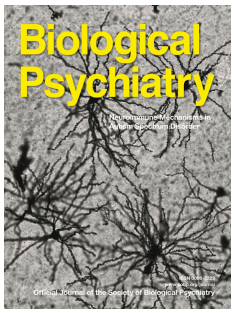
Journal Pre-proof



Navigating the social environment in adolescence: The role of social brain development

Jack L. Andrews, Saz Ahmed, Sarah-Jayne Blakemore

PII: S0006-3223(20)31920-X

DOI: https://doi.org/10.1016/j.biopsych.2020.09.012

Reference: BPS 14339

To appear in: *Biological Psychiatry*

Received Date: 20 February 2020

Revised Date: 4 September 2020

Accepted Date: 14 September 2020

Please cite this article as: Andrews J.L., Ahmed S. & Blakemore S.-J., Navigating the social environment in adolescence: The role of social brain development, *Biological Psychiatry* (2020), doi: https://doi.org/10.1016/j.biopsych.2020.09.012.

Navigating the social environment in adolescence: The role of social brain development

Short title: Adolescent brain development and social cognition

Jack L. Andrews[1*], Saz Ahmed[1*] & Sarah-Jayne Blakemore[1, 2]

[1] Institute of Cognitive Neuroscience, University College London, UK
[2] Department of Psychology, University of Cambridge, UK

*Joint first authors

Corresponding author:
Professor Sarah-Jayne Blakemore
Department of Psychology, University of Cambridge
Downing St, Cambridge, CB2 3EB
Email: sjblakemore@psychol.cam.ac.uk

Abstract

Successful navigation of the social environment is dependent on a number of social cognitive processes, including mentalising and resistance to peer influence. These processes continue to develop during adolescence, a time of significant social change, and are underpinned by regions of the social brain that continue to mature structurally and functionally into adulthood. In this review, we describe how mentalising, peer influence and emotion regulation capacities develop to aid the navigation of the social environment during adolescence. Heightened susceptibility to peer influence and hypersensitivity to social rejection in adolescence increase the likelihood of both risky and prosocial behaviour in the presence of peers. Developmental differences in mentalising and emotion regulation, and the cortico-subcortical circuits that underpin these processes, might put adolescents at risk of developing mental health problems. We suggest how interventions aimed at improving prosocial behaviour and emotion regulation abilities hold promise in reducing the risk of poor mental health as adolescents navigate the changes in their social environment.

Keywords: adolescence, social brain, social cognition, mentalising, peer influence, emotion regulation

Introduction

Adolescence is defined as the period of life between 10 and 24 years and is characterised by biological, psychological and social change(1). In addition, adolescence is a time of heightened susceptibility to mental health problems, with approximately 75% of adult mental health disorders first appearing before the age of 24(2). Changes in the social environment around the time of puberty often include the transition from primary to secondary educational settings, an increase in number of peers and an increase in time spent with peers relative to family(3). These changes present new challenges, including the need to integrate into large and unstable social networks and the possibility of peer rejection(4). Peer judgements influence adolescents' social and self-worth(5), and young people who adapt well to these changes by gaining a stable social position have better physical and psychological health outcomes(6,7).

Social cognitive processes, such as taking others' perspectives, regulating emotions and managing peer influence, facilitate successful social transition into adulthood. Navigating the social environment is reliant on successfully employing these processes, and training certain abilities, such as emotion regulation, might foster resilience to mental health problems in adolescence. Here, we review research on the development of social cognitive processes, and structural and functional changes in the social brain, during adolescence.

Social brain development in adolescence

During development the brain undergoes substantial structural change in terms of grey and white matter volume, surface area and cortical thickness(8–10). MRI studies have shown that cortical grey matter volume increases during childhood, reaches a peak in late childhood, and declines during the second decade of life, eventually stabilising in the mid-twenties(9,11–13). At the same time, there is a linear increase in white matter volume across childhood and adolescence(9,11,12). It is important to note that these findings are based on averages, and the precise timing and trajectories of structural development of different regions show substantial variation between individuals in terms of intercept (e.g. overall volume) and slope of change(14,15). One source of individual variation in adolescence is puberty and it has been proposed that brain development is influenced by the sex hormones that control the onset of and progression through puberty(16–19). For example, pubertal maturation is associated with developmental changes in subcortical brain volume, beyond chronological age(20,21). The neural and psychosocial changes that are associated with progression through puberty can also produce mental health vulnerabilities (Pfeifer and Allen, this issue).

Some of the latest maturing brain regions are found within the 'social brain,' the network of regions involved in recognising, understanding and interpreting social cues from others. Brain areas involved in these complex components of social cognition include the dorsomedial prefrontal cortex (mPFC), anterior cingulate cortex (ACC), inferior frontal gyrus, posterior superior temporal sulcus (pSTS), anterior temporal cortex (ATC), amygdala and anterior insula(22). Several regions within the social brain network are involved in mentalising, the ability to interpret mental states, the feelings and actions of others(22) (see Figure 1).

Longitudinal neuroimaging studies have shown that regions within the social brain undergo substantial and protracted structural development. For example, grey matter volume and cortical thickness in pSTS, TPJ and dmPFC decreases from childhood into the early twenties, whilst in the ATC, cortical thickness increases until early adulthood and grey matter volume increases until adolescence(23) (see Figure 2). Subcortical regions involved in the generation of emotions and emotion regulation, which is the ability to effectively manage and respond to an emotional experience(24), also show developmental change during adolescence. For example, the amygdala, which has vast connections with regulatory regions such as the PFC and ACC, increases in volume by around 7% between late childhood and mid-adolescence, with no significant change after around 14 years on average(15) (note there were significant individual differences in this pattern).

In addition to changes in brain structure, adolescence is a time of significant functional maturation of regions involved in social cognitive processes. These functional changes are reflected in studies exploring task-based activation patterns and functional connectivity between different brain regions. One longitudinal resting state functional connectivity study showed that, in contrast to the strong connectivity between brain regions implicated in sensory and motor function by age 14 (which became stronger from 14-26 years), disruptive development occurred in regions involved in higher order processes. These included frontoparietal and limbic networks activated by tasks related to memory, mentalising and social cognition, with weak connections becoming stronger and strong connections becoming weaker(25). This suggests that disruptive maturation of functional connectivity might support the development of more sophisticated mentalising, social cognition, executive functioning and emotion regulation skills as adolescents mature into independent adults.

Social cognitive development in adolescence

Mentalising/Perspective taking. Mentalising enables us to understand the minds and predict the behaviour of other people(26). Developmental functional neuroimaging studies have compared the neural processing of mentalising across age, usually by asking participants to think about other people's mental states. The TPJ, STS and precuneus showed increased activity when young adolescents(27–29) listened to sections of a story describing a character's thoughts (requiring mental state attribution) compared to sections of the same story that described the physical environment (not requiring mental state attribution), and this activity correlated with behavioural performance(29). They also demonstrated increasing specialisation of the TPJ for mental state content with age(27,29). Other studies have also found age differences in the activation of the dmPFC and TPJ during mentalising tasks (see review 37). Several studies have found that activity in the dmPFC during mentalising tasks decreases between adolescence and adulthood (see review 22). Other studies have found that adults recruit the TPJ more strongly than do adolescents when responding to scenarios relating to their own intentions versus physical events(31). Previous work in adults has found that the TPJ is recruited more by tasks requiring reasoning about others' intentions and beliefs (cognitive theory of mind)(39), while the mPFC is recruited more by inferences about emotions and preferences (affective theory of mind)(40–42). The studies suggest that the pattern of recruitment of these brain regions during mentalising changes across adolescence.

It has been suggested that adolescents, relative to adults, might be using different cognitive strategies when thinking about others' intentions(22). One theory proposes that, during social tasks, adolescents use cognitive strategies that rely more on explicit reflection about the self and others, subserved by the dmPFC, whereas adults rely more on the automatic processing of social scripts, subserved by the temporal lobes(22). This might enable adults to process social information side by side with other demanding tasks more efficiently than adolescents. This hypothesis was tested and the prediction supported by superimposing a working memory task on a mentalising task(36). Studies are needed to further investigate this and replicate the limited number of developmental studies on mentalising, particularly given the relatively small sample sizes and the dearth of longitudinal developmental studies(37).

The ability to take other people's perspectives is especially important when integrating into new social contexts and choosing which peers to align oneself with. In one study, whilst both adolescents and adults recruited the dmPFC when taking the someone else's perspective into account, adolescents - but not adults - also recruited the dmPFC when no social cues were present (i.e. when no perspective taking was required)(38). This suggests

that adolescents recruit regions of the brain involved in mental state attribution in non-social contexts where mentalising is not required(38,36,39). Behavioural studies have demonstrated that the ability to take someone else's perspective is still maturing during adolescence(40–42), with children and adolescents making more errors than adults on perspective taking tasks(47,48, see review 50). It has been suggested that inhibitory control, which is still maturing during adolescence, may be contributing to these differences(41)

To date, few studies have examined functional connectivity during mentalising or perspective taking tasks. In one such study, adolescents (aged 11-18 years), relative to adults, showed greater functional connectivity between regions of the social brain (vmPFC and pSTS/TPJ) when thinking about social emotions, such as guilt, relative to basic emotions, such as disgust, possibly indicating changes in the functional integration of the mentalising network across development(44). However, other studies have found no age effects in functional connectivity within mentalising brain regions during a false belief task(35) (in 9 to 13-year-olds) and a social evaluation task(52) (in 8 to 16-year-olds). The different age ranges studied and variations in the tasks employed may account for these differences in findings.

Mentalising, the peer environment and mental health. Adolescents who perform less well on tasks that involve inferring mental states from vignettes and film clips depicting social situations are more likely to report higher levels of loneliness and peer rejection, independent of age and socioeconomic status, compared with adolescents who show better mentalising performance(46). Neuroimaging studies have reported increased activity in the mPFC, precuneus and TPJ (areas within the mentalising network) in response to peer rejection(47,48). Social network studies, in which individuals' peer relationships are mapped, have related an adolescent's social network position to brain activation during tasks involving social information. In one such study involving 16-17 year old boys, participants with higher brokerage – the ability to connect with otherwise disconnected others - exhibited greater activity within mentalising brain regions when presented with divergent feedback from peers when recommending computer games(49). These findings suggest that the mentalising network is more strongly recruited in risky social situations characterised by risk of rejection or dissimilarity to one's peers. Further, inter-individual variation in activity in brain regions involved in the processing of social exclusion and mentalising following social exclusion predicted increased risk taking in the presence of a peer one week later(50). The authors suggested that adolescents who showed this heightened neural response in the face of social

rejection may be more likely to change their behaviour in the presence of peers in order to avoid further exclusion and to promote affiliation with peers.

In one study, increased functional connectivity between social brain regions involved in mentalising during experimentally induced social exclusion, relative to social inclusion, was observed in adolescents(51). Stronger connectivity within the mentalising network during exclusion was found among participants with less dense real world social networks, characterised by fewer connections among one's friends(51). The authors suggested that this increased functional coupling between mentalising regions following social exclusion might support the ability to consider the intentions of others, including people who are excluding the participant. Mentalising may therefore be involved in the successful adaptation to stressful social interactions. In turn, there is meta-analytic evidence of a reciprocal relationship between poor mentalising abilities and mood disorders, such that impairments in mentalising may be both a cause and effect of internalising disorders(59–61, see review 62). In one study of adolescents with depression, reduced mentalising was associated with the severity of depression(56). The stress-reward-mentalising model accounts for these findings by suggesting that child and adolescent depression emerges from the interaction between impairments in stress-regulatory, reward and mentalising systems(57).

Emotion regulation. It has been proposed that the ability to regulate one's emotions in response to unfavourable social cues can buffer against negative mental health outcomes (see review 32). One study showed that adolescents who are accepted by their peers exhibit more adaptive emotion regulation and are at lower risk for internalising symptoms(58). It has been hypothesised that the protracted development of the PFC (which is typically recruited during emotion regulation) renders adolescents less able to regulate their emotions successfully(24), putting them at greater risk for anxiety and stress related disorders (see review 66). Behavioural and neuroimaging studies of typically developing young people suggest that emotion regulation capacities develop substantially across adolescence(60,61), with an increased use of adaptive emotion regulation strategies with age(62–64). Age-related improvements in emotion regulation have been associated with reduced amygdala activation(65,66) and inverse coupling (i.e., negative functional connectivity) between the vmPFC and amygdala(65). By mid-adolescence, most individuals exhibit inverse amygdala–PFC connectivity (compared with positive connectivity in childhood), with stronger inverse connectivity associated with lower symptoms of non-clinical anxiety(67,68). Disruptions in the same cortico-limbic circuitry during emotion regulation have been implicated in anxiety

and depression in adolescents(73,76–78, see review 79). For example, in female adolescents, positive amygdala-vlPFC connectivity during an emotion regulation task predicted future symptoms of anxiety over the subsequent nine months(69). It was also related to greater self-reported rumination and mediated the association between self-reported rumination and depressive symptoms following a social exclusion task(73).

The ability to mentalise is considered a key factor in the development of adequate emotion regulation abilities(74). Studies have also shown that adolescents' vulnerability to peer influence may be mediated by neural mechanisms that underlie emotion regulation, attentional control and mentalising(60,65,75). These findings suggest that individual variation in emotion regulation abilities might contribute to risky decision making in the presence of peers. Furthermore, poor emotion regulation predicts greater participation in risky behaviours(76,77) and that the mere exposure to peers online can alter self-regulation(78). Negative social situations, such as being socially excluded, require increased emotion regulation and this has been found to recruit regulation networks in adolescents(79). Social exclusion might also require an increased effort to understand the intentions of others (e.g. "why did they reject me?"); however, further research is needed to explore this.

Between the ages of 12 and 15 years, less adaptive and more maladaptive emotion regulation strategies, such as rumination, avoidance and suppression, are observed in comparison to other age groups(62). Training adaptive emotion regulation skills may therefore be useful in this age group (see meta-analysis 87) and may represent a fruitful avenue for helping adolescents deal with stressful social situations and reduce the risk of developing depression and anxiety(24,70,81). Several studies have shown positive short-term effects of instructed emotion regulation strategies such as distraction, acceptance and cognitive reappraisal, which is reinterpreting a negative event in a more positive way, in early and mid-adolescence,(82–84) and in clinically anxious youth aged 8-17 years(85).

Further research is needed to investigate mechanisms of psychological interventions targeting emotion regulation abilities and whether training emotion regulation skills mediates the impact of psychological interventions on internalising symptoms. Emotion regulation intervention studies with larger samples and in adolescents with psychiatric conditions are needed to investigate the long-term benefits of emotion regulation training and how the neurocognitive development of social cognitive skills such as mentalising contribute to the use of particular emotion regulation strategies.

Peer influence in adolescence

Adolescents are particularly susceptible to peer influence, especially in risky contexts. Research has begun to explore the positive influence peers can have on prosocial decisions, and even on prosocial risk-taking, as described below.

Peer influence on risk behaviour. Belonging to a peer group that engages in antisocial or illegal behaviour is a risk factor for engagement in a number of negative health risk behaviours (86). Evidence from longitudinal social network studies has demonstrated that this clustering of risk behaviour is associated with both peer selection and peer socialisation, in which an individual's behaviour changes over time as a result of shifts in the behaviour of friends(87). Furthermore, engagement in certain risk-taking behaviours may be dependent on status such that both high and low status individuals tend to take more risks, but with different intentions (e.g. to maintain or gain status)(88). Longitudinal studies have shown that risk taking is predicted by whether or not an adolescent endorses societal stereotypes of adolescent risk taking, via activity in cognitive control related brain regions such as the vlPFC(89). This suggests that stereotypes about adolescents may play a role in how the brain responds to risk-taking during adolescence(90).

Multiple theoretical models have been proposed to explain the mechanisms underlying peer influence effects on risk-taking and have distinguished between social motivation, reward sensitivity and distraction(91). Whilst not mutually exclusive, each model emphasises different neurodevelopmental processes that lead to altered decision-making processes during adolescence. For example, social motivation models are underpinned by developmental changes observed in the social brain(22,23) and emphasise the importance of the social value given to peer group norms in guiding decision making processes(92). Conversely, reward sensitivity and distraction models highlight the continued maturation of cognitive control and its neural systems during adolescence. The reward sensitivity, mismatch or dual systems model (93–95) is based on the notion that regions of the brain that support cognitive control (e.g. the PFC) mature at slower rates than brain regions implicated in reward processing (e.g., the nucleus accumbens and ventral striatum) and emotional reactivity (e.g. the amygdala). This model is also used to explain the weaker emotion regulation abilities of adolescents compared with adults(24) (note that these models have been critiqued; e.g. 106). Value-based explanations of adolescent decision making(97), which account for the subjective value placed on each component of a decision, are consistent with social motivation models whereby a high value is placed on peer group norms and this guides behaviour during adolescence(92). This is supported by a study showing that young

adolescents (aged 12-14 years) are more influenced by the risk perception of teenagers than adults(98), and that this susceptibility to peer influence is even stronger when they believe that other teenagers rate a situation as *more risky* than they initially thought it was(99).

During adolescence, the social context might influence the reward value associated with engagement in risk behaviours(100). Studies using simulated driving tasks show that adolescents are more likely than adults to take risks when in the presence of their friends compared to when driving alone(100–103). This is particularly pronounced when their 'passengers' reported a strong preference for risk taking(104) and when participants reported lower resistance to peer influence following social exclusion(105). Adolescents exhibited greater activity in brain regions implicated in reward processing, including the ventral striatum and nucleus accumbens, when taking driving risks in the presence of peers(100,101). Adolescents' neural sensitivity to a response inhibition task was found to predict their risk-taking behaviour under peer influence: higher activation in the ventral striatum and PFC was associated with engaging in fewer risks, on a simulated driving game, when paired with cautious peers(103). This suggests that stronger inhibitory activity in a cognitive control task predicts the extent to which adolescents are influenced by the social norms of a safety-promoting peer. These studies demonstrate that, during adolescence, the presence of peers might increase the reward value of a risky decision, supporting both social motivation and reward sensitivity models (see reviews 116,117).

Adolescents and young adults take more risks on the Balloon Analogue Risk Taking (BART) task, in which participants inflate a virtual balloon for a monetary reward, when encouraged by a peer compared with either being alone or in a peer's presence but without encouragement(108) or when other participants were perceived to be making high risk choices versus low risk choices(109). This is consistent with findings showing that adolescents follow suit when observing peers make safe, risk averse and prosocial decisions (91,104,110–112). Neural responses during the BART task were associated with adolescents' behavioural conformity to safe versus risky peer influence during a driving task one week later, and connectivity between the ventral striatum, insula and ACC predicted safer driving under risky peer influence (113).

Young people who are affiliated with antisocial peers are more prone to developing externalising disorders such as antisocial behaviour and substance use during adolescence(114–117). One study found that the genetic influence on adolescent externalising disorders was greater when the degree of antisocial peer affiliation was higher, and smaller when it was lower(118). This finding that antisocial peers appear to activate

genetic risk for externalising problems was only found in adolescents aged 17, and not at ages 20, 24, or 29 years (see also meta-analyses 129,130). This suggests that adolescence is a critical period for the development of externalising problems, particularly in adverse social environments.

Avoiding the social risk of rejection. Adolescents demonstrate hypersensitivity to the negative effects of social rejection(121–123), and it has been proposed that adolescents are more likely to take health and legal risks in the presence of their peers in order to avoid the social risk of rejection(30,124). Social rejection is associated with disruptions in emotion regulation(125) and is a risk factor for adolescent-onset mood disorders, such as depression and anxiety(126–128). Therefore adolescents who engage in health risk behaviours, such as smoking, when this is the peer group norm may be doing so in order to avoid the social risk of rejection, even when such risks carry negative health or legal consequences(124). In one study, adolescents reported greater concern about engaging in social risks (such as defending an unpopular opinion) relative to adults. Additionally, greater concern for social risk was more strongly related to rejection sensitivity and depressed mood in adolescents (11-17 years) compared to adults(129). Making decisions that increase one's social value by reducing their risk of social rejection might be adaptive for adolescents, for whom social status and high quality friendships are known to be related to better psychological and physical health outcomes(6,7). Thus, deciding to engage in risk behaviours such as smoking or binge drinking when in a peer group setting, while not being an objectively 'good' decision, might not necessarily be an irrational choice for adolescents when these risk behaviours are valued by the peer group. Reducing their social risk by engaging in behaviours, risky or otherwise, that increase their social status and reduce their risk of rejection may be especially important for adolescents.

Peer influence on prosocial behaviour. Whilst adolescents are particularly susceptible to peer influence in risky contexts, experimental research has begun to explore the positive influence peers can have on prosocial decisions (see review 140). Adolescents are more likely to volunteer in the community if they observe peers doing so(131) and, compared with adults, adolescents are more likely to be influenced by others towards (hypothetical) prosocial behaviours(111). As with risk-taking behaviour, high-status peers(131) and very close friends(132) tend to be more influential. In incentivised charitable donation tasks and public goods games, adolescents who observed peers being generous were more likely to be

generous themselves(112) and exhibited higher activity in social brain regions, such as the dmPFC, TPJ, precuneus and STS, during prosocial decision-making(112,130,133). In a study of older adolescents (mean age 20) participants recruited brain regions involved in self-control and mentalising when making decisions to donate money to family members. Greater functional coupling between self-control and mentalising regions with the ventral striatum was observed in participants who reported stronger familial obligation(134). This suggests that prosocial behaviour may be reliant on the development of brain regions involved in mentalising and self-control, in addition to reward-related regions.

Recent studies have investigated 'prosocial risk-taking', namely, taking risky decisions with the intention of helping others, for example standing up to someone who is bullying a friend (see reviews 145,146). Findings show that, unlike potentially harmful risk taking behaviours such as reckless driving or illicit drug taking, prosocial risk taking is not associated with impulsivity or risk taking on experimental tasks, and is instead associated with lower reward sensitivity, higher punishment sensitivity and greater school engagement(137). These findings demonstrate that positive, as opposed to negative, risk taking may be beneficial for adolescents.

The evidence that peers have the capacity to influence decisions towards positive outcomes can inform interventions aimed at improving health and well-being during adolescence(112,130,138). Indeed, studies that target social norms through peer-led interventions during adolescence have shown positive outcomes across a number of domains, including a 25% reduction in bullying over one year in middle schools(139) and a sustained reduction in the uptake of regular smoking in students aged 12-13 years for two years after the intervention's delivery(140). Therefore, peers might influence adolescent adherence to a variety of interventions. This area of research is becoming increasingly important in understanding the context in which interventions are most successful.

Conclusion

Adolescence is a time of social transition, during which a number of socio-cognitive processes such as mentalising, resistance to peer influence and emotion regulation continue to mature, alongside the brain systems that support them. Adolescents are especially vulnerable to mental health problems and risk factors in the social environment, such as peer rejection, which contribute to this vulnerability. Interventions aimed at improving prosocial behaviour, and emotion regulation abilities, particularly in ambiguous or risky social situations, may hold promise in reducing some of this vulnerability towards poor mental health.

Figure legends

Figure 1. The mentalising network. Regions that are involved in mentalising include the dorsal medial prefrontal cortex (dmPFC) and temporoparietal junction (TPJ) (27,141–143), which are involved in inferring mental states; the precuneus, a region often co-activated with the mPFC during perspective taking with mental imagery tasks; the posterior superior temporal sulcus (pSTS) (144,145), which is involved in face perception; and the anterior temporal cortex (ATC) (146) which is involved in representing and retrieving social knowledge (see meta-analysis (147). Figure adapted from (22).

Figure 2. Best fitting models of structural brain development across adolescence, shown for each region of interest (combined hemispheres). Each model is fitted to the middle 80% of the sample (ages 9–22 years for mBA10; corresponding to dmPFC), TPJ and pSTS; ages 11–24 years for ATC). The lighter lines show the fitted models applied to females only, and the darker lines show the fitted models applied to males only. Solid lines indicate the fitted model was significant $p < 0.05$, whereas dashed lines indicate the fitted model was not significant ($p \geq 0.05$). Taken from (23).

Financial disclosure: The authors have no biomedical financial interests or potential conflicts of interest.

Acknowledgments

SJB is funded by Wellcome, the Jacobs Foundation and the University of Cambridge. SA is funded by a Wellcome grant to SJB. JLA is funded by a Medical Research Council studentship.

References

1. Sawyer SM, Azzopardi PS, Wickremarathne D, Patton GC (2018): The age of adolescence. *Lancet Child Adolesc Health* 2: 223–228.

2. Kessler RC, Angermeyer M, Anthony JC, De Graaf R, Demyttenaere K, Gasquet I, *et al.* (2007): Lifetime prevalence and age-of-onset distributions of mental disorders in the World Health Organization's World Mental Health Survey Initiative. *World Psychiatry* 6: 168–176.

3. Blakemore S-J (2019): Adolescence and mental health. *Lancet Lond Engl* 393: 2030–2031.

4. Burnett Heyes S, Jih Y-R, Block P, Hiu C-F, Holmes EA, Lau JYF (2015): Relationship reciprocation modulates resource allocation in adolescent social networks: Developmental effects. *Child Dev* 86: 1489–1506.

5. Birkeland MS, Breivik K, Wold B (2014): P Peer acceptance protects global self-esteem from negative effects of low closeness to parents during adolescence and early adulthood. *J Youth Adolesc* 43: 70–80.

6. Almquist Y (2009): Peer status in school and adult disease risk: a 30-year follow-up study of disease-specific morbidity in a Stockholm cohort. *J Epidemiol Community Health* 63: 1028–1034.

7. van Harmelen A-L, Kievit RA, Ioannidis K, Neufeld S, Jones PB, Bullmore E, *et al.* (2017): Adolescent friendships predict later resilient functioning across psychosocial domains in a healthy community cohort. *Psychol Med* 47: 2312–2322.

8. Giedd JN, Blumenthal J, Jeffries NO, Castellanos FX, Liu H, Zijdenbos A, *et al.* (1999): Brain development during childhood and adolescence: a longitudinal MRI study. *Nat Neurosci* 2: 861–863.

9. Tamnes CK, Herting MM, Goddings A-L, Meuwese R, Blakemore S-J, Dahl RE, *et al.* (2017): Development of the cerebral cortex across adolescence: A multisample study

of inter-related longitudinal changes in cortical volume, surface area, and thickness. *J Neurosci Off J Soc Neurosci* 37: 3402–3412.

10. Vijayakumar N, Allen NB, Youssef G, Dennison M, Yücel M, Simmons JG, Whittle S (2016): Brain development during adolescence: A mixed-longitudinal investigation of cortical thickness, surface area, and volume. *Hum Brain Mapp* 37: 2027–2038.
11. Aubert-Broche B, Fonov VS, García-Lorenzo D, Mouiha A, Guizard N, Coupé P, *et al.* (2013): A new method for structural volume analysis of longitudinal brain MRI data and its application in studying the growth trajectories of anatomical brain structures in childhood. *NeuroImage* 82: 393–402.
12. Mills KL, Goddings A-L, Herting MM, Meuwese R, Blakemore S-J, Crone EA, *et al.* (2016): Structural brain development between childhood and adulthood: Convergence across four longitudinal samples. *NeuroImage* 141: 273–281.
13. Wierenga L, Langen M, Ambrosino S, van Dijk S, Oranje B, Durston S (2014): Typical development of basal ganglia, hippocampus, amygdala and cerebellum from age 7 to 24. *NeuroImage* 96: 67–72.
14. Foulkes L, Blakemore S-J (2016): Is there heightened sensitivity to social reward in adolescence? *Curr Opin Neurobiol* 40: 81–85.
15. Mills KL, Goddings A-L, Clasen LS, Giedd JN, Blakemore S-J (2014): The developmental mismatch in structural brain maturation during adolescence. *Dev Neurosci* 36: 147–160.
16. Vijayakumar N, Op de Macks Z, Shirtcliff EA, Pfeifer JH (2018): Puberty and the human brain: Insights into adolescent development. *Neurosci Biobehav Rev* 92: 417–436.
17. Lenroot RK, Gogtay N, Greenstein DK, Wells EM, Wallace GL, Clasen LS, *et al.* (2007): Sexual dimorphism of brain developmental trajectories during childhood and adolescence. *NeuroImage* 36: 1065–1073.

18. Peper JS, Hulshoff Pol HE, Crone EA, van Honk J (2011): Sex steroids and brain structure in pubertal boys and girls: a mini-review of neuroimaging studies. *Neuroscience* 191: 28–37.

19. Sowell ER, Trauner DA, Gamst A, Jernigan TL (2002): Development of cortical and subcortical brain structures in childhood and adolescence: a structural MRI study. *Dev Med Child Neurol* 44: 4–16.

20. Goddings A-L, Mills KL, Clasen LS, Giedd JN, Viner RM, Blakemore S-J (2014): The influence of puberty on subcortical brain development. *NeuroImage* 88: 242–251.

21. Wierenga LM, Bos MGN, Schreuders E, vd Kamp F, Peper JS, Tamnes CK, Crone EA (2018): Unraveling age, puberty and testosterone effects on subcortical brain development across adolescence. *Psychoneuroendocrinology* 91: 105–114.

22. Blakemore S-J (2008): The social brain in adolescence. *Nat Rev Neurosci* 9: 267–277.

23. Mills KL, Lalonde F, Clasen LS, Giedd JN, Blakemore S-J (2014): Developmental changes in the structure of the social brain in late childhood and adolescence. *Soc Cogn Affect Neurosci* 9: 123–131.

24. Ahmed SP, Bittencourt-Hewitt A, Sebastian CL (2015): Neurocognitive bases of emotion regulation development in adolescence. *Dev Cogn Neurosci* 15: 11–25.

25. Váša F, Romero-Garcia R, Kitzbichler MG, Seidlitz J, Whitaker KJ, Vaghi MM, *et al.* (2020): Conservative and disruptive modes of adolescent change in human brain functional connectivity. *Proc Natl Acad Sci U S A* 117: 3248–3253.

26. Olsson A, Ochsner KN (2008): The role of social cognition in emotion. *Trends Cogn Sci* 12: 65–71.

27. Saxe RR, Whitfield-Gabrieli S, Scholz J, Pelphrey KA (2009): Brain regions for perceiving and reasoning about other people in school-aged children. *Child Dev* 80: 1197–1209.

28. Mukerji CE, Lincoln SH, Dodell-Feder D, Nelson CA, Hooker CI (2019): Neural correlates of theory-of-mind are associated with variation in children's everyday social cognition. *Soc Cogn Affect Neurosci* 14: 579–589.

29. Gweon H, Dodell-Feder D, Bedny M, Saxe R (2012): Theory of mind performance in children correlates with functional specialization of a brain region for thinking about thoughts. *Child Dev* 83: 1853–1868.

30. Blakemore S-J, Mills KL (2014): Is adolescence a sensitive period for sociocultural processing? *Annu Rev Psychol* 65: 187–207.

31. Blakemore S-J, den Ouden H, Choudhury S, Frith C (2007): Adolescent development of the neural circuitry for thinking about intentions. *Soc Cogn Affect Neurosci* 2: 130–139.

32. Schlaffke L, Lissek S, Lenz M, Juckel G, Schultz T, Tegenthoff M, *et al.* (2015): Shared and nonshared neural networks of cognitive and affective theory-of-mind: a neuroimaging study using cartoon picture stories. *Hum Brain Mapp* 36: 29–39.

33. Sebastian CL, Fontaine NMG, Bird G, Blakemore S-J, De Brito SA, McCrory EJP, Viding E (2012): Neural processing associated with cognitive and affective Theory of Mind in adolescents and adults. *Soc Cogn Affect Neurosci* 7: 53–63.

34. Shamay-Tsoory SG (2011): The neural bases for empathy. *Neurosci Rev J Bringing Neurobiol Neurol Psychiatry* 17: 18–24.

35. Leopold A, Krueger F, dal Monte O, Pardini M, Pulaski SJ, Solomon J, Grafman J (2012): Damage to the left ventromedial prefrontal cortex impacts affective theory of mind. *Soc Cogn Affect Neurosci* 7: 871–880.

36. Mills KL, Dumontheil I, Speekenbrink M, Blakemore S-J (2015): Multitasking during social interactions in adolescence and early adulthood. *R Soc Open Sci* 2: 150117.

37. Foulkes L, Blakemore S-J (2018): Studying individual differences in human adolescent brain development. *Nat Neurosci* 21: 315–323.

38. Dumontheil I, Hillebrandt H, Apperly IA, Blakemore S-J (2012): Developmental differences in the control of action selection by social information. *J Cogn Neurosci* 24: 2080–2095.

39. van den Bos W, van Dijk E, Westenberg M, Rombouts SARB, Crone EA (2011): Changing brains, changing perspectives: the neurocognitive development of reciprocity. *Psychol Sci* 22: 60–70.

40. Dumontheil I, Apperly IA, Blakemore S-J (2010): Online usage of theory of mind continues to develop in late adolescence. *Dev Sci* 13: 331–338.

41. Symeonidou I, Dumontheil I, Chow W-Y, Breheny R (2016): Development of online use of theory of mind during adolescence: An eye-tracking study. *J Exp Child Psychol* 149: 81–97.

42. van den Bos E, de Rooij M, Sumter SR, Westenberg PM (2016): Continued development of recursive thinking in adolescence: Longitudinal analyses with a revised recursive thinking test. *Cogn Dev* 37: 28–41.

43. Crone EA, Fuligni AJ (2020): Self and others in adolescence. *Annu Rev Psychol* 71: 447–469.

44. Burnett S, Blakemore S-J (2009): Functional connectivity during a social emotion task in adolescents and in adults. *Eur J Neurosci* 29: 1294–1301.

45. McCormick EM, van Hoorn J, Cohen JR, Telzer EH (2018): Functional connectivity in the social brain across childhood and adolescence. *Soc Cogn Affect Neurosci* 13: 819–830.

46. Devine RT, Hughes C (2013): Silent films and strange stories: theory of mind, gender, and social experiences in middle childhood. *Child Dev* 84: 989–1003.

47. Beyer F, Münte TF, Krämer UM (2014): Increased neural reactivity to socio-emotional stimuli links social exclusion and aggression. *Biol Psychol* 96: 102–110.

48. Meyer ML, Masten CL, Ma Y, Wang C, Shi Z, Eisenberger NI, Han S (2013): Empathy for the social suffering of friends and strangers recruits distinct patterns of brain activation. *Soc Cogn Affect Neurosci* 8: 446–454.

49. O'Donnell MB, Bayer JB, Cascio CN, Falk EB (2017): Neural bases of recommendations differ according to social network structure. *Soc Cogn Affect Neurosci* 12: 61–69.

50. Falk EB, Cascio CN, Brook O'Donnell M, Carp J, Tinney FJ, Bingham CR, *et al.* (2014): Neural responses to exclusion predict susceptibility to social influence. *J Adolesc Health* 54: S22–S31.

51. Schmälzle R, Brook O'Donnell M, Garcia JO, Cascio CN, Bayer J, Bassett DS, *et al.* (2017): Brain connectivity dynamics during social interaction reflect social network structure. *Proc Natl Acad Sci U S A* 114: 5153–5158.

52. Fischer-Kern M, Tmej A (2019): Mentalization and depression: Theoretical concepts, treatment approaches and empirical studies - an overview. *Z Psychosom Med Psychother* 65: 162–177.

53. Li S, Zhang B, Guo Y, Zhang J (2015): The association between alexithymia as assessed by the 20-item Toronto Alexithymia Scale and depression: A meta-analysis. *Psychiatry Res* 227: 1–9.

54. Nolte T, Guiney J, Fonagy P, Mayes LC, Luyten P (2011): Interpersonal stress regulation and the development of anxiety disorders: an attachment-based developmental framework. *Front Behav Neurosci* 5: 55.

55. Luyten P, Campbell C, Allison E, Fonagy P (2020): The mentalizing approach to psychopathology: state of the art and future directions. *Annu Rev Clin Psychol* 16: 297–325.

56. Murri MB, Ferrigno G, Penati S, Muzio C, Piccinini G, Innamorati M, *et al.* (2017): Mentalization and depressive symptoms in a clinical sample of adolescents and young adults. *Child Adolesc Ment Health* 22: 69–76.

57. 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. *Clin Psychol Rev* 64: 87–98.

58. Kim J, Cicchetti D (2010): Longitudinal pathways linking child maltreatment, emotion regulation, peer relations, and psychopathology. *J Child Psychol Psychiatry* 51: 706–716.

59. Powers A, Casey BJ (2015): The Adolescent brain and the emergence and peak of psychopathology. *J Infant Child Adolesc Psychother* 14: 3–15.

60. McRae K, Gross JJ, Weber J, Robertson ER, Sokol-Hessner P, Ray RD, *et al.* (2012): The development of emotion regulation: an fMRI study of cognitive reappraisal in children, adolescents and young adults. *Soc Cogn Affect Neurosci* 7: 11–22.

61. Silvers JA, McRae K, Gabrieli JDE, Gross JJ, Remy KA, Ochsner KN (2012): Age-related differences in emotional reactivity, regulation, and rejection sensitivity in adolescence. *Emot Wash DC* 12: 1235–1247.

62. Cracco E, Goossens L, Braet C (2017): Emotion regulation across childhood and adolescence: evidence for a maladaptive shift in adolescence. *Eur Child Adolesc Psychiatry* 26: 909–921.

63. Gullone E, Hughes EK, King NJ, Tonge B (2010): The normative development of emotion regulation strategy use in children and adolescents: a 2-year follow-up study. *J Child Psychol Psychiatry* 51: 567–574.

64. Zimmermann P, Iwanski A (2014): Emotion regulation from early adolescence to emerging adulthood and middle adulthood: Age differences, gender differences, and emotion-specific developmental variations. *Int J Behav Dev* 38: 182–194.

65. Silvers JA, Insel C, Powers A, Franz P, Helion C, Martin RE, *et al.* (2017): vlPFC-vmPFC-amygdala interactions underlie age-related differences in cognitive regulation of emotion. *Cereb Cortex* 27: 3502–3514.

66. Stephanou K, Davey CG, Kerestes R, Whittle S, Pujol J, Yücel M, *et al.* (2016): Brain functional correlates of emotion regulation across adolescence and young adulthood. *Hum Brain Mapp* 37: 7–19.

67. Gee DG, Humphreys KL, Flannery J, Goff B, Telzer EH, Shapiro M, *et al.* (2013): A developmental shift from positive to negative connectivity in human amygdala-prefrontal circuitry. *J Neurosci Off J Soc Neurosci* 33: 4584–4593.

68. Kujawa A, Wu M, Klumpp H, Pine DS, Swain JE, Fitzgerald KD, *et al.* (2016): Altered development of amygdala-anterior cingulate cortex connectivity in anxious youth and young adults. *Biol Psychiatry Cogn Neurosci Neuroimaging* 1: 345–352.

69. Davis MM, Miernicki ME, Telzer EH, Rudolph KD (2019): The contribution of childhood negative emotionality and cognitive control to anxiety-linked neural dysregulation of emotion in adolescence. *J Abnorm Child Psychol* 47: 515–527.

70. Platt B, Campbell CA, James AC, Murphy SE, Cooper MJ, Lau JYF (2015): Cognitive reappraisal of peer rejection in depressed versus non-depressed adolescents: functional connectivity differences. *J Psychiatr Res* 61: 73–80.

71. Perlman G, Simmons AN, Wu J, Hahn KS, Tapert SF, Max JE, *et al.* (2012): Amygdala response and functional connectivity during emotion regulation: A study of 14 depressed adolescents. *J Affect Disord* 139: 75–84.

72. Young KS, Sandman CF, Craske MG (2019): Positive and negative emotion regulation in adolescence: Links to anxiety and depression. *Brain Sci* 9.

73. Fowler CH, Miernicki ME, Rudolph KD, Telzer EH (2017): Disrupted amygdala-prefrontal connectivity during emotion regulation links stress-reactive rumination and adolescent depressive symptoms. *Dev Cogn Neurosci* 27: 99–106.

74. Fonagy P, Gergely G, Jurist E, Target M (2005): *Affect Regulation, Mentalization, and the Development of Self*, 1st ed. New York: Other Press.

75. Aron AR, Robbins TW, Poldrack RA (2004): Inhibition and the right inferior frontal cortex. *Trends Cogn Sci* 8: 170–177.

76. Magar ECE, Phillips LH, Hosie JA (2008): Self-regulation and risk-taking. *Personal Individ Differ* 45: 153–159.

77. Perino MT, Guassi Moreira JF, McCormick EM, Telzer EH (2019): Apples to apples? Neural correlates of emotion regulation differences between high- and low-risk adolescents. *Soc Cogn Affect Neurosci* 14: 827–836.

78. King KM, McLaughlin KA, Silk J, Monahan KC (2018): Peer effects on self-regulation in adolescence depend on the nature and quality of the peer interaction. *Dev Psychopathol* 30: 1389–1401.

79. Jankowski KF, Batres J, Scott H, Smyda G, Pfeifer JH, Quevedo K (2018): Feeling left out: depressed adolescents may atypically recruit emotional salience and regulation networks during social exclusion. *Soc Cogn Affect Neurosci* 13: 863–876.

80. Schäfer JÖ, Naumann E, Holmes EA, Tuschen-Caffier B, Samson AC (2017): Emotion regulation strategies in depressive and anxiety symptoms in youth: A meta-analytic review. *J Youth Adolesc* 46: 261–276.

81. Schweizer S, Leung JT, Kievit R, Speekenbrink M, Trender W, Hampshire A, Blakemore S-J (2019): Protocol for an app-based affective control training for adolescents: proof-of-principle double-blind randomized controlled trial. *Wellcome Open Res* 4: 91.

82. Wante L, Van Beveren M-L, Theuwis L, Braet C (2018): The effects of emotion regulation strategies on positive and negative affect in early adolescents. *Cogn Emot* 32: 988–1002.

83. Hilt LM, Pollak SD (2012): Getting out of rumination: Comparison of three brief interventions in a sample of youth. *J Abnorm Child Psychol* 40: 1157–1165.

84. Rood L, Roelofs J, Bögels SM, Arntz A (2012): The effects of experimentally induced rumination, positive reappraisal, acceptance, and distancing when thinking about a stressful event on affect states in adolescents. *J Abnorm Child Psychol* 40: 73–84.

85. De Witte NAJ, Sütterlin S, Braet C, Mueller SC (2017): Psychophysiological correlates of emotion regulation training in adolescent anxiety: Evidence from the novel PIER task. *J Affect Disord* 214: 89–96.

86. Fergusson DM, Swain-Campbell NR, Horwood LJ (2002): Deviant peer affiliations, crime and substance use: a fixed effects regression analysis. *J Abnorm Child Psychol* 30: 419–430.

87. Henneberger AK, Gest SD, Zadzora KM (2019): Preventing adolescent substance use: A content analysis of peer processes targeted within universal school-based programs. *J Prim Prev* 40: 213–230.

88. Ennett ST, Faris R, Hipp J, Foshee VA, Bauman KE, Hussong A, Cai L (2008): Peer smoking, other peer attributes, and adolescent cigarette smoking: A social network analysis. *Prev Sci* 9: 88–98.

89. Qu Y, Pomerantz EM, McCormick E, Telzer EH (2018): Youth's conceptions of adolescence predict longitudinal changes in prefrontal cortex activation and risk taking during adolescence. *Child Dev* 89: 773–783.

90. Choudhury S (2010): Culturing the adolescent brain: what can neuroscience learn from anthropology? *Soc Cogn Affect Neurosci* 5: 159–167.

91. Ciranka S, van den Bos W (2019): Social influence in adolescent decision-Making: A formal framework. *Front Psychol* 10: 1915.

92. Crone EA, Dahl RE (2012): Understanding adolescence as a period of social-affective engagement and goal flexibility. *Nat Rev Neurosci* 13: 636–650.

93. Casey BJ, Galván A, Somerville LH (2016): Beyond simple models of adolescence to an integrated circuit-based account: A commentary. *Dev Cogn Neurosci* 17: 128–130.

94. Shulman EP, Smith AR, Silva K, Icenogle G, Duell N, Chein J, Steinberg L (2016): The dual systems model: Review, reappraisal, and reaffirmation. *Dev Cogn Neurosci* 17: 103–117.

95. Steinberg L (2010): A dual systems model of adolescent risk-taking. *Dev Psychobiol* 52: 216–224.

96. Meisel SN, Fosco WD, Hawk LW, Colder CR (2019): Mind the gap: A review and recommendations for statistically evaluating Dual Systems models of adolescent risk behavior. *Dev Cogn Neurosci* 39: 100681.

97. Pfeifer JH, Berkman ET (2018): The development of self and identity in adolescence: neural evidence and implications for a value-based choice perspective on motivated behavior. *Child Dev Perspect* 12: 158–164.

98. Knoll LJ, Magis-Weinberg L, Speekenbrink M, Blakemore S-J (2015): Social influence on risk perception during adolescence. *Psychol Sci* 26: 583–592.

99. Knoll LJ, Leung JT, Foulkes L, Blakemore S-J (2017): Age-related differences in social influence on risk perception depend on the direction of influence. *J Adolesc* 60: 53–63.

100. Chein J, Albert D, O'Brien L, Uckert K, Steinberg L (2011): Peers increase adolescent risk taking by enhancing activity in the brain's reward circuitry. *Dev Sci* 14: F1–F10.

101. Gardner M, Steinberg L (2005): Peer influence on risk taking, risk preference, and risky decision making in adolescence and adulthood: An experimental study. *Dev Psychol* 41: 625.

102. Shepherd JL, Lane DJ, Tapscott RL, Gentile DA (2011): Susceptible to social influence: risky "driving" in response to peer pressure. *J Appl Soc Psychol* 41: 773–797.

103. Cascio CN, Carp J, O'Donnell MB, Tinney FJ, Bingham CR, Shope JT, *et al.* (2015): Buffering social influence: neural correlates of response inhibition predict driving safety in the presence of a peer. *J Cogn Neurosci* 27: 83–95.

104. Bingham CR, Simons-Morton BG, Pradhan AK, Li K, Almani F, Falk EB, *et al.* (2016): Peer passenger norms and pressure: experimental effects on simulated driving among teenage males. *Transp Res Part F Traffic Psychol Behav* 41: 124–137.

105. Peake SJ, Dishion TJ, Stormshak EA, Moore WE, Pfeifer JH (2013): Risk-taking and social exclusion in adolescence: neural mechanisms underlying peer influences on decision-making. *NeuroImage* 82: 23–34.

106. Do K, Prinstein MJ, Eva H Telzer (In Press): Neurobiological susceptibility to peer influence in adolescence. *In K.C. Kadosh (Ed.), Handbook of Developmental Cognitive Neuroscience.* Oxford University Press.

107. Defoe IN, Semon Dubas J, Romer D (2019): Heightened adolescent risk-taking? Insights from lab studies on age differences in decision-making. *Policy Insights Behav Brain Sci* 6: 56–63.

108. Reynolds EK, MacPherson L, Schwartz S, Fox NA, Lejuez CW (2014): Analogue study of peer influence on risk-taking behavior in older adolescents. *Prev Sci Off J Soc Prev Res* 15: 842–849.

109. Tomova L, Pessoa L (2018): Information about peer choices shapes human risky decision-making. *Sci Rep* 8: 1–9.

110. Braams BR, Davidow JY, Somerville LH (2019): Developmental patterns of change in the influence of safe and risky peer choices on risky decision-making. *Dev Sci* 22: e12717.

111. Foulkes L, Leung JT, Fuhrmann D, Knoll LJ, Blakemore S-J (2018): Age differences in the prosocial influence effect. *Dev Sci* 21: e12666.

112. Hoorn J van, Dijk E van, Meuwese R, Rieffe C, Crone EA (2016): Peer influence on prosocial behavior in adolescence. *J Res Adolesc* 26: 90–100.

113. Pei R, Lauharatanahirun N, Cascio CN, O'Donnell MB, Shope JT, Simons-Morton BG, *et al.* (2020): Neural processes during adolescent risky decision making are associated with conformity to peer influence. *Dev Cogn Neurosci* 100794.

114. Andrews JA, Tildesley E, Hops H, Li F (2002): The influence of peers on young adult substance use. *Health Psychol Off J Div Health Psychol Am Psychol Assoc* 21: 349–357.

115. Dishion TJ, Owen LD (2002): A longitudinal analysis of friendships and substance use: bidirectional influence from adolescence to adulthood. *Dev Psychol* 38: 480–491.

116. Monahan KC, Steinberg L, Cauffman E (2009): Affiliation with antisocial peers, susceptibility to peer influence, and antisocial behavior during the transition to adulthood. *Dev Psychol* 45: 1520–1530.

117. Samek DR, Goodman RJ, Erath SA, McGue M, Iacono WG (2016): Antisocial peer affiliation and externalizing disorders in the transition from adolescence to young adulthood: Selection versus socialization effects. *Dev Psychol* 52: 813–823.

118. Samek DR, Hicks BM, Keyes MA, Iacono WG, McGue M (2017): Antisocial peer affiliation and externalizing disorders: Evidence for Gene × Environment × Development interaction. *Dev Psychopathol* 29: 155–172.

119. Bergen SE, Gardner CO, Kendler KS (2007): Age-related changes in heritability of behavioral phenotypes over adolescence and young adulthood: a meta-analysis. *Twin Res Hum Genet Off J Int Soc Twin Stud* 10: 423–433.

120. Rhee SH, Waldman ID (2002): Genetic and environmental influences on antisocial behavior: a meta-analysis of twin and adoption studies. *Psychol Bull* 128: 490–529.

121. Sebastian CL, Tan GCY, Roiser JP, 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: 686–694.

122. Sebastian C, Viding E, Williams KD, Blakemore S-J (2010): Social brain development and the affective consequences of ostracism in adolescence. *Brain Cogn* 72: 134–145.

123. Will G-J, van Lier PAC, Crone EA, Güroğlu B (2016): Chronic childhood peer rejection is associated with heightened neural responses to social exclusion during adolescence. *J Abnorm Child Psychol* 44: 43–55.

124. Blakemore S-J (2018): Avoiding social risk in adolescence: *Curr Dir Psychol Sci* 27: 116-122.

125. McLaughlin KA, Hatzenbuehler ML, Hilt LM (2009): Emotion dysregulation as a mechanism linking peer victimization to internalizing symptoms in adolescents. *J Consult Clin Psychol* 77: 894–904.

126. Beeri A, Lev-Wiesel R (2012): Social rejection by peers: a risk factor for psychological distress. *Child Adolesc Ment Health* 17: 216–221.

127. Pickering L, Hadwin JA, Kovshoff H (2019): The role of peers in the development of social anxiety in adolescent girls: A systematic review. *Adolesc Res Rev* 1-22

128. Slavich GM, O'Donovan A, Epel ES, Kemeny ME (2010): Black sheep get the blues: A psychobiological model of social rejection and depression. *Neurosci Biobehav Rev* 35: 39–45.

129. Andrews JL, Foulkes LE, Bone JK, Blakemore S-J (2020): Amplified concern for social risk in adolescence: development and validation of a new measure. *Brain Sci* 106: 397.

130. Telzer EH, van Hoorn J, Rogers CR, Do KT (2018): Social influence on positive youth development: A developmental neuroscience perspective. *Adv Child Dev Behav* 54: 215–258.

131. Choukas-Bradley S, Giletta M, Cohen GL, Prinstein MJ (2015): Peer influence, peer status, and prosocial behavior: An experimental investigation of peer socialization of adolescents' intentions to volunteer. *J Youth Adolesc* 44: 2197–2210.

132. Padilla-Walker LM, Fraser AM, Black BB, Bean RA (2015): Associations between friendship, sympathy, and prosocial behavior toward friends. *J Res Adolesc* 25: 28–35.

133. Chierchia G, Pi-Sunyer BP, Blakemore S-J (2020): Prosocial influence and opportunistic conformity in adolescents and young adults. *Psychol Sci.*

134. Telzer EH, Masten CL, Berkman ET, Lieberman MD, Fuligni AJ (2011): Neural regions associated with self control and mentalizing are recruited during prosocial behaviors towards the family. *NeuroImage* 58: 242–249.

135. Do KT, Guassi Moreira JF, Telzer EH (2017): But is helping you worth the risk? Defining Prosocial Risk Taking in adolescence. *Dev Cogn Neurosci* 25: 260–271.

136. Duell N, Steinberg L (2019): Positive risk taking in adolescence. *Child Dev Perspect* 13: 48–52.

137. Duell N, Steinberg L (2020): Differential correlates of positive and negative risk taking in adolescence. *J Youth Adolesc* 49: 1162–1178.

138. Andrews JL, Foulkes L, Blakemore S-J (2020): Peer influence in adolescence: Public-health implications for COVID-19. *Trends Cogn Sci* 24: 585-587.

139. Paluck EL, Shepherd H, Aronow PM (2016): Changing climates of conflict: A social network experiment in 56 schools. *Proc Natl Acad Sci* 113: 566–571.

140. Campbell R, Starkey F, Holliday J, Audrey S, Bloor M, Parry-Langdon N, *et al.* (2008): An informal school-based peer-led intervention for smoking prevention in adolescence (ASSIST): a cluster randomised trial. *The Lancet* 371: 1595–1602.

141. Carter RM, Bowling DL, Reeck C, Huettel SA (2012): A distinct role of the temporal-parietal junction in predicting socially guided decisions. *Science* 337: 109–111.

142. Saxe R, Kanwisher N (2003): People thinking about thinking people. The role of the temporo-parietal junction in "theory of mind." *NeuroImage* 19: 1835–1842.

143. Frith CD (2007): The social brain? *Philos Trans R Soc Lond B Biol Sci* 362: 671–678.

144. Pelphrey KA, Viola RJ, McCarthy G (2004): When strangers pass: processing of mutual and averted social gaze in the superior temporal sulcus. *Psychol Sci* 15: 598–603.

145. Puce A, Perrett D (2003): Electrophysiology and brain imaging of biological motion. *Philos Trans R Soc Lond B Biol Sci* 358: 435–445.

146. Olson IR, McCoy D, Klobusicky E, Ross LA (2013): Social cognition and the anterior temporal lobes: a review and theoretical framework. *Soc Cogn Affect Neurosci* 8: 123–133.

147. Schurz M, Radua J, Aichhorn M, Richlan F, Perner J (2014): Fractionating theory of mind: a meta-analysis of functional brain imaging studies. *Neurosci Biobehav Rev* 42: 9–34.