promoting effect of spaced trials on LTM formation. The Behavioral Tagging hypothesis and ERKs1/2 kinases involved in spaced learning may be important targets for enhancing learning and memory.

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Topic: AS13 Emotion, Memory and Cognition

SURPRISING REWARD DOWNSHIFT ACTIVATES THE LATERAL HABENULA, BUT NOT THE MEDIAL HABENULA, AS MEASURED IN TERMS OF C-FOS EXPRESSION

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Convergent results suggest that lateral habenula (LHb) activity reduces reward value and enhances aversive learning. Electrical stimulation of LHb neurons reduces sucrose intake and cocaine/morphine seeking, whereas LHb lesions attenuate taste aversion learning and avoidance of predator odor, retard appetitive extinction, and interfere with appetitive conditioned inhibition training. However, the role of the LHb in consummatory successive negative contrast (cSNC), an animal model of acute anxiety/frustration induced by reward loss, remains unknown. We hypothesized that a surprising reward downshift would enhance activity in the LHb. Three groups of rats received access to sucrose during eleven 5-min sessions. Group 32-2 had access to 32% sucrose for 10 sessions followed by a downshift to 2% sucrose on session 11. Groups 2-2 and 32-32 (unshifted controls) had access to 2% and 32% sucrose, respectively, in each of 11 sessions. After session 11, all animals were perfused and brains were prepared for immunohistochemistry of c-Fos expression, a marker of neuronal depolarization. There was less sucrose consumption on session 11 in Group 32-2 than in Groups 2-2 and 32-32—the cSNC effect ($p < 0.04$). Cell density was elevated in the lateral and medial sections of the LHb in Group 32-2, relative to unshifted groups ($ps < 0.02$). No group differences were observed in the medial habenula ($p > 0.60$). These results suggest that the LHb is involved in the cSNC effect, but its precise function remains to be determined, whether it affects cSNC by detecting the mismatch between obtained and expected rewards (reward relativity) or triggers negative emotion (frustrative nonreward) elicited by the reward loss event. Further studies involving integrated assessment of c-Fos in a wide range of brain regions, including the LHb, may clarify the fit of the LHb activity in the connectome underlying the response to reward downshift.

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LOSS-OF-FUNCTION ZEBRAFISH (DANIO RERIO) FOR GENES ASSOCIATED WITH WILLIAMS SYNDROME SHOW ALTERED QUANTITY DISCRIMINATION

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Numerosity, the ability to discriminate sets of items, has an evolutionary conserved mechanisms in all vertebrates studied. People with Williams syndrome (WS), a rare multigenic condition caused by hemideletion of 7q11.23, show altered number cognition abilities (e.g., dyscalculia). Assessing the contribution of specific genes in animal models could help discern their contribution to the WS-associated numerical impairments. Here, we used a group size preference behavioural assay previously developed by our lab to screen the contribution of two of the genes affected in WS to the quantitative abilities of juvenile zebrafish (*Danio rerio*). We used loss-of function zebrafish for: a) *baz1b*, a chromatin remodeller implicated in neural crest development; and b) *fzd9b*, codifying for a transmembrane protein receptor from the frizzled family and associated with anxiety-predisposition. The contrasts of shoals of conspecifics assessed were 2 versus 5, 2 versus 4 and 2 versus 3. In both lines, mutant zebrafish failed to show a preference for the larger group of conspecifics when confronted with the contrast of 2 versus 5. Additionally, in the *fzd9b* line, heterozygous fish were able to show a preference for the larger group in the contrast of 2 versus 3 while WT did not. Our data suggests that lack of functional *baz1b* and *fzd9b* reduces the ability of juvenile zebrafish to discriminate a larger from a smaller shoal of conspecifics. Lack of these genes seems to mostly impair their ability to approximate large numerosities while that of small numerosities is largely conserved. These data agree with previous research in humans with WS and highlight the importance to further characterise the genetic basis of dyscalculia in WS and associated disorders.

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