to be an attractive system for understanding how the brain responds to threats and activates defenses that help maintain homeostasis.

COMPETING FINANCIAL INTERESTS
The author declares no competing financial interests.

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Social nudges: utility conferred from others

David V Smith & Mauricio R Delgado

Observing the choices of others adds utility to the chosen option. The additional utility conferred by others' choices is encoded by the ventromedial prefrontal cortex and explains the idiosyncratic effects of social influence.

Other individuals can profoundly influence our decisions. From purchasing a new car to ordering lunch at a restaurant, our knowledge of what others chose in similar situations affects our own choices. These social nudges can shape the courses of our lives, leading us to make better or worse decisions. For example, when having lunch with friends, you may have a desire to order the bacon cheeseburger but you may conform to a healthier salad option if that is what your friends order. Although psychologists have long recognized the importance of conformity, it remains unclear why some individuals are more likely to conform.

In this issue of *Nature Neuroscience*, Chung *et al.*[1] investigated conformity in the context of decisions involving uncertainty. The decisions that have the most effect on our future involve elements of uncertainty: for example, questioning whether that new car is right for you. In these situations, social information often influences our decision. But it does so in an asymmetric manner, leading some people to take the risk and buy the car and other people to take the safe choice and wait for a better deal.

Chung *et al.*[1] hypothesized that the influence of a social nudge on decisions involving uncertainty critically depends on how others influence the perceived utility of the chosen option. To test this intriguing hypothesis, they developed a simple gambling task. On each trial, participants choose between two gambles that differ in objective uncertainty. In this task, a 'safer' gamble carries less payoff variance (for example, 40% chance of $33 versus 60% chance of $23), whereas a 'riskier' gamble carries more payoff variance (for example, 40% chance of $57 versus 60% chance of $2). When the probability of a high payoff is low, the difference in expected value favors the safer option. However, as the probability of a high payoff increases, participants switch to the riskier option. If you switch relatively early, you would be considered risk seeking; if you switch relatively late, you would be considered risk averse (**Fig. 1**).

In a clever twist, Chung *et al.*[1] created a powerful social context by informing participants that some decisions would be made publicly in front of peers who were concurrently performing the task. This elegant design feature allowed Chung *et al.*[1] to focus on trials in which the participant made their choice after observing the choices of two peers. Exposure to this social information had dramatic influences on risk-taking behavior. Observing peers choose risky options encouraged participants to choose the risky option. Likewise, observing peers choose safe options encouraged participants to choose the safe option. Critically, if these effects are a result of the social context, and not merely the addition of information, then similar conformity effects should not be observed when the player observes the choices of a nonsocial control (for example, a computer). In line with this expectation, Chung *et al.*[1] found, in a separate behavioral experiment, that observing the choices of a computer had no influence on risk-taking behavior, thereby confirming the social nature of the observed effect.

The distinction between social and nonsocial contexts has been observed in several studies[2]. For example, recent work has suggested that social decisions are uniquely tied to computations in the temporal-parietal junction (TPJ)[3]. Striatal responses to reward are also sensitive to social context, with increasing social closeness predicting heightened striatal responses to monetary rewards[4]. In addition, winning a video game against a human opponent relative to a computerized opponent selectively increases responses in the striatum and the ventromedial prefrontal cortex (VMPFC)[5]. Building on these observations, other studies have demonstrated that the VMPFC responds to social feedback[6,7] and computes the subjective value of social information[8].

Consistent with these reports, Chung *et al.*[1] found that VMPFC responses encoded the subjective value of the chosen gamble. However, unlike in previous studies, they also developed a computational model to demonstrate that the VMPFC encodes the added utility conferred by others' choices and predicts the likelihood of conforming to those choices. These results critically depended on participants' attitudes toward risk. Specifically, a risk-averse participant was more likely to conform to a safe influence, whereas a risk-seeking participant was more likely to conform to a risky influence.

Interestingly, when participants conformed contrary to their attitudes toward risk

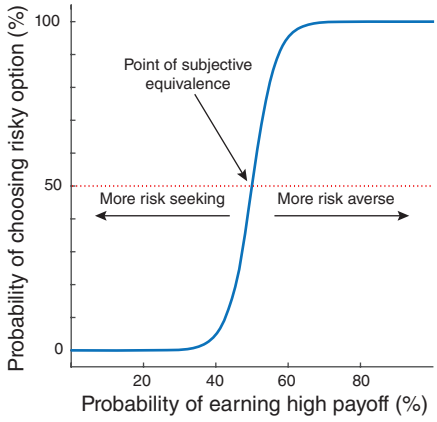

Figure 1 Individuals vary in their willingness to choose risky options. The probability of choosing the risky option increases as the probability of earning the high payoff increases. The point at which a participant is indifferent between the risky and safe option indicates that person's individual preference for risk.

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(for example, a risk-averse participant succumbing to the influence of risk-seeking peers), activation in anterior insula and dorsal anterior cingulate cortex (dACC) increased. Although Chung *et al.*[1] interpret these responses as reflecting conflict between individual desires and group pressures, another recent study suggested that dACC integrates decision variables necessary for collective decisions[9]. These differing accounts of dACC function in social decision-making need not be mutually exclusive. For instance, recent work has demonstrated that the dACC can support different functions through distinct patterns of responses[10].

The study from Chung *et al.*[1] also raises questions that could inform models of social influence and conformity. For example, recent work has indicated that the mere presence of a peer boosts responses in neural networks implicated in attention, but not motivation[11]. These findings, when coupled with the results from Chung *et al.*[1], raise the question of whether attention and motivation affect social nudges through similar or distinct pathways. In addition, several studies have shown that functional connectivity between VMPFC and TPJ increases during social valuation[12] and social competition[13]. These observations raise the intriguing possibility that VMPFC responses to the other-conferred utility observed by Chung *et al.*[1] may partially depend on interactions with other regions such as TPJ. Characterizing how VMPFC functions as part of a larger network of interconnected regions could help advance models of individual differences, potentially clarifying the mechanisms that contribute to social nudges.

Chung *et al.*[1] provide a sophisticated account of how social information is integrated with individual preferences to guide behavior in the presence of a social nudge. Given the ubiquity of social influence, these findings have a wide range of implications. For example, understanding the mechanisms of social nudges could provide an opportunity to clarify the role of peer pressure in educational settings. In such settings, teachers could group students according to individual preferences so that social information can be used to maximize academic performance[14]. In addition, these new findings could also have implications for financial markets by curbing the deleterious effects of herd mentality[15]. These examples highlight the promise that may arise from a new mechanistic understanding of how we respond to social nudges.

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Lysosomes to combat Parkinson's disease

Ole Isacson

A study finds the transcription factor Lmx1b to be necessary in adults for preventing degeneration of midbrain dopamine neurons and implicates it in lysosomal function and regulation in these neurons.

The dopaminergic neurons of the midbrain (mDA neurons) are highly metabolic, have extensive synaptic connections and are under constant high oxidative stress[1,2]. When these neurons malfunction and eventually die, people will develop the movement disorder signs and symptoms of Parkinson's disease (PD). Although PD incidence increases markedly with population aging, the genes and factors that generate the mDA neurons are first expressed during development and then maintained at low levels in adulthood. Given that these neurons are postmitotic at birth, these functions must be optimized from early development in the fetal brain to adult and throughout aging. How is this accomplished, and what are the critical mechanisms that can enable mDA neurons to survive a lifetime? To understand what sustains mDA neurons, we need to start at the beginning, at their birth. Master transcription factors orchestrate the construction of the midbrain, and, by the first trimester, the cellular and physiological character of individual mDA neurons is fully determined. Could the establishment of these early transcription factors affect the chance that these neurons will survive adult pathological challenges and age? Laguna *et al.*[3] now show that when one of these developmental factors, LIM homeobox transcription factor 1β (Lmx1b), is ablated in mouse mDA neurons after the neuron is born, these neurons degenerate in adulthood. They also observed Lmx1b reductions in human PD.

The specific actors in these midbrain developmental cellular events are nuclear transcription factors such as Nurr1, Pitx3, Lmx1a, Lmx1b, Otx2, Foxa1 and Foxa2 (ref. 4). There have been hints that each of these factors can act both in development and adulthood to regulate functions critical to mDA neurons' survival[1,4,5]. Nurr1 controls levels of almost all DA transmitter–synthesizing enzymes, independently of cell type and mDA degeneration[4,6]. Developmental absence of Pitx3 results in a loss of the mDA neurons, whereas reductions of Otx2 and FoxA2 can result in a loss of function or susceptibility to degeneration of the mDA neurons[4,5,7]. However the relevance of these model studies to PD has been hard to interpret because the transcription factor losses have occurred *in utero* or during development, not in a cell- or region-restricted manner or during adulthood. To overcome some of these problems, Laguna *et al.*[3] used Cre recombinase under the control of the dopamine transporter (DAT) locus (*Slc6a3*), which is not active until after birth of all mDA neurons is complete, to ablate the transcription factors Lmx1a or Lmx1b in transgenic mice late in development. Expression of Lmx1a is known to be necessary for the production of specific mDA neurons

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