A key component of revenge: The "love" hormone

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Association of brain networks mediates propensity for revenge. A study of intergroup conflict in humans reveals stronger activation of the medial prefrontal cortex and higher levels of oxytocin increase the tendency to retaliate against out-group members.

Emotional contagion highly mediates humans' emotional experiences, this is to say that people are affected by the transmission of someone else's emotions (Thornton and Tamir 2017). Although this mechanism of contagion helps to socially interact, it can be detrimental to society when it involves the transmission of anger (Qu et al. 2016). The concept of people belonging to an ingroup (identified by the same characteristics) than to an outgroup (differing in identity) intensifies these emotions. Moreover, along with enhancing emotions, the tendency of revenge towards the other group as a response to their offenses is consequently strengthened (Pereira and van Prooijen 2018). The neurobiological responses to intergroup conflict still haven't been completely understood, nor the neural mechanism that drives revenge desire. Due to its worldwide relevance, of social dynamics and conflict, revenge propensity's neurobiological mechanism needs to be understood. In their paper published in *eLife*, "A neurobiological association of revenge propensity during intergroup conflict", Han et al. (2020) demonstrated that humans had a higher propensity to harm everyone outside their ingroup when their levels of endogenous oxytocin (OT) were elevated and mediated by the medial prefrontal cortex (mPFC) as a response of an outgroup member inflicting pain on an ingroup person. This association of the mPFC, OT, and revenge could potentially explain the neural underpinning that promotes the process of conflict contagion. Oxytocin is a hormone that gets the name "love hormone" from its influence on social bonding, trust, and conformism (Hertz et al. 2016). In the investigation of Han et al. (2020), they tested for endogenous OT levels—synthesized within the organism—in humans' saliva. Alternatively, in chimpanzees, OT was related to intergroup conflict and hostility towards outgroup members (Samuni et al. 2017). Furthermore, when OT is administered to humans, it seems to significantly activate the region of the mPFC (Skvortsova et al. 2020). This brain region promotes favoritism to an ingroup member over an outgroup member (Lin et al. 2018). This preference has prevented scientists from finding the real cause of revenge, whether it is negative outgroup behavior towards an ingroup member or a result of ingroup favoritism even in the absence of conflict. To control the bias created by favoritism, researchers classified the participants—healthy adults—into two groups (revenge and control) that matched in emotions, attitudes, and behaviors; however, they differ in the reason to punish the outgroup member. They also created the neural-behavior paradigm: the revenge group had to watch a competitive game, while two ingroup and outgroup members—both confederates—gave each other electric shocks. In addition, the control group had a computer that gave the shocks. Han et al. used these groups to test their hypothesis that the revenge group's OT levels would be higher than those in the control group—due to experiencing intergroup conflict— and thus, OT would mediate the tendency to mistreat the outgroup. Researchers mixed this paradigm with the functional magnetic resonance imaging (fMRI) neuroimaging technique. This method records the specific brain structures activated in a person during a certain task or experience (Roy et al. 2018). Simultaneously, Han et al. (2020) collected saliva samples to test their OT levels at three different points of the experiment: when the conflict started, when it ended, and after 15 minutes of conflict. They found that in the revenge group, after experiencing the intergroup conflict, their OT levels were significantly higher than those for the control group. Furthermore, their levels seemed to continuously increase throughout the other stages, thus suggesting a connection between intergroup conflict and high OT levels during intergroup conflict. Later on, they aimed to investigate the neural activation produced by OT. They analyzed fMRI scans taken after the second time - while also showing pictures of ingroup members with painful and non-painful facial expressions - and concluded that there was a greater activation, for both, in the mPFC. Researchers examined the association between the mPFC activity and the propensity of both groups to undertake vengeful behavior. They obtained that those in the revenge group had a higher potential to punish the outgroup members without caring whether they were directly involved or not in the conflict (punishing or just watching). Moreover, these results were followed by associating the levels of OT with the activation of mPFC in the experiment for both groups (revenge and control) towards the perception of ingroup suffering. Han et al. (2020) demonstrated that there was a significantly stronger association of mPFC activity with higher OT levels in the revenge group (vs. control group). This association suggested that the intergroup conflict made the OT levels increase and enhance its connection to the mPFC activity, which was a response to perceived ingroup pain in a conflict with an outgroup member. Researchers further these conclusions and aimed to investigate whether this association was linked to revenge propensity after experiencing the conflict. The researchers found that the increase in OT levels after experiencing the conflict and the tendency for punishment towards the outgroup was mediated by the activity of the mPFC. All in all, they showed that having ingroup identity alters OT function. Instead of promoting a bonding environment, it made them negatively respond to an outgroup during intergroup conflict. Similarly, the activation of mPFC was also related to higher OT levels and revenge propensity. Thus, it was concluded that the association between these is opening the understanding of the neurobiological explanation of the desire for revenge (Fig. 1).

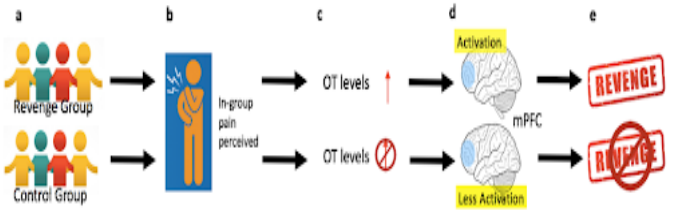

Figure 1. Humans' neurobiological association of mPFC and endogenous oxytocin during intergroup conflict. **(a)** Two groups of participants played a competitive game, ingroup vs. outgroup. Painful and non-painful electric shocks were given by a computer (control) or each other (revenge). **(b)** Each group watched ingroup getting painful shocks, and then pictures of them with a painful or non-painful facial expression. **(c)** The levels of OT increased significantly for the revenge group (vs. control group) that watched their ingroup get punished by an outgroup member. **(d)** fMRI scans showed higher activation of mPFC for the revenge group (vs. control). **(e)** The revenge group was most likely to undertake vengeful behavior (vs. control group). Han et al.'s (2020) research model helped them conclude their hypothesis. They aimed to investigate the neurobiological responses to intergroup conflict. Notably, they were able to establish an association between mPFC and endogenous OT levels both mediated by intergroup conflict and subsequently leading to revenge propensity. However, their results for punishment tendency rely greatly on participants' self-reports, and therefore it is difficult to determine real vengeful behavior. For a long time, the presence of intergroup conflict in the world has been a predominant factor of conflict contagion across communities. Even though this study investigated only one brain association, further studies need to delve deeper into other neural networks underlying revenge behavior. For example, pursuing research on serotonin, which is linked to the amygdala and mediating moral judgment and behavior (Crockett et al. 2010). By furthering knowledge about the neurobiology of intergroup conflict, people will gain insight into the importance of social identity, group dynamics, and how it connects to the brain— all leading to developing strategies to overcome group conflicts.