Chapter 3

Adolescent brain development and the development of mentalizing

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Adolescence is a time marked by important biological and psychosocial changes. Whilst in the past, adolescence was typically seen as a time of increased risk for the development of psychopathology, there is now increasing consensus, particularly within the mentalizing approach to development, that adolescence also provides new opportunities for growth and resilience and is associated with marked plasticity (Luyten, Campbell, Allison, & Fonagy, 2020). This chapter is written with this double focus in mind in an attempt to redress the balance and consider adolescence as a pivotal stage in development in explaining both risk and resilience.

The focus in this chapter will be on neurobiological findings concerning the development of mentalizing, our capacity to understand oneself and others as motivated by intentional mental states. We discuss neuroimaging evidence suggesting that adolescence is associated with the functional and structural reorganisation of three distinct, although highly related, biobehavioural systems, i.e. the stress-regulation, attachment/reward and mentalizing systems, as well as other related neural systems such as systems for cognitive control. These reorganisations occur at a time when the establishment of new and more complex relationships and a sense of agency and autonomy are two central and major developmental tasks (see Figure 1).

From an evolutionary perspective, the following three basic biobehavioural systems have evolved to enable humans to adapt to our complex social environment: (a) a system that has developed to deal with distress following threat (the stress/threat system); (b) a system that is centrally involved in producing rewarding experiences associated with the formation of interpersonal relationships (i.e., infant–parent, parent–infant, romantic attachment, and other

attachment relationships) on the one hand, and experiences of agency and autonomy on the other (the reward system); and (c) a mentalizing or social cognition system, which underpins the human species-specific capacity to understand oneself and others in terms of intentional mental states (e.g., feelings, desires, wishes, attitudes, and values), a quintessential capacity in our complex interpersonal world (Fonagy & Luyten, 2018; Luyten & Fonagy, 2018).

The implications of the reorganisations in these three biobehavioural systems in adolescence, and their role in explaining both vulnerability and resilience, are vast and deserve our attention. They are essential to understand for every clinician working with adolescents. As we will show, research into the neurobiological changes that affect the neural circuits underpinning social cognition during adolescence has important implications for clinical work with adolescents, and particularly for those suffering from mental health issues. This chapter will guide us through this significant research.

We will discuss developmental cascades set in motion as a result of excessive and/or age-inappropriate stress, typically resulting in impairments in reward sensitivity and in the capacity for mentalizing (see Figure 1). Together, these impairments affect normative developmental tasks, typically leading to vicious cycles marked by a disturbed capacity for relatedness and agency/autonomy and associated mentalizing problems. Yet, throughout, we emphasise that even marked 'impairments' in mentalizing and attachment are in the eye of the beholder. From the perspective of the young person, they represent attempts at adaptation to a specific (interpersonal) environment. For instance, in an environment characterised by marked emotional neglect and violence, the use of instrumental aggression can be seen as an understandable strategy to ensure status and, in extreme circumstances, survival (Fonagy & Luyten, 2018). Similarly, for young people growing up in a context of abuse, the inhibition of mentalizing, typically combined with high levels of epistemic mistrust in others, are an understandable response to protect the self from further abuse and disappointment (Luyten,

Campbell, Allison, et al., 2020). In this context, we also warn against overly simplistic interpretations of neurobiological findings implying a direct parallel between findings that suggest a reorganisation of neurobiological systems involved in emotion regulation in adolescence and typical impulsive and risk-taking behaviours in adolescence.

The stress system: dealing with adversity

We begin our journey through the rapidly accumulating literature on neurobiological changes in adolescence by a discussion of findings concerning the impact of stress and adversity on the developing adolescent brain. Most, if not all, psychological disorders are best conceptualised as developmental, stress-related disorders, with elevated and/or age-inappropriate stress playing a major role in their onset and perpetuation. In adolescence, as noted, major changes in sociocultural expectations occur leading to increased stress (Auerbach, Admon, & Pizzagalli, 2014; Davey, Yücel, & Allen, 2008; Forbes & Dahl, 2012; Spear, 2000) in the domains of both relatedness and agency/autonomy (see Figure 2). On the one hand, adolescence entails that peer and romantic relationships become increasingly important, which is expressed, for instance, in increased rejection sensitivity. On the other hand, demands for achievement, reflected in increased sensitivity to failure, intensify. The emergence of pubertal sexuality and (relational and instrumental) aggression, and associated bodily changes, provide further challenges in both domains.

The human biobehavioural stress system is a complex system of neural structures that is involved in detecting, integrating, and responding to threat. While the hypothalamic—pituitary—adrenal (HPA) axis system and the sympathetic nervous system make up the core structures of the stress system, the amygdala, hippocampus, and areas in the prefrontal cortex (PFC), including the anterior cingulate cortex, orbitofrontal cortex, and medial PFC (MPFC),

also play import roles in this network (McEwen, 2007). Together, these structures serve *allostasis*, the capacity to continuously adapt to ever-changing circumstances (McEwen, 2007). When this fails, *allostatic load* ensues and the individual's self-regulatory capacities increasingly begin to fail (McEwen, 2000).

Research indicates that there is an even broader network of physiological systems responsible for establishing and maintaining allostasis, serving the fight/flight/freeze response in the face of acute stress (Gunnar & Quevedo, 2007; McEwen, 2007). This network includes the autonomic nervous system and the metabolic system, gut, kidneys, and immune system, each with their relatively distinct biomediators (e.g., cortisol, sympathetic and parasympathetic transmitters, metabolic hormones, and cytokines, respectively). These findings emphasise the embodied nature of adversity, and the intertwining of physical and mental health, particularly in those individuals with a history of early adversity.

Consistent with these views, there is now relatively good evidence to suggest that early adverse experiences may result in permanent alterations in the stress system associated with an earlier age of onset of psychopathology, greater symptom severity, higher levels of comorbidity, a greater risk for suicide and a poorer response to treatment across different psychiatric conditions (Teicher & Samson, 2013). High levels of stress, particularly during 'critical time windows' (Heim, Plotsky, & Nemeroff, 2004) in which the stress system is highly sensitive to environmental factors, typically result in HPA-axis overactivity (i.e., a constant state of fight/flight) and a consequent increased vulnerability to stressors throughout life (Kertes, Gunnar, Madsen, & Long, 2008). In humans, this critical time window in which the HPA axis is particularly sensitive to programming effects extends into early adulthood (Lupien, McEwen, Gunnar, & Heim, 2009). This leads to a cascade of physical and psychological consequences given the intricate relation between the stress system and other key biological and psychosocial systems, such as problems with sleep and attention,

difficulties with motor control, coordination, learning difficulties and social and relational problems, as well as problems related to the immune system, pain-regulating systems, the metabolic system, and the reproductive system (Eiland & Romeo, 2013; Lupien et al., 2009). Specific genetic variation may make individuals more sensitive to environmental factors ('differential susceptibility hypothesis') (Ellis, Boyce, Belsky, Bakermans-Kranenburg, & van Ijzendoorn, 2011).

Core structures of the stress system, including the amygdala and hippocampus, undergo major structural and functional reorganisation in adolescence: the amygdala and hippocampus increase in volume and reach their peak volume in adolescence, and their central functional role in adolescence is also demonstrated by studies suggesting that these areas show greater activation, compared to children or adults in various stress tasks. The PFC, in turn, is subject to cortical thinning during adolescence as a result of synaptic pruning and programmed cell death (Mutlu et al., 2013; Shaw et al., 2008).

Similarly, adolescence is associated with a considerable increase in HPA axis reactivity to stress, compared to children and adults (Casey, Getz, & Galvan, 2008). Adolescents have been shown to have both higher basal stress levels and heightened reaction to stress, and social rejection and academic stressors in particular (Masten et al., 2009; Sebastian, Viding, Williams, & Blakemore, 2010; Sebastian et al., 2011), consistent with the greater demands for autonomy, agency and achievement and the growing importance peer and romantic relationships (Auerbach et al., 2014) (see also Figure 2).

There is now also good evidence to suggest that young people who are at increased risk for the development of psychopathology also unwittingly generate in part their own stressful environment (i.e., active stress-generation or evocative person-environment effects) (Hammen, 2005). Several unhealthy and maladaptive behaviours such as smoking and unhealthy eating, risky sexual behaviours, including (sexual) revictimisation, self-harm, and

violence in (intimate) relationships (Afifi et al., 2009; Anda et al., 2006) further contribute to a vicious cycle leading to increased vulnerability to psychopathology.

The reward system: regulating attachment and agency/autonomy

Two areas of reward are central in adolescence: relationships, with a developmental shift towards a growing importance of peer and romantic relationships (including sexuality), and agency/achievement (including instrumental aggression) (see Figure 2) (Blatt, 2008; Luyten, 2017). Both areas are often intertwined, as social status often increases relational attractiveness and vice versa. Yet, at least in Western cultures, boys place slightly more emphasis on agency/autonomy, while in girls there is a somewhat greater emphasis on attachment (Luyten & Blatt, 2013; Spear, 2000).

A mesocorticolimbic dopaminergic system underpins experiences of reward in the human brain. The mesolimbic pathways originate from the ventral tegmental area and project to ventral striatal regions and the hippocampus and amygdala. A mesocortical pathway consists of projections to the PFC and anterior cingulate cortex (Nestler & Carlezon, 2006; Pizzagalli, 2014; Russo & Nestler, 2013; Spear, 2000). Biomediators in these pathways involve dopamine, oxytocin, vasopressin, opioids and cannabinoids. They mediate feelings of acceptance, support, agency and validation on the one hand, and the emotional pain associated with social loss and rejection, which is increased in adolescence, particularly in women (Hsu et al., 2015; Spear, 2000).

The reward system plays a central role in the development and functioning of the stress system (Hostinar, Sullivan, & Gunnar, 2014; Strathearn, 2011; Swain et al., 2014). Developmentally, the attachment behavioural system is activated when faced with threat, leading to proximity seeking with an attachment figure. The capacity of attachment figures to offer marked, mirroring responses will help the child to develop the capacity to downregulate

stress and to trust others as a source of comfort and support. The reward system underpins these affective responses as secure attachment experiences buffer the effects of stress in early development, resulting in 'adaptive hypoactivity' of the HPA axis (Gunnar & Quevedo, 2007). Hence, for children growing up in secure attachment contexts, others become rewarding and, at the same time, feelings of agency and autonomy develop and are equally experienced as rewarding. Hence, the capacity for self-regulation and co-regulation mutually reinforce each other. By contrast, in children growing up with unavailable, unresponsive or abusive attachment figures, capacities for co-regulation and self-regulation are typically impaired. These children either begin to excessively rely on attachment hyperactivating strategies in an attempt to find downregulation of distress, or attachment deactivating strategies, leading to so-called compulsive autonomy (i.e., the belief that one has to be able to deal with distress without the help of others). For insecurely attached individuals, relying on others is not rewarding, as there is always the underlying belief that others will not be available. On the biological level, insecure attachment is therefore associated with increased vulnerability for stress, as expressed in dysfunctions of the stress system (Auerbach et al., 2014; Pizzagalli, 2014; Strathearn, 2011).

The abovementioned biomediators are centrally involved in these processes. Oxytocin, for instance, has been shown to increase affiliative behaviour when faced with distress, and thus effective co-regulation of stress with others. Oxytocin furthermore reduces behavioural and neuroendocrinological responses to stress (Neumann, 2008) via downregulation of the HPA system (Feldman, 2017), and enhances mentalizing and trust in others (Domes, Heinrichs, Michel, Berger, & Herpertz, 2007; Heinrichs & Domes, 2008). Importantly, these positive effects of oxytocin seem limited to in-group members, whereas increased levels of oxytocin appear to lead to increased distrust, and less cooperative behaviour in relation to out-group members, even in securely attached individuals (Bartz, Zaki, Bolger, & Ochsner, 2011).

Individuals with an insecure attachment history show decreased basal oxytocin levels and in these individuals oxytocin administration leads to decreased trust and cooperative behaviour, and increased cortisol response to stress, even in relation to in-group members (Bartz et al., 2011; Feldman, 2017). Hence, attachment experiences are not perceived as rewarding, and by contrast intensify distress. These findings are critically important for clinicians' understanding of their encounter with insecurely attached young people (and particularly those with a history of abuse). For these young people relationships, including the therapeutic relationship, are not experienced as rewarding, but as a source of distress. At best, relationships with others (including mental health professionals) are experienced as ambivalent, and there is often a thin line between their wish and need to be understood and cared for, and overstimulation of the attachment system (and the ensuing loss of mentalizing associated with such overstimulation discussed below). Hence, clinicians may easily overwhelm the young person with warmth and support which, however well-intentioned, is experienced as aversive and even dangerous. Mentalization-Based Treatment therefore typically titrates interventions to take into account the negative effects of attachment activation on mentalizing (Bateman & Fonagy, 2019).

The reorganisation of the reward system in adolescence might have direct behavioural effects in adolescence, but the precise mechanisms and their effects on adolescent behaviour are still relatively poorly understood. Research findings based on studies with both animals and humans, do suggest, however, that adolescence is characterised by the lowest levels of dopamine in striatal regions and the highest levels of dopamine in prefrontal regions, resulting in a so-called "mini-reward deficiency syndrome" in adolescence (Spear, 2007). Any clinician working with adolescents is familiar with this 'syndrome': adolescents may be easily bored and, at the same time, frustration of their strong needs for belongingness and/or achievement and the status that is associated may give rise to intense feelings of rejection and failure, or a

combination of both, and subsequent frustration and aggression. These experiences may lead to different compensatory behaviours such as risk-taking, substance abuse and/or oppositional behaviour, which provides an explanation for the high comorbidity observed between internalising and externalising problems in adolescence (Davey et al., 2008; Spear, 2000).

As noted, the precise mechanisms involved remain elusive. It has been suggested that the hypo-responsivity to reward in adolescence may be evolutionarily adaptive: the low incentive value of reward supports adolescents' explorative tendencies, autonomy strivings and relational and sexual desires. In combination with high levels of phasic dopamine in response to reward, this might indeed explain the normative 'Sturm und Drang' typically associated with adolescence (Davey et al., 2008; Luciana, 2013). Yet, the observed impairments in reward sensitivity in adolescence might also be the consequence of downregulation through high levels of dopamine in the PFC (which inhibit mentalizing, see also below), resulting from increased stress in adolescence (Pizzagalli, 2014; Spear, 2000). Furthermore, increased representational capacities in adolescence may lead rewards such as love and status to be increasingly experienced as temporally distant as adolescence is typically associated with a psychosocial moratorium, limiting the adolescent's feelings of agency and autonomy.

Particularly in vulnerable young people, a greater need for reward, together with the perception that reward experiences are temporally distant, may further increase distress. In combination with ensuing mentalizing impairments, discussed below, this may lead to compensatory behaviours (e.g., substance abuse, violence, or social withdrawal). Because this further decreases the incentive value of relationships with others who may provide corrective experiences, a vicious cycle ensues, characterised by social isolation or excessive reliance on peers that struggle with similar issues. In these circumstances, it often becomes extremely difficult to be able to re-calibrate one's mind in interaction with others.

As noted, this may be particularly difficult in those young people with a history of adversity. Hence, the absence of resilience, associated with social learning driven by epistemic trust, may be key, particularly if increased social pressures for agency and relatedness are not balanced by an environment that supports these two fundamental developmental tasks, creating a toxic mixture for many young people (Debbané, 2015; Escofet, 2012). Yet, from an evolutionary perspective, the insecure attachment strategies that young people develop under such circumstances are better seen as attempts at coping with the (perceived) unavailability, unresponsiveness, or intrusiveness of attachment figures and the broader sociocultural context more generally (Belsky & Fearon, 2008; Ein-Dor, Mikulincer, Doron, & Shaver, 2010; Simpson & Belsky, 2008). Hence, to put it somewhat provokingly, the brain adapts itself to a specific environment in order to optimise survival and social functioning. Although this attempt at adaptation may be far from optimal from a normative perspective, once a certain minimally satisfying state of allostasis has been reached, the brain stops any further iterations in attempting to find a better solution. It is here that psychosocial interventions may be useful. This brings us to the importance of mentalizing in adolescence and in any intervention that aims at behavioural change in young people.

The mentalizing system: understanding oneself and others

Although it is largely unknown why humans developed the capacity for mentalizing, it represented a major leap forward as this capacity enables complex communication and collaboration typical of human social systems. Furthermore, the development of advanced capacities for mentalizing, which are largely or completely absent in most other animal species, also enabled the capacity for self-consciousness and even further, the capacity for imagination and, as a consequence, for transcending physical reality (see Davey et al., 2008).

Each of these capacities, however, also paradoxically increases vulnerability for psychopathology. Self-consciousness also led to the emergence of so-called self-conscious emotions (e.g., embarrassment, regret, shame, and guilt) which, although in essence adaptive, may become maladaptive when chronic and/or excessive. Moreover, the capacity for imagination not only allows humans to envision an ideal state of the self, but also to become painfully aware of the discrepancy between the actual and ideal self-states. More generally, the quintessential role of social embeddedness in the fabric of human social organisation may lead to feelings of utter isolation and loneliness in those who lack skills required for human collaboration and communication (Luyten, Campbell, & Fonagy, 2020).

These issues are central during adolescence (Crone & Dahl, 2012), which might explain, at least in part, the increase in the prevalence of psychopathology in this developmental stage. Hence, while changes in the brain, and the emergence of full mentalizing throughout adolescence, allow for increasingly complex communication, collaboration and imagination, they also increase vulnerability in biologically and/or environmentally less fortunate young people (Sharp, Vanwoerden, & Wall, 2018).

As is well-known, mentalizing is multi-dimensional (see Table 1). From a neural perspective, the different dimensions of mentalizing are subserved by a complex set of neural systems (Luyten & Fonagy, 2015).

As with the stress and reward system, neural systems involved in mentalizing undergo significant reorganisation in adolescence. Synaptogenesis (the formation of new synapses between neurons), synaptic pruning (the eradication of unused synapses) and myelination (the myelin-coating of neurons which enhances the transmission speed of electrical impulses) (Blakemore, 2008, 2018) lead to significant structural and functional changes in mentalizing regions. For instance, both the synaptic reorganisation, in which excess synapses are being eliminated, and the increase in myelination of remaining synapses, most likely explain the

decrease in grey cortical matter and increase in white cortical matter observed in fMRI studies in adolescents.

It is assumed that these processes increase the efficiency of brain networks implicated in mentalizing, and particularly the involved cortical regions (Blakemore, 2008, 2018), which in turn are thought to foster the development of social cognition in adolescence, enabling the development of a more differentiated and integrated sense of self and identity on the one hand, and relatedness on the other (Fonagy & Luyten, 2016; Sharp et al., 2018).

However, it is hypothesised that in the midst of these cortical changes, mentalizing skills of adolescents may be temporarily disrupted, also referred to as the mentalizing or pubertal 'dip' in social cognition (Blakemore, 2008). These findings have led to an ongoing discussion whether the development of social cognition in adolescence follows a linear (with levels of social cognition simply increasing with age) or a non-linear trajectory (with levels of mentalizing actually decreasing in mid-adolescence because of the reorganisation of mentalizing networks). Some studies that focused on externally-based mentalizing indeed found such a "dip" in the accuracy in mentalizing (Carey, Diamond, & Woods, 1980; Diamond, Carey, & Back, 1983; Tonks, Williams, Frampton, Yates, & Slater, 2007) or reaction time in mentalizing tasks (McGivern, Andersen, Byrd, Mutter, & Reilly, 2002) in mid-pubertal adolescents (12 to 13 years old). This pubertal dip in mentalizing has been be related to hormonal changes.

Studies suggesting a pubertal dip in mentalizing because of the neural reorganisation of brain areas involved in mentalizing are appealing because they may provide a straightforward explanation for the observed increase in both internalising and externalising problems in adolescence, and particularly the increase in acting out behaviour, such as self-harm, risk-taking behaviours, substance abuse, and violence in general (Sharp et al., 2018).

However, research in this area is far from conclusive as many other studies on externally-based mentalizing found a systematic linear increase in mentalizing skills across adolescence in similar face and emotion recognition tasks as used in research suggesting a pubertal dip (van Rooijen, Junge, & Kemner, 2018; Vetter, Drauschke, Thieme, & Altgassen, 2018; Vetter, Leipold, Kliegel, Phillips, & Altgassen, 2013) with no indication of age, pubertal or peer-bias effects (i.e. that adolescents may simply be better in recognising faces of peers simply because they interact more with peers). Additionally, there is no strong evidence for a pubertal dip in research on other mentalizing dimensions, such as in the development of Theory-of-Mind (Keulers, Evers, Stiers, & Jolles, 2010; Vetter et al., 2013), perspective taking (Choudhury, Blakemore, & Charman, 2006) and empathy (van Rooijen et al., 2018), with studies consistently suggesting a gradual linear increase in mentalizing skills. However, more longitudinal studies are needed before firmer conclusions can be drawn, particularly as changes in mentalizing in adolescence might be heavily influenced by changes in cognitive functioning (including executive functioning and effortful control). Indeed, studies in this area suggest increasing cognitive and impulse control with age during adolescence.

Hence, although the jury is still out on the course of the development of mentalizing in adolescence, the rapid changes that are characteristic of adolescence both in the domains of relatedness and autonomy/achievement present considerable challenges to mentalizing in young people, even in normative development. Hence, with increasing arousal, it should not be surprising that adolescents may often lose the ability for controlled mentalizing, and often switch to more rapid, automatic mentalizing, and eventually non-mentalizing modes of thinking about the self and others.

This seems particularly true for those young people with a history of early adversity, as adversity not only undermines mentalizing capacities, but also the capacity for epistemic trust and thus the capacity to re-calibrate one's mind when faced with new challenges (Luyten,

Campbell, Allison, et al., 2020; Luyten & Fonagy, 2019). As a result, these young people are increasingly cut off from corrective experiences and might end up in complete social isolation and loneliness and/or seek social recalibration of the mind with peers that suffer from similar problems. With the current availability of social media, the latter strategy in particular may rapidly spiral out of control. The inability to make sense of changes that come with the advent of adolescence may lead to excessive mentalizing (hypermentalizing) and/or the avoidance of mentalizing (hypomentalizing). Both can be seen as a defensive strategy to avoid thinking about the painful nature of these experiences and both may explain in part the mini reward deficiency syndrome characteristic of adolescence. Hence, yet again, from the perspective of the adolescent, these represent adaptation strategies, whilst the outside world typically tends to consider these strategies as pathological and abnormal.

Conclusions

Although there is no one-to-one relation between brain functioning and behaviour, knowledge of the adolescent brain is essential in our understanding of their subjective experience. Indeed, it is the essence of the mentalizing approach that mentalizing allows us to understand the experience of the other, and thus see the world from their perspective. Yet research on mentalizing has also taught us that this capacity is limited and that misunderstanding of others is commonplace. This is probably especially true for any adult trying to understand the adolescent and his or her brain. While adolescents often have major impairments in understanding how brain and environment, and their interactions, determine their sense of self and others, it behoves us to show modesty when it comes down to our own capacity to understand these influences in a human being that sees the world often very differently from how we do.

References

- Afifi, T. O., MacMillan, H., Cox, B. J., Asmundson, G. J. G., Stein, M. B., & Sareen, J. (2009). Mental health correlates of intimate partner violence in marital relationships in a nationally representative sample of males and females. *Journal of Interpersonal Violence*, 24(8), 1398-1417. doi:10.1177/0886260508322192
- Anda, R., Felitti, V., Bremner, J., Walker, J., Whitfield, C., Perry, B., . . . Giles, W. (2006). The enduring effects of abuse and related adverse experiences in childhood. *European Archives of Psychiatry and Clinical Neuroscience*, 256(3), 174-186.
- Auerbach, R. P., Admon, R., & Pizzagalli, D. A. (2014). Adolescent depression: Stress and reward dysfunction. *Harvard Review of Psychiatry*, 22(3), 139-148. doi:10.1097/hrp.00000000000000034
- Bartz, J. A., Zaki, J., Bolger, N., & Ochsner, K. N. (2011). Social effects of oxytocin in humans: Context and person matter. *Trends in Cognitive Sciences*, *15*(7), 301-309. doi:10.1016/j.tics.2011.05.002
- Bateman, A., & Fonagy, P. (2019). *Handbook of mentalizing in mental health practice* (2nd ed.). Washington, DC: American Psychiatric Press.
- Belsky, J., & Fearon, P. R. M. (2008). Precursors of attachment security. In J. Cassidy & P. R. Shaver (Eds.), *Handbook of attachment theory and research* (2nd ed., pp. 295-316). New York, NY: Guilford Press.
- Blakemore, S. J. (2008). The social brain in adolescence. *Nature Reviews. Neuroscience*, 9(4), 267-277. doi: 10.1038/nrn2353
- Blakemore, S.-J. (2018). *Inventing ourselves: The secret life of the teenage brain*. London, UK: Doubleday/Penguin.
- Blatt, S. J. (2008). *Polarities of experience: Relatedness and self definition in personality development, psychopathology, and the therapeutic process.* Washington, DC: American Psychological Association.
- Carey, S., Diamond, R., & Woods, B. (1980). Development of face recognition: A maturational component? *Developmental Psychology*, 16(4), 257-269. doi: 10.1037/0012-1649.16.4.257
- Casey, B. J., Getz, S., & Galvan, A. (2008). The adolescent brain. *Developmental Review*, 28(1), 62-77. doi:10.1016/j.dr.2007.08.003
- Choudhury, S., Blakemore, S. J., & Charman, T. (2006). Social cognitive development during adolescence. *Social Cognitive and Affective Neuroscience*, 1(3), 165-174. doi: 10.1093/scan/nsl024
- Crone, E. A., & Dahl, R. E. (2012). Understanding adolescence as a period of social-affective engagement and goal flexibility. *Nature Reviews. Neuroscience*, *13*(9), 636-650. doi:10.1038/nrn3313
- Davey, C. G., Yücel, M., & Allen, N. B. (2008). The emergence of depression in adolescence: Development of the prefrontal cortex and the representation of reward. *Neuroscience & Biobehavioral Reviews*, 32(1), 1-19. doi:10.1016/j.neubiorev.2007.04.016
- Debbané, M. (2015). *Adolescent attachment: From brain to culture*. Paper presented at the European Society of Child and Adolescent Psychiatry, Madrid, Spain.
- Diamond, R., Carey, S., & Back, K. J. (1983). Genetic influences on the development of spatial skills during early adolescence. *Cognition*, 13(2), 167-185. doi:10.1016/0010-0277(83)90021-5

- Domes, G., Heinrichs, M., Michel, A., Berger, C., & Herpertz, S. C. (2007). Oxytocin improves "mind-reading" in humans. *Biological Psychiatry*, 61(6), 731-733. doi:10.1016/j.biopsych.2006.07.015
- Eiland, L., & Romeo, R. D. (2013). Stress and the developing adolescent brain. *Neuroscience*, 249(0), 162-171. doi:10.1016/j.neuroscience.2012.10.048
- Ein-Dor, T., Mikulincer, M., Doron, G., & Shaver, P. R. (2010). The attachment paradox: How can so many of us (the insecure ones) have no adaptive advantages? *Perspectives on Psychological Science*, *5*(2), 123-141. doi:10.1177/1745691610362349
- Ellis, B. J., Boyce, W. T., Belsky, J., Bakermans-Kranenburg, M. J., & van Ijzendoorn, M. H. (2011). Differential susceptibility to the environment: An evolutionary—neurodevelopmental theory. *Development and Psychopathology*, 23(01), 7-28. doi:doi:10.1017/S0954579410000611
- Escofet, P. (2012). *Le microcosme juvénile: Sociabilité adolescente, école et violences*. Gollion, Switzerland: Infolio.
- Feldman, R. (2017). The neurobiology of human attachments. *Trends in Cognitive Sciences*, 21(2), 80-99. doi:10.1016/j.tics.2016.11.007
- Fonagy, P., & Luyten, P. (2016). A multilevel perspective on the development of borderline personality disorder. In D. Cicchetti (Ed.), *Developmental psychopathology. Vol. 3:*Maladaptation and psychopathology (3rd ed., pp. 726-792). New York, NY: John Wiley & Sons.
- Fonagy, P., & Luyten, P. (2018). Conduct problems in youth and the RDoC approach: A developmental, evolutionary-based view. *Clinical Psychology Review*, 64, 57-76. doi:10.1016/j.cpr.2017.08.010
- Forbes, E. E., & Dahl, R. E. (2012). Research Review: Altered reward function in adolescent depression: What, when and how? *Journal of Child Psychology and Psychiatry*, 53(1), 3-15. doi:10.1111/j.1469-7610.2011.02477.x
- Gunnar, M., & Quevedo, K. (2007). The neurobiology of stress and development. *Annual Review of Psychology*, 58(1), 145-173. doi:10.1146/annurev.psych.58.110405.085605
- Hammen, C. (2005). Stress and depression. *Annual Review of Clinical Psychology, 1*(1), 293-319. doi:10.1146/annurev.clinpsy.1.102803.143938
- Heim, C., Plotsky, P. M., & Nemeroff, C. B. (2004). Importance of studying the contributions of early adverse experience to neurobiological findings in depression. *Neuropsychopharmacology*, 29(4), 641-648. doi: 10.1038/sj.npp.1300397
- Heinrichs, M., & Domes, G. (2008). Neuropeptides and social behaviour: Effects of oxytocin and vasopressin in humans. *Progress in Brain Research*, 170, 337-350. doi:10.1016/S0079-6123(08)00428-7
- Hostinar, C. E., Sullivan, R. M., & Gunnar, M. R. (2014). Psychobiological mechanisms underlying the social buffering of the hypothalamic-pituitary-adrenocortical axis: A review of animal models and human studies across development. *Psychological Bulletin*, *140*(1), 256-282. doi:10.1037/a0032671
- Hsu, D. T., Sanford, B. J., Meyers, K. K., Love, T. M., Hazlett, K. E., Walker, S. J., . . . Zubieta, J. K. (2015). It still hurts: Altered endogenous opioid activity in the brain during social rejection and acceptance in major depressive disorder. *Molecular Psychiatry*, 20(2), 193-200. doi:10.1038/mp.2014.185
- Kertes, D. A., Gunnar, M. R., Madsen, N. J., & Long, J. D. (2008). Early deprivation and home basal cortisol levels: a study of internationally adopted children. *Development and Psychopathology*, 20(2), 473-491. doi: 10.1017/S0954579408000230

- Keulers, E. H. H., Evers, E. A. T., Stiers, P., & Jolles, J. (2010). Age, sex, and pubertal phase influence mentalizing about emotions and actions in adolescents. *Developmental Neuropsychology*, *35*(5), 555-569. doi: 10.1080/87565641.2010.494920
- Lupien, S. J., McEwen, B. S., Gunnar, M. R., & Heim, C. (2009). Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nature Reviews. Neuroscience*, 10(6), 434-445. doi:10.1038/nrn2639
- Luyten, P. (2017). Personality, psychopathology, and health through the lens of interpersonal relatedness and self-definition. *Journal of the American Psychoanalytic Association*, 65(3), 473-489. doi:doi:10.1177/0003065117712518
- Luyten, P., & Blatt, S. J. (2013). Interpersonal relatedness and self-definition in normal and disrupted personality development: Retrospect and prospect. *American Psychologist*, 68(3), 172-183. doi:10.1037/a0032243
- Luyten, P., Campbell, C., Allison, E., & Fonagy, P. (2020). The mentalizing approach to psychopathology: State of the art and future directions. *Annual Review of Clinical Psychology*, *16*(1), null. doi:10.1146/annurev-clinpsy-071919-015355
- Luyten, P., Campbell, C., & Fonagy, P. (2020). Borderline personality disorder, complex trauma, and problems with self and identity: A social-communicative approach. *Journal of Personality*, 88(1), 88-105. doi:10.1111/jopy.12483
- Luyten, P., & Fonagy, P. (2015). The neurobiology of mentalizing. *Personality Disorders: Theory, Research and Treatment,* 6(4), 366-379.
- Luyten, P., & Fonagy, P. (2018). The stress-reward-mentalizing model of depression: An integrative developmental cascade approach to child and adolescent depressive disorder based on the Research Domain Criteria (RDoC) approach. *Clinical Psychology Review*, 64, 87-98. doi:10.1016/j.cpr.2017.09.008
- Luyten, P., & Fonagy, P. (2019). Mentalizing and trauma. In A. Bateman & P. Fonagy (Eds.), *Handbook of mentalizing in clinical practice* (2nd ed., pp. 79-99. Washington, DC: American Psychiatric Press.
- Masten, C. L., Eisenberger, N. I., Borofsky, L. A., Pfeifer, J. H., McNealy, K., Mazziotta, J. C., & Dapretto, M. (2009). Neural correlates of social exclusion during adolescence: Understanding the distress of peer rejection. *Social Cognitive and Affective Neuroscience*, 4(2), 143-157. doi:10.1093/scan/nsp007
- McEwen, B. S. (2007). Physiology and neurobiology of stress and adaptation: Central role of the brain. *Physiological Reviews*, 87(3), 873-904. doi:10.1152/physrev.00041.2006
- McGivern, R. F., Andersen, J., Byrd, D., Mutter, K. L., & Reilly, J. (2002). Cognitive efficiency on a match to sample task decreases at the onset of puberty in children. *Brain and Cognition*, 50(1), 73–89. doi: 10.1016/s0278-2626(02)00012-x
- Mutlu, A. K., Schneider, M., Debbane, M., Badoud, D., Eliez, S., & Schaer, M. (2013). Sex differences in thickness, and folding developments throughout the cortex. *NeuroImage*, 82, 200-207. doi:10.1016/j.neuroimage.2013.05.076
- Nestler, E. J., & Carlezon, W. A., Jr. (2006). The mesolimbic dopamine reward circuit in depression. *Biological Psychiatry*, *59*(12), 1151-1159. doi:10.1016/j.biopsych.2005.09.018
- Neumann, I. D. (2008). Brain oxytocin: A key regulator of emotional and social behaviours in both females and males. *Journal of Neuroendocrinology*, 20(6), 858-865. doi:10.1111/j.1365-2826.2008.01726.x
- Pizzagalli, D. A. (2014). Depression, stress, and anhedonia: Toward a synthesis and integrated model. *Annual Review of Clinical Psychology*, *10*, 393-423. doi:10.1146/annurev-clinpsy-050212-185606
- Russo, S. J., & Nestler, E. J. (2013). The brain reward circuitry in mood disorders. *Nature Reviews. Neuroscience*, 14(9), 609-625. doi:10.1038/nrn3381

- Sebastian, C., Viding, E., Williams, K. D., & Blakemore, S.-J. (2010). Social brain development and the affective consequences of ostracism in adolescence. *Brain and Cognition*, 72(1), 134-145. doi:10.1016/j.bandc.2009.06.008
- Sebastian, C. L., Tan, G. C., Roiser, J. P., Viding, E., Dumontheil, I., & Blakemore, S.-J. (2011). Developmental influences on the neural bases of responses to social rejection: Implications of social neuroscience for education. *Neuroimage*, *57*(3), 686-694. doi:10.1016/j.neuroimage.2010.09.063
- Sharp, C., Vanwoerden, S., & Wall, K. (2018). Adolescence as a sensitive period for the development of personality disorder. *Psychiatric Clinics of North America*, 41(4), 669-683. doi:https://doi.org/10.1016/j.psc.2018.07.004
- Shaw, P., Kabani, N. J., Lerch, J. P., Eckstrand, K., Lenroot, R., Gogtay, N., . . . Wise, S. P. (2008). Neurodevelopmental trajectories of the human cerebral cortex. *Journal of Neuroscience*, 28(14), 3586-3594. doi:10.1523/jneurosci.5309-07.2008
- Simpson, J. A., & Belsky, J. (2008). Attachment theory within a modern evolutionary framework. In J. Cassidy & P. R. Shaver (Eds.), *Handbook of attachment: Theory, research, and clinical applications* (2nd ed., pp. 131-157). New York, NY: Guilford Press.
- Spear, L. (2007). The developing brain and adolescent-typical behavior patterns: An evolutionary approach. In D. Romer & E. F. Walker (Eds.), *Adolescent psychopathology and the adolescent brain* (pp. 9-30). New York, NY: Oxford University Press.
- Spear, L. P. (2000). The adolescent brain and age-related behavioral manifestations. *Neuroscience & Biobehavioral Reviews*, 24(4), 417-463. doi:10.1016/S0149-7634(00)00014-2
- Strathearn, L. (2011). Maternal neglect: Oxytocin, dopamine and the neurobiology of attachment. *Journal of Neuroendocrinology*, 23(11), 1054-1065. doi:10.1111/j.1365-2826.2011.02228.x
- Swain, J. E., Kim, P., Spicer, J., Ho, S. S., Dayton, C. J., Elmadih, A., & Abel, K. M. (2014). Approaching the biology of human parental attachment: Brain imaging, oxytocin and coordinated assessments of mothers and fathers. *Brain Research*, *1580*, 78-101. doi:10.1016/j.brainres.2014.03.007
- Teicher, M. H., & Samson, J. A. (2013). Childhood maltreatment and psychopathology: A case for ecophenotypic variants as clinically and neurobiologically distinct subtypes. *American Journal of Psychiatry*, *170*(10), 1114-1133. doi: 10.1176/appi.ajp.2013.12070957
- Tonks, J., Williams, W. H., Frampton, I., Yates, P., & Slater, A. (2007). Assessing emotion recognition in 9–15-years olds: Preliminary analysis of abilities in reading emotion from faces, voices and eyes. *Brain Injury*, 21(6), 623-629. doi: 10.1080/02699050701426865
- van Rooijen, R., Junge, C. M. M., & Kemner, C. (2018). The interplay between gaze following, emotion recognition, and empathy across adolescence; a pubertal dip in performance? *Frontiers in Psychology*, *9*, 127. doi: 10.3389/fpsyg.2018.00127
- Vetter, N. C., Drauschke, M., Thieme, J., & Altgassen, M. (2018). Adolescent basic facial emotion recognition is not influenced by puberty or own-age bias. *Frontiers in Psychology*, *9*, 956. doi: 10.3389/fpsyg.2018.00956
- Vetter, N. C., Leipold, K., Kliegel, M., Phillips, L. H., & Altgassen, M. (2013). Ongoing development of social cognition in adolescence. *Child Neuropsychology*, 19(6), 615-629. doi: 10.1080/09297049.2012.718324