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Neural Correlates of Social Pain in Psychological Disorders: Implications for Educational Settings

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Abstract

Pain has long been defined as a multidimensional construct; in past research, not only have the physical and sensory aspects of pain been investigated, but also the cognitive and emotional aspects, which include the experience of social pain. This experience is generally accepted to be very distressing and can have adverse effects on one's mental health, especially for those with neurological disorders. In my paper, I examine the effects of social pain on brain activity in individuals with autism spectrum disorder (ASD) and borderline personality disorder (BPD) as compared to those who are neurotypical. This research finds that while neurotypical individuals show neural hyperactivity in key brain regions during social exclusion, individuals with ASD show hypoactivity during exclusion, and BPD patients show hyperactivity under both inclusion and exclusion conditions. This information provides useful tools for educators and has practical applications for teaching and interpersonal management strategies.

Introduction

Researchers conceptualize social pain as the feeling of pain resulting from negative social experiences. Social pain can come in the form of neglect, rejection from a social group, bullying, or the loss of a friend or family member (MacDonald & Leary, 2005). These events can be severely distressing - indeed, painful - despite the lack of any tangible threat to one's personal health or safety. While there is variability in the amount of social pain we feel, certain threats to our sense of connection can be felt more than others. Socially mediated pain can be experienced to such a great extent that one is not only extremely distressed but also incapacitated.

Initial attempts to understand the experience of social pain drew on its similarity to physical pain. Along those lines, psychologists argue that social pain, like physical pain, may serve an adaptive evolutionary function. Theoretically, social and physical pain are connected due to the fact that perceiving social exclusion to be painful was helpful from an evolutionary perspective (MacDonald & Leary, 2005). In human's evolutionary past, social connections were crucial to survival, and adults benefitted in many ways from having a connection to a social group (Eisenberger, 2012). Those who were a part of a social group received priority access to resources such as food and protection from predators. Therefore, feeling "hurt" when separated from social groups may have evolved as a way to prevent such separation and related physical hardship.

Empirically, neuroscience research has discovered commonalities in neural pathways which connect the experiences of physical and social pain. Studies on animals and humans have shown that physical and social pain rely on the same neurobiological and neural substrates (Eisenberger, 2012). Research on this topic is typically conducted via experiments accompanied by fMRI scans in order to examine areas of the brain that are activated due to social pain and whether these brain regions are similarly activated during physical pain.

While it is clear that physical pain is tied neurally to social pain, studying this connection is only one approach to understanding the experience of social pain. An alternative approach involves examining brain activity present during social threats in individuals who are not neurotypical (e.g., those with autism, social anxiety, or borderline personality disorder). This approach offers opportunities for new insights that would not emerge from a singular focus on neurotypical individuals. In particular, this approach allows researchers the opportunity to compare and contrast the specific neurological activity experienced by people with different types of psychological disorders. Given some disorders involve hypersensitivity to and misinterpretation of social cues and others involve difficulty detecting social cues, the neurological mechanisms underlying the experience of social pain may yield a similar pattern of differences. This research has implications for future studies and for educating those who work with these individuals, and it is important to look into this topic not only to gain more knowledge about these disorders, but also to better understand and assist individuals with the disorders.

The goal of this literature review is to explore the ways in which individuals with neurological disorders experience social pain as compared to neurotypical individuals and what differences, if any, the fMRI scans show. I begin by describing brain regions and functions as well as examining research demonstrating the neurological basis for social pain among neurotypical individuals. Next, I explore ways in which individuals with neurological disorders such as autism and borderline personality disorder experience social pain. I then examine similarities and differences in brain regions and their activation during experiences of social pain and use that information to draw conclusions and discuss important implications for this research.

Brain Regions Associated with Social Pain

Researchers have defined several areas of the brain relevant to processing and reacting to social information. In some cases, research literature focuses on brain regions applicable to particular situations (e.g., social acceptance or exclusion) or population groups (e.g., neurotypical or neurodiverse) being studied. In other cases, the literature centers on interconnections between brain regions that support social processing. For example, research on the social brain area focuses in on brain areas inclusive of the superior temporal sulcus (STS) and its adjacent areas, such as the middle temporal gyrus (MTG), fusiform gyrus (FG), amygdala, medial prefrontal cortex (MPFC), and inferior frontal gyrus (IFG) (Kim et al., 2015).

Literature has examined various brain regions and their functions in relation to social pain and social information processing. Below are some common terms that will be used throughout the paper (Carlson & Birkett, 2017). These terms are organized by two major brain areas which are associated with social information processing.

Prefrontal Cortex:

The prefrontal cortex is thought to be responsible for modulating and integrating feelings of pain, whether it be social or physical.

- Ventrolateral Prefrontal Cortex (vPFC): Linked with emotional regulation. Associated with descending pain modulation (i.e., when active, pain is alleviated).
- Medial Prefrontal Cortex (mPFC): Involved in social cognition, working memory for emotional responses, and behavior.
- Dorsomedial Prefrontal Cortex (dmPFC): Processes sense of self, integrates social impressions, as well as fear and anxiety information processing. Involved in self-referential mentalizing about social knowledge.
- Inferior Frontal Gyrus (IFG): Implements reappraisal strategies, responsible for reappraising the intentions of others.

Limbic System:

The limbic system is composed of the cingulate gyrus, the anterior thalamus, the hypothalamus and mammillary bodies, the cingulate cortex, the hippocampus, and the amygdala. The limbic system contains several brain areas which underlie emotional processing and complex socio-cognitive abilities.

- Amygdala: Essential to the ability to feel emotions and perceive them in others. Involved in emotional processing, especially of fear.

  Cingulate Cortex Region

  The cingulate cortex is part of the limbic system and is thought to be involved in the identification and interpretation of several different types of pain.

  - Cingulate Gyrus: Involved in regulating emotions and behavior. Predicts and avoids negative consequences.

- Dorsal Anterior Cingulate Cortex (dACC): Important for acute pain perception, executive control, conflict monitoring, and salience processing.
- Subgenual Anterior Cingulate Cortex (sgACC): Regulates emotion. Hypoactivity in this region causes feelings of sadness.
- Ventral Anterior Cingulate Cortex (vACC): Related to the effect of negative social feedback. Involved in social decision making.
- Posterior Cingulate Cortex: Involved in the perception of social dynamics, mentalizing, and memory.

Miscellaneous Areas:

These brain regions are implicated later in the paper but cannot be organized in the sections mentioned above.

- Ventral Striatum: Linked with emotional regulation and reward processing. Processes only positive emotions (Negative emotions are processed in the dorsal striatum).
- Anterior Insula (AI): Active in response to pain and to expressions of fear and disgust. Associated with negative emotional experience.
- Precuneus: Involved in memory recollection, integration of information, and affective responses to pain.
- Fusiform Gyrus (FG): Underlies our ability to process faces. Has been implicated as part of the broader social brain area.
- Superior Temporal Sulcus: Important in detection of social cues and is active during cognitive empathy. Has been implicated as part of the broader social brain area.

Social Pain Amongst Neurotypical Individuals

The term “neurotypical” is used to describe individuals who display typical developmental, intellectual and cognitive abilities. These individuals generally think, perceive and behave in ways that are considered to be “normal” by the general public. It is important to note that the neurotypical experience of the world is not the only one or the only correct one. The term “neurodiverse” is used to describe individuals who experience neurologically atypical patterns of thought or behavior. In keeping with existing research approaches, this paper focuses on neurodiversity by comparing neurotypical patterns of brain activity in response to social pain to neurodiverse patterns.

Pain has long been defined as a multidimensional construct; in past research, not only have the physical and sensory aspects of pain been investigated, but also the cognitive and emotional aspects of pain. Traditional models have focused on the transactional aspect of pain where pain causes a problem which then results in changes of mood in the subject and prompts a response from others (Evers et al., 2003). This model has since been expanded by research which focuses on the experience of physical pain being connected neurally to the experience of social pain.

This phenomenon was first recorded using functional magnetic resonance imaging (fMRI) studies in the early 2000’s which discovered that brain regions traditionally associated with the salience of physical pain, such as the dorsal anterior cingulate cortex (dACC), ventrolateral prefrontal cortex (VPFC), and the anterior insula (AI), show similar activation patterns in response to paradigms designed to evoke social pain (Eisenberger et al., 2003). Recent studies have shown that dACC and anterior insula activity is heightened when reliving the experience of social pain (Meyer et al., 2016). For the purposes of this paper, emphasis will be placed on brain activity observed during social exclusion and social pain rather than physical pain.

The general paradigm used in recent studies examining neural activity during social exclusion involves inducing social threat through an experimental manipulation, during which brain activity is recorded using an fMRI scan. Most studies induce social threat by manipulating the experience of social exclusion. Many of these studies use the program “Cyberball”, an online ball-tossing game commonly used in the social psychological literature on ostracism, social exclusion, and rejection (Williams, 2007). Cyberball is alterable, and many neuroscience studies employ a within-subjects design where participants experience both the acceptance and exclusion conditions. While playing Cyberball, participants are involved in a ball tossing game with two or three other computerized players that are controlled by the programmer. Experimenters can manipulate the speed of the game, how often the ball is tossed to the player (inclusion vs exclusion), and iconic representation, among other features (Williams, 2007). Studies with similar methods facilitate valuable comparisons between neurotypical and neurodivergent patterns of brain activity.

In the very first study to explore the neural basis of social exclusion (Eisenberger et al., 2003), participants played a game of CyberBall with two other computer simulated players. In the first round, participants played normally with the two other players, meaning all players, whether real or computerized, received the ball ⅓ of the time and subsequently experienced feelings of inclusion. In the second round, however, participants were socially excluded by the two computer players and they stopped receiving the ball after receiving it only twice at the beginning of game play. The fMRI revealed that during social exclusion, the dACC and anterior insula showed greater activity as compared to when the participants were being included.These regions are associated with the experience of social pain.

Since the publication of Eisenberger et al. (2003), subsequent studies examining neural correlates of social exclusion have replicated their findings of increased activity in the dACC and anterior insula in response to being socially excluded. Later studies have also found a positive correlation between neural activity in those areas and feelings of social distress, low self esteem, and anxious attachment (Eisenberger, 2012). Conversely, those who are less sensitive to social exclusion, namely people high in attachment avoidance, have shown a decrease in dACC and anterior insula activity (DeWall et al., 2012).

Activity in the dACC and anterior insula is not only elicited by social exclusion, but also by visual cues that suggest the subject of the image is rejecting the participant. In a study conducted by Kross et al. (2007), participants were shown rejection-themed paintings while undergoing an fMRI. The results showed heightened activity in the dACC and anterior insula upon viewing disapproving faces in the paintings, which suggests that the feelings of social exclusion can result not only from the actual experience of being socially excluded but also from imagining negative evaluation from others.

In sum, the dACC and anterior insula are consistently activated in response to social exclusion experienced by neurotypical individuals. This section lays a framework for the rest of the paper and acts as a tool for comparison purposes. This information will be important as we look to compare and contrast brain activity across several disorders. While there are varying degrees of neurological diversity, this paper will focus on the neurodiversity experienced in individuals with autism and borderline personality disorder during social exclusion.

Social Pain Amongst Individuals with Autism

Autism, or autism spectrum disorder (ASD), is defined by the DSM-5 as containing a wide range of conditions characterized by deficits in social communication or interaction and repetitive behaviors or actions (American Psychiatric Association, 2013). It is important to note that there is not just one type of autism but rather several subtypes, and because autism is a spectrum disorder, each individual with autism has a diverse set of strengths and challenges.

ASD is characterized by core symptoms such as persistent deficits in social communication and social interaction across multiple contexts, and restrictive, repetitive patterns of behavior (American Psychiatric Association, 2013). The combination of delays in verbal communication skills, difficulty reading non-verbal cues, and feeling overwhelmed by sensory inputs make it difficult for these individuals to develop and apply social skills. Those with autism report significantly more loneliness and fewer friendships that last for shorter amounts of time. Individuals with ASD do not necessarily prefer to be alone, but rather experience higher levels of loneliness due to lack of social skills (Jobe & White, 2006). This social dysfunction can manifest in many behaviors, from avoiding all social interactions to monopolizing conversations (Applied Behavior Analysis Edu, n.d.). Due to lack of research on each specific subtype of autism, I will be taking a broader approach and evaluating studies that conceptualize autism more generally.

A study conducted by Griemel and colleagues (Greimel et al., 2010) asked 15 boys with ASD and two control groups to view emotional faces presented to them, infer the emotional state of the face, and judge their own emotional response to the face. When inferring the emotional state of the faces, those with ASD showed decreased fusiform gyrus activation compared to controls suggesting they experience difficulties when attempting to process the emotional states of faces. When asked to infer their own emotional response to the faces, inferior frontal gyrus activity was decreased, suggesting difficulties with implementing reappraisal strategies. In other words, individuals with ASD have trouble reassessing the intentions of others after already deciding what they think those intentions are (Greimel et al., 2010).

Similarly, in a study conducted by Kim et al (2015), a fMRI was performed to investigate brain activation during the processing of happy, fearful, and neutral faces in 17 children with ASD. Upon visualising emotional face stimuli, children with ASD displayed reduced activity in areas of the social brain relevant to social cognition. Specifically, less activity was seen in the amygdala, superior temporal sulcus (STS), and inferior frontal gyrus (IFG). These results suggest that children with ASD experience different levels of processing social and emotional experiences neurally. Although these studies do not speak to the experience of social exclusion directly, they provide evidence of the neuropsychological mechanisms that may contribute to the social interaction difficulties people with ASD may face.

Interestingly, hypoactivation of brain regions is not only elicited by the perception of emotional faces, but also by social exclusion. In a study conducted in 2011 (Masten et al., 2011), 19 adolescents with ASD and 17 typically developing controls played a game of Cyberball in which they were ostensibly excluded by peers as they were receiving an fMRI scan. Compared to controls, individuals with ASD showed decreased activity in the subgenual anterior cingulate cortex (sgACC) and anterior insula (AI) which are regions associated with the distressing aspect of social exclusion. Less activity was also seen in the ventrolateral prefrontal cortex (vPFC) and the ventral striatum, areas which were previously linked with regulation of distress responses during peer exclusion. That is, research directly examining neurological activation during social exclusion finds that additional brain regions involved in emotional regulation are hypoactivated among people with ASD.

Throughout all of the aforementioned studies it has been shown that individuals with autism spectrum disorder typically display hypoactivation in the social areas of their brains. The neural regions related to the processing of empathy and mentalization are included in the social brain area which shows abnormality in the imaging studies of ASD patients. Since hypoactivation is present upon viewing emotional faces and when processing social exclusion, it is reasonable to hypothesize that those with ASD have difficulty detecting and coping with social rejection.

Social Pain Amongst Individuals with Borderline Personality Disorder

The diagnostic criteria of the DSM-IV-TR describes borderline personality disorder (BPD) as a mental health disorder which includes self-image issues, emotional management problems, and patterns of unstable relationships with others. Those with

BPD often experience strong emotional swings, impulsive behavior, severe anger, and an unclear self-image (American Psychiatric Association, 2013). Patients meeting criteria for BPD show a tendency to misinterpret social situations, feel an intense fear of loss or abandonment, and reveal difficulties in repairing trust after experiencing disappointment (Lis & Bohus, 2013). This pattern of symptomatology raises the question: What is the neural basis for social pain among people with BPD?

One study conducted by Wrege and colleagues (2019) strives to answer this question. The study utilized the Cyberball paradigm to examine social exclusion among 39 patients with BPD and 29 controls. Results showed that, compared to controls, socially excluded BPD patients reported lower feelings of belonging as well as hyperactivation in the anterior cingulate cortex (ACC), medial prefrontal cortex (mPFC), and right precuneus. ACC hyperactivation aligns with the subjective detection of exclusion and feelings of social pain, while PFC activation is associated with its regulation (Wrege et al. 2019) .

A study conducted by Ruocco et al. (2010) investigated social exclusion within an interaction involving real people by utilizing a new social exclusion paradigm inspired by Cyberball. Using a pack of playing cards, participants were instructed to take turns distributing cards to the other players from a stack of cards placed face down in the middle of the table. When a player received a card, they received a turn to give a card to another player. Subjects were told that whoever received the queen of hearts would get a small token, so those with more cards were more likely to win at the end of the game. During the exclusion condition, subjects received seven cards from all other players and then stopped receiving cards for the rest of the game. It was found that in patients with BPD, social exclusion elicited hyperactivation of the mPFC. The activation within this brain region increased with an increase in self-reported rejection and abandonment fears. The authors concluded that this result might be an indication of a possible dysfunction of the frontolimbic circuitry, an area inclusive of the frontal lobe and limbic system. The mPFC has several connections with limbic structures and hyperactivity in the mPFC is thought to play a role in regulating activity in the amygdala.

Research has found that social inclusion can also elicit hyperactivity of certain brain regions in BPD patients. The Cyberball paradigm was used in a study conducted by Maljeko and colleagues (2018) in which individuals with BPD participated in inclusion, exclusion, and passive watching (control) conditions. Compared to the control condition, the inclusion condition elicited hyperactivity in the dorsomedial prefrontal cortex (dmPFC) and the posterior cingulate cortex (PCC). Considering the contribution of these brain regions to emotional conflict monitoring, self-referential processes and mentalizing, these alterations seem plausible considering the frequent internal assumption among people with BPD that others will reject them.

In another study conducted by Dosmalla et al. (2014), 20 BPD patients and 20 controls played the Cyberball game in exclusion and inclusion conditions. During each condition, BPD patients showed hyperactivation of the dorsal anterior cingulate cortex (dACC) and mPFC, but the most activity was seen during the inclusion condition. This was thought to occur because subjects felt continually uncertain on what to expect behaviorally from the co-players which prompted them to put extra effort into monitoring their behavior and looking for social rejection cues. These results suggest that individuals with BPD have a tendency to hypermentalize during social encounters and therefore have difficulty discriminating between social situations.

In another experiment using Cyberball conducted by Brown and colleagues (2017), neural activity was examined during conditions of exclusion and inclusion in patients with BPD and controls without BPD. During exclusion, enhanced activation was shown in the ventral anterior cingulate cortex (vACC), and during inclusion, enhanced activation was shown in the dorsomedial prefrontal cortex (dmPFC) and anterior insula. Again, these regions implicate the presence of hypermentalization during social exclusion in BPD patients.

Consistently, BPD patients experience neural hyperactivity under both inclusion and exclusion conditions. This is unique and gives more insight into the neural processing that underlies this disorder. Hyperactivity under inclusion conditions likely stems from the fear of rejection commonly found in BPD. In other words, upon inclusion, individuals with BPD may be skeptical and pay very close attention to the social cues of others to make sure they are not being rejected, causing hyperactivity in relevant regions of the brain. Conversely, under exclusion conditions, individuals with BPD are more sensitive and take social rejection very hard, which explains hyperactivity in regions related to social information processing and emotional regulation.

Synthesis and Conclusions

In this paper, I have summarized several studies investigating the neural correlates of social pain among neurotypical individuals, individuals with autism spectrum disorder, and individuals with borderline personality disorder. Through the use of fMRI scans, these studies have revealed some consistent patterns of neural activation. Here I will discuss the similarities and differences between these fMRI scans.

In neurotypical individuals, a pattern of hyperactivity is seen in the dorsal anterior cingulate cortex (dACC) and anterior insula in response to paradigms designed to elicit social pain (Eisenberger 2012). The dACC and anterior insula have been shown to contribute primarily to the affective or unpleasant component of physical pain and are also activated during social pain. This makes sense considering that social pain and physical pain have common neural pathways and rely on the same neurobiological substrates.

Conversely, individuals with autism spectrum disorder show hypoactivity in some of the same areas. While playing Cyberball, less activity was seen in the subgenual anterior cingulate cortex (sgACC), anterior insula, ventrolateral prefrontal cortex (vPFC), and ventral striatum (Masten et al., 2011). The subACC and AI are involved in the distressing component of peer rejection while the vPFC and ventral striatum are involved in regulating these distress responses. This hypoactivation could be the case for several reasons which will be discussed later on.

Stepping back, a core process of social interaction with another person is empathic ability, and previous studies have examined the dysfunction of brain regions (amygdala, medial prefrontal cortex, and insula) associated with empathic ability in ASD (Oberman, 2007). When shown emotional faces, those with ASD showed reduced activity in the amygdala, inferior frontal gyrus (IFG), and fusiform gyrus (FG) (Griemel et al., 2010; Kim et al., 2015). These are all areas of the social brain relevant to social cognition and suggest that those with ASD are less likely to be able to understand emotional cues from others and therefore struggle with empathy. This difficulty understanding emotional cues helps to explain why those with ASD show hypoactivity in certain areas of their brain during exclusion. Hypoactivity in the amygdala, IFG, and FG when processing emotional faces corresponds to hypoactivity when being socially excluded. In sum, the inability to process emotional faces may serve as an explanation for hypoactivity during social exclusion in those with ASD.

Neural correlates related to obtaining information about the observed emotion of another person and feeling empathy are neuronal populations called mirror neurons. Dysfunction of the IFG is one of the fundamental deficits of ASD, and is one region which contains mirror neurons (Williams et al, 2006). This difficulty understanding the emotional states of others is one explanation for why during social exclusion, those with ASD show hypoactivity in regions which are hyperactive in neurotypical individuals.

The amygdala is the main area of the social brain and encompasses parts of the limbic system. It is involved in processing emotions, particularly fear, and activity in the amygdala is shown to be reduced when viewing fearful faces in those with ASD (Kim et al., 2015). The fusiform gyrus is a part of the social brain along with the amygdala and underlies our ability to process faces. Activity in the fusiform gyrus was shown to be reduced when individuals with ASD interpreted the emotional state of others’ faces (Greimel et al., 2010). This supports previous studies which suggest that those with ASD have trouble interpreting the emotional states of others which could lead to difficulties when attempting to process the experience of social exclusion.

We have seen that while neurotypical individuals tend to show hyperactivity in brain regions related to social processing, those with ASD show hypoactivity in some of the same areas. One hypothesis to explain this phenomenon proposed by Masten and colleagues (2011) suggests that adolescents with ASD may be more habituated to the feeling of being rejected since that is something they experience frequently, resulting in a dampening of neural responses due to familiarity. Another hypothesis is that individuals with ASD may expect rejection when interacting with peers and have learned through experience that they are not as likely to be included.

The Cyberball paradigm has also been used in studies investigating neural activity during social exclusion in patients with borderline personality disorder (BPD). Interestingly, BPD patients show brain hyperactivity under both inclusion and exclusion conditions. In general, areas that were active during social exclusion were the ventral anterior cingulate cortex, medial prefrontal cortex, precuneus. Areas active during social inclusion were the dorsomedial prefrontal cortex, posterior cingulate cortex, anterior insula, and dorsal anterior cingulate cortex. (Brown et al., 2017; Dosmalla et al. 2014; Maljeko et al. 2018; Ruocco et al. 2010; Wrege et al., 2019).

Hyperactivity in the ventral anterior cingulate cortex, medial prefrontal cortex, and precuneus is associated with social exclusion in BPD patients (Brown et al., 2017; Ruocco et al., 2010; Wrege et al., 2019). These areas are involved in social cognition, responses to negative social experiences, and integration of social information. One central characteristic of borderline personality disorder is an extreme fear of rejection, which tends to cause very negative reactions when the individual is excluded or rejected. Therefore, hyperactivation of these areas during social exclusion is due to the fact that those with BPD experience a very strong emotional reaction to rejection.

Given the findings regarding hyperactivity during inclusion were surprising, it is worth discussing these in more detail. Hyperactivity of the dorsomedial prefrontal cortex (dmPFC) in response to social inclusion in BPD patients suggests that this inclusion requires the subject to work hard to integrate social cues and process their own emotional response (Brown et al., 2017). The posterior cingulate cortex has similar functions to the dmPFC and is also active in response to social inclusion. This contributes to the idea that individuals with BPD tend to hypermentalize in response to social inclusion due to the fact that they are so fearful of rejection that they generally always think they are being rejected, even when that is not the case. In their study, Brown and colleagues theorize, “For BPD patients, social exclusion could be the situation they anticipate as a result of regularly biased processing in real life. The experimental condition of social inclusion could, therefore, be more unexpected for BPD patients who may then change their subjective perception to match expectations and to stabilize negative beliefs” (Brown et al., 2017, pg. 7). The activation of the dmPFC is thought to work as an effort to resolve this discrepancy between experience and expectations.

Activity in the dACC and anterior insula in response to social inclusion in BPD patients (Dosmalla et al., 2014) is the opposite of what is seen in neurotypical individuals. These are areas which are typically active in response to negative, not positive, social and physical experiences. This supports the idea that BPD patients hypermentalize during both negative and positive social situations.

Indeed, the BPD research deviates from both the neurotypical and ASD research in that BPD patients experience neural hyperactivity under both inclusion and exclusion conditions. This information allows for more insight into the neural processing that underlies this disorder. Hyperactivity under inclusion conditions likely stems from the fear of rejection commonly found in BPD. In other words, upon inclusion, individuals with BPD may be skeptical and pay very close attention to the social cues of others to make sure they are not being rejected, causing hyperactivity in relevant regions of the brain. Conversely, under exclusion conditions, individuals with BPD are more sensitive and take social rejection very hard, which explains hyperactivity in regions related to social information processing and emotional regulation.

In sum, we have learned that neural activation of brain regions associated with social and emotional processing in general and the experience and regulation of social pain more specifically differs across different psychological disorders. Whereas neurotypical individuals show hyperactivity during social exclusion, those with ASD show hypoactivity during social exclusion, and BPD patients show hyperactivity during both social exclusion and inclusion.

Implications for Social Behavior in Educational Settings

Although this research has a number of practical implications, there are two I would like to explore here. First, this research can be helpful in learning how to most effectively assist individuals with these disorders navigate social situations within educational settings. Second, this research can be helpful for those who interact with these individuals (such as peers or faculty) to best understand how their actions are affecting those who are neurodiverse. Teachers who might not have a broad knowledge of neuroscience will also be able to benefit from this information and apply it to their teaching strategies.

Autism is currently considered to be one of the most common developmental disorders and is the most common childhood neurological disorder (Autism Society Canada, 2005). The Center for Disease Control's Autism and Developmental Disabilities Monitoring Network (ADDM) estimates that 1 in every 68 children will be diagnosed with autism (Centers for Disease Control and Prevention, 2004), so it stands to reason that many of these children will be students in a general education classroom. According to the Individuals with Disabilities Education Improvement Act (IDEIA) of 2004, children with disabilities are urged to be educated with their nondisabled peers as much as possible, providing a more inclusive environment (Individuals with Disabilities Education Improvement Act 2004). For this reason, it is not only important for special education teachers to learn more information about the social processing of these children, but is also important for general education teachers. This information should urge teachers to reconsider how they are working with these students and it would be beneficial for them to include students with autism alongside their neurotypical peers as much as possible in inclusive conditions.

For children with high functioning ASD, social problems become evident with peers as early as preschool, and these problems become more pronounced over time as typical peer social interactions become more complex (Chamberlain et al., 2007). Evidence suggests that children with ASD have the fewest number of friendships of all disability groups (Rowley et al., 2012). The National Research Council's guidelines in Educating Children with Autism state that educational objectives should focus on social skills which facilitate participation in familial, educational, and community settings (National Research Council, 2001). Additionally, research has shown that children with autism can greatly benefit in many areas of their lives from interventions such as social skill groups and video modeling (DeRosier et al., 2011). These interventions have been proven to be effective in improving social skills such as social responses, awareness of emotions, social problem solving, and expression of emotions. Educators and other faculty who work with these children should speak with their parents and urge them to consider interventions for their child.

As discussed in Synthesis and Conclusions, children with autism tend to expect social exclusion and therefore brain hypoactivity is induced in response to this exclusion. Since the majority of this social exclusion comes from peers, it is important that educators are aware of this and actively make changes to attempt to decrease the frequency at which this occurs. This may mean implementing lesson plans on bullying, or making sure that children with autism interact as much as possible with their neurotypical peers and that those interactions are monitored.

It is difficult to diagnose borderline personality disorder in children because similar symptoms may evolve into one of several disorders as the child develops (Chanen et al., 2012). However, by the time the child reaches adolescence or adulthood, it is much easier to accurately diagnose this disorder. Children who are later diagnosed with BPD are often more sensitive to criticism, become upset at changes in routine or plans, and are more easily frustrated.

As a teacher, some ways to alleviate these symptoms include making sure that any criticism is delivered in a kind-hearted manner and that it is always constructive. As previously discussed, individuals with BPD show neural hyperactivity even when being included due to fear of rejection, so it is important to make sure the child does not feel like they are being rejected by their teacher. Another suggestion for teachers might be to write out a detailed schedule for the day that students can easily read and make sure to stick to that plan. For children and adolescents with BPD, knowing the lesson plan ahead of time will give them a heads up about activities that involve social interaction or social evaluation so that they can establish plans for smooth exchanges.

Although everyone experiences social pain, the neurological expression of social pain can be quite different depending on a person's degree of neurodiversity. This paper has examined and discussed similarities and differences in neural activity during social pain among individuals who are neurotypical compared to individuals with ASD and BPD and discussed implications for educational settings. It is important that, as a society, we recognize these differences and actively work toward making changes to positively impact neurodiverse individuals in their social experiences throughout their educational career.

References

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). https://doi.org/10.1176/appi.books.9780890425596

Autism Society Canada. (2005). *What is autism spectrum disorder*? Retrieved from http://www.insightpsychological.com/Library/ASD%20factsheet.pdf

Brown, R. C., Plener, P. L., Groen, G., Neff, D., Bonenberger, M., & Abler, B. (2017). Differential neural processing of social exclusion and inclusion in adolescents with non-suicidal self-injury and young adults with borderline personality disorder. *Frontiers in Psychiatry, 8*, 267-272. https://doi.org/10.3389/fpsyt.2017.00267

Carlson, N. R. & Birkett, M. A. (2017). *Physiology of Behavior* (12th ed.). Pearson.

Centers for Disease Control and Prevention. (2014). *Autism spectrum disorder data and statistics – autism and developmental disabilities monitoring network*. National Center on Birth Defects and Developmental Disabilities. Retrieved from https://www.cdc.gov/ncbddd/autism/data.html.

Chamberlain, B., Kasari, C., & Rotheram-Fuller, E. (2007). Involvement or isolation? The social networks of children with autism in regular classrooms. *Journal of Autism and Developmental Disorders*, *37(2)*, 230–242. https://doi.org/10.1007/s10803-006-0164-4

Chanen, A. M., & Kaess, M. (2012). Developmental pathways to borderline personality disorder. *Current Psychiatry Reports, 14(1)*, 45–53. https://doi.org/10.1007/s11920-011-0242-y

DeRosier, M. E., Swick, D. C., Davis, N. O., McMillen, J. S., & Matthews, R. (2011). The efficacy of a social skills group intervention for improving social behaviors in children with high functioning autism spectrum disorders. *Journal of Autism and Developmental Disorders, 41(8),* 1033–1043. https://doi.org/10.1007/s10803-010-1128-2

DeWall, C. N., Masten, C. L., Powell, C., Combs, D., Schurtz, D. R., & Eisenberger, N. I. (2012). Do neural responses to rejection depend on attachment style? An fMRI study. *Social Cognitive and Affective Neuroscience*, *7(2)*, 184–192. https://doi.org/10.1093/scan/nsq107

Domsalla, M., Koppe, G., Niedtfeld, I., Vollstädt-Klein, S., Schmahl, C., Bohus, M., & Lis, S. (2014). Cerebral processing of social rejection in patients with borderline personality disorder. *Social Cognitive and Affective Neuroscience*, *9(11)*, 1789–1797. https://doi.org/10.1093/scan/nst176

Eisenberger, N. I., Lieberman, M. D., & Williams, K. D. (2003). Does rejection hurt? An FMRI study of social exclusion. *Science (New York, N.Y.), 302(5643)*, 290–292. https://doi.org/10.1126/science.1089134

Eisenberger, N. I. (2012). Broken hearts and broken bones. *Current Directions in Psychological Science, 21(1)*, *42–47*. https://doi.org/10.1177/0963721411429455

Eisenberger N. I. (2012). The pain of social disconnection: Examining the shared neural underpinnings of physical and social pain. *Nature Reviews Neuroscience, 13(6)*, 421–434. https://doi.org/10.1038/nrn3231

Evers, A. W., Kraaimaat, F. W., Geenen, R., Jacobs, J. W., & Bijlsma, J. W. (2003). Pain coping and social support as predictors of long-term functional disability and pain in early rheumatoid arthritis. *Behaviour Research and Therapy, 41(11)*, 1295–1310. https://doi.org/10.1016/s0005-7967(03)00036-6

Greimel, E., Schulte-Rüther, M., Kircher, T., Kamp-Becker, I., Remschmidt, H., Fink, G. R., Herpertz-Dahlmann, B., & Konrad, K. (2010). Neural mechanisms of empathy in adolescents with autism spectrum disorder and their fathers. *NeuroImage*, *49(1)*, 1055–1065. https://doi.org/10.1016/j.neuroimage.2009.07.057

Individuals with Disabilities Education Improvement Act Amendments of 2004, 20 U.S.C. § 1401 et seq.

Jobe, L., & White, S. (2007). Loneliness, social relationships, and a broader autism phenotype in college students. *Personality and Individual Differences, 42*, 1479-1489. https://doi.org/10.1016/j.paid.2006.10.021.

Kim, S. Y., Choi, U. S., Park, S. Y., Oh, S. H., Yoon, H. W., Koh, Y. J., Im, W. Y., Park, J. I., Song, D. H., Cheon, K. A., & Lee, C. U. (2015). Abnormal activation of the social brain network in children with autism spectrum disorder: An FMRI study. *Psychiatry Investigation*, 12(1), 37–45. https://doi.org/10.4306/pi.2015.12.1.37

Kross, E., Egner, T., Ochsner, K., Hirsch, J., & Downey, G. (2007). Neural dynamics of rejection sensitivity. *Journal of Cognitive Neuroscience*, *19(6)*, 945–956. https://doi.org/10.1162/jocn.2007.19.6.945

Lis, S., & Bohus, M. (2013). Social interaction in borderline personality disorder. *Current Psychiatry Reports, 15(2)*, 1-7. https://doi.org/10.1007/s11920-012-0338-z

MacDonald, G., & Leary, M. R. (2005). Why does social exclusion hurt? The relationship between social and physical pain. *Psychological Bulletin*, *131(2)*, 202–223. https://doi.org/10.1037/0033-2909.131.2.202

Malejko, K., Neff, D., Brown, R., Plener, P. L., Bonenberger, M., Abler, B., & Graf, H. (2018). Neural correlates of social inclusion in borderline personality disorder. *Frontiers in Psychiatry*, *9*, 653-657. https://doi.org/10.3389/fpsyt.2018.00653

Masten, C. L., Colich, N. L., Rudie, J. D., Bookheimer, S. Y., Eisenberger, N. I., & Dapretto, M. (2011). An fMRI investigation of responses to peer rejection in adolescents with autism spectrum disorders. *Developmental Cognitive Neuroscience*, *1(3)*, 260–270. https://doi.org/10.1016/j.dcn.2011.01.004

Meyer, M. L., Williams, K. D., & Eisenberger, N. I. (2015). Why social pain can live on: Different neural mechanisms are associated with reliving social and physical pain. *PloS one, 10(6)*, e0128294. https://doi.org/10.1371/journal.pone.0128294

National Research Council. (2001). Educating children with autism. Committee on educational interventions for children with autism. *Division of Behavioral and Social Sciences and Autism*. Washington, DC: National Academies Press.

Oberman, L. M., & Ramachandran, V. S. (2007). The simulating social mind: the role of the mirror neuron system and simulation in the social and communicative deficits of autism spectrum disorders. *Psychological Bulletin*, *133(2),* 310–327. https://doi.org/10.1037/0033-2909.133.2.310

Rowley, E., Chandler, S., Baird, G., Simonoff, E., Pickles, A., Loucas, T., & Charman, T. (2012). The experience of friendship, victimization and bullying in children with an autism spectrum disorder: Associations with child characteristics and school placement. *Research in Autism Spectrum Disorders*, *6*, 1126–1134. https://doi.org/10.1016/j.rasd.2012.03.004.

Ruocco, A. C., Medaglia, J. D., Tinker, J. R., Ayaz, H., Forman, E. M., Newman, C. F., Williams, J. M., Hillary, F. G., Platek, S. M., Onaral, B., & Chute, D. L. (2010). Medial prefrontal cortex hyperactivation during social exclusion in borderline personality disorder. *Psychiatry Research: Neuroimaging*, *181(3)*, 233–236. https://doi.org/10.1016/j.pscychresns.2009.12.001

Why Do Autistic People Have Issues with Social Skills? (n.d.). Retrieved from https://www.appliedbehavioranalysisedu.org/why-do-autistics-have-issues-with-social-skills/

Williams, J. H., Waiter, G. D., Gilchrist, A., Perrett, D. I., Murray, A. D., & Whiten, A. (2006). Neural mechanisms of imitation and 'mirror neuron' functioning in autistic spectrum disorder. *Neuropsychologia, 44(4)*, 610–621.

Williams, K. D., & Jarvis, B. (2006). Cyberball: A program for use in research on interpersonal ostracism and acceptance. *Behavior Research Methods*, *38(1),* 174–180. https://doi.org/10.3758/bf03192765

Williams, K. D. (2007). Ostracism. *Annual Review of Psychology, 58(1)*, 425–452. https://doi.org/10.1146/annurev.psych.58.110405.085641

Wrege, J. S., Ruocco, A. C., Euler, S., Preller, K. H., Busmann, M., Meya, L., Schmidt, A., Lang, U. E., Borgwardt, S., & Walter, M. (2019). Negative affect moderates the effect of social rejection on frontal and anterior cingulate cortex activation in borderline personality disorder. *Cognitive, Affective, & Behavioral Neuroscience*, *19(5),* 1273–1285. https://doi.org/10.3758/s13415-019-00716-0