely demonstrates that delta activity increases (particularly at the frontal leads) are associated with cognitively impaired states such as MCI and AD (Koenig et al., 2005, Rossini et al., 2007, Babiloni et al., 2010, Dimpfel, 2014) and arithmetic performance (Fernández et al., 1995, Lees et al., 2016). Opposing results have been found in the beta frequency, where research has indicated that activity reductions (particularly at the parietal leads (Missonnier et al., 2007, Pijnenburg et al., 2004)) are associated with broad performance impairments (Lees et al., 2016) or impaired cognitive states (Huang et al., 2000, Stam et al., 2003, Pachou et al., 2008, Aurtenetxe et al., 2013). Finally, gamma activity was also associated with judgement performance, as it has been in previous research (Lees et al., 2016). Furthermore, broader EEG based cognitive research has associated cognitive impairment with both increases in gamma activity (Tallon-Baudry et al., 1998, Cho et al., 2006, van Deursen et al., 2008, Lee et al., 2010) as well as decreases in gamma activity (Koenig et al., 2005, Pachou et al., 2008, Moretti et al., 2009, Aurtenetxe et al., 2013). Therefore, based on previous literature and the present results, it could be reasonably suggested that changes in frontal pole, central and temporal delta activity,

temporal beta activity, as well as temporo-parietal and parietal gamma activity, could represent altered judgement performance, and may prove to be useful in prediction of such performance alterations in this cognitive domain.

5.5 Limitations, Future Directions & Conclusions

5.5.1 Limitations

Whilst the cognitive assessment implemented in the present analysis was comprehensive, the cognitive tasks utilised were either relatively basic or largely targeted a singular domain. Future research should look to make use of more complex cognitive assessments of singular domains, and/or tasks that assess multiple related domains, as these may prove to be more applicable to examining everyday cognitive function. Further, some cognitive capabilities, for example, learning, spatial awareness, and long-term memory were not assessed in the present analysis, and would be well suited for future examinations of the predictive capability of EEG for cognitive function.

5.5.2 Future directions

Projecting ahead, research examining the predictive capability of EEG for cognitive impairment may consider the utilisation of event-related evoked potentials, EEG coherence analysis, and/or oscillatory variables as viable complementary sources of information to the spectral frequency analysis utilised in this study. Furthermore, the hybridisation of EEG data with other electrophysiological monitoring methods previously associated to cognitive performance, such as, heart rate variability, could strengthen the predictive capabilities of any algorithm developed. Similarly, hybridisation of EEG data with traditional spatial imaging modalities like MRI to gain a

greater insight into both the spatial and temporal changes in neurodegeneration could prove to be a fruitful avenue of future research.

Additionally, in terms of data analysis strategy, utilisation of non-linear modelling may also provide an avenue forward. Furthermore, combining both linear and non-linear modelling with more complicated and detailed analytical techniques, such as general estimating equations, support vector machines, and neural networks, may hold great promise for refining the current results and future algorithm development.

With respect to the development of predictive algorithms, the examination of spectral activity ratios, for example, theta/gamma ratio, and/or reactivity ratios, that is, the change in theta/change in gamma, may prove useful as additional input variables. Further, research could benefit from implementing the present regression equations into an offline testing and validation procedure, as this would allow for a concrete examination of the predictive capability of the proposed equations. Finally, future research should investigate the possibility of placing these equations (or any proposed algorithm) into real time analysis, whereby prediction could be performed on an ongoing basis as the EEG data is captured, hence, improving the applicability and utilisation of any such algorithm.

5.5.3 Conclusions

In conclusion, the present analysis examined the predictive capability of whole brain EEG activity for cognitive performance and suggested that both global and domain specific cognitive performance of nurses and non-health professionals may be predicted (with varying degrees of success) by a unique combination of EEG variables. More specifically, a combination of fast wave activity variables in the alpha, beta and theta frequency bands predicted global cognitive performance. Moreover, performance in the

attention domain was predicted by alpha, beta and gamma activity with good success, while memory domain performance was predicted with moderate to strong success by a combination of delta, gamma and theta activities. Finally, judgement domain performance was similarly predicted by a combination of delta, beta and gamma variables. These form a series of results that enable the acceptance of the second hypothesis of the present research, (*'EEG variables will be able to predict both global and domain specific cognitive performance')*.

It is possible that these proposed models may prove useful in the prediction of early cognitive impairment, and may enable better diagnosis, treatment and management of cognitive impairment.

Chapter 6 – Conclusions

Stress and anxiety have both been shown to impact neural health (Sapolsky, 1996) and connectivity (Bishop, 2009), and various cognitive functions (Kivimäki and Lusa, 1994, Ashcraft, 2002, Tollenaar et al., 2008, Schwabe and Wolf, 2010, Renden et al., 2015). However, there is a strong need for such research in the health professions (LeBlanc, 2009, Lees and Lal, 2017), as patient care quality can be affected (Sveinsdóttir et al., 2006). Further, in some instances health professionals are the difference between life and death, and operating at anything less than optimal is of genuine concern.

The first aim of the present research was *"to investigate the associations between stress, and anxiety, and cognitive performance in nurses and non-health professionals"*; stress and anxiety are both associated with changes in cognitive performance of non-health professionals and nurses; however, the nature of the relationship between stress, anxiety, and cognitive performance may not be as expected.

In the first experiment (Chapter 3), stress was significantly and positively associated with memory domain performance for the nurse group; a result that aligns with previous research (Kirschbaum et al., 1996, Sandström et al., 2005, Luethi et al., 2008). Additionally, a significant and negative associated between stress and memory performance was found for the non-health professional group; similarly, in the second experiment (Chapter 4), a higher stress level was associated with impaired memory performance in nurses. Examining all the memory related results together suggests that the relationship between stress and memory may be best reflected by the inverted-U hypothesis, as has been previously suggested (Sandi, 2013). The second experiment (Chapter 4) also revealed that stress was associated with improved judgement domain performance in non-health professionals. Similarly, across the two experiments, anxiety

was also associated with impaired memory performance in both samples groups; a set of results that are well established in existing literature (Savage et al., 2000, Ashcraft, 2002, Lautenbacher et al., 2002).

Likewise, global cognitive performance of both groups was also associated with anxiety; however, between the groups the relationship was dimorphic with performance improving in nurses and declining in non-health professionals. Moreover, both experiments revealed that a number of EEG activity changes were associated with both stress and anxiety. Stress was associated with changes in the delta, theta, beta and gamma activity bands, and anxiety was associated with changes in primarily the delta and gamma bands, but also the alpha band. Together, these results allow the first hypothesis (*"Stress and anxiety will be associated with global and/or domain specific cognitive performance in both nurses and non-health professionals"*) of the present research to be accepted, as both stress and anxiety were associated with global and/or domain specific cognitive performance variables, including EEG variables.

Furthermore, the second hypothesis of the present research was *"the two sample groups (nurses and non-health professionals) will vary and present unique relationships between stress/anxiety and cognitive performance"*. Different cognitive impact profiles presented for the two sample groups (e.g. stress was positively correlated to memory performance in nurses, but negatively correlated to memory performance in non-health professionals), as well as the two experiments conducted e.g. stress was positively correlated with memory performance of nurses in one experiment (Chapter 3) and negatively correlated in the other (Chapter 4). Based upon these findings, the second hypothesis of the present research was accepted, and it could be suggested that the experienced level of stress and/or anxiety is a modulating factor; with mild levels being

associated to enhanced performance improvement, and moderate levels associated with impaired performance.

Lastly, EEG data was used as an objective physiological marker of cognitive performance. Therefore, the second aim of the present study was *"to investigate the associations between electroencephalographic variables and global and domain specific cognitive performance in nurses and non-health professionals"*. The present results suggest that both global and domain specific cognitive performance are correlated with various EEG variables, and that cognitive function may be predicted by a unique combination of these EEG variables.

More specifically, global cognitive performance was strongly predicted by combination of variables from the high frequency activities such as alpha, beta and gamma. With respect to domain specific performance, attention domain performance was predicted by alpha, beta and gamma activity variables with good success. Further, memory domain performance was reasonably well predicted by a combination of delta, gamma and theta frequency band variables. Finally, judgement domain performance was similarly predicted by a combination of delta, beta and gamma variables. These results, partially confirm the third present hypothesis (*"Electroencephalographic variables will be associated with both global and domain specific cognitive performance; with the higher frequency bands being the most likely implicated."*), and align with previous research; however, further validation of the regression equations and/or the development and validation of an independent algorithm would be worthwhile.

In conclusion, the findings of the present research demonstrate the multifaceted nature of the relationships between stress, anxiety and cognitive performance, where it is possible to observe both improvement and impairment of cognitive performance. In

addition, it also determines a unique profile of the cognitive impact of stress and anxiety, for both non-health professionals and nurses. Understanding and exploring these profiles, may enable the development and implementation of targeted and potentially industry specific management, monitoring and/or intervention strategies that could be utilised to preserve the health and performance of nurses, ensure quality of patient care, and reduce the incidence of adverse medical events. Moreover, EEG activity appears to be a promising predictor for early cognitive impairment, and may prove useful in the diagnosis of MCI and dementia, and subsequently, allow earlier management and treatment, and improve quality of life.

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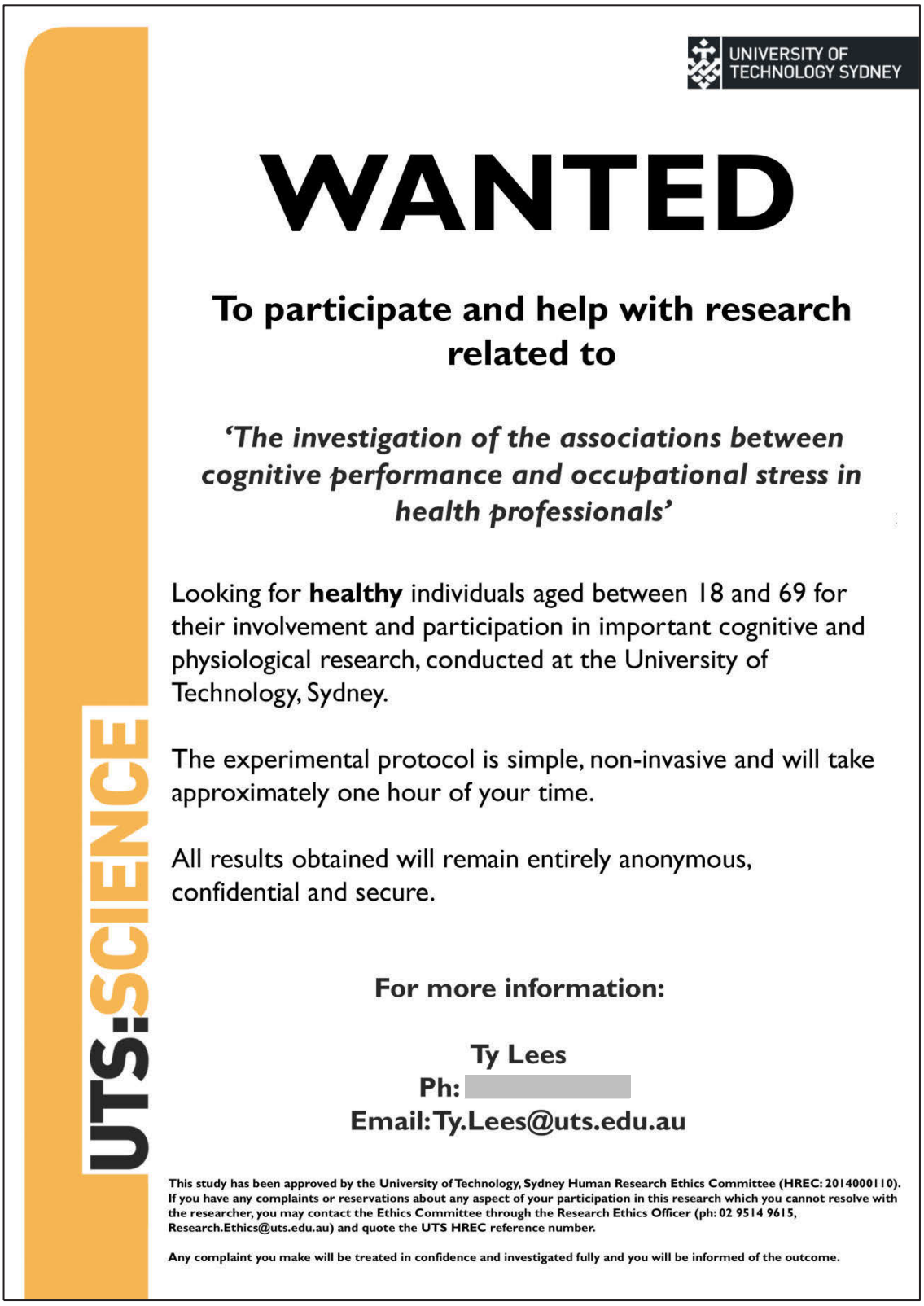
XI. Appendices

1. Recruitment Posters

1.1 Non-health professionals



1.2 Nurses

UNIVERSITY OF TECHNOLOGY SYDNEY

WANTED

To participate and help with research related to

'The investigation of the associations between cognitive performance and stress in health professionals'

Looking for currently active **nurses** aged between 18 and 69 for their involvement and participation in important cognitive and physiological research, conducted at the University of Technology, Sydney.

The experimental protocol is simple, non-invasive and will take approximately one hour of your time. All participants will be paid **$50** for their successful involvement.

All results obtained will remain entirely anonymous, confidential and secure.

For more information:

Ty Lees
Ph: [redacted]
Email: Ty.Lees@uts.edu.au

UTS:SCIENCE

This study has been approved by the University of Technology, Sydney Human Research Ethics Committee (HREC: 201400110). If you have any complaints or reservations about any aspect of your participation in this research which you cannot resolve with the researcher, you may contact the Ethics Committee through the Research Ethics Officer (ph: 02 9514 9615, Research.Ethics@uts.edu.au) and quote the UTS HREC reference number. Any complaint you make will be treated in confidence and investigated fully and you will be informed of the outcome.

2. Consent Form

UNIVERSITY OF TECHNOLOGY SYDNEY

UNIVERSITY OF TECHNOLOGY, SYDNEY
CONSENT FORM

I _____ agree to participate in the research project ***'Investigating cognitive and behavioural (stress and fatigue) associations in health professionals and/or healthy/general samples'*** (Approval no: UTS HREC REF NO. 2014000110) being conducted by Ty Lees at the Neuroscience Research Unit, University of Technology, Sydney (UTS). Funding for this research has been provided by the School of Medical and Molecular Biosciences (UTS).

I understand that the purpose of this study is to explore any associations of human physiology to brain function as this has implications for the future development of algorithms and/or management programs.

I also understand that participation in this research will involve minimal risk and/or inconvenience to me. Further, I am aware that participation in this study will include measurements of blood pressure and brain activity as well as the completion of questionnaires on lifestyle, brain (cognitive) function, behaviour, shiftwork and coping. Finally, I understand that I may be invited to participate in non-invasive salivary cortisol testing.

I also understand the study will involve screening for blood pressure and there is the possibility that I may be found to have high blood pressure. If my blood pressure is greater than 140/90 mmHg, I will be advised to consult a doctor. If my blood pressure is greater than 160/100 mmHg prior to commencing the study I will not be included in the study. If my blood pressure is greater than 160/100 mmHg at any time during the study, the study will be stopped. In both latter cases, I will be offered to be escorted to a doctor and/or advised to consult a doctor.

I am aware that I can contact Ty Lees on 0423355251 or the supervisor Associate Professor Sara Lal ((02) 9514-1592 or Sara.Lal@uts.edu.au) if I have any concerns regarding the research. Further, I am aware that I am able to withdraw my participation from this research project at any time, without consequences, and without providing a reason.

I agree that Ty Lees has sufficiently answered my questions.

I agree that the data gathered in this project may be published in a form that does not identify me in any way. All salivary samples will be used for the purpose of this study only and will be disposed after use.

_____ ____/____/____
Signature (participant)

_____ ____/____/____
Signature (researcher or delegate)

NOTE:
This study has been approved by the University of Technology, Sydney Human Research Ethics Committee. If you have any complaints or reservations about any aspect of your participation in this research which you cannot resolve with the researcher, you may contact the Ethics Committee through the Research Ethics Officer (ph: 02 9514 9772; Research.Ethics@uts.edu.au) and quote the UTS HREC reference number. Any complaint you make will be treated in confidence and investigated fully and you will be informed of the outcome.

3. Emergency Protocol

EMERGENCY PROTOCOL: GENERAL

General Emergency Protocol:

**** ALWAYS DIAL SECURITY FIRST****

UTS Contacts
1) Dial/Call UTS Security: **dial "6" on an internal UTS phone or 95141192**
2) Dial/Call 000
3) Dial/Call Student medical Services (95141177)
4) Dial/Contact supervisor: Sara Lal (95141592) or other authority in the Department

Also if required:

Contact details for UTS medical center:
Student Services Unit
Tower Building 1, Level 6, UTS
Ph: 95141177

Hours of opening:
Monday: 8:30am-5:30pm
Tuesday: 8:30am- 5:15pm
Wednesday: 8:30am-5:00pm
Thursday: 8:30am-3:45pm
Friday: 8:30am-4:45pm
Saturday and Sunday: Closed

Note: hours of opening are approximations

Contact details for external medical center:
Broadway General Practice
Level 1 Broadway Shopping Center
Ph: 92815085

Hours of opening:
Monday–Wednesday: 8:30am-7pm
Thursday: 8:30-8pm
Friday: 8:30am-7pm
Saturday: 9am-6pm
Sunday: 10am-6pm

Addition to all research study protocols

Student/Researcher protocol

Inclusion criteria

Currently based on lifestyle questionnaire (Craig et al., 1996) per parent ethics approval for the study. That is to qualify for the study (per the lifestyle questionnaire) participants have to meet the following inclusion criteria: no severe concomitant disease, no history of alcoholism and drug abuse and no psychosis, psychological and intellectual problems likely to limit compliance.
Now also add the following:

Before commencement of any human related research study, after the participant/volunteer has had a 10-minute sitting (rest) period, take 3 sitting BP measurements from the arm. A standard sphygmomanometer or a validated and reliable digital BP monitor (Omron etc.) should be used to take BP measurements.

After the measurements, **if the average of the 3 BP readings are > 160/100 mmHg or > 160 mmHg for systolic alone or >100 mmHg for diastolic BP alone, the participant will not be included in the research study and will be thanked for their time.**
Student/researcher will/must advise participant of their BP and will/must urge them to seek medical attention.

Similarly 3 BP readings are to be taken at the end of the study (if the participant qualified and underwent the study.

NOTE:
According to the Australian Heart Foundation (AHF) (www.heartfoundation.org.au) new hypertension guidelines (2008):
Normal BP: < 120/80 mmHg
High to Normal BP: 120-139/80-89 mmHg
Grade 1 (mild) hypertension: 140-159/90-99 mmHg
Grade 2 (moderate) hypertension: 160-179/100-109
Grade 3 (severe) hypertension: ≥ 180/110 mmHg

According to the AHF guidelines subjects with blood pressure (BP) values >160/100 mmHg (160/100 mmHg is the lower level of moderate hypertension) will not be included in the study and will be thanked for their time. This also means that **subjects with severe hypertension (with BP ≥180/110 mmHg) will never be included in a study**.

BP level of 160/100 mmHg is considered in the lowest range of the moderate hypertension range according to the Australian Heart Foundation (New Hypertension Guidelines, 2008).

Note: In any case if BP levels of >140/90mmHg (lowest range of mild hypertension) are recorded, advise the participant to consult their GP.

Grounds for not commencing a study, terminating a study or withdrawing a participant

Accompanying the participant for Medical Assessment

As mentioned above: study will not commence if:
- BP levels outside the criteria stipulated above (if the average of the 3 BP readings are > 160/100 mmHg or > 160 mmHg for systolic alone or >100 mmHg for diastolic BP alone, the participant will not be included in the research)
- or per exclusion criteria based on the Lifestyle questionnaire (Craig et al., 1996).

Prior to any study ask the participants to identify whether they are experiencing any pain, discomfort or not feeling well or have a headache. In all these situations the study will not continue the participant will not be included in the study and will be thanked for their time.

Prior to participation in the study you must inform the participants that if at any time during the study they experience any discomfort or not feel well or a headache commences, they must inform you (the instructor/researcher) present in the lab and the study will be immediately ceased and not continue and the participant will be thanked for their time. In this situation, you will advise and/or offer to guide (accompanied by the researcher should the participant prefer this) to consult the UTS medical center or the nearest medical center stipulated in the emergency protocol posted in the lab; or consult a medical center of their choice should they prefer the latter. You, the researcher will offer to accompany the participant to the nearest medical center should the above situation arise.

In case of an Emergency

Refer to emergency protocols displayed in the research labs.

General Emergency Protocol
To be followed if there is an emergency during working/business hours (8:30am-5pm).

NOTE: All Security personnel have an up-to-date first aid certificate, and in the event of an emergency, they should be your first point of contact.

Summary Record after each research study

You must make notes on each study immediately after its completion by filling out the form below.

Summary record of the research study

To be completed immediately after each lab study

Date: _____

Name of researcher: _____

Name of participant: _____

1. Provide a brief summary of the study (tick one of the following):
   - the study went smoothly
   - there were some issues
   - there were major issues

2. Researchers general account and summary of the study- detail in a few lines or more:

_____
_____
_____
_____
_____
_____

3. Was there any 'out of the ordinary' event or issue in this lab study? Yes/No

If Yes, provide more details:

_____
_____
_____
_____
_____
_____

4. Was there an emergency situation in the lab? Yes/No

If Yes, provide more details:

_____
_____
_____
_____
_____
_____

Note:
If you answered Yes to Question 3, you must notify a senior researcher and/or supervisor immediately.
If you answered Yes to Question 4, (you SHOULD have followed the emergency protocol and you MUST fill out an 'Incident Form', See http://www.ehs.uts.edu.au/emergency/accident.html).
Attach more pages if required.