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Opinion Article

The Role of GLP-1 and Insulin in Metabolic Sensing

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DESCRIPTION

In order to endure and prosper in the face of selection pressure, there must learn associations that arise from the sensory data they take in from a changing environment. By means of associative learning, sensory inputs acquire a driving force that empowers their brains to guide their activities and, consequently, modify their behaviour in order to preserve the fitness of an organism. According to historical assumptions, associative learning primarily depends on the body's sensory systems providing information about the outside world, which the brain then has to understand in order to choose a conduct. But both human and non-human behaviour is incredibly flexible, skillfully adjusting to both internal and external demands.

For example, maintaining energy balance necessitates that modify behaviour to meet our physiological requirements. Thus, in order to maintain homeostasis, brain must receive, process, and prioritise physiological signals. In order to achieve this, signals that are physiologically important and related to metabolism are sent from the brain to the peripheral *via* parallel pathways. Eventually, these signals are combined with sensory cues from the outside world to produce motivated behaviour and stimulate the desire for food. More specifically, motivated behavioural responses and the learning of new outcome associations are induced by metabolic sensing of homeostatic state, which can modify the value of stimuli, actions and facilitate the quick detection of physiologically relevant sensory cues (from the body and the external environment).

At the neuronal level, motivation is regulated and actions are reinforced through Dopamine (DA)-dependent plasticity by dopamine neurons located in the ventral midbrain and their projection targets. Learning from rewards depends on the mesoaccumbens route, which is the DA projection from the Nucleus Accumbens (NAc) to the Ventral Tegmental Area (VTA). Reward prediction errors, which are significant learning signals in computational theories that formalise the neurobiological implementation of motivated behaviour in algorithms for reconstructing a reward distribution from experience, are, in fact, encoded by VTA dopaminergic neurons. The discrepancy between the actual and predicted values of an action's results is known as a reward prediction error.

Moreover, one must consider the relative prediction's precision when evaluating the magnitude of a mistake. To maximise learning, the influence of the prediction error should be down or up weighted relative to higher-order statistical features of the learnt associations, such as the variance of the outcome or the volatility of its anticipation. The mesoaccumbens DA pathway in humans and monkeys exhibits adaptive encoding of prediction mistakes, which aligns with theoretical models of adaptive learning.

Prediction errors signal the need to revise preexisting beliefs about incoming sensory inputs in the broader framework of associative learning, in addition to the previously mentioned benefits. In order to provide a need-appropriate result evaluation and adaptively direct choice behaviour, it is necessary to assess the importance of these inputs in light of the current physiological situation. Recently, the mesoaccumbens pathway has disclosed as a prominent candidate for this contextualization of the learning process to metabolic sensing of homeostatic state; in relation to food intake, VTA DA neurons are highly modulated by peripheral orexigenic and anorexigenic peptides and are susceptible to the postestive effects of food and the nutritional value of food cues.

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