INVITED REVIEW

The social re-orientation of adolescence: a neuroscience perspective on the process and its relation to psychopathology

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ABSTRACT

Background. Many changes in social behavior take place during adolescence. Sexuality and romantic interests emerge during this time, and adolescents spend more time with peers and less time with parents and family. While such changes in social behavior have been well documented in the literature, relatively few neurophysiological explanations for these behavioral changes have been presented.

Method. In this article we selectively review studies documenting (a) the neuronal circuits that are dedicated to the processing of social information; (b) the changes in social behavior that take place during adolescence; (c) developmental alterations in the adolescent brain; and (d) links between the emergence of mood and anxiety disorders in adolescence and changes in brain physiology occurring at that time.

Results. The convergence of evidence from this review indicates a relationship between development of brain physiology and developmental changes in social behavior. Specifically, the surge of gonadal steroids at puberty induces changes within the limbic system that alters the emotional attributions applied to social stimuli while the gradual maturation of the prefrontal cortex enables increasingly complex and controlled responses to social information.

Conclusions. Observed alterations in adolescent social behavior reflect developmental changes in the brain social information processing network. We further speculate that dysregulation of the social information processing network in this critical period may contribute to the onset of mood and anxiety disorders during adolescence.

INTRODUCTION

Dramatic changes in social behavior occur during adolescence, yet the neurobiological underpinnings of these changes have largely been neglected. Detailing how adolescent changes in social perception, social emotion, and social cognition are instantiated in neural circuits will enhance our understanding of development in two ways. First, while evidence indicates that changes in social behavior are influenced by both socio-cultural (see Greenfield et al. 2003, for example) and congenitally determined forces (such as an individual’s hormonal milieu), both must impact on brain circuits in order to alter behavior. Thus adolescent social re-orientation may serve as a naturally occurring model for the study of developmental changes in brain–behavior relations. Second, considering the important role aberrant emotional responses to social encounters play in adolescent mood and anxiety disorders, identifying the brain circuits that underlie these processes under both normal and pathological conditions will increase our ability to treat these disorders.

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The three nodes mediating social information processing

In social species, social stimuli receive intensive neuronal processing. For example, primates are able to detect remarkably subtle differences in both static and changeable aspects of facial stimuli; and this occurs at a relatively early point in the visual processing stream (Perrett et al. 1982; Haxby et al. 2002; Pietrini et al. 2004). The early detection and categorization of a stimulus as ‘social’ is followed by neuronal processing that then integrates the stimulus into a larger emotional and cognitive framework (Adolphs, 2001). This process involves bidirectional interactions between areas devoted to social detection and those devoted to affective (limbic regions) and higher order cognitive processing (prefrontal cortical regions). The network of brain systems that are devoted to the extensive processing of social stimuli has been detailed in several previous theories (e.g. Allison et al. 2000; Adolphs, 2001; Haxby et al. 2002; Gallagher & Frith, 2003; Lee et al. 2004). For the purposes of this paper we will present an admittedly oversimplified model of the social information processing network (SIPN) that incorporates many aspects of previously presented models. In our model SIPN is broken down into three basic nodes: the detection node, the affective node and the cognitive-regulatory node. The nodes are based upon similarity of function and developmental timing.

The detection node is dedicated to categorizing a stimulus as social and deciphering its basic ‘social’ properties. The detection node includes the inferior occipital cortex, inferior regions of the temporal cortex, the intraparietal sulcus and a region in the fusiform gyrus known as the fusiform face area, that contribute to categorizing a stimulus as social (Perrett et al. 1982; Haxby et al. 2002). An additional region along the superior temporal sulcus (STS) seems to be involved in the processing of biological movement (Allison et al. 2000; Vaina et al. 2001; Haxby et al. 2002; Puce & Perrett, 2003; Jellemann et al. 2004). Finally, areas in the anterior portion of the temporal cortex have been implicated in social recognition (Adolphs, 2001; Gallagher & Frith, 2003). Collectively these regions carry out perceptual functions such as determining whether a stimulus is animate or not, whether it is a conspecific, what it is doing or intends to do, and what its individual identity is.

Once a stimulus has been categorized as social and its basic properties identified, it is further processed in the affective node. The affective node is composed primarily of regions engaged by reward or punishment. This includes the amygdala, ventral striatum, septum, bed nucleus of the stria terminalis, hypothalamus, and under some conditions, the orbitofrontal cortex. Here the stimulus is imbued with emotional significance. Such determinations as whether the stimulus should be approached or avoided are made in this node. This node also modulates various autonomic and cognitive processes (such as cardiovascular control and attention allocation) in order to organize the systemic response to the social stimulus. Several regions in the affective node have been found to display increased activity when adult subjects viewed emotionally provocative social stimuli such as their own children or romantic partners (Bartels & Zeki, 2001; Leibenluft et al. 2004; Nitschke et al. 2004) individuals perceived as attractive (Aharon et al. 2001; Kampe et al. 2001; O’Doherty et al. 2003), erotic stimuli (Karama et al. 2002; Hamann et al. 2004), or faces displaying fearful or threatening expressions (Whalen et al. 1998; Baird et al. 1999; Killgore & Yurgelun-Todd, 2001; Winston et al. 2002).

Finally, social stimuli undergo processing in a network dedicated to more complex cognitive and mentalistic operations in a node we refer to as the cognitive-regulatory node. Three basic processes occur in the cognitive-regulatory node. The first is perceiving the mental state of other individuals [so-called theory-of-mind (TOM) operations]. TOM processes are driven largely by activity within the paracingulate area or dorsomedial prefrontal cortex (MPFC; Gallagher & Frith, 2003). The second operation is the inhibition of prepotent responses as regulated by the ventral prefrontal cortex. The final operation carried out in the cognitive-regulatory node is the generation of goal-directed behaviors. This necessitates devising a multi-step plan (a sequence of behavioral responses) and selectively executing the plan with the aim of achieving a goal. Goal-directed behavior relies on interactions between the dorsal and ventral prefrontal cortices. In the context of complex
social environments, the ability to control the expression of emotional tendencies in the service of goal achievement represents a particularly important skill. That is, in a social context, individuals need to be able to put the brakes on appetitive, fearful, or angry behavior involving conspecifics (Blair & Cipolotti, 2000), and put immediate motivations into a larger goal-oriented structure. Thus, the cognitive-regulatory node comprises the medial and dorsal prefrontal frontal cortex and portions of the ventral prefrontal cortex, including the orbitofrontal cortex (Mitchell et al., 2002; Gallagher & Frith, 2003; Kringlebach & Rolls, 2003). Note that portions of the ventral prefrontal cortex play a role in both direct affective attributions as well as cognitive representation and regulation of affective responses (Rolls, 2004). Therefore, the ventral prefrontal cortex has been placed in both nodes in our model.

In general our model posits that social stimuli are processed in the three nodes in a sequential manner. However, the nodes do not work in isolation. Rather, they function as an interactive network. A social stimulus that is imbued with a great deal of affective significance will generate feedback from the affective node onto the detection node where further perceptual processing will take place. Likewise efforts to monitor the frame of mind of another individual (as in TOM tasks) requires interactions between perceptual processes carried out in the detection node and cognitive framing that occurs in the cognitive-regulatory node. Similar bi-directional interactions occur between the cognitive-regulatory and affective nodes particularly during inhibitory operations. Thus, there is a primary directionality to the SIPN, from perceptual to affective to cognitive, but the system also functions as a distributed network (see Fig. 1).

**Developmental changes in the SIPN during adolescence**

Several dramatic changes occur in social behavior during adolescence. Three prominent changes are the emergence of sexuality, an increase in peer orientation, and a decrease in parental and family orientation (Larson & Richards, 1994; Halpern et al. 1997, 1998; Steinberg & Morris, 2001). These changes are not unique to humans, as social re-structuring is a near universal developmental phenomenon among socially living species, and is usually associated with emergent sexuality (Pusey, 1987; Monard et al. 1996; Perrin & Mazalov, 1999). Our thesis is that adolescent social re-orientation can be attributed to alterations in processing of social stimuli within the SIPN that occur as a result of hormones, the maturation of neuronal processes, and learning (i.e. the laying down of new local networks within this system as a result of experience).

**The detection node**

Developmental studies of brain processing in humans have generally found that brain systems in the detection node mature quite early in life. For example, newborn infants show face specific patterns of evoked potentials (Halit et al., 2003), and a recent neuroimaging study found that faces elicited activation of the fusiform face area in 2-month-olds (Tzourio-Mazoyer et al. 2002). Children as young as 3 years are able to reliably distinguish biological from non-biological motion, and maximum accuracy is achieved on this measure by the age of 5 years (Pavlova et al. 2001). Thus, the ability to perform categorical and perceptual processes on social stimuli using the detection node of the SIPN matures well before adolescence, and, to our knowledge, no changes in this node have been documented during the adolescent period. Thus, this SIPN theory possesses some level of specificity. Adolescent changes in social cognition are hypothesized to be less closely tied to development than changes in the other nodes. Specifically, in both of the other two nodes, marked changes during the adolescent period are likely to occur as a result of the emergence of gonadal hormones in the brain and the gradual maturation of neural networks.

**The affective node**

The regions that make up the affective node are densely innervated by gonadal steroid receptors and undergo both functional and anatomical reorganization during puberty (Geidd et al. 1997; McEwen, 2001; Osterlund & Hurd, 2001; Romeo et al. 2002; Stevens, 2002). Gonadal steroids alter brain function in a variety of ways, one of which is to regulate other neurotransmitter systems. Many neurotransmitter systems thought to be involved in social responsiveness, such as dopamine, serotonin,
endogenous opioids, oxytocin, and vasopressin are so regulated by gonadal steroids (De Vries et al. 1992; McCarthy, 1995; Rubinow & Schmidt, 1996; Epperson et al. 1999; McEwen, 2001; Osterlund & Hurd, 2001). Gonadal steroids may alter affective processing in more direct ways as well (Baulieu, 1998; McEwen, 2001).

At the behavioral level, there is clear evidence from both human and animal studies that gonadal steroids alter responsiveness to social stimuli. Perhaps the most convincing evidence from human studies is the effects of androgens on sexual responsiveness. In both males and females, higher levels of androgen in circulation result in increased capacity of a social stimulus to elicit urges for sexual approach (Halpern et al. 1997, 1998; Anderson et al. 1999a). Recent studies have also shown that the affective response elicited by male faces varies across the menstrual cycle in women, presumably reflecting the influence of fluctuating levels of gonadal steroids in the central nervous system (Penton-Voak et al. 1999). There is also evidence that circulating gonadal steroid levels may be related to the affective responses elicited by infant-related stimuli in both mothers and fathers (Fleming et al. 1997, 2002; Storey et al. 2000). Although the literature is less consistent, several studies have also found evidence of links between gonadal steroid levels and dominance behavior (Mazur & Booth, 1998; Rowe et al. 2004), and between pubertal status and conflict with parents (Steinberg, 1987). These findings
suggest that gonadal steroids may influence the affective attributions that are made to social stimuli within the SIPN. The anatomical distribution of gonadal hormone receptors, and the functional roles of the different nodes within the SIPN, suggests that the affective node of the SIPN is likely the primary site of action for these hormonal effects.

While human studies have focused largely on detecting correlations between gonadal hormone levels and affective responses to social stimuli, studies have elucidated more specific effects of gonadal hormones on circumscribed brain regions within the SIPN. In general, studies find that gonadal steroids exert a strong influence on such social processes as sexual behavior, maternal behavior, social bonding, and social memory (Fleming & Corter, 1995; Insel, 1997; Hull et al. 1999; Pfaff et al. 2002; Winslow & Insel, 2004). In general, these hormonal effects are mediated by functional changes in the affective node that are indirectly related to gonadal hormone levels in circulation. For example, circulating levels of estrogen can increase oxytocin receptor density within the amygdala, septum, nucleus accumbens, and bed nucleus of the stria terminalis of female rats, and the oxytocin receptor density in these regions may be related to the amount of nurturant behavior bestowed upon infants (Champagne et al. 2001).

A similar pattern of results has been observed in a variety of models (Fleming & Corter, 1995; Hull et al. 1999; Pfaff et al. 2002; Winslow & Insel, 2004). Some studies have also shown that hormonal effects are most important during initial encounters with a novel stimulus, and after behavioral patterns have been established they become relatively independent of hormonal manipulations (Fleming & Corter, 1995). This suggests that the adolescent period may be a particularly important and malleable one in establishing patterns of social behavior. Thus, in general, gonadal hormones have important effects on how structures within the affective node respond to social stimuli, and will ultimately influence the emotional and behavioral responses elicited by a social stimulus during adolescence.

The cognitive-regulation node

The importance of the prefrontal cortex in social information processing has been revealed by studies of individuals with damage to this area. These patients display impairments in social awareness and social decision making and often have psychopathic behavioral tendencies (Anderson et al. 1999b; Blair & Cipolotti, 2000). Moreover, in individuals whose prefrontal regions are damaged early in development, dysfunctions in social cognition seem to be particularly severe and may interfere with the ability to generate abstract knowledge about appropriate social and moral expectations (Anderson et al. 1999b).

Detailed morphometric studies have shown that portions of the prefrontal cortex, including orbitofrontal, ventrolateral, and medial prefrontal regions, do not reach maturity until early adulthood, typically in the late teens or early twenties (Sowell et al. 1999; Casey et al. 2000; Gogtay et al. 2004). Consistent with these observations, behavioral studies demonstrate that performance on tasks that require inhibitory control, such as the go–no go paradigm, does not plateau until mid to late adolescence, and behavioral performance on such tasks is related to both functional and morphometric development in the prefrontal cortex (Casey et al. 2000; Gogtay et al. 2004).

In contrast to the affective node, developmental changes in the cognitive node do not appear to be as closely tied to gonadal hormones as those in the affective node. The development that does occur in the cognitive node is a result of increased myelination and pruning of existing synaptic networks. This developmental process occurs throughout the brain but is protracted in the prefrontal regions (Casey et al. 2000; Gogtay et al. 2004). Since pruning involves survival of local network connections through competitive, use-dependent strengthening, it is a developmental process which is similar in many ways to learning. The protracted nature of pruning within prefrontal regions may, therefore, facilitate the incorporation of experience into neurobiological development. Thus, in contrast to developmental changes in the affective node, developmental changes within the cognitive node are likely to be slow, iterative, and independent of hormonal status. However, interaction between the affective and cognitive nodes may result in secondary effects of hormones within the cognitive node.
What can neuroimaging tell us about developmental alterations in the SIPN?

The fact that alterations in social behavior occur during adolescence is incontrovertible. Further, like all behavioral shifts, these alterations must be mediated within the central nervous system. The model presented above is based upon existing knowledge of development, social behavior, and brain physiology. It represents, we believe, a reasonable outline of how behavioral development may be implemented in the brain. According to our model, the surge of gonadal hormones during puberty alters the emotional processing of social stimuli, as instantiated in the affective as opposed to the detection or cognitive-regulatory nodes. Concurrently, although on a slightly different time scale, maturational changes and experience alter the brain’s ability to regulate responses to social stimuli and integrate them into a larger framework. However, this outline is speculative, and requires experimental confirmation, especially in humans. In particular, the details of exactly what changes occur in the SIPN and how they are brought about remain poorly understood.

We believe that non-invasive neuroimaging techniques which permit probing of neural activity while subjects engage in social information processing offer an excellent new means for directly assessing the brain changes that we postulate to occur during human adolescence.

For example, a recent study from our laboratory highlights differences between adolescents and young adults in activation patterns in both the affective and the cognitive-regulatory nodes while processing social stimuli. While passively viewing faces with fearful emotional expressions, adolescents exhibited greater activation than adults in the amygdala, orbitofrontal cortex, and anterior cingulate. This finding suggests that when attention is unconstrained, adolescents may be more sensitive to the emotional properties of a social stimulus. However, when we asked subjects to switch their attention between a salient emotional property (how afraid does it make you feel?) and a non-emotional property (how wide is the nose?), we found that adults, but not adolescents, were able to selectively engage and disengage the orbitofrontal cortex (Monk et al. 2003). These findings highlight interacting, developmental alterations in both the affective and cognitive-regulatory nodes of the SIPN (see Fig. 2).

Based on the developmental literature, we would expect several other changes to take place within the affective node of the SIPN during the adolescent period. For example, peers should be perceived as more rewarding in middle and late adolescence than in late childhood, and the reverse pattern should emerge with parents. At a neural level we would predict that as adolescence progresses, cues of peers would induce increased activation in the affective node. This enhanced affective processing would also probably exert stronger influences on attention and memory processes which could be probed with tasks such as the dot probe, which assesses attentional engagement (Mogg et al. 1998; Pine et al. in press) and the N-back, which assesses working memory (Pochon et al. 2002) inside the scanner.

Likewise, a similar pattern would likely emerge in neuronal activations induced by cues of potential mating partners. That is, opposite-sexed peers should be afforded greater approach value and cognitive resources than same-sexed peers (at least in heterosexual youth). As outlined above, recent neuroimaging studies have
shown these processes can be studied within the fMRI environment (Bartels & Zeki, 2000; Aharon et al. 2001; Kampe et al. 2001; O'Doherty et al. 2003; Leibenluft et al. 2004; Nitschke et al. 2004), involve structures within the affective node of the SIPN, and may be greatly modified by the presence of gonadal hormones.

Another phenomenon that has been noted in the developmental literature is a hypersensitivity to acceptance and rejection by peers during adolescence (Brown, 1986; O'Brien & Bierman, 1988; Larson & Richards, 1994). Neuroimaging tasks could be designed to probe responses to simulated instances of social acceptance or inclusion, and social rejection or exclusion (Eisenberger et al. 2003). We hypothesize that activations associated with motivation, self-esteem, acceptance and rejection in a social context will be augmented in the affective node of the SIPN during the adolescent period, and extreme activation may be related to psychopathology.

While the tasks described above are tailored to assess alterations in the processing of social stimuli within the affective node, we predict that changes occur in the cognitive-regulatory node as well. As mentioned above, we have begun to use event-related fMRI to probe adolescents’ responses to faces with varying emotional expressions. Our initial findings have indicated that, when exposed to hostile or fearful faces, young adolescents are less able to modulate activity within the affective node of the SIPN under varying task demands than are adults (Monk et al. 2003). These findings suggest that developmental changes occur in the ability to regulate responses to social stimuli – a process we have ascribed to maturation of the cognitive-regulatory node. We and others have also observed sex-specific changes in the interaction between the affective and cognitive nodes in face emotion tasks (Killgore et al. 2001; McClure et al. 2004). Such developmental changes may reflect interactions between affective and cognitive nodes across development.

Finally there is a surprising lack of literature in both the neuroimaging and behavioral domains on adolescent changes in mentalizing or TOM tasks. Much of the developmental work in the TOM literature has focused on early stages of development (Frith & Frith, 2003). The ability to take someone else’s perspective first emerges in early childhood. However, several recent imaging studies of mentalizing have implicated a region in the dorsomedial cortex as being central to this ability (Gallagher & Frith, 2003). This region continues to undergo developmental changes well into late adolescence or early adulthood (Sowell et al. 1999; Casey et al. 2000), implying that subtle changes in the ability to mentalize may continue to develop during adolescence. Recently, fMRI tasks have been developed in which the subject directly interacts with another individual, or believes he or she is interacting with another individual (McCabe et al. 2001; Rilling et al. 2002; Eisenberger et al. 2003). These tasks involve complex social interactions such as cooperation, predicting the performance of another individual, and possibly empathy, all of which represent components of mentalizing and TOM. Conducting neuroimaging studies with such tasks on adolescents may reveal subtle and late-emerging changes in the neuronal systems that are engaged during such complex social processes.

Brain development, SIPN, and affective disorders

The incidence of affective and specific anxiety disorders rises dramatically during adolescence (Pine et al. 1998, 2002; Hankin & Abramson, 2001; Costello et al. 2002). This includes depression, social phobia, and panic disorder, as a result this time period is viewed as potentially formative in the developmental course of mood and anxiety disorders (Pine et al. 1998, 2002). While a number of factors probably contribute to the emergence of psychiatric symptoms during adolescence, several lines of investigation indicate that changes in the processing of social stimuli within the SIPN may be particularly important. First, adolescence clearly represents a period of heightened emotional responsiveness to social stimuli and socially related events (Larson & Richards, 1994; Rudolph & Hammen, 1999; Steinberg & Morris, 2001). The large-scale changes in social behavior that occur during adolescence are mediated by strong emotional motivations. In some individuals the strong emotions cross the line from normalcy into psychopathology. The intensified emotional potency of interpersonal interactions during...
this period may result in heightened sensitivity to negative interpersonal events which may be particularly relevant to psychopathology (Rudolph & Hammen, 1999; Hankin & Abramson, 2001). Indeed, rejection by romantic partners and peers is a strong predictor of behavioral and psychiatric difficulties during adolescence, including initial depressive episodes (Hecht et al. 1998; Monroe et al. 1999); substance use and externalizing behavior (Pristin & La Greca, 2004); and school adjustment (Zettergren, 2003). Difficulties in social relationships also represent a major risk factor for adolescent suicide and suicide attempts (Gould et al. 2003). Interestingly, suicidality is more often related to problems with parents during early adolescence, and to problems with romantic relationships during later adolescence (Gould et al. 2003), suggesting that this phenomenon may parallel changes in the affective significance placed upon different social exchanges within the SIPN. Moreover, there is some evidence that the association between stress and emotion is particularly strong in adolescent girls (Pine et al. 2002). This raises questions as to the role of hormones and their impact on the affective node in this process. Thus, the regions within the SIPN are involved not only in social change but can also be the focal point of adolescent psychopathology.

A second reason that neural processing of social stimuli may be particularly relevant to psychopathology during adolescence is the added stress that is placed on the SIPN during a period of social re-orientation. The importance of a social support network as a buffer against both social and non-social stressors, such as academic achievement, has been well documented in the literature. This is true throughout development, but adolescence represents a period in which the social network itself changes. During adolescence, family often becomes a less prominent factor in the lives of children, while peers become more so (Steinberg & Morris, 2001). Thus, in order to maintain a social support network, adolescents need to integrate themselves into a new network. This social integration relies heavily on the SIPN. Adolescents who are compromised in their ability to perceive social processes and ascribe appropriate emotional significance to social events, or who have difficulty mentalizing or regulating their behavior in a social context, may end up with impoverished social networks during this transitional period, and therefore be more susceptible to the pathogenic effects of stress.

Another aspect of SIPN development that may be particularly relevant to emergent psychopathology during the adolescent period is the apparent mismatch in the development of the affective and cognitive-regulatory nodes. While rapid and dramatic changes occur in the affective node of the SIPN at the onset of puberty, resulting in powerful emotional urges for sexual behavior independence and the formation of new social bonds (Steinberg & Morris, 2001), the maturation of the cognitive node lags behind by several years (Beauregard et al. 2001; Casey et al. 2000; Ochsner et al. 2002; Schaefer et al. 2002). Thus, in individuals who have powerful emotional responses to social stimuli the ability to regulate, contextualize, plan, or inhibit newly emergent and highly motivated behavior in a context-appropriate manner is far from mature. This mismatch may lead to behavioral difficulties for some individuals.

Finally, as noted above, the functioning of the SIPN during adolescence may provide new insights into the emergence of gender difference in mood and anxiety disorders. The approximate 2:1 female preponderance of mood and anxiety disorders first emerges during adolescence (Angold et al. 1998; Cyranowski et al. 2000; Hankin & Abramson, 2001; Kessler et al. 2003), and may relate to gender differences in social processing or stress responsivity that also change during adolescence (Killgore et al. 2001; Pine, 2001; McClure et al. 2004). Gender differences in social behavior are apparent throughout the lifespan, but many of these differences become accentuated during puberty. Females tend to rely more on social support mechanisms to buffer stress, and may be more sensitive to social rejection than are males (Rudolph & Hammen, 1999; Cyranowski et al. 2000; Taylor et al. 2000; Stroud et al. 2002). Recent epidemiological studies, however, have revealed that the relationship between gender and susceptibility to social stress may be more complicated and one may need to take into consideration the kind of social stress encountered by the different genders (Kendler et al. 2001). These findings suggest that under many
conditions, compromised functioning of the SIPN may be more detrimental to the psychological well-being of women than of men, and that these gender differences may emerge during adolescence.

Interestingly recent studies have reported abnormalities in SIPN structures in adolescents suffering from mood or anxiety disorders. Morphometric studies have found anatomical alterations in the superior temporal gyrus, ventral prefrontal cortex, and amygdala of adolescents with a variety of mood and anxiety disorders (De Bellis et al. 2002a, b; Blumberg et al. 2003; DelBello et al. 2004). Alterations have also been reported in frontal white-matter volume and in choline levels in the orbitofrontal cortex of depressed adolescents (Steingard et al. 2000, 2002). In addition to these static differences, functional abnormalities in amygdala responsiveness to social stimuli has also been observed in depressed and anxious adolescents (Thomas et al. 2001). Given the extensive interconnections between the amygdala and orbitofrontal regions one might also expect to see alterations in the ventral frontal regions as well. Thus, we hypothesize that abnormalities in amygdala function, as well as abnormalities in other structures that the SIPN comprises will impact social cognition and behavior during adolescence, resulting in aberrant socio-emotional responding, compromised perception of social stimuli, difficulties with complex social behavioral processes and will often be an important vector for psychopathology in some individuals (Tse & Bond, 2003).

Finally, there is a critical need for development of novel fMRI tasks that engage ecologically valid aspects of social regulation. At a behavioral level we hypothesize that adolescent females with mood and anxiety disorders will exhibit particularly strong negative reaction to perceived social rejection on such tasks. Moreover, these responses should be reflected in the affective node.

CONCLUSIONS

Many changes occur in the social behavior of adolescents. These behavioral changes are a reflection of alterations in neuronal processes that are brought about by hormonal restructuring, maturation and learning. Since recent neuroimaging studies have begun to reveal the brain systems involved in decoding and integrating social information, the time is ripe to probe the changes that take place in this network during the adolescent period. Such studies are likely to yield important new insights into the mechanistic changes in neuronal architecture that drive large-scale changes in adolescent behavior. Furthermore, the relationship between affective disorders and dysfunctional social behavior strongly suggests an important relationship between social information processing systems and psychiatric illness—particularly mood and anxiety disorders. While the model presented here is at present overly broad and at places probably also oversimplified, we believe it sets up a theoretical perspective from which more refined and empirically based observations can emerge.

ACKNOWLEDGMENTS

The authors thank Dr Lawrence Steinberg for providing useful commentary on an earlier version of this manuscript; Dr Christopher Monk and Lidia Kibiuk for assistance with the figures; and three anonymous reviewers for providing substantive and helpful critiques. This manuscript is the product of the authors’ review of existing literature and was entirely supported by NIMH intramural funding.

DECLARATION OF INTEREST

None.

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