tDCS excites cortical activity and cathodal inhibits it. However, when it comes to the cortical activity, this polarity holds dominance only in the motor area. In other areas, there are conflicting opinions on the impact of it. The possible source of this inhomogeneity is related to the limitation of previous studies. First, a large number of studies applied tDCS on various cognitive areas even without targeting the region of stimulation. Even though the stimulation is the same, different mediums lead to different impacts. Without the understanding of the cortical structure, it is hard to interpret the result of the stimulation. Second, many studies tested only one of two polarities. It leads us unable to discern the tDCS effects from measurement variability.

To overcome this problem, we tested the effect of anodal and cathodal stimulation on the same condition in the human visual cortex, of which structure is the most well-known in the human brain. We also developed an experimental protocol that enables us to dissect the tDCS effects and other noise variabilities from the measurement.

By applying this protocol, we demonstrated that the polarity of electrical current does not determine the excitation nor inhibition of cortical activity. Instead, both polarities of tDCS induce changes in the cortical population response to dynamic stimuli and cathodal was superior in scale. Our findings imply that the tDCS manifest its impact in a much more complicated way than previously reported.

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P10.81

The role of basolateral amygdala parvalbumin neurons in the blocking of Pavlovian fear

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Principal (PN) basolateral amygdala (BLA) neurons are essential for the acquisition, extinction and expression of simple forms of Pavlovian conditioning. During these simple forms of learning, PN activity is tightly regulated by parvalbumin-expressing (PV) BLA interneurons. However, both fear learning and the activity of PNs, are influenced prediction error: the discrepancy between the actual and expected outcomes of a conditioning trial. Whether, when, and how activity of BLA PV neurons contribute to fear prediction errors remains poorly understood. Here, we used PV Cre rats to address this. We used fibre photometry to record activity of BLA PV neurons during the associative blocking of learned fear. We show that the US-related activity of these neurons varies with prediction error. Then we used optogenetics to study the causal role of BLA PV neurons in gating fear learning in response to prediction errors and we found that optogenetic inhibition of PV+ neurons around moments of an expected shock restored prediction error and prevented the associative blocking of Pavlovian fear. Our findings suggest that prediction error acts to regulate fear learning via BLA PV neuronal gating of PN activity.

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P10.82

Contributions of the sound symbolism to acquisition of new word-meaning association

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Sound symbolism is the idea that a sound itself gives a meaning and impression. For example, phoneme /o/ gives bigger impression than /i/. The phenomenon was observed from infants and children, and relationship between the effect of sound symbolism and language learning has been discussed. The purpose of this study was to find out whether sound symbolism contribute word-meaning association learning. We created pseud words and line-drawing symbols with different roundness as auditory and visual stimulus, respectively. The stimulus words consisting of 3 mora were pronounced by native Japanese speakers. The perceptual roundness of both auditory and visual stimulus were evaluated in 9 levels, and the score were later used to create auditory-visual stimulus sets. The results confirmed that phoneme /k/, /t/, /s/ had angular impression and /m/, /n/ had round impression. Three experimental groups were created, and each learned different sets of word meaning (drawing symbol) pairs. First group was trained with congruent stimulus set, in which words of angular impression were paired with angular shapes. Second group was trained with incongruent stimulus set, in which words of angular impression were paired with round shapes. The last group was trained with stimulus set with randomized combination. The subjects in each group learned the associations for 5 days under functional magnetic resonance imaging (fMRI) scanning. Their learning performance was tested just after the training and again a week late to check consolidation of the learning. Our results will be the first step toward understanding of behavioral and neural basis of the sound symbolism in language learning.

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P10.83

E-vapour inhalation – How does it affect memory?

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Tobacco smoking is well-known to affect cognitive behaviour, particularly short-term memory; whilst high-fat diet (HFD) consumption has similar adverse impact on cognitive behaviour. E-cigarettes gained popularity by purporting to be a safer nicotine delivery system than cigarettes (fewer toxicants and thus presumed lower cancer producing effects). However, the impact of e-vapour inhalation on cognition is unknown as is the contribution of HFD. In this study, we aimed to investigate whether e-vapour inhalation interacts with HFD to affect short-term memory function and neural integrity in a mouse model. Balb/c mice (7 weeks) were fed a HFD (43% fat, 20 kJ/g, $n = 30$) for 10 weeks to induce obesity with chow as control (14% fat, 14 kJ/g, $n = 30$). From weeks 11–16, a sub-group of mice ($n = 15$) in each dietary group was exposed to e-vapour (18 mg/mL nicotine, tobacco flavour) twice

daily. Novel objective recognition test (NOR) was performed in week 15. Half of the brain hemisphere was collected for Western blotting for phosphorylated (p)-Tau protein level, and the other half was fixed for immunohistochemistry of the hippocampus for the apoptotic markers active caspase-3 and TUNEL. HFD consumption alone did not impair the performance during NOR, although p-Tau was reduced in the whole brain ($p < 0.05$), and Caspase 3 and TUNEL were increased in the hippocampus. In mice exposed to e-vapour, short-term memory function was significantly impaired, and was independent of the diet. E-vapour exposure significantly increased p-Tau levels, Caspase 3 and TUNEL positive cells in chow-fed mice but reduced p-Tau levels in HFD mice. In conclusion, long term e-vapour exposure led to impaired cognitive function independent of diet. Although HFD consumption alone increased p-Tau level and cell death, short-term memory function was not affected. There was no interaction between e-vapour exposure and HFD consumption on cognition in the current setting.

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P11.01

Inactivation of ATM and DNA polymerase b results in cerebellar ataxia

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Several factors including XRCC1 (a scaffolding protein) are involved in Base Excision Repair (BER) pathway for damaged bases of DNA. Defective BER factors cause human genetic syndromes such as 'Spinocerebellar ataxia with axonal neuropathy 1' (SCAN1, the responsible gene; *TDP1*) and 'Ataxia with oculomotor apraxia type 1' (AOA1, gene; *APTX1*), suggesting that there is a strong connection between ataxia and BER defects. Selective inactivation of *Xrcc1* (its binding partners include TDP1 and APTX1) during murine neurodevelopment resulted in interneuron loss in the cerebellum and severe cerebellar ataxia (in association with *Atm* inactivation). ATM is a serine/threonine kinase and one of the immediate responders to DNA damage to regulate DNA damage responses. Its mutation leads to human genetic disease; Ataxia Telangiectasia. Recently, the human *XRCC1* mutant patient with ataxia has been reported. In order to find out the causative gene for the neural phenotypes observed in the conditional *Xrcc1* animal, we knocked out conditionally DNA polymerase β (*Polb*, BER factor and XRCC1 binding partner) during brain development using a *Nestin-cre* line. Interestingly *Polb* animals displayed neuronal defects in the cerebrum, not in the cerebellum that is different from the *Xrcc1* animal. Even though *Polb/Atm* double knockout (DKO) animals showed severe ataxia, the neuropathology of the cerebellum was completely different from that of *Xrcc1/Atm* DKO animals with similar ataxia. The analysis of gene expression profiling revealed that Inositol 1,4,5-Trisphosphate Receptor Type 1 (*Itpr1*) was the most affected gene in the Purkinje cells of the *Polb/Atm* DKO cerebellum. The *ITPR1* is the responsible gene for 'Spinocerebellar ataxia 15/16'. These data suggest that ITPR1 might be one of the responsible factors during the initial stage of cerebellar ataxia due to genomic instability.

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P11.02

Impact of *Rsf1* deficiency on DNA damage response in the nervous system

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Maintaining genome stability is one of the most important factors for proper neural development, so DNA repair mechanisms are crucial for this organ. DNA repair factor mutations could lead to human genetic diseases. Among these genetic diseases, Ataxia telangiectasia (A-T) due to the mutated *ATM* gene affects the brain particularly the cerebellum, resulting in ataxia. ATM, a serine/threonine kinase, responds immediately to DNA strand breaks and regulates DNA damage responses (DDR) including apoptosis and cell cycle arrest by phosphorylation of numerous downstream substrates including Remodeling and Spacing Factor 1 (RSF1) and p53. RSF1 associated with SNF2H, as one of ATP-dependent chromatin remodelers, is involved in chromatin remodeling, spacing and gene expression. However, the connection between ATM and RSF1 in the nervous system is not fully understood. To investigate the role of ATM and RSF1 in the nervous system, particularly ATM dependent DDR during brain development, we have generated the *Rsf1* conditional knockout mouse model using a *Nestin-Cre* to delete the *Rsf1* gene selectively during neurogenesis, and found out RSF1 is dispensable for brain development, yet the *Rsf1* deficient developing brains are fully resistant to exogenously induced DNA damage. Furthermore, we observed that neuronal cells with double inactivation of *Atm* and *Rsf1* showed abnormal p53 activation which was absent in *Atm* null neuronal cells upon DNA strand breaks, suggesting that the crosstalk between ATM and RSF1 is necessary for genomic stability against excessive DNA damage during neurogenesis.

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P11.03

Timely inhibitory circuit formation by Abl1 regulates innate olfactory behaviors in the mouse

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Over half of the interneurons in the mouse olfactory bulb (OB) are developed during the first week after birth, and dominantly connect to excitatory tufted cells near the superficial granule cell layer (sGCL), unlike late-born interneurons. However, the molecular mechanisms underlying the temporal specification have not been identified. Here, we discover the role of Abelson Tyrosine-Protein Kinase 1 (Abl1) in the temporal development of early-born OB interneurons. Lentiviral knockdown of Abl1 disrupts sGCL-specific circuit of the early-born interneurons by integratory and functional defects, resulting in olfactory hyper-sensitivity. From a proteomics approach, we find that Doublecortin (Dcx) is phosphorylated by Abl1, and which contributes to the stabilization of Dcx, thereby regulating microtubule dynamics. Finally, Dcx overexpression rescues Abl1-knockdown-induced anatomic or functional defects. In summary, we suggest that the specific signaling of Abl1-Dcx in early-born interneurons facilitates the temporal development of sGCL circuit for