



The Neurobiology of Empathy: the Development, Mechanism, and Dysfunction of Empathy

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Empathy is defined as “the ability to recognize, understand, and share the emotions and perspectives of another person”. While empathy is not linked to any specific emotion, it is the confluence of conscious thought, emotional intelligence, and a range of neurochemical processes that allow us to reflect the ups and downs of human life. Often thought to be one of the greatest strengths of human nature, we take for granted the ease at which we can read social situations and mirror the feelings of our friends, families, and total strangers alike, strengthening societal bonds. However, how are we able to mimic other’s feelings, understand each other’s motivations, and relate on a deeply personal level?

Development of Empathy

Signs of empathy develop at just 7 months old, suggesting an innate predisposition for empathy development. The ability to be empathetic is closely linked to the prefrontal cortex, maturing along with the PFC and our higher thinking and impulse control (Sultan & Kahan, 2025). Because of this, childhood is the most critical period when empathy is taught and developed, with adverse childhood experiences, or ACEs, being the primary risk factor for underdeveloped or non-existent empathy, as well as the development of mental disorders defined as “cluster B” disorders in the DSM. While the capacity for empathy appears biologically primed, with twin studies showing a 30-60% heritability estimate, parents must still model social interactions to build up empathetic relationships in their children (Sultan & Kahan, 2025). Empathy’s expression is further shaped by cultural norms, socialization, and individual differences, further expanding on the idea that empathy is only partly influenced by genetics that environment then continues to build upon throughout life.

For example, empathetic expression is commonly thought to be higher in females than males, with women being portrayed as emotional and nurturing to men’s dissociation and higher cognitive thinking. In a study when participants were asked to recognize the facial queues of emotions in images of other people, females had quicker and more accurate results than males (Hampson et al., 2006). While this is only one method of studying empathy, there is a clear difference in empathetic ability between sexes, and it remains a balance between small biological differences and social expectations, with young female toddlers expressing more prosocial interest and willing to comfort others in pain. This gender/sex gap only expands as we age, leading scientists to believe that empathy is both biologically and socially driven, with sex differences not merely culturally driven byproducts (Christov-Moore et al., 2014).

Neural and Chemical Mechanisms

At the neural level, empathy relies on two key regions: the anterior insula, which activates both when a person feels pain or disgust and when they witness those same feelings in others, and the medial prefrontal cortex (mPFC), which underlies cognitive empathy and perspective-taking (Bernhardt & Singer, 2012). This maps onto the broader division between the two major classifications of empathy: affective empathy (feeling what someone else feels) and cognitive empathy (understanding and relating to another's feelings/mental state).

Chemically, the neuropeptide oxytocin enhances social bonding and emotional recognition, and works alongside arginine vasopressin to regulate attachment and fear responses. Genetic variation in the oxytocin receptor gene (OXTR) modulates empathic performance: individuals carrying the OXTR rs2268493 C allele show greater connectivity between the nucleus accumbens, the brain's reward hub, and the mPFC, a pattern linked to stronger empathic performance (Li et al., 2023). Dopamine similarly influences cognitive empathy through the enzyme dopamine beta-hydroxylase (DBH), which converts dopamine into norepinephrine. A promoter polymorphism in the DBH gene, -1021C/T, accounts for measurable variance in empathic ability, with the high-activity CC genotype associated with greater empathy than the CT or TT genotypes (Gong et al., 2014).

Narcissism and Empathy

Narcissism (or Narcissistic Personality Disorder) is a mental health disorder that is characterized by a "grandiose sense of self importance", and the idea that they are "special" and can only be understood by high status people/ institutions. Individuals with NPD often display lack of empathy & interpersonally exploitative behaviors. Many theorists view grandiosity as a defensive structure, likely built around an underlying fragile/ shame prone self, oft. rooted in early relational wounds. While the exact cause of NPD hasn't been identified yet, there is concrete evidence pointing that genetics and environment play roles in its development. Twin studies suggest heritable characteristics (no specific gene has been identified yet). There are differences in brain structure (reduced grey matter in areas is tied to emotional regulation/ empathy processing. With NPD and other "cluster B" personalities in the DSM-5 (such as antisocial & borderline), traumatic childhood experiences often act as an "on switch" for these underlying conditions. According to multiple studies, ACEs are the primary risk factor for the development of NPD in adulthood. Dysfunctional household environments and parenting practices compound the association between ACEs and pathological narcissism (Ross et al., 2024).

Conclusion

Empathy operates not as a singular trait, but as a complex synthesis of neurochemical pathways, structural brain architecture, and environmental influences. From early indicators in infancy to the functional specialization of the anterior insula and medial prefrontal cortex, the



human capacity for perspective-taking and emotional mirroring is deeply rooted in our biology. However, biological predisposition does not alone account for empathetic ability. As seen from multiple studies, childhood experiences and environment similarly play a large role in the development of empathy. Adverse Childhood experiences often act as a trigger for Cluster-B personality disorders, further proving that predisposition alone does not guarantee healthy empathetic function. Ultimately, empathy relies on a continuous communication between genetics and environment; mapping these underlying neural mechanisms not only clarifies the foundations of prosocial behavior, but also provides insight into the neurobiological origin of severe social and personality dysfunctions.

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