Studying individual differences in human adolescent brain development

Lucy Foulkes and Sarah-Jayne Blakemore*

UCL Institute of Cognitive Neuroscience, 17 Queen Square, London WC1N 3AR, UK

*Corresponding author: s.blakemore@ucl.ac.uk

Abstract

Adolescence is a period of social, psychological and biological development. During adolescence, relationships with others become more complex, peer relationships are paramount and there is significant development of social cognition. These psychosocial changes are paralleled by structural and functional changes in the brain. Existing research in adolescent neurocognitive development has focussed largely on averages, but this obscures meaningful individual variation in development. In this Perspective, we propose that the field should now move towards studying individual differences. We start by discussing individual variation in structural and functional brain development. To illustrate the importance of considering individual differences in development, we consider three sources of variation that contribute to neurocognitive processing: socioeconomic status, culture and peer environment. To assess individual differences in neurodevelopmental trajectories, large-scale longitudinal datasets are required. Future developmental neuroimaging studies should attempt to characterise individual differences to move towards a more nuanced understanding of neurocognitive changes during adolescence.

Introduction

Adolescence, the stage of life that begins with puberty and ends with adult independence, is a period of profound social, psychological and biological change. It is a time of social reorientation, during which adolescents spend more time with peers[1] and peers increasingly affect adolescents' self-concept, wellbeing and behaviour[2–5]. Several key aspects of social cognition continue to develop during adolescence[6,7]. Compared with adults, adolescents demonstrate heightened effects of peer influence on risk taking[8], risk perception[9,10] and reasoning[11], hypersensitivity to social exclusion[12,13], and reduced use of other people's perspective in decision making[14]. In parallel with these psychosocial changes, adolescence is characterised by biological changes, including the hormonal and physical changes that characterise puberty and substantial development of the brain.

The field of human adolescent neurocognitive development has expanded rapidly over the past two decades, and the field is now rich with neuroimaging studies demonstrating significant structural and functional development of the brain during this period of life. Most of these studies have focused on average brain development, and this group-based approach is useful because it improves signal-to-noise ratio and increases statistical power in studies that often have relatively small sample sizes[15]. However, adolescence is not the same for everyone. There are striking individual differences in both behavioural and biological development. By averaging across participants, we are not addressing the fact that adolescents, and their brains, develop in meaningfully different ways. In this paper, we review some of the literature on individual differences in adolescent development and propose that addressing individual variation is an important next step for the field of adolescent neuroscience.

We start by examining evidence for individual differences in adolescent brain development, and then describe the emerging evidence base that individual differences in socioeconomic status (SES), culture and peer environment contribute to variation in adolescent brain development and behaviour. There are many other factors that influence neurocognitive development; these three factors were selected as examples to illustrate the importance of looking at individual differences in adolescence. For the purpose of this Perspective, *SES* is defined as an individual's social and economic position in relation to others. In children and adolescents, SES is typically based on family income and/or parental education. *Culture* is defined here as a system of social norms, beliefs and values that are shared by a large group of people[16]. Cross-cultural studies may compare groups of individuals across countries or different cultures within a country. Finally, *peer environment* is defined here as the relationships and interactions a person experiences with people of a similar age. At the end of this paper, we make recommendations for studying individual differences in neurocognitive development during adolescence.

Brain development at an individual level

The human brain undergoes significant structural change during adolescence, in terms of grey matter volume, surface area and cortical thickness, as well as white matter volume and microstructure[17–19]. Recent analyses have shown that trajectories of structural development across the cortex are remarkably consistent in four longitudinal cohorts of child, adolescent and young adult participants from three different countries[18,20]. Cortical grey matter volume increases in early childhood[21], and volume and thickness decline at an accelerated pace in frontal, parietal and temporal cortices throughout adolescence, levelling off in the twenties[18]. Cerebral white matter increases linearly throughout childhood and adolescence[18,20].

Subcortical regions also undergo structural development in adolescence, with substantial heterogeneity in average trajectories across regions[22,23]. One study used a mixed cross-sectional and longitudinal design with 147 participants aged 7-24 years, 53 of whom were scanned two or more times[22]. Averaging across the cohort, some structures decreased in grey matter volume as age increased (caudate, putamen, nucleus accumbens), whilst others showed an inverted U-shaped trajectory (amygdala, cerebellum, hippocampus, pallidum and thalamus; see Figure 1[22]). A recent accelerated longitudinal study of 270 participants aged 8-28 years, with up to three scans each, indicated that there are distinct developmental trajectories within subregions of the hippocampus[23].

[Figure 1 here]

Inspection of the raw data in all these studies reveals large variance in structural development trajectories in both cortical and subcortical regions (see Figure 2[18]). It is likely that both the intercepts (overall level, e.g. volume) and slopes (i.e. trajectories) are subject to individual differences. However, few studies have statistically evaluated individual differences, and those that do tend to model subject level intercepts only, not slopes. This is partly due to constraints in existing data sets: in order to model individual differences in trajectories, scans from the same individual at multiple time points are needed. The majority of existing cohort data sets are from studies that have employed accelerated longitudinal designs, in which multiple single cohorts, each starting at a different age, are scanned two or more times within a relatively narrow age range. The scarcity of data from individual participants over several time points over an extended period of time (from late childhood to early adulthood), and the

relatively small sample sizes, have generally precluded the possibility of statistically modelling individual differences.

[Figure 2 here]

One study attempted to address this by examining the relative development of three brain regions: the prefrontal cortex (PFC), the amygdala and the nucleus accumbens[24]. The age at which each brain region matured was defined as a stabilisation of grey matter volume (note this is just one way of defining brain maturity and there are other possibilities[25]). Maturation was assessed using two analyses: one averaged across participants and the other analysed trajectories at an individual level. The analysis that averaged across participants showed that each region undergoes a slightly different developmental pattern of grey matter volume. Grey matter volume in the amygdala increased until mid-adolescence when it stopped changing; there was a shallow decline in volume in the nucleus accumbens throughout adolescence; and there was a substantial and protracted decline in the PFC throughout adolescence. However, inspecting individual trajectories revealed that this pattern did not apply uniformly to all participants (Figure 3[24]). Instead, there was wide individual variation in patterns of brain development, with some individuals showing very different maturity rates between regions, while others showed no difference. This study included 152 scans from 33 participants out of the very large NIMH cohort (all participants required at least three scans spanning late childhood, adolescence and early adulthood, and those scans needed to be of sufficiently high quality in the three regions of interest). The individual trajectories were not statistically evaluated, but were instead visually inspected by three independent researchers[24]. Despite

these limitations, this analysis suggests that structural development is not uniform across adolescents and differs both in terms of intercept and slope.

[Figure 3 here]

Functional MRI studies employing paradigms that assess different cognitive and social-emotional processes have demonstrated that, on average, neural activity also shows age-related changes during adolescence (e.g.[26,27]). However, few studies have assessed whether adolescents show individual differences in these trajectories. The majority of fMRI studies compare age groups in cross-sectional designs; there are very few longitudinal studies assessing the same individuals over multiple time points on the same task[28]. This is partly because of challenges associated with longitudinal fMRI studies, including the difficulty in disentangling genuine age-related changes from test-retest reliability error[29,30]. Cross-sectional developmental fMRI studies of, for example, risk-taking show significant individual differences[31], as do the small number of longitudinal fMRI studies that have been conducted (e.g.[27,32]), indicating that functional activity may also have different developmental trajectories across different adolescents.

Many different genetic and environmental factors play a role in determining individual brain developmental trajectories (both structural and functional), including puberty stage, gender, nutrition and the social, family and school environment. To illustrate the impact different environments can have on individual neurocognitive development, in the next sections we discuss examples of three social environmental sources of individual differences: SES, culture and the peer environment.

Socioeconomic status

The socioeconomic environment in which a child grows up has a significant effect on many aspects of development, including physical and mental health, and the way in which the brain develops[33,34]. In one cross-sectional study of 1099 individuals aged three to 20 years, number of years of parental education was associated with larger cortical surface area in many brain regions involved in language, reading, social cognition, executive functions and spatial skills[35]. Family income was logarithmically associated with cortical surface area: for individuals from lower income families, small increments in income were associated with larger differences in surface area relative to the same increments in higher income families[35]. Another study with 5 to 18 year olds showed an interaction between SES and age on grey matter volume in the amygdala and hippocampus (see Figure 4[36]). For individuals with the highest SES, older age was associated with *increased* left inferior frontal gyrus and superior temporal gyrus volume, while for individuals with the lowest SES, older age was associated with *decreased* volume in these areas. These studies demonstrate that SES affects brain development, but our understanding of this relationship is incomplete. SES might moderate the way in which participants complete a cognitive task, leading to differences in brain structure and function, or directly mediate the relationship between brain development and cognitive outcomes, and/or affect brain development via distal factors such as chronic stress or nutrition[34]. Although the exact relationship is unclear, the two studies described above illustrate the importance of combining SES with age to obtain a more nuanced understanding of individual differences in adolescent development. Future studies should attempt to characterise the mechanisms through which SES affects brain development.

[Figure 4 here]

In adolescent and young adult samples, SES has been associated with neural response to social cognition tasks. In one study, 12-13 year olds underwent fMRI whilst passively viewing emotional faces. Adolescents' SES (measured by household income and parental education) was negatively associated with activity in both the dorsomedial PFC and amygdala whilst viewing angry faces (see Figure 5[37]). Muscatell and colleagues also investigated the effect of self-reported social status on brain activity associated with mentalising, the process of attributing mental states to others[37]. Undergraduate students aged 18 to 24 years old (late adolescence and early adulthood) viewed photos of faces, purportedly of other students, and read first-person passages supposedly written by the person in the photograph – this was the mentalising condition[37]. In the non-mentalising condition, participants were asked to view and read about inanimate objects. Participants reported their perceived social status: where on a hierarchy they saw themselves relative to their university peers with respect to wealth, education and job prospects. This is a subjective report of social status that is related to SES, which is typically assessed with objective measures of a person's standing relative to their peers (e.g. family income)[37]. The results showed that self-reported social status was associated with differences in activation during this task. Lower self-reported status was associated with heightened activity in the medial prefrontal cortex, precuneus and left posterior superior temporal sulcus in the mentalising condition[37]. However, the studies tested single age groups, so the developmental trajectory of neural processing during these tasks, and their relationship with SES, is not known. A number of studies have shown that children with low SES (measured by family income) perform less well in mentalising tasks (e.g.[38]), but to our knowledge the neural correlates of this have not been assessed. Together, the studies provide initial evidence that SES is associated with neurocognitive performance in social tasks in childhood and adolescence. Future studies could assess wider age ranges, ideally from late childhood to early adulthood, to provide a

more complete picture of how individual differences in SES affect the neural correlates of mentalising across development. This is an important question as studies have shown that mentalising performance[14,39,40] and the brain regions it relies on[6], continue to develop throughout adolescence.

[Figure 5 here]

Individual differences in SES are also associated with the neural response to social exclusion, which is often assessed using an online ball-throwing game called Cyberball. In this paradigm, the participant plays a game of catch with two online (fictitious) players[13,41,42]. In the first round, the other players throw the ball to the participant and involve him/her in the game (social inclusion). In the second round, the other players initially throw the participant the ball but then stop, and only throw it to each other for the rest of the game (social exclusion). In adolescence, there is affective and neural hypersensitivity to social exclusion in this game (e.g.[43]). For example, adolescents who experienced social exclusion in the Cyberball task (relative to social inclusion) showed increased activation in the anterior insula (AI) and subgenual anterior cingulate cortex (sgACC), and this activation was positively correlated with self-reported distress[12]. However, one study with 16-17 year old males showed that this pattern of activation was moderated by SES[44]. Participants played Cyberball while undergoing fMRI, and then played a driving simulator game in which social conformity (engaging in risky behaviour suggested by a confederate) was assessed. For individuals with low SES, as measured by fathers' education level, increased activity in a number of regions was associated with increased conformity in the driving game, including the ACC, AI, ventral striatum (VS), ventromedial and dorsomedial prefrontal cortices (vmPFC, dmPFC) and

temporal parietal junction (TPJ). For those with high SES, increased activity in these regions was associated with decreased conformity[44]. The authors highlight that these areas have previously been implicated in affect (ACC, AI), reward (VS, vmPFC) and mentalising (TPJ, vmPFC), but it is not clear why SES would moderate the relationship between activity in these regions and subsequent levels of social conformity.

Together, these studies demonstrate that SES is linked to differences in brain structure during development and neural activity during social cognitive tasks in adolescence. It is not routine for SES to be analysed in cognitive neuroscience studies of adolescent development, but these results suggest that it could be linked to meaningful individual differences, and should be taken into account[34].

Culture

Adolescents around the world grow up in very different cultures, each of which has a specific framework of customs, beliefs and expectations of adolescent behaviour[45]. Societal expectations of adolescence differ widely between different cultures: some expect and enable young people to remain in full-time education and live with caregivers throughout the teenage years and into the twenties; in others, young people are expected to become financially independent from a much younger age, and to start their own families as soon as they reach sexual maturity[45]. Despite these large differences in societal expectations, there are some remarkable similarities in adolescent behavioural development across cultures, in terms of self-regulation (the ability to monitor and control one's behaviour and emotions) and sensation seeking (the desire to experience novelty and take risks)[46]. Across most of the 11 countries included in this study, self-regulation improved linearly during adolescence and plateaued in the mid-twenties, whereas sensation seeking increased between late childhood and adolescence, was highest in the late teens, and then declined throughout the twenties[46].

However, the pattern was not uniform across countries. Cross-cultural disparity was more pronounced in a study assessing differences in adolescent risk taking in the same 11 countries[47]. Participants aged 10-30 completed self-report questionnaires of health and antisocial risk taking and two experimental tasks: the Stoplight task, which assesses risks taken in a driving simulator game, and the Balloon Analogue Risk Task (BART), in which money is gained for inflating a balloon and lost if the balloon bursts, which it can do at any point[47]. There were variations in trajectories across countries. For example, risk taking on the Stoplight driving task showed a quadratic and linear pattern across age in India, Jordan and the Philippines, a linear and quadratic pattern across age in China, Italy and the United States, a negative linear trajectory across age in Colombia, and no association with age in Cyprus, Kenya, Sweden and Thailand[47]. The results indicate that the varying cultures in which adolescents grow up can lead to individual differences in their behavioural development, but the neurocognitive development that underlies these differences is not known.

Cultural neuroscience is an emerging field that assesses the relationship between culture and brain structure and function, and studies in adult groups have demonstrated differences in neural activity across cultures when completing a range of cognitive tasks (e.g.[45]). However, few studies have investigated cultural differences in the development of the adolescent brain, despite recognition that this is a critical future direction for cultural neuroscience[48] and understanding that adolescents hold very different societal roles across cultures[45]. One of the few adolescent studies in this area was an fMRI study that asked White and Latino American adolescents to play a game to earn money for themselves or for their family, and showed that giving to the family was associated with different patterns of brain activity in the two cultural groups[49]. Although there was comparable behavioural performance between the two groups, White participants showed more activity in the VS, dorsal striatum (DS) and ventral tegmental area (VTA) when winning money for themselves compared to winning for their

family[49]. In contrast, Latino participants showed similar (VS) or increased (DS, VTA) activity when winning for their family than for themselves[49]. The authors hypothesise that this difference in activation may reflect cultural differences in how much time adolescents spend helping their families, such as caring for siblings or assisting with household tasks. American adolescents from Latino backgrounds spend more time helping their families than those from European backgrounds[50], possibly because adolescents from different cultures have varying degrees of family obligation – the sense of duty felt towards helping their family[51]. In support of this, in the fMRI study, activity in the VS, DS and VTA when winning for family was positively associated with self-reported enjoyment and satisfaction when helping the family (for both cultural groups)[49].

Individual differences in family obligation have also been associated with risk taking. One study of 14-16 year olds from Mexican backgrounds found that those with higher levels of family obligation were less likely to take risks in the Balloon Analogue Risk Task (adolescents from other backgrounds were not assessed)[52]. The study also found that family obligation values were associated with reduced activity in the VS when the participants received monetary reward (for themselves)[52]. These studies suggest that cultural differences in family relationships may be linked to significant neurocognitive differences and risk taking in adolescents.

There are cultural differences in susceptibility to peer influence in adolescence. It is well established in Western samples that, relative to adults, adolescents are especially susceptible to peer influence[9,10,53]. To date, there have been mixed findings on the impact of culture on peer influence. Some studies have showed that peer substance use influences adolescents' own substance use across a range of industrialised cultures (Hong Kong[54]; USA/UK[55]). One study directly compared adolescents from the US and China and found that in both countries adolescents' smoking is equally strongly influenced by peer smoking[56]. Within US samples,

however, several older studies have demonstrated that peer influence is a predictor of smoking in White adolescents but not Black adolescents[57], and a stronger predictor of smoking for White adolescents than other ethnic groups including Asian and Latino adolescents[58] and Pacific Islanders[59]. This may be because in some cultures conformity to family norms is paramount, and family attitudes might have a stronger influence on smoking behaviour than peers[58].

Future research should explore the possible neurocognitive mechanisms underlying these cultural differences in adolescents' susceptibility to social influence, and broaden the focus away from only smoking behaviour. In a study of Mexican-American 16-18 year olds[60], a task assessing susceptibility to social influence (measured by how much participants changed their ratings of artworks after seeing likeability ratings from others) elicited activity in regions associated with mental state reasoning (medial prefrontal cortex, temporal parietal junction) and self-control (ventrolateral prefrontal cortex). However, the study did not include adolescents from other cultural groups. A study of 14-18 year old American adolescents (ethnicity not reported) found that increased risk-taking in the presence of peers was modulated by increased activation in the VS, a region that has been implicated in reward processing[8]. However, this study did not assess cultural differences in the neural response to peer influence on risk-taking. A speculative possibility is that adolescents from cultures that show reduced susceptibility to peer influence may exhibit higher activation in brain regions associated with self-control, and/or reduced activation in reward-related regions, when making decisions in the presence of peers. It is also unclear how culture affects susceptibility to peer influence across age, as existing studies have typically focused on adolescent age groups only, or used wider age groups but not reported cultural differences. For example, a decrease in social influence from late childhood (age 8-10) to adulthood (age 25+) has been reported[9,10,53], but ethnicity was not analysed in these studies, so it is unclear whether this

linear decrease is uniformly true for all cultures. The studies on smoking indicate that adolescents of different ethnicities may be differently influenced by peer smoking[57–59], but it is unclear how these cultures affect the *trajectory* of social influence across age.

Peer environment

During adolescence, individuals develop an increasingly complex network of relationships with their peers[61]. The pattern of interactions that an adolescent has with his or her peers varies between individuals. First, adolescents differ with respect to how frequently they are victimised by their peers: some adolescents are never bullied, whilst others report a chronic history of being rejected and victimised[62–65]. Second, adolescents vary both in the number of friends they have and the quality of those friendships, such as the extent to which they feel understood and supported by their friends[66]. This has a significant impact on their mental health and well-being[62–66] and can affect both their behavioural and neural responses to social interactions[61]. As such, peer relationships are an important source of individual variation that should be assessed when investigating neurocognitive development in adolescence.

Adolescents with a history of repeated rejection by peers (as measured by retrospective self-report) show a different neural response to social exclusion assessed with the Cyberball paradigm[67]. Specifically, compared with stably accepted adolescents (no history of peer rejection), chronically rejected adolescents display higher activity in the dACC during social exclusion[67]. One study found that 14-16 years old girls with a history of being victimized had higher levels of risk-taking in a simulated driving task, as well as increased activation during risky decisions (amygdala, mPFC, medial posterior parietal junction, posterior parietal junction, TPJ and VS), compared with girls who had experienced low levels of peer victimisation[68]. Social exclusion has also been associated with subsequent risk taking in

typical samples[69]. A second study showed that adolescents with self-reported lower levels of resistance to peer influence were especially likely to take risks in driving games after being socially excluded and this was mediated by neural activity in the right TPJ[70]. Differences in neural activity after Cyberball are also linked to symptoms of psychopathology: in one study of adolescent girls, activation during social exclusion in the dACC, sgACC and AI was associated with depression and social anxiety symptoms, and this link was stronger in individuals who had been chronically victimized compared to those who had not[71].

Conversely, a positive social environment can have protective long-term benefits for an adolescent. For example, one study with 14-24 year olds found that self-reported friendship quality support predicted better psychosocial functioning one year later[66]. In another study, positive peer relationships reduced the association between negative parenting practices and later antisocial behaviour (e.g. getting in fights) in young adolescents[72] and reduced the association between peer conflict and risk taking[73].

The fMRI and behavioural studies reviewed here indicate that an adolescent's peer environment can affect their development in both negative and positive ways. Others have argued that individual differences in neurobiology can determine how sensitive an adolescent is to their social context, indicating that identical social environments might affect different individuals in different ways[61,74]. For example, adolescents who are particularly hypervigilant to social threat cues may be at risk of developing social anxiety disorder or other internalising problems[75]. Together, this research highlights that individual differences in peer environment should be measured to understand better why adolescents respond differently in neurocognitive tasks assessing social interactions.

Limitations

There are several limitations of the current paper. First, many factors not reviewed here also play a critical role in individual variation in adolescent development. Other social environmental factors that influence adolescent development in addition to the three we highlight here include parenting style[74,76–78], sibling number and relationships[79] and school environment[80,81]. Another important source of variation is puberty status. Most studies have analysed structural trajectories as a function of age, but chronological age and puberty stage are not tightly associated in late childhood and early adolescence: there is substantial individual variation in puberty development. Studies that have included an estimate of puberty (such as Tanner stage) have demonstrated variance in structural and functional brain development over and above chronological age alone (e.g.[82–84]). As such, we recognise that the three social environmental factors here likely have interactive effects with pubertal stage to determine brain development in adolescence.

Second, like all environmental factors, the three reviewed here do not exert their influence in isolation from each other; there are important interrelations between them. For example, there are significant cultural differences in the prevalence of adolescents reporting peer victimisation (for instance, there are relatively high levels in Baltic countries[85]) and the risk of being victimised is increased in low SES adolescents[86]. Indicators of SES are strongly associated with ethnicity[87].

A third limitation is that environmental factors act in concert with genetics to affect development in a number of ways. Social context can trigger, or protect from, a genetic risk factor[88]. One developmental example is that the family environment can *interact* with a child's genes to influence the neural, behavioural and mental health consequences of maltreatment[89]. Carriers of the MAOA-l allele who have suffered maltreatment in childhood are more likely than individuals who do not carry this allele to develop antisocial behaviour

disorders, possibly because the MAOA-l allele is associated with hyper-responsiveness in brain regions that detect threat and reduced activation in brain regions responsible for emotional control[90]. This leads MAOA-l carriers who have been maltreated to be especially susceptible to later reactive aggression and violence[90]. Genes and the social environment can also be *correlated* with one another. For example, a shy child might elicit different behaviour from their family and peers[91]. Thus, there are complex interactions and correlations between an individual's genes, pubertal status and the environment in which he or she grows up, which are important to take into account when considering adolescent brain development.

It is important to note that there are issues inherent in the current imaging technology that limit the extent to which individual brain development can be investigated[15], which have contributed to the aforementioned limitations in the field. For example, precisely because of individual differences in brain structure and function, it is difficult to be confident that functionally equivalent regions are identified across subjects, and to account for individual differences in the haemodynamic response function[15].

Suggestions for future research

Studies of adolescent brain development typically report group-based averages, which highlight important changes in development across this period. Future studies should consider within-group variance in order to obtain a more nuanced picture of adolescent neurocognitive development. There are a number of issues that need to be addressed in order to conduct studies of adolescent neurocognitive development at an individual level, and here we make a number of recommendations to guide this research field.

First, large sample sizes are required in order to have sufficient power to explore individual differences. One way to manage the requirement for large sample sizes is to utilise publically

available datasets (e.g.[92,93]), such as the Human Connectome Project[94], and the Adolescent Brain Cognitive Development study[95], although the large majority of currently available data are cross-sectional and from adults. Data sharing amongst scientists investigating adolescent brain development should be encouraged. Second, in order to track individual development across time, longitudinal designs are required[96,97]. Third, the age ranges studied need to be larger than are typically included in developmental studies, ideally spanning late childhood to early adulthood, in order to assess the entire developmental period of adolescence. Using large, longitudinal samples is especially important when assessing subcortical regions, to minimise the possibility that apparent differences in individual trajectories are due to noise. Fourth, data on relevant individual difference variables should be collected and analysed as variables of interest, for instance by extracting longitudinally modelled individual slopes or latent change scores[98] and/or using group variability measures[99]. A final suggestion is that future research should identify the specific neural systems affected by individual difference variables, in order to draw together the currently disparate findings involving a number of brain regions and systems. By combining all of these recommendations, we can start to build a truly comprehensive picture of how the brain changes across adolescence, and the individual variables that affect the trajectory of development.

Conclusion

The past 20 years has seen a rapid expansion of research into adolescent brain development. This research has largely focussed on group-based means, enabling us to draw conclusions about average adolescent development. However, adolescents are a heterogeneous group, with different trajectories of brain development and patterns of behaviour. To progress the field, sources of individual differences should be assessed as variables of interest, and not treated as statistical noise. Taking into account individual differences is particularly important

if findings from neuroscience studies are to have real life relevance, for example, in the areas of public health and education. In these domains, a one-size-fits all approach might not be appropriate. For example, the research reviewed here suggests that socioeconomic status, culture and peer environment are three sources of variance that affect neurocognitive development in adolescence, and this in turn might have implications for how different adolescents learn in school or respond to public health advertising. Individual variability should be taken into account as we continue to refine our understanding of the adolescent brain.

Acknowledgements

The authors are funded by the Wellcome Trust and the Jacobs Foundation. We thank Dr C. Tamnes for his helpful comments on an earlier draft of the manuscript.

Author contributions

LF and SJB contributed equally to the writing of this Perspective.

Competing Financial Interests Statement

The authors have no competing financial interests.

References

1. Larson, R. & Richards, M. H. Daily companionship in late childhood and early adolescence: changing developmental contexts. *Child Dev.* **62,** 284–300 (1991).
2. O'Brien, S. F. & Bierman, K. L. Conceptions and perceived influence of peer groups: Interviews with preadolescents and adolescents. *Child Dev.* **59,** 1360 (1988).
3. Harrell, A. W., Mercer, S. H. & DeRoisier, M. E. Improving the social-behavioral adjustment of adolescents: The effectiveness of a social skills group intervention. *J. Child Fam. Stud.* **18,** 378–387 (2009).
4. Gorrese, A. & Ruggieri, R. Peer attachment and self-esteem: A meta-analytic review. *Personal. Individ. Differ.* **55,** 559–568 (2013).
5. Oldehinkel, A. J., Rosmalen, J. G. M., Veenstra, R., Dijkstra, J. K. & Ormel, J. Being admired or being liked: Classroom social status and depressive problems in early adolescent girls and boys. *J. Abnorm. Child Psychol.* **35,** 417–427 (2007).
6. Blakemore, S.-J. The social brain in adolescence. *Nat. Rev. Neurosci.* **9,** 267–277 (2008).
7. Blakemore, S.-J. & Mills, K. L. Is adolescence a sensitive period for sociocultural processing? *Annu. Rev. Psychol.* **65,** 187–207 (2014).
8. Chein, J., Albert, D., O'Brien, L., Uckert, K. & Steinberg, L. Peers increase adolescent risk taking by enhancing activity in the brain's reward circuitry. *Dev. Sci.* **14,** F1–F10 (2011).
9. Knoll, L. J., Magis-Weinberg, L., Speekenbrink, M. & Blakemore, S.-J. Social influence on risk perception during adolescence. *Psychol. Sci.* **26,** 583–592 (2015).
10. Knoll, L. J., Leung, J. T., Foulkes, L. & Blakemore, S.-J. Age-related differences in social influence on risk perception depend on the direction of influence. *J. Adolesc.* **60,** 53–63 (2017).

11. Wolf, L. K., Bazargani, N., Kilford, E. J., Dumontheil, I. & Blakemore, S.-J. The audience effect in adolescence depends on who's looking over your shoulder. *J. Adolesc.* **43,** 5–14 (2015).
12. Masten, C. L. *et al.* Neural correlates of social exclusion during adolescence: Understanding the distress of peer rejection. *Soc. Cogn. Affect. Neurosci.* **4,** 143–157 (2009).
13. Sebastian, C., Viding, E., Williams, K. D. & Blakemore, S.-J. Social brain development and the affective consequences of ostracism in adolescence. *Brain Cogn.* **72,** 134–145 (2010).
14. Dumontheil, I., Apperly, I. A. & Blakemore, S.-J. Online usage of theory of mind continues to develop in late adolescence. *Dev. Sci.* **13,** 331–338 (2010).
15. Dubois, J. & Adolphs, R. Building a science of individual differences from fMRI. *Trends Cogn. Sci.* **20,** 425–443 (2016).
16. Rohner, R. P. Toward a conception of culture for cross-cultural psychology. *J. Cross-Cult. Psychol.* **15,** 111–138 (1984).
17. Giedd, J. N. *et al.* Brain development during childhood and adolescence: A longitudinal MRI study. *Nat. Neurosci.* **2,** 861–863 (1999).
18. Tamnes, C. K. *et al.* Development of the cerebral cortex across adolescence: A multisample study of inter-related longitudinal changes in cortical volume, surface area, and thickness. *J. Neurosci.* **37,** 3402–3412 (2017).

This study analysed longitudinal data from 388 individuals aged between 8 and 30 years from four large cohorts in three countries, the United States, the Netherlands and Norway (854 total scans). In all four groups, there were decreases in grey matter volume across the cortex throughout adolescence, with the largest decreases occurring in the prefrontal, parietal and temporal cortices.

19. Vijayakumar, N. *et al.* Brain development during adolescence: A mixed-longitudinal investigation of cortical thickness, surface area, and volume. *Hum. Brain Mapp.* **37,** 2027–2038 (2016).
20. Mills, K. L. *et al.* Structural brain development between childhood and adulthood: Convergence across four longitudinal samples. *NeuroImage* **141,** 273–281 (2016).
21. Gilmore, J. H. *et al.* Longitudinal development of cortical and subcortical gray matter from birth to 2 years. *Cereb. Cortex* **22,** 2478–2485 (2012).
22. Wierenga, L. *et al.* Typical development of basal ganglia, hippocampus, amygdala and cerebellum from age 7 to 24. *NeuroImage* **96,** 67–72 (2014).
23. Tamnes, C. K., Bos, M. G. N., van de Kamp, F. C., Peters, S. & Crone, E. A. Longitudinal development of hippocampal subregions from childhood to adulthood. (2017). doi:10.1101/186270

This paper assessed the structural development of subregions within the hippocampus. Data were from a large accelerated longitudinal study (N=270, 678 scans) of 8-28 year olds. The study found heterogeneity of trajectories across region, with some showing early volume increases and others showing nonlinear decreases in volume.

24. Mills, K. L., Goddings, A.-L., Clasen, L. S., Giedd, J. N. & Blakemore, S.-J. The developmental mismatch in structural brain maturation during adolescence. *Dev. Neurosci.* **36,** 147–160 (2014).
25. Somerville, L. H. Searching for signatures of brain maturity: What are we searching for? *Neuron* **92,** 1164–1167 (2016).
26. Crone, E. A., van Duijvenvoorde, A. C. K. & Peper, J. S. Annual research review: Neural contributions to risk-taking in adolescence - developmental changes and individual differences. *J. Child Psychol. Psychiatry* **57,** 353–368 (2016).

27. Simmonds, D. J., Hallquist, M. N. & Luna, B. Protracted development of executive and mnemonic brain systems underlying working memory in adolescence: A longitudinal fMRI study. *NeuroImage* **157,** 695–704 (2017).
28. Crone, E. A. & Elzinga, B. M. Changing brains: How longitudinal functional magnetic resonance imaging studies can inform us about cognitive and social-affective growth trajectories. *Wiley Interdiscip. Rev. Cogn. Sci.* **6,** 53–63 (2015).
29. Herting, M. M., Gautam, P., Chen, Z., Mezher, A. & Vetter, N. C. Test-retest reliability of longitudinal task-based fMRI: Implications for developmental studies. *Dev. Cogn. Neurosci.* (2017). doi:10.1016/j.dcn.2017.07.001
30. King, K. M. *et al.* Longitudinal modeling in developmental neuroimaging research: Common challenges, and solutions from developmental psychology. *Dev. Cogn. Neurosci.* (2017). doi:10.1016/j.dcn.2017.11.009

This provides an overview of issues involved in conducting longitudinal structural and functional studies to measure brain development across age. Suggested analytical approaches are demonstrated on simulated data, and the underlying code is available for other researchers to access.

31. Sherman, L., Steinberg, L. & Chein, J. Connecting brain responsivity and real-world risk taking: Strengths and limitations of current methodological approaches. *Dev. Cogn. Neurosci.* (2017). doi:10.1016/j.dcn.2017.05.007
32. Ordaz, S. J., Foran, W., Velanova, K. & Luna, B. Longitudinal growth curves of brain function underlying inhibitory control through adolescence. *J. Neurosci.* **33,** 18109–18124 (2013).
33. Hackman, D. A. & Farah, M. J. Socioeconomic status and the developing brain. *Trends Cogn. Sci.* **13,** 65–73 (2009).

34. Farah, M. J. The neuroscience of socioeconomic status: Correlates, causes, and consequences. *Neuron* **96,** 56–71 (2017).

35. Noble, K. G. *et al.* Family income, parental education and brain structure in children and adolescents. *Nat. Neurosci.* **18,** 773–778 (2015).

This cross-sectional study shows an association between SES and cortical surface area across age. Data were from a cohort of 1099 individuals aged 3 to 20 years old. There was a significant interaction between SES, age and surface area, highlighting the importance of including SES in studies investigating the development of brain structure.

36. Noble, K. G., Houston, S. M., Kan, E. & Sowell, E. R. Neural correlates of socioeconomic status in the developing human brain. *Dev. Sci.* **15,** 516–527 (2012).

37. Muscatell, K. A. *et al.* Social status modulates neural activity in the mentalizing network. *NeuroImage* **60,** 1771–1777 (2012).

38. Tompkins, V., Logan, J. A. R., Blosser, D. F. & Duffy, K. Child language and parent discipline mediate the relation between family income and false belief understanding. *J. Exp. Child Psychol.* **158,** 1–18 (2017).

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

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

41. Abrams, D., Weick, M., Thomas, D., Colbe, H. & Franklin, K. M. On-line ostracism affects children differently from adolescents and adults. *Br. J. Dev. Psychol.* **29,** 110–123 (2011).

42. Sebastian, C. L. *et al.* Developmental influences on the neural bases of responses to social rejection: Implications of social neuroscience for education. *NeuroImage* **57,** 686–694 (2011).
43. Vijayakumar, N., Cheng, T. W. & Pfeifer, J. H. Neural correlates of social exclusion across ages: A coordinate-based meta-analysis of functional MRI studies. *NeuroImage* **153,** 359–368 (2017).
44. Cascio, C. N., O'Donnell, M. B., Simons-Morton, B. G., Bingham, C. R. & Falk, E. B. Cultural context moderates neural pathways to social influence. *Cult. Brain* **5,** 50–70 (2017).
45. Choudhury, S. Culturing the adolescent brain: What can neuroscience learn from anthropology? *Soc. Cogn. Affect. Neurosci.* **5,** 159–167 (2010).
46. Steinberg, L. *et al.* Around the world, adolescence is a time of heightened sensation seeking and immature self-regulation. *Dev. Sci.* e12532 (2017). doi:10.1111/desc.12532
47. Duell, N. *et al.* Age patterns in risk taking across the world. *J. Youth Adolesc.* (2017). doi:10.1007/s10964-017-0752-y
48. Miller, J. G. & Kinsbourne, M. Culture and neuroscience in developmental psychology: Contributions and challenges. *Child Dev. Perspect.* **6,** 35–41 (2012).
49. Telzer, E. H., Masten, C. L., Berkman, E. T., Lieberman, M. D. & Fuligni, A. J. Gaining while giving: An fMRI study of the rewards of family assistance among White and Latino youth. *Soc. Neurosci.* **5,** 508–518 (2010).

This is one of the few fMRI studies to compare neural activity in adolescents of different cultures, in this case White and Latino Americans. When winning money for their family, Latino participants showed more activation in brain regions that have been implicated in reward processing. The paper demonstrates that individual differences in culture can be associated with different patterns of neural activity.

50. Telzer, E. H. & Fuligni, A. J. Daily family assistance and the psychological well-being of adolescents from Latin American, Asian, and European backgrounds. *Dev. Psychol.* **45,** 1177–1189 (2009).
51. Fuligni, A. J., Tseng, V. & Lam, M. Attitudes toward family obligations among American adolescents with Asian, Latin American, and European backgrounds. *Child Dev.* **70,** 1030–1044 (1999).
52. Telzer, E. H., Fuligni, A. J., Lieberman, M. D. & Galván, A. Meaningful family relationships: neurocognitive buffers of adolescent risk taking. *J. Cogn. Neurosci.* **25,** 374–387 (2013).
53. Steinberg, L. & Monahan, K. C. Age differences in resistance to peer influence. *Dev. Psychol.* **43,** 1531–1543 (2007).
54. Loke, A. & Mak, Y. Family process and peer influences on substance use by adolescents. *Int. J. Environ. Res. Public. Health* **10,** 3868–3885 (2013).
55. D'Amico, E. J. & McCarthy, D. M. Escalation and initiation of younger adolescents' substance use: The impact of perceived peer use. *J. Adolesc. Health* **39,** 481–487 (2006).
56. Unger, J. B. *et al.* Peer influences and access to cigarettes as correlates of adolescent smoking: A cross-cultural comparison of Wuhan, China, and California. *Prev. Med.* **34,** 476–484 (2002).
57. Headen, S. W., Bauman, K. E., Deane, G. D. & Koch, G. G. Are the correlates of cigarette smoking initiation different for black and white adolescents? *Am. J. Public Health* **81,** 854–858 (1991).
58. Landrine, H., Richardson, J. L., Klonoff, E. A. & Flay, B. Cultural diversity in the predictors of adolescent cigarette smoking: The relative influence of peers. *J. Behav. Med.* **17,** 331–346 (1994).

59. Unger, J. B. *et al.* Ethnic variation in peer influences on adolescent smoking. *Nicotine Tob. Res.* **3,** 167–176 (2001).
60. Welborn, B. L. *et al.* Neural mechanisms of social influence in adolescence. *Soc. Cogn. Affect. Neurosci.* **11,** 100–109 (2016).
61. Lamblin, M., Murawski, C., Whittle, S. & Fornito, A. Social connectedness, mental health and the adolescent brain. *Neurosci. Biobehav. Rev.* **80,** 57–68 (2017).
62. Arseneault, L., Bowes, L. & Shakoor, S. Bullying victimization in youths and mental health problems: 'Much ado about nothing'? *Psychol. Med.* **40,** 717–729 (2010).
63. Copeland, W. E., Wolke, D., Angold, A. & Costello, E. J. Adult psychiatric outcomes of bullying and being bullied by peers in childhood and adolescence. *JAMA Psychiatry* **70,** 419–426 (2013).
64. Takizawa, R., Maughan, B. & Arseneault, L. Adult health outcomes of childhood bullying victimization: Evidence from a five-decade longitudinal British birth cohort. *Am. J. Psychiatry* **171,** 777–784 (2014).
65. Singham, T. *et al.* Concurrent and longitudinal contribution of exposure to bullying in childhood to mental health: The role of vulnerability and resilience. *JAMA Psychiatry* **74,** 1112–1119 (2017).
66. van Harmelen, A.-L. *et al.* Adolescent friendships predict later resilient functioning across psychosocial domains in a healthy community cohort. *Psychol. Med.* **47,** 2312–2322 (2017).
67. Will, G.-J., van Lier, P. A. C., Crone, E. A. & Güroğlu, B. Chronic childhood peer rejection is associated with heightened neural responses to social exclusion during adolescence. *J. Abnorm. Child Psychol.* **44,** 43–55 (2016).
68. Telzer, E. H., Miernicki, M. E. & Rudolph, K. D. Chronic peer victimization heightens neural sensitivity to risk taking. *Dev. Psychopathol.* **10,** 1–14 (2017).

This fMRI study compared adolescents with a history of chronic peer victimisation to those with no history of being victimised. The participants with a history of victimisation took more risks in a risk-taking task and also showed heightened activation in a number of regions during the task, showing how individual differences in peer environment are associated with behavioural and neural differences.

69. Falk, E. B. *et al.* Neural responses to exclusion predict susceptibility to social influence. *J. Adolesc. Health* **54,** S22–S31 (2014).
70. Peake, S. J., Dishion, T. J., Stormshak, E. A., Moore, W. E. & Pfeifer, J. H. Risk-taking and social exclusion in adolescence: Neural mechanisms underlying peer influences on decision-making. *NeuroImage* **82,** 23–34 (2013).
71. Rudolph, K. D., Miernicki, M. E., Troop-Gordon, W., Davis, M. M. & Telzer, E. H. Adding insult to injury: Neural sensitivity to social exclusion is associated with internalizing symptoms in chronically peer-victimized girls. *Soc. Cogn. Affect. Neurosci.* **11,** 829–842 (2016).
72. Lansford, J. E., Criss, M. M., Pettit, G. S., Dodge, K. A. & Bates, J. E. Friendship quality, peer group affiliation, and peer antisocial behavior as moderators of the link between negative parenting and adolescent externalizing behavior. *J. Res. Adolesc.* **13,** 161–184 (2003).
73. Telzer, E. H., Fuligni, A. J., Lieberman, M. D., Miernicki, M. E. & Galván, A. The quality of adolescents' peer relationships modulates neural sensitivity to risk taking. *Soc. Cogn. Affect. Neurosci.* **10,** 389–398 (2015).
74. Schriber, R. A. & Guyer, A. E. Adolescent neurobiological susceptibility to social context. *Dev. Cogn. Neurosci.* **19,** 1–18 (2016).

75. Caouette, J. D. & Guyer, A. E. Gaining insight into adolescent vulnerability for social anxiety from developmental cognitive neuroscience. *Dev. Cogn. Neurosci.* **8,** 65–76 (2014).
76. Darling, N. & Steinberg, L. Parenting style as context: An integrative model. *Psychol. Bull.* **113,** 487–496 (1993).
77. Kerr, M., Stattin, H. & Özdemir, M. Perceived parenting style and adolescent adjustment: Revisiting directions of effects and the role of parental knowledge. *Dev. Psychol.* **48,** 1540–1553 (2012).
78. Kim-Spoon, J., Maciejewski, D., Lee, J., Deater-Deckard, K. & King-Casas, B. Longitudinal associations among family environment, neural cognitive control, and social competence among adolescents. *Dev. Cogn. Neurosci.* **26,** 69–76 (2017).
79. Harper, J. M., Padilla-Walker, L. M. & Jensen, A. C. Do siblings matter independent of both parents and friends? Sympathy as a mediator between sibling relationship quality and adolescent outcomes. *J. Res. Adolesc.* **26,** 101–114 (2016).
80. Bonell, C. *et al.* Initiating change locally in bullying and aggression through the school environment (INCLUSIVE): A pilot randomised controlled trial. *Health Technol. Assess.* **19,** 1–110 (2015).
81. Luengo Kanacri, B. P. *et al.* Longitudinal relations among positivity, perceived positive school climate, and prosocial behavior in Colombian adolescents. *Child Dev.* **88,** 1100–1114 (2017).
82. Goddings, A.-L. *et al.* The influence of puberty on subcortical brain development. *NeuroImage* **88,** 242–251 (2014).
83. Herting, M. M. & Sowell, E. R. Puberty and structural brain development in humans. *Front. Neuroendocrinol.* **44,** 122–137 (2017).

84. Motta-Mena, N. V. & Scherf, K. S. Pubertal development shapes perception of complex facial expressions. *Dev. Sci.* **20,** e12451 (2017).
85. Craig, W. *et al.* A cross-national profile of bullying and victimization among adolescents in 40 countries. *Int. J. Public Health* **54,** 216–224 (2009).
86. Tippett, N. & Wolke, D. Socioeconomic status and bullying: A meta-analysis. *Am. J. Public Health* **104,** e48–e59 (2014).
87. Williams, D. R., Priest, N. & Anderson, N. B. Understanding associations among race, socioeconomic status, and health: Patterns and prospects. *Health Psychol.* **35,** 407–411 (2016).
88. Shanahan, M. J. & Hofer, S. M. Social context in gene-environment interactions: Retrospect and prospect. *J. Gerontol. B. Psychol. Sci. Soc. Sci.* **60 Spec No 1,** 65–76 (2005).
89. Byrd, A. L. & Manuck, S. B. MAOA, childhood maltreatment, and antisocial behavior: Meta-analysis of a gene-environment interaction. *Biol. Psychiatry* **75,** 9–17 (2014).
90. McCrory, E., De Brito, S. A. & Viding, E. Research review: The neurobiology and genetics of maltreatment and adversity. *J. Child Psychol. Psychiatry* **51,** 1079–1095 (2010).
91. Knafo, A. & Jaffee, S. R. Gene-environment correlation in developmental psychopathology. *Dev. Psychopathol.* **25,** 1–6 (2013).
92. Kaufmann, T. *et al.* Delayed stabilization and individualization in connectome development are related to psychiatric disorders. *Nat. Neurosci.* **20,** 513–515 (2017).
93. Rosenberg, M. D. *et al.* A neuromarker of sustained attention from whole-brain functional connectivity. *Nat. Neurosci.* **19,** 165–171 (2016).
94. Van Essen, D. C. *et al.* The WU-Minn Human Connectome Project: An overview. *NeuroImage* **80,** 62–79 (2013).

95. Volkow, N. D. *et al.* The conception of the ABCD study: From substance use to a broad NIH collaboration. *Dev. Cogn. Neurosci.* (2017). doi:10.1016/j.dcn.2017.10.002
96. Madhyastha, T. *et al.* Current methods and limitations for longitudinal fMRI analysis across development. *Dev. Cogn. Neurosci.* (2017). doi:10.1016/j.dcn.2017.11.006
97. Mills, K. L. & Tamnes, C. K. Methods and considerations for longitudinal structural brain imaging analysis across development. *Dev. Cogn. Neurosci.* **9,** 172–190 (2014).
98. Kievit, R. A. *et al.* Developmental cognitive neuroscience using Latent Change Score models: A tutorial and applications. *Dev. Cogn. Neurosci.* (2017). doi:10.1016/j.dcn.2017.11.007
99. Wierenga, L. M. *et al.* A key characteristic of sex differences in the developing brain: Greater variability in brain structure of boys than girls. *Cereb. Cortex* 1–11 (2017). doi:10.1093/cercor/bhx154

Figure Legends

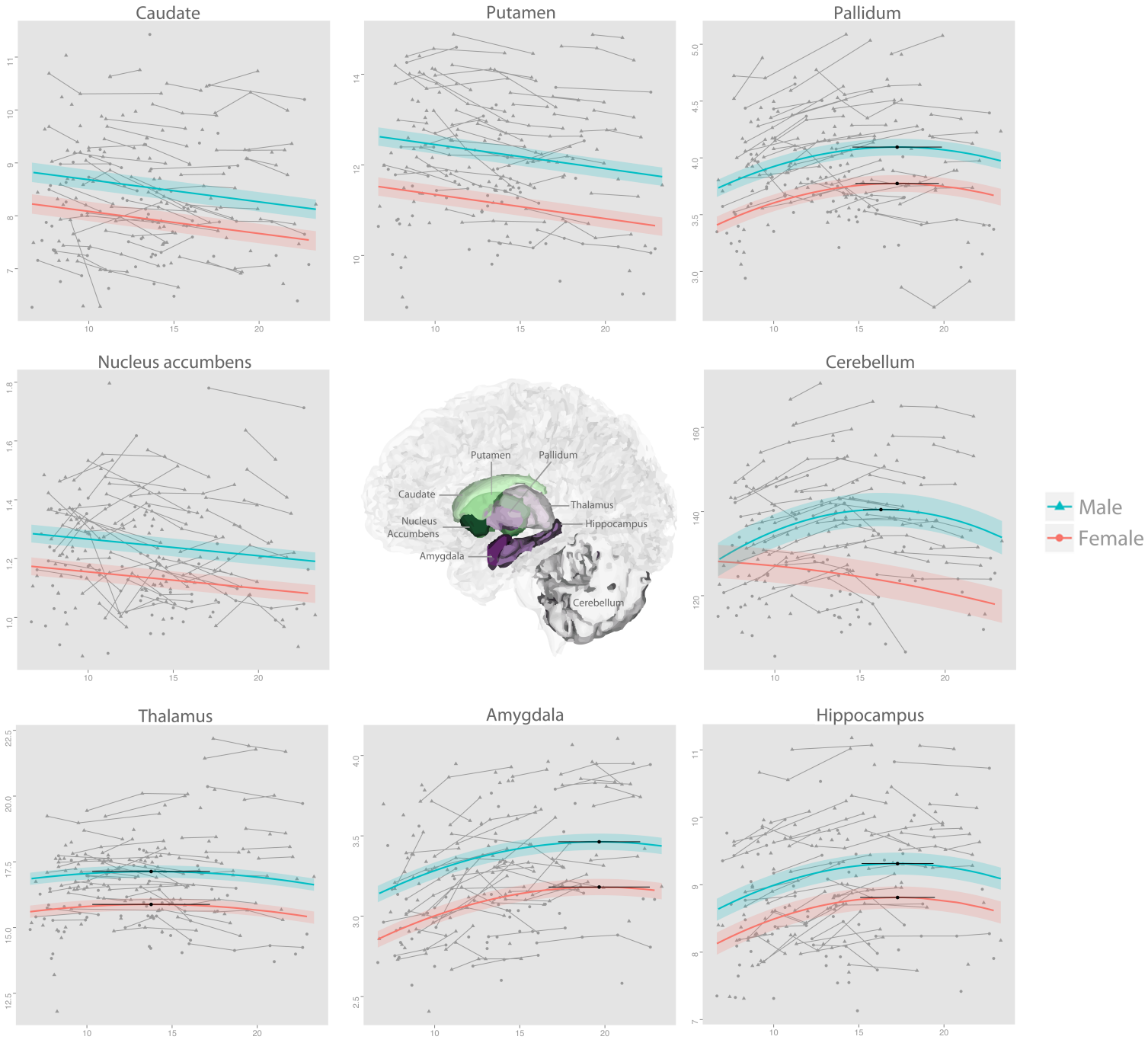
Figure 1. Developmental trajectories for total grey matter volume: Age 7.0–23.3 years old. Mean volume in cm^3 (y-axis) by age in years (x-axis) is shown for males (n = 94, blue) and females (n = 53, red). The shade around the regression lines represents the 95% confidence interval of the intercept. Reproduced from[22]

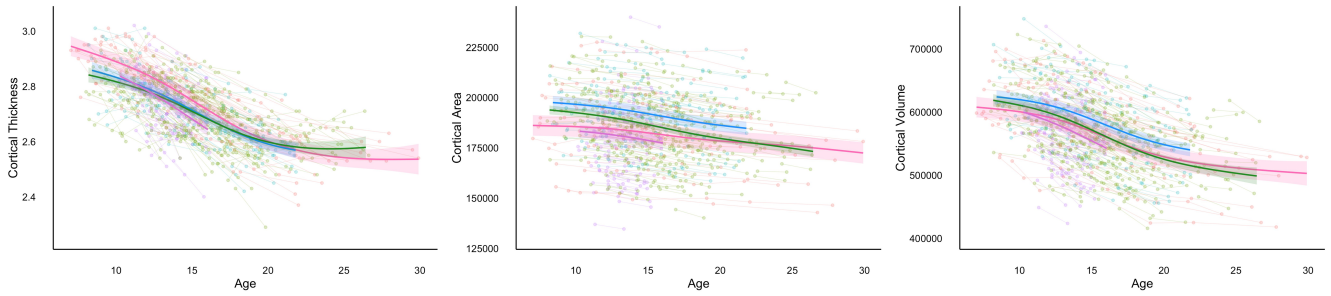
Figure 2. Developmental trajectories for global cortical measures for four different cohorts: Child Psychiatry Branch (pink), Pittsburgh (purple), Neurocognitive Development (blue) and Braintime (green). Spaghetti plots of mean cortical thickness, total cortical surface area, and total cortical volume, controlling for sex. The coloured lines represent the GAMM fitting while the lighter coloured areas correspond to the 95% confidence intervals. Reproduced from[18]

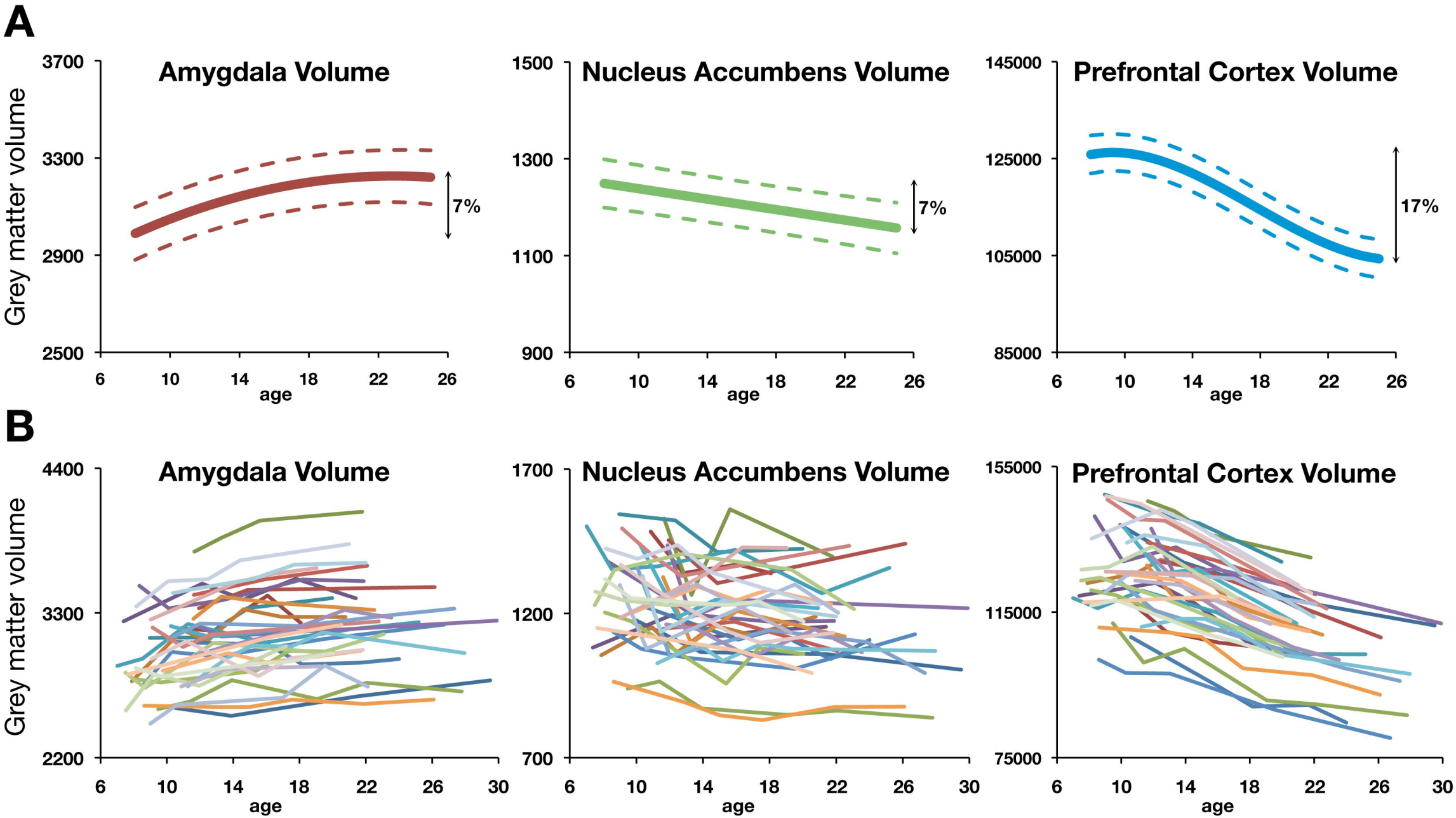
Figure 3. The top row shows the best fitting group models for average developmental trajectories in grey matter volume in the amygdala, nucleus accumbens and prefrontal cortex from 33 participants scanned at least three times between late childhood and early adulthood; dashed lines indicate 95% CI. The bottom row shows individual data from the 33 participants. Reproduced from[24]

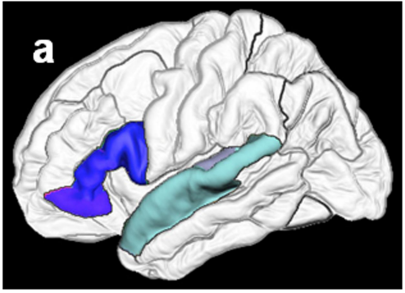
Figure 4. SES x Age interaction in left temporal gyrus and left inferior frontal gyrus volume. Reproduced from[36].

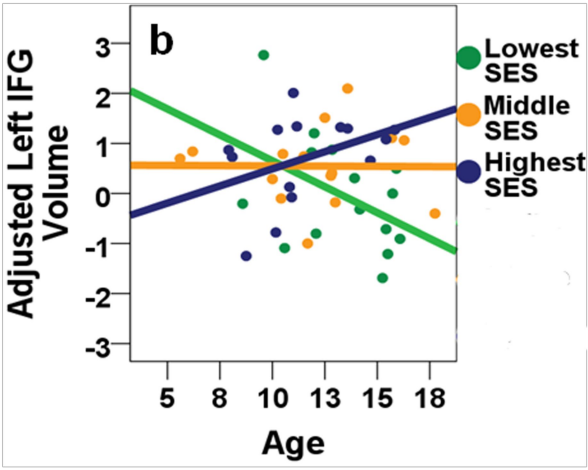
Figure 5. Activation in the dmPFC (panel a) and amygdala (panel b) that correlated negatively with SES during the viewing of angry faces vs. fixation. Reproduced from[37].

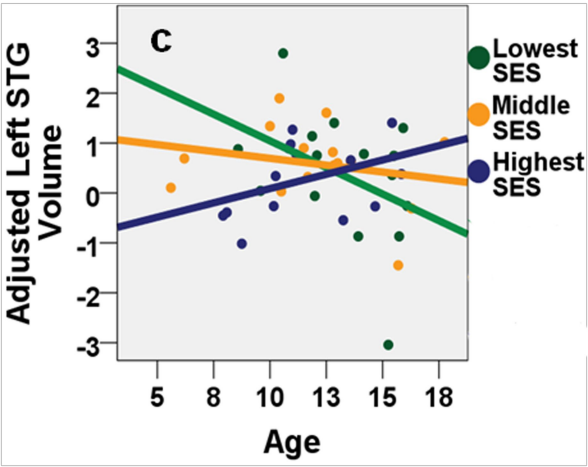

Caudate
Putamen
Pallidum
Nucleus accumbens
Putamen
Pallidum
Caudate
Thalamus
Nucleus
Accumbens
Hippocampus
Amygdala
Cerebellum
Cerebellum
Male
Female
Thalamus
Amygdala
Hippocampus

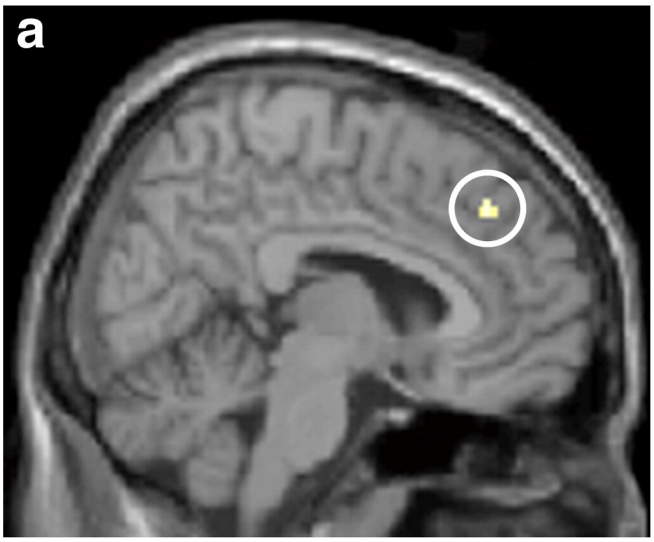

Cortical Thickness
3.0
2.8
2.6
2.4
10
15
20
25
30
Age
Cortical Area
225000
200000
175000
150000
125000
10
15
20
25
30
Age
Cortical Volume
700000
600000
500000
400000
10
15
20
25
30
Age

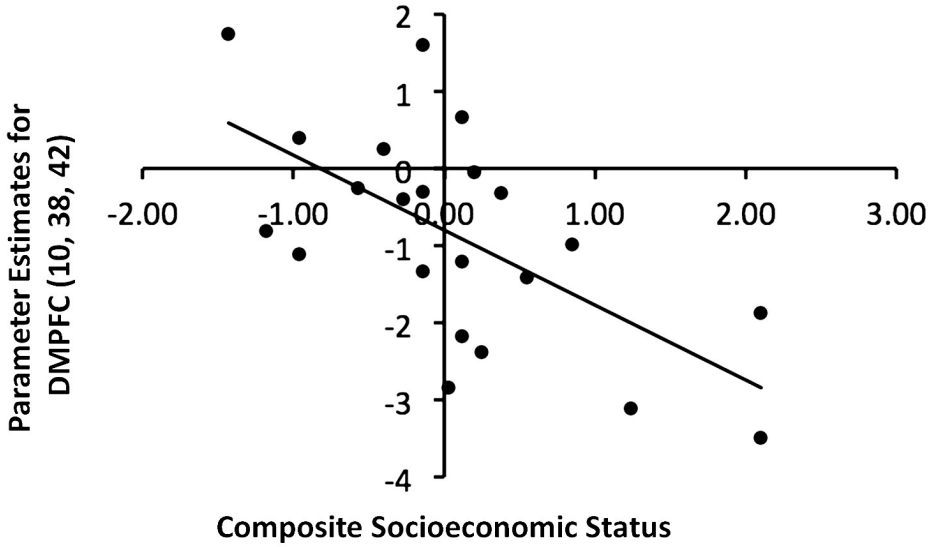

A
Amygdala Volume
Nucleus Accumbens Volume
Prefrontal Cortex Volume
Grey matter volume
3700
3300
2900
2500
1500
1300
1100
900
145000
125000
105000
85000
7%
7%
17%
6
10
14
18
22
26
age
B
Amygdala Volume
Nucleus Accumbens Volume
Prefrontal Cortex Volume
Grey matter volume
4400
3300
2200
1700
1200
700
155000
115000
75000
6
10
14
18
22
26
30
age

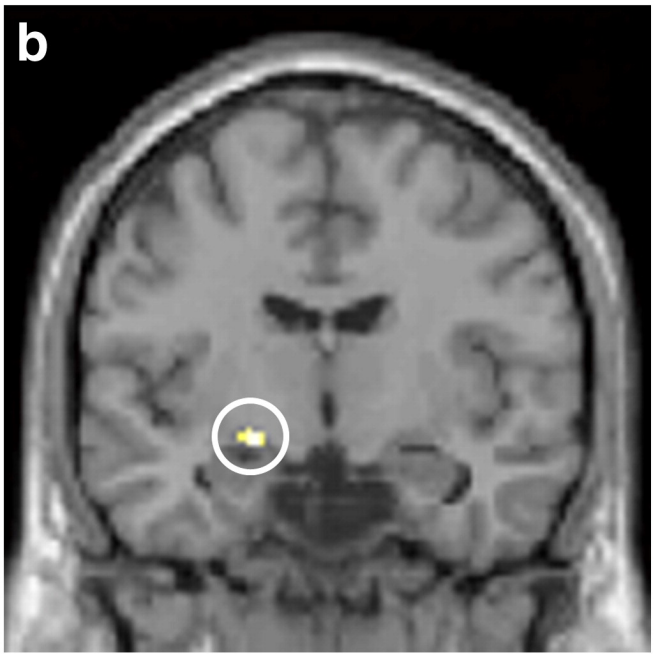

a

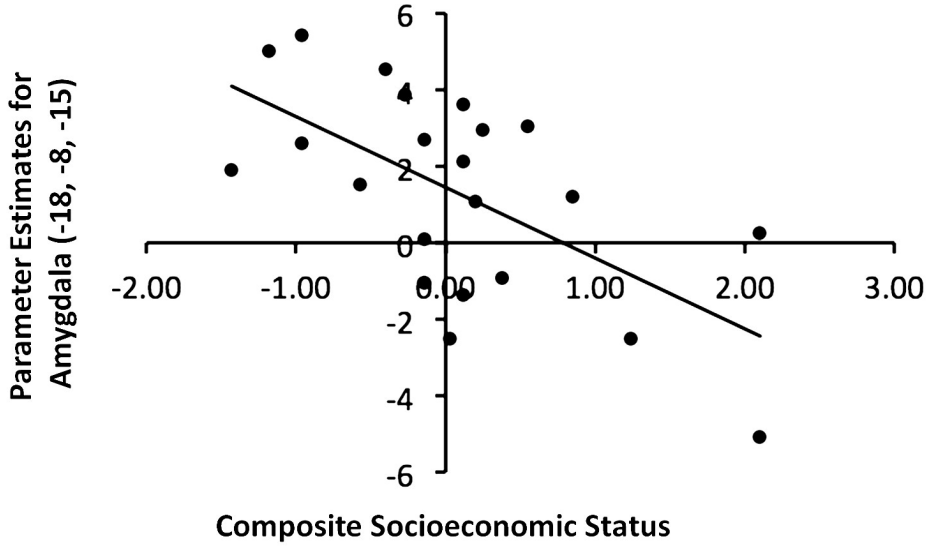

b
Adjusted Left IFG Volume
3
2
1
0
-1
-2
-3
5
8
10
13
15
18
Age
Lowest SES
Middle SES
Highest SES


c
Adjusted Left STG Volume
3
2
1
0
-1
-2
-3
5
8
10
13
15
18
Age
Lowest SES
Middle SES
Highest SES


a


Parameter Estimates for DMPFC (10, 38, 42)
2
1
0
-1
-2
-3
-4
-2.00
-1.00
0.00
1.00
2.00
3.00
Composite Socioeconomic Status


b


Parameter Estimates for Amygdala (-18, -8, -15)
6
4
2
0
-2
-4
-6
-2.00
-1.00
0.00
1.00
2.00
3.00
Composite Socioeconomic Status