

Social influence and the brain: persuasion, susceptibility to influence and retransmission

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Social influence is an important topic of research, with a particularly long history in the social sciences. Recently, social influence has also become a topic of interest among neuroscientists. The aim of this review is to highlight current research that has examined neural systems associated with social influence, from the perspective of being influenced as well as influencing others, and highlight studies that link neural mechanisms with real-world behavior change beyond the laboratory. Although many of the studies reviewed focus on localizing brain regions implicated in influence within the lab, we argue that approaches that account for networks of brain regions and that integrate neural data with data beyond the laboratory are likely to be most fruitful in understanding influence.

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Introduction

Social influence is omnipresent, occurring through implicit observation of cultural norms, face-to-face and mediated interpersonal communication, as well as mass mediated communication. Even though individuals are often unaware of the power of social influence, research shows its effects on behavior in a wide variety of circumstances [1]. The mechanisms driving social influence thus remain of high interest in diverse fields including psychology, sociology, communications, health, political science, marketing, and economics.

Recently, neuroscientists have begun to contribute to our understanding of social influence, especially with respect to underlying mechanisms that are not necessarily accessible with traditional self-report methodologies (Figure 1; for reviews see: [2–4]). For example, neuroimaging

enables examination of mental processes in real time and reduces the need to rely exclusively on participant introspection [5]. This review highlights recent advances in neuroscience research on social influence, examining the core processes believed to be associated with susceptibility to influence, as well as successfully influencing others. To connect the study of influence with the broader social and cognitive neuroscience literature, we summarize evidence for overlap between neural systems implicated in conflict detection, positive valuation, social cognition, and self-related processing in the context of social influence. We conclude with a discussion of new insights and methods within social and cognitive neuroscience and computational social science disciplines that promise to advance our understanding of influence moving forward.

Susceptibility to social influence

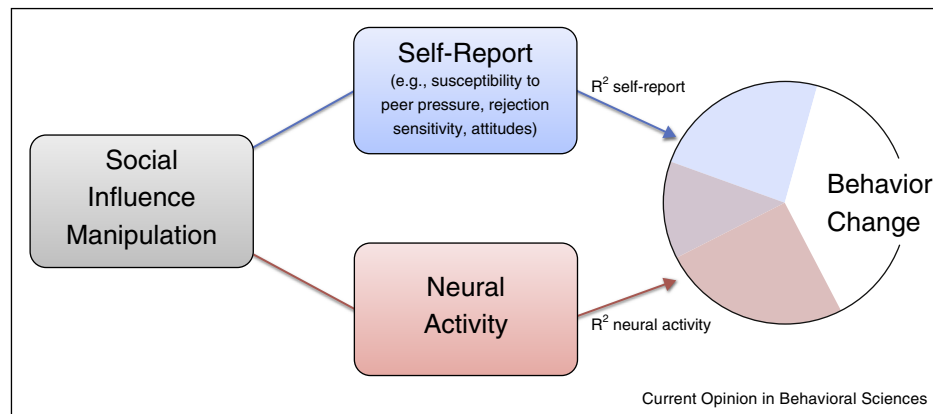
Building on a long history of social sciences studying compliance and conformity (for a review, see [1]), a growing body of research has documented neural correlates of attitude and behavior change in response to social norms or peer pressure. Converging evidence emphasizes overlap with brain systems associated with conflict detection and valuation in susceptibility to social influence [4].

Conflict detection and distress of misalignment with the group

Social psychologists have suggested that one core function of compliance and conformity is to maintain group harmony [1]. This account suggests that attitude and behavior change in response to social influence require the ability, whether conscious or unconscious, to detect conflicts between one's current behavior, preference or choice and those of others. The perception of being misaligned with others may elicit distress [6,7], which can motivate behavioral and attitudinal adjustments to realign with the group [8]. In this context, conformity may be enacted to gain group acceptance or support, which are also key to survival in evolutionary contexts [9].

The dorsal anterior cingulate cortex (dACC) is one key brain region implicated in conflict monitoring and detection [10–17], and early studies of influence demonstrated that updating behavior in response to misalignment with the group is associated with increased activity within this region [6,18], as well as in anterior insula (AI), a region hypothesized to encode the discomfort of being misaligned with the group [6,7]. To further test the causal role of brain regions hypothesized to be involved in conflict

Figure 1



Example heuristic model demonstrating the use of multiple methodologies to understand unique variance in behavior change in response to social influence manipulations (modified from; [2]).

monitoring and detection in social influence, researchers used transcranial magnetic stimulation (TMS) to down-regulate the posterior medial frontal cortex (pmFC), overlapping with dACC, during a social influence task. This manipulation reduced conformity to social influence, possibly by interrupting key processes relevant to reinforcement learning, and hence social conformity [19].

Extending to behaviors beyond the neuroimaging lab, individual differences in reactivity to social exclusion within dACC, AI, and subgenual cingulate predicted susceptibility to risky social influence in teens in a driving context one week after data were collected within these hypothesized regions using fMRI [20*]. Taken together, these studies are consistent with the idea that sensitivity to social conflict and distress in form of anticipated or actual ‘social pain’ may contribute to conformity, such that individuals may conform to avoid negative social consequences and promote social bonding [3,8].

Valuation

In addition to conflict detection, social influence may derive power from positive value placed on social relationships [21]. Expected or experienced reward of social belonging or approval from others is thought to motivate conformity [22]. The ventral striatum (VS) and ventromedial prefrontal cortex (VMPFC) are known to respond to a wide variety of rewarding stimuli, including primary and secondary rewards [23]; VMPFC is known to convert various types of value (e.g., monetary and social) into a common scale which allows individuals to anticipate overall benefits of a stimulus based on diverse types of information (e.g., [24]). In studies of social conformity, neural activity within VS and VMPFC have been implicated in updating preferences to be in line with group opinions [25–27], which may reflect anticipated social rewards of group alignment. Some authors have also

interpreted this to suggest that participants internalize what is valued by peers and come to value attitude objects rated positively by others more highly.

Moderators of neural conformity effects

The brain systems reviewed above do not work in isolation and neural activity during social influence can also be moderated according to social context. For example, research has demonstrated that neural underpinnings of social influence are modulated by message source variables (such as communicator expertise and/or celebrity status) [29] and in-group versus out-group status [30*]. Furthermore, research examining peer influence and risk behaviors among adolescents suggests that developmental factors modulate neural processes key to influence; for example, the mere presence of another peer is associated with increased activity in hypothesized reward regions (VS, orbitofrontal cortex) during the decision-making process in adolescents (compared to adults), which in turn is associated with increased risk-taking [31]. Likewise, social norms expressed by adolescent peer confederates (risky vs cautious) interact with individual differences in neural regions associated with response inhibition (including the right inferior frontal gyrus and basal ganglia) to predict later risk-taking behavior in adolescents, suggesting that neural resources may be used differently in different social contexts [32].

Social context may also modulate neural correlates of conformity. For example, one study that directly tested neural differences between public and private conformity found that brain regions hypothesized to be involved in conflict detection (dACC) during compliance decisions were associated with public compliance, while amygdala and hippocampus activity was associated with private opinion changes [28]. Additional research is needed to convincingly demonstrate whether neural activity in each

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