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The nucleus accumbens, dopamine, and social learning

Description

[su_box title="Key excerpt" box_color="#000000?]The nucleus accumbens has a significant role in the cognitive processing of motivation, aversion, reward (i.e., incentive salience, pleasure, and positive reinforcement), and reinforcement learning (e.g., Pavlovian-instrumental transfer).[/su_box]

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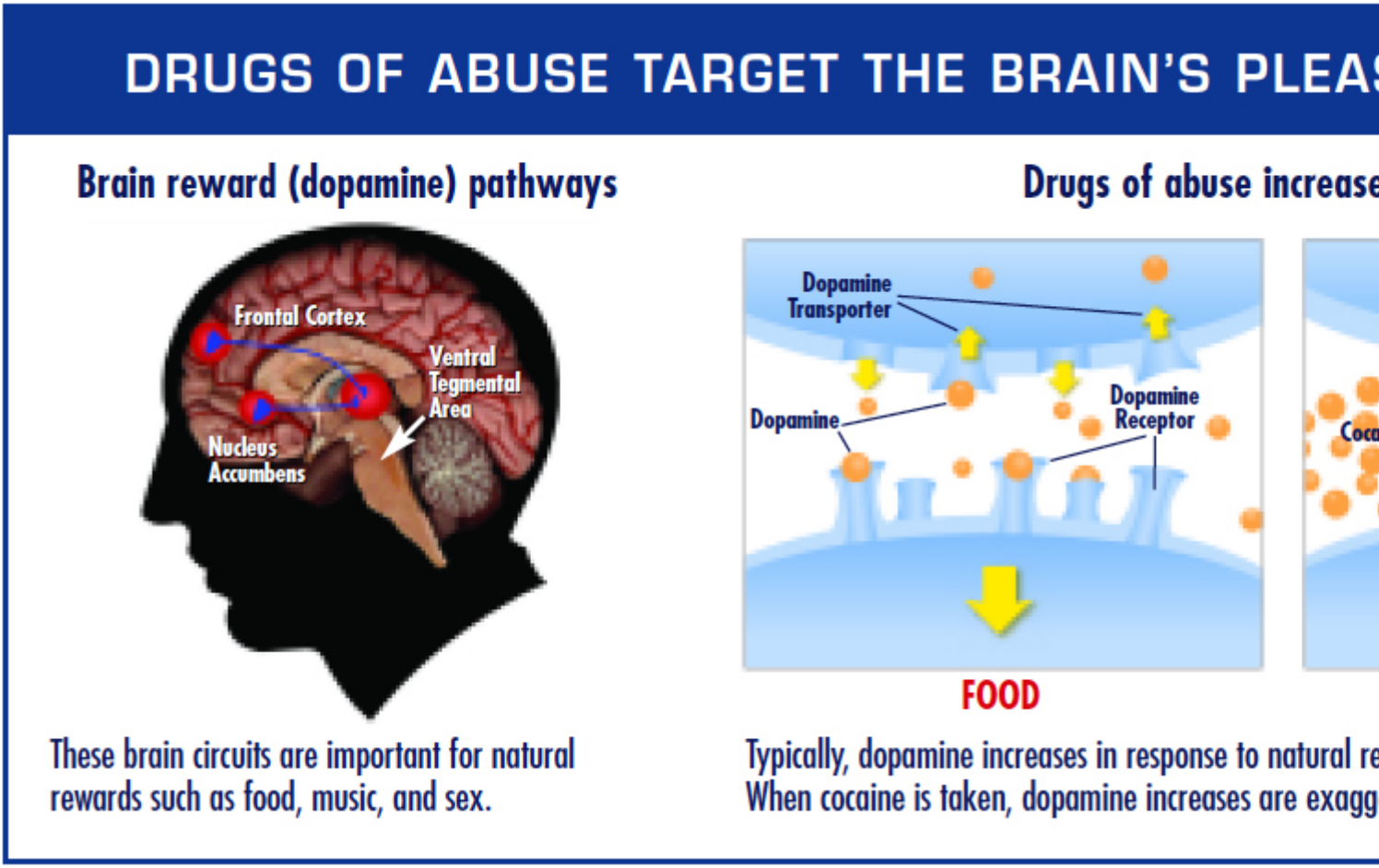
Sagittal MRI slice with highlighting (red) indicating the nucleus accumbens.

The nucleus accumbens (NAc or NAcc), also known as the accumbens nucleus, or formerly as the nucleus accumbens septi (Latin for nucleus adjacent to the septum) is a region in the basal forebrain rostral to the preoptic area of the hypothalamus.[1] The nucleus accumbens and the olfactory tubercle collectively form the ventral striatum. The ventral striatum and dorsal striatum collectively form the striatum, which is the main component of the basal ganglia.The dopaminergic neurons of the mesolimbic pathway project onto the GABAergic medium spiny neurons of the nucleus accumbens and olfactory tubercle. Each cerebral hemisphere has its own nucleus accumbens, which can be divided into two structures: the nucleus accumbens core and the nucleus accumbens shell. These substructures have different morphology and functions.



Different NAcc subregions (core vs shell) and neuron subpopulations within each region (D1-type vs D2-type medium spiny neurons) are responsible for different cognitive functions. As a whole, the nucleus accumbens has a significant role in the cognitive processing of motivation, aversion, reward (i.e., incentive salience, pleasure, and positive reinforcement), and reinforcement learning (e.g., Pavlovian-instrumental transfer); hence, it has a significant role in addiction. In addition, part of the nucleus accumbens core is centrally involved in the induction of slow-wave sleep. The nucleus accumbens plays a lesser role in processing fear (a form of aversion), impulsivity, and the placebo effect. It is involved in the encoding of new motor programs as well.

en.wikipedia.org/wiki/Nucleus_accumbens

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Further References

Dölen, G., Darvishzadeh, A., Huang, K. W., & Malenka, R. C.. (2013). Social reward requires coordinated activity of nucleus accumbens oxytocin and serotonin. Nature

Plain numerical DOI: 10.1038/nature12518
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“Social behaviours in species as diverse as honey bees and humans promote group survival but often come at some cost to the individual. although reinforcement of adaptive social interactions is ostensibly required for the evolutionary persistence of these behaviours, the neural mechanisms by which social reward is encoded by the brain are largely unknown. here we demonstrate that in mice oxytocin acts as a social reinforcement signal within the nucleus accumbens core, where it elicits a presynaptically expressed long-term depression of excitatory synaptic transmission in medium spiny neurons. although the nucleus accumbens receives oxytocin-receptor-containing inputs from several brain regions, genetic deletion of these receptors specifically from dorsal raphe nucleus, which provides serotonergic (5-hydroxytryptamine; 5-ht) innervation to the nucleus accumbens, abolishes the reinforcing properties of social interaction. furthermore, oxytocin-induced synaptic plasticity requires activation of nucleus accumbens 5-ht1b receptors, the blockade of which prevents social reward. these results demonstrate that the rewarding properties of social interaction in mice require the coordinated activity of oxytocin and 5-ht in the nucleus accumbens, a mechanistic insight with implications for understanding the pathogenesis of social dysfunction in neuropsychiatric disorders such as autism.”
Trezza, V., Damsteegt, R., Achterberg, E. J. M., & Vanderschuren, L. J. M. J.. (2011). Nucleus Accumbens -Opioid Receptors Mediate Social Reward. Journal of Neuroscience

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“Positive social interactions are essential for emotional well-being and proper behavioral development of young individuals. here, we studied the neural underpinnings of social reward by investigating the involvement of opioid neurotransmission in the nucleus accumbens (nac) in social play behavior, a highly rewarding social interaction in adolescent rats. intra-nac infusion of morphine (0.05-0.1 ?g) increased pinning and pouncing, characteristic elements of social play behavior in rats, and blockade of nac opioid receptors with naloxone (0.5 ?g) prevented the play-enhancing effects of systemic morphine (1 mg/kg, s.c.) administration. thus, stimulation of opioid receptors in the nac was necessary and sufficient for morphine to increase social play. intra-nac treatment with the selective ?-opioid receptor agonist [d-ala(2),n-mephe(4),gly(5)-ol]enkephalin (damgo) (0.1-10 ng) and the ?-opioid receptor antagonist cys-tyr-d-trp-arg-thr-pen-thr-nh(2) (ctap) (0.3-3 ?g) increased and decreased social

play, respectively. the ?-opioid receptor agonist dpdpe ([d-pen(2),d-pen(5)]-enkephalin) (0.3-3 ?g) had no effects, whereas the ?-opioid receptor agonist u69593 (n-methyl-2-phenyl-n-[(5r,7s,8s)-7-(pyrrolidin-1-yl)-1-oxaspiro[4.5]dec-8-yl]acetamide) (0.01-1 ?g) decreased social play. intra-nac treatment with ?-endorphin (0.01-1 ?g) increased social play, but met-enkephalin (0.1-5 ?g) and the enkephalinase inhibitor thiorphan (0.1-1 ?g) were ineffective. damgo (0.1-10 ng) increased social play after infusion into both the shell and core subregions of the nac. last, intra-nac infusion of ctap (3 ?g) prevented the development of social play-induced conditioned place preference. these findings identify nac ?-opioid receptor stimulation as an important neural mechanism for the attribution of positive value to social interactions in adolescent rats. altered nac ?-opioid receptor function may underlie social impairments in psychiatric disorders such as autism, schizophrenia, or personality disorders."
Day, J. J., Roitman, M. F., Wightman, R. M., & Carelli, R. M.. (2007). Associative learning mediates dynamic shifts in dopamine signaling in the nucleus accumbens. Nature Neuroscience

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"The ability to predict favorable outcomes using environmental cues is an essential part of learned behavior. dopamine neurons in the midbrain encode such stimulus-reward relationships in a manner consistent with contemporary learning models, but it is unclear how encoding this translates into actual dopamine release in target regions. here, we sampled dopamine levels in the rat nucleus accumbens on a rapid (100 ms) timescale using electrochemical technology during a classical conditioning procedure. early in conditioning, transient dopamine-release events signaled a primary reward, but not predictive cues. after repeated cue-reward pairings, dopamine signals shifted in time to predictive cue onset and were no longer observed at reward delivery. in the absence of stimulus-reward conditioning, there was no shift in the dopamine signal. consistent with proposed roles in reward prediction and incentive salience, these results indicate that rapid dopamine release provides a reward signal that is dynamically modified by associative learning."
Wise, R.. (1989). Brain Dopamine And Reward. Annual Review of Psychology

Plain numerical DOI: 10.1146/annurev.psych.40.1.191
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"While the evidence is strong that dopamine plays some fundamental and special role in the rewarding effects of brain stimulation, psychomotor stimulants, opiates, and food, the exact nature of that role is not clear. one thing is clear: dopamine is not the only reward transmitter, and dopaminergic neurons are not the final common path for all rewards. dopamine antagonists and lesions of the dopamine systems appear to spare the rewarding effects of nucleus accumbens and frontal cortex brain stimulation (simon et al 1979) and certainly spare the rewarding effects of apomorphine (roberts & vickers 1988). it is clear that reward circuitry is multisynaptic, and since dopamine cells do not send axons to each other or receive axons from each other, dopamine can at best serve as but a single link in this circuitry. if dopamine is not a final common path for all rewards, could it be an intermediate

common path for most rewards? some workers have argued against such a view, but at present they must do so on incomplete evidence. for example, phillips (1984) has argued that there must be multiple reward systems, functionally independent and organized in parallel with one another. his primary evidence, however, is the fact that brain stimulation is rewarding at different levels of the nervous system. as we have seen in the case of midline mesencephalic stimulation, the location of the electrode tip in relation to the dopamine cells and fibers tells us little about the role of dopamine in brain stimulation reward. it seems clear that the ventral tegmental dopamine system plays a critical role in midline mesencephalic reward, despite the distance from the electrode tip to the dopamine cells where morphine causes its dopamine-dependent facilitory effects or to the dopamine terminals where low-dose neuroleptics presumably cause theirs. until pharmacological challenge has been extended to the cases discussed by phillips, we can only speculate as to the role of dopamine in each of those cases. in the cases where pharmacological challenge has been examined, only nucleus accumbens and frontal cortex have been found to have dopamine-independent reward sites. it is not consistent with the dopamine hypothesis that dopamine-independent reward sites should exist in these areas, since any reward signals carried to nucleus accumbens or frontal cortex by dopamine fibers would-unless we are to believe that reward ‘happens’ at these sites-have to be carried to the next stage of the c…”

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