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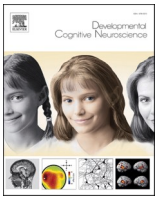

Puberty and risky decision-making in male adolescents

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ABSTRACT

Pubertal development is a potential trigger for increases in risk-taking behaviours during adolescence. Here, we sought to investigate the relationship between puberty and neural activation during risky decision-making in males using functional magnetic resonance imaging (fMRI). Forty-seven males aged 12.5–14.5 years completed an fMRI risk-taking task (BART) and reported their tendencies for risky decision-making using a self-report questionnaire. Puberty was assessed through self-reported pubertal status and salivary testosterone levels. Testosterone concentration, but not physical pubertal status, was positively correlated with self-reported risk-taking behaviour, while neither was correlated with BART performance. Across the whole sample, participants had greater activation of the bilateral nucleus accumbens and right caudate on trials when they made a successful risky decision compared to trials when they made a safe choice or when their risky decision was unsuccessful. There was a negative correlation between pubertal stage and brain activation during unsuccessful risky decision-making trials compared within unsuccessful control trials. Males at a lower stage of pubertal development showed increased activation in the left insula, right cingulate cortex, dorsomedial prefrontal cortex (dmPFC), right putamen and right orbitofrontal cortex (OFC) relative to more pubertally mature males during trials when they chose to take a risk and the balloon popped compared to when they watched the computer make an unsuccessful risky decision. Less pubertally mature males also showed greater activation in brain regions including the dmPFC, right temporal and frontal cortices, right OFC, right hippocampus and occipital cortex in unsuccessful risky trials compared to successful risky trials. These results suggest a puberty-related shift in neural activation within key brain regions when processing outcomes of risky decisions, which may reduce their sensitivity to negative feedback, and in turn contribute to increases in adolescent risk-taking behaviours.

1. Introduction

During adolescence, individuals become more autonomous and begin to make decisions independently of adults (Blakemore and Robbins, 2012; Steinberg, 2008). Epidemiological data show that this period is associated with increases in health risk behaviours including drug and alcohol consumption and risky sexual behaviours compared to childhood, and many laboratory studies report increased adolescent risk-taking using self-report measures and cognitive tasks (Braams et al., 2015; Crone et al., 2016; Duell et al., 2018; Defoe et al., 2015; Rosenbaum and Hartley, 2019). One potential driver for this increased risky decision-making propensity is puberty (Collado-Rodriguez et al., 2014; Schulz and Sisk, 2016; Sisk and Foster, 2004). Males who enter puberty earlier than their peers have an increased risk of tobacco, alcohol and marijuana use during adolescence (Cance et al., 2013; Kaltiala-Heino et al., 2011; Patton et al., 2004), while elevated levels of puberty-related hormones have been associated with alcohol use and aggressive risk-taking (de Water et al., 2013; Vermeersch et al., 2008). This study aims to investigate the relationship between puberty and neural activation during decision-making in adolescent males.

The act of decision-making involves multiple component processes (Blakemore and Robbins, 2012). It has been hypothesized that differing developmental trajectories of the brain regions involved in these processes might underlie some of the increased risky decision-making associated with adolescence (Casey et al., 2011; Shulman et al., 2016). Specifically, the 'dual systems hypothesis' proposes that subcortical

Abbreviations: NAcc, Nucleus accumbens; OFC, orbitofrontal cortex; BART, Balloon Analog Risk-taking Task.

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brain regions involved in reward processing develop earlier in adolescence than prefrontal cortical regions involved in cognitive control (Casey et al., 2011; Shulman et al., 2016), although see (Crone and Dahl, 2012; Pfeifer and Allen, 2016). Neuroimaging studies investigating the role of puberty in risky decision-making have predominantly focused on reward processing ((Braams et al., 2015; Forbes et al., 2010; Op de Macks et al., 2011, 2016; van Duijvenvoorde et al., 2014; Alarcón et al., 2017) see (Goddings et al., 2019) for review). Testosterone concentration was positively associated with nucleus accumbens (NAcc) activation during reward processing in a longitudinal study of 299 8–27 year-old males and females using a coin-toss gambling task (Braams et al., 2015), and in 10–16 year-olds on receiving a reward in a jackpot gambling task (Op de Macks et al., 2011), although there was no association with physical pubertal development in a longitudinal analysis of a subset of these participants (van Duijvenvoorde et al., 2014). A further study of 12–17 year-old males and females also showed a positive association between testosterone levels and right NAcc activation during reward processing in win vs. no-win Wheel of Fortune decision-making trials (Alarcón et al., 2017). In contrast, a study using a gambling card task in 11–13 year-olds reported a positive association between testosterone levels and caudate activation in males when *anticipating* a reward, but a negative association between testosterone and caudate activation in male and females when processing reward outcomes (Forbes et al., 2010). Finally, a study using the jackpot task in 11–13 year-old females found that testosterone levels were positively associated with risk-taking, mediated by increased medial orbitofrontal cortex (OFC), but not NAcc, activation, while higher oestradiol levels were associated with increased NAcc activation, which in turn related to decreased risk-taking (Op de Macks et al., 2016). A second key system involved in adolescent risky decision-making in the influential dual systems hypothesis, alongside the reward system, is cognitive control (Casey et al., 2011; Shulman et al., 2016; Vijayakumar et al., 2018). Relatively few studies have investigated pubertal development and its relationship with cognitive control maturation, and the available studies have focused on other aspects of cognitive control, including working memory (Alarcón et al., 2014) and inhibition (Schulte et al., 2020).

The aim of the current study was to examine whether brain activation during risky decision-making is related to pubertal development. We investigated whether activation of regions involved in risky decision-making, considering both regions specific to reward processing and regions associated with cognitive control, are related to physical or hormonal pubertal development. Since males and females experience differential timing of pubertal onset (Mendle et al., 2019), different physiological hormones exposure (Bordini and Rosenfield, 2011a), and develop different secondary sexual characteristics (Bordini and Rosenfield, 2011b), we chose to limit our study to male participants, and recruited study participants within a narrow age range (12.5–14.5 years), when males can be at all pubertal stages, to maximise the ability to assess pubertal differences in behaviour and neural activation independent of age.

We opted to use a modified version of the Balloon Analog Risk-taking Task (BART; adapted from (Lejuez et al., 2002)) and collected self-reported levels of risk-taking behaviours. Performance on the BART risk-taking task has been shown to increase in the second decade of life and then plateau in the early twenties in lab environments (Duell et al., 2018; Peper et al., 2018; Humphrey and Dumontheil, 2016). BART task performance has been correlated with the propensity for real-life risk--taking behaviours in adults and adolescents, including smoking (Lejuez et al., 2005), alcohol use (MacPherson et al., 2010), marijuana use (Hanson et al., 2014) and risky driving (Vaca et al., 2013), although not all studies replicate these associations (Dean et al., 2011; Hulvershorn et al., 2015). The exact probabilities associated with positive and negative outcomes with risky decisions are not explicit on the BART task, and there is evidence that suggests this uncertainty, which reflects the uncertainty of real-life decision-making, may better capture adolescents' brain responses to risk than tasks where the probabilities are explicit (Korucuoglu et al., 2020). The BART task allows the comparison of trials where participants actively make decisions versus passively observe a decision made by the computer, trials where the participants choose to be 'safe' versus choose to take a 'risk' and trials where a decision to take a risk that results in a positive versus a negative outcome.

Our first set of hypotheses concerns the comparison of active decision-making to the control condition of following the computer's choices. First, compared with passively observing risk-taking, actively deciding whether to take a risk in the BART has been associated with greater activation in a number of brain regions including the striatum, OFC, insula, anterior cingulate cortex (ACC), the inferior frontal gyrus (IFG), bilateral parietal cortices and the dorsolateral and medial prefrontal cortex (dlPFC and mPFC) (Korucuoglu et al., 2020; Qu et al., 2015; Rao et al., 2008; Schonberg et al., 2012). Based on these findings, we expected to see greater activation in these regions across the whole group for $\text{decision}_{\text{expand}}$ trials, when participants were deciding to take a risky rather than a safe choice, compared with $\text{control}_{\text{expand}}$ trials, where the computer is 'making' the decision to take a risk. Second, the sequential decision-making design of the BART task means that there are greater risks and rewards associated with each balloon as it increases in size. Previous studies have found that activation in some of these regions, including the insula and the OFC, increases parametrically during active decision-making as a function of the relative size of the balloon being inflated (Korucuoglu et al., 2020; Schonberg et al., 2012). We expected to replicate this finding across the whole group of participants. Parametric analyses were only undertaken for the inflate decision since the design of the BART means that there are many inflate trials with different balloon sizes, while there are fewer stop and pop events. Third, the brain regions associated with making a safe choice are similar to those activated when making a risky decision, in particular the bilateral ACC, IFG, superior frontal gyrus (SFG), bilateral parietal cortices and the striatum (Korucuoglu et al., 2020). We expected to see greater activation in these regions across participants when actively making a safe choice compared to observing a safe choice made by the computer. Finally, previous studies have not compared unsuccessful active risky decision-making trials to trials where the negative outcome follows a computer's risky decision-making. A large number of brain regions are activated when observing the negative outcome of an active decision, including middle and inferior temporal regions, lateral occipital regions, lateral OFC, insula, middle frontal lobe, and both the inferior and superior parietal lobe (Korucuoglu et al., 2020; Schonberg et al., 2012). We hypothesised that these regions would show greater activation during unsuccessful trials following an active decision than following the computer's decision, across the whole group.

Our second set of hypotheses related to differences in brain activation during the active decision-making condition. We expected that trials in which participants opted to take a risk would be associated with greater activation of regions involved in reward processing (e.g. NAcc and OFC) compared with trials when participants decided to stop inflating the balloon (safe trials (Op de Macks et al., 2018), while safe decisions would be associated with greater activation in cognitive control regions, including the PFC, ACC and insula than risky decisions. Within risky trials, we expected greater neural activation in reward processing brain regions, particularly the NAcc and OFC, on successful trials compared to unsuccessful trials (McCormick and Telzer, 2017). Conversely, we predicted that activation of the superior frontal gyrus, mPFC and ACC would be greater in unsuccessful trials than successful ones since these regions are associated with distinguishing between positive and negative feedback, and with evaluating action outcomes (Schonberg et al., 2012; McCormick and Telzer, 2017; Paulsen et al., 2015a).

Our final set of analyses involved comparing brain activation across the contrasts outlined above as a function of pubertal status, to investigate whether the activations were related to pubertal development. Testosterone levels and physical pubertal status are overlapping but distinct measures of puberty and may therefore show different

correlations with brain activation during the decision-making task. The mechanisms underlying the impact of puberty on cognitive brain development are still poorly understood, and are likely to include both direct and indirect effects. Testosterone levels measure a single time-point of hormonal status, providing information on hormone concentration on the day of testing as well as a general indicator of an individual's overall pubertal development. Physical Tanner stage measures serve as an indicator of an individual's cumulative exposure to sex steroid hormones including testosterone, and take into account the visibility of pubertal changes e.g. body hair development, which may impact on a young person's experiences and behaviours. Previous studies have shown associations between both testosterone and physical pubertal status and brain activation in the striatum and OFC during reward processing in 8–17 year-olds (Braams et al., 2015; Op de Macks et al., 2011; Alarcón et al., 2017). Based on these findings, we would expect to find that males with higher levels of testosterone and/or more advanced physical pubertal status would show increased activation in reward processing areas (particularly the striatum and the vmPFC), in the $decision_{inflate}$ vs $control_{inflate}$ and $decision_{inflate}$ vs. $decision_{stop}$ contrasts (see full task description in the Methods below). Few studies of the cognitive control aspects of risky decision-making in males have incorporated measures of pubertal development, and studies looking at pubertal development and decision-making have often undertaken analyses only focussed on reward processing-associated regions of interest. There is, however, evidence of associations between puberty and the structural development of key brain regions associated with decision-making, particularly the ACC, the parietal lobe and the dlPFC (Vijayakumar et al., 2018). We therefore undertook an exploratory analysis of associations between pubertal development and brain activation during the BART task across the whole brain, rather than focussing on specific regions of interest. We were specifically interested in identifying any pubertal differences in key brain regions associated with decision-making and cognitive control that are activated during the BART task, including the insula, ACC, parietal lobe, dlPFC and mPFC (Qu et al., 2015; Rao et al., 2008; Schonberg et al., 2012).

2. Methods

2.1. Participants

Fifty healthy male participants aged 12.5–14.5 years were recruited from XXX and the local region via advertisements posted around the university campus and letters sent to local schools. The functional magnetic resonance imaging (MRI) data of two participants were incorrectly acquired, and one participant was completely removed from the analysis due to excessive movement (see below and Supplementary Materials), leaving data from 47 participants for analysis. Diffusion tensor imaging data for these same participants have been published previously (Menzies et al., 2015). All participants spoke English as their native language and had normal or corrected to normal vision. Participants assented to the study, and written informed consent was obtained from a parent or legal guardian. Potential participants were excluded based on a self- or parent-reported history of prematurity (<34 weeks gestation), previous neurosurgery, a known neurological, psychiatric or endocrine disorder, taking any medications known to significantly impact on hormone levels or any contraindications to MRI. IQ was measured using a two-subtest version of the Wechsler Abbreviated Scale of Intelligence (Wechsler, 1999). Subjects received £ 10 for attending the testing session and earned a further £ 5–10 depending on their performance (see below for details). The study was approved by the XXX University Research Ethics Committee. Demographic details including age, IQ, testosterone level and risk-taking scores are described in Table 1.

Table 1
Participant demographics and BART performance data shown for the whole group with fMRI data (N=47). Significant correlations with each of Tanner stage and testosterone (with no covariates) are highlighted in bold. [a] One participant did not complete the pubertal self-assessment, leaving n=46.[b] Testosterone concentration could not be estimated for four participants, leaving n=43.[c] One participant did not complete the CARE questionnaire, leaving n=46. [d] The BART decision balloon stopping point SD is the mean and SD of each participant's standard deviation of stopping point and provides an indicator of variability between participants.

	Whole group N=47	Pearson correlation with Tanner stage		Pearson correlation with testosterone	
	Mean ± SD (Range)	r	p	r	p
Age (years)	**13.6 ± 0.4 (12.7-14.3)**	**0.51**	**<0.001**	**0.38**	**0.01**
Testosterone pg/ml[b]	**89.6 ± 36.7**	**0.52**	**<0.001**	**-**	**-**
IQ	112 ± 11 (88-140)	0.27	0.07	0.15	0.3
CARE questionnaire[c]	2.9 ± 1.2 (1.0-6.8)	-0.02	0.9	**0.39**	**0.01**
BART Total balloon trials	87 ± 27 (40-174)	-0.09	0.5	-0.02	0.9
BART Proportion of decision balloons/run	0.5 (0.48-0.52)	-0.16	0.3	-0.25	0.1
BART Number of $decision_{stop}$ balloons	29 ± 15 (13–84)	-0.16	0.3	-0.02	0.9
BART Number of $decision_{pop}$ balloons	13 ± 5 (3–23)	0.21	0.2	-0.08	0.6
BART Decision balloons - mean stopping point[d]	4.5 ± 1.3 (1.6-6.8)	0.23	0.1	0.08	0.6
BART Decision balloon stopping point SD[d]	1.5 ± 0.6 (0.1-3.6)	-0.04	0.8	0.08	0.6

2.2. Puberty assessments

Two independent indicators of pubertal development were obtained for each participant to capture different aspects of puberty. First, a self-reported assessment of physical puberty status was completed on the day of testing. In private, participants viewed gender-specific line drawings of pubertal stages of gonadal and pubic hair development with short written descriptions (Taylor et al., 2001) and were asked to rate which picture most resembled their current stage of development. Such self-report measures have been shown to be a valid method for pubertal development assessment, with adolescents being reasonably accurate observers (Shirtcliff et al., 2009). Gonadal development and pubic hair developmental stages were averaged to construct a single 'average Tanner stage' variable (range 1–5).

Second, testosterone concentrations were measured using salivary hormone assays. These assays have been shown to be reliable and to correlate highly with serum levels (Granger et al., 1999), and were used to avoid invasive testing. Upon waking on the morning of their scan, before 9 am, each participant collected up to 3.5 mL passive (unstimulated) drool samples of saliva after rinsing their mouths with water, and before brushing their teeth, eating or drinking anything (except water). On arrival at the testing centre, parents and participants were asked to verify that they had followed these instructions. Participants transported their samples to the testing centre on the day of collection in an ice-filled insulated polystyrene box. A subset of participants (n = 4) did not provide saliva samples on the day of scanning; for these participants, saliva samples were collected within one week of the MRI session. Samples were stored at –80 °C and analysed as a single batch by Salimetrics Europe Ltd (http://www.salimetrics.com/). Duplicate assays for

testosterone were performed for each participant. Oestradiol and dehydroepiandrosterone (DHEA) concentrations were also acquired but were not included in the current analysis. The testosterone range of sensitivity was 1–600 pg/mL. The average intra-assay coefficient of variation was 1.4%, and mean values were used for all analyses. Four participants had insufficient volume saliva samples for analysis, leaving 43 participants.

2.3. Cognitive appraisal of risky events (CARE) risk-taking questionnaire

Participants completed the self-assessment CARE risk-taking questionnaire, a tool designed for, and previously used in research into, risk behaviour in young people (Fromme et al., 1997; Galvan et al., 2007). This asks participants their likelihood of undertaking 30 activities over the subsequent six months using a Likert 1–7 scale (1 = Not at all likely, 7 = Extremely likely) in six subscales: sports (4 items); sexual behaviour

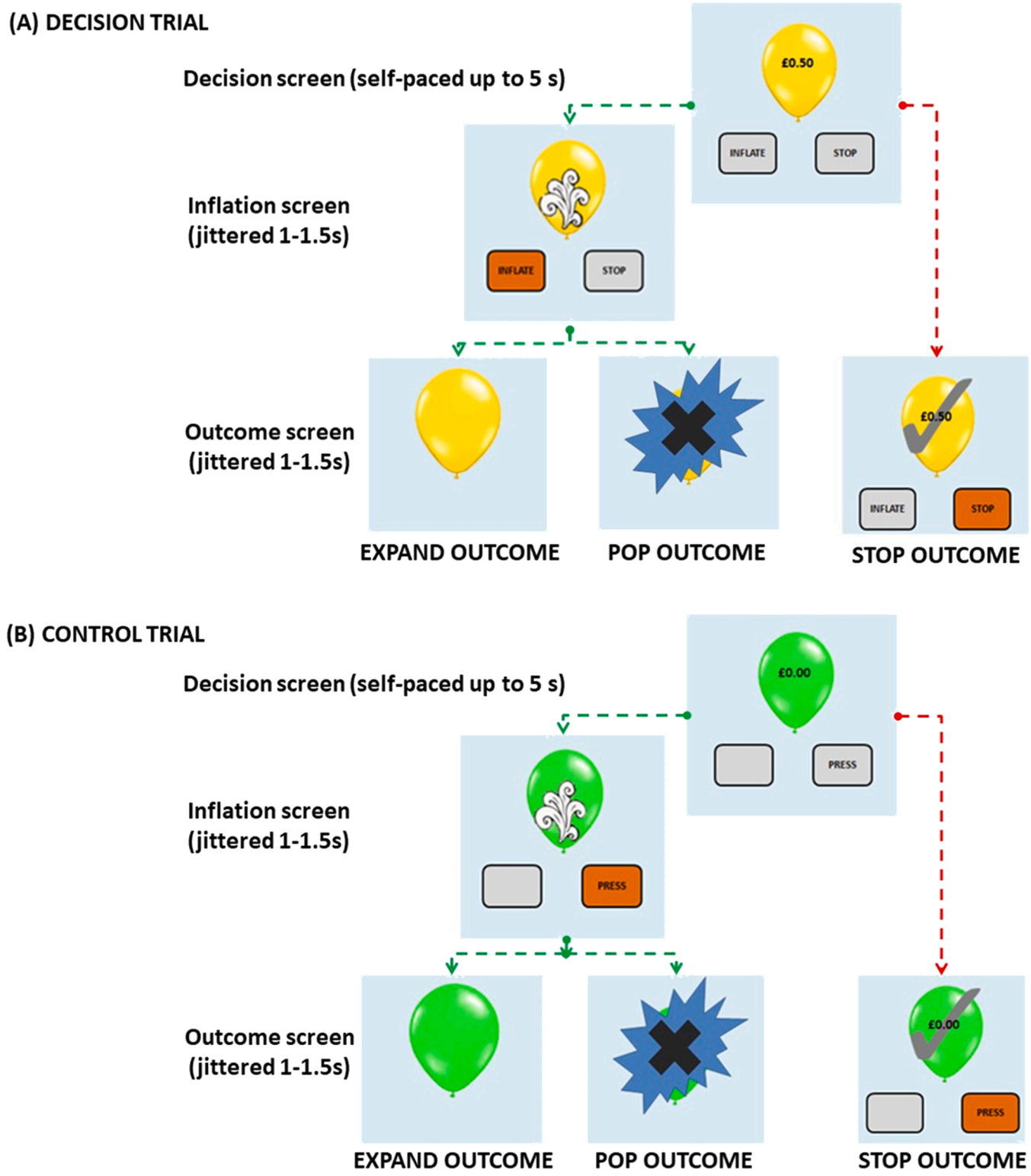

Fig. 1. The modified BART task showing (A) a decision trial and (B) a control trial.

(5 items); alcohol use (4 items); illicit drug use (4 items); aggressive/illegal behaviours (8 items); and academic risk-taking (5 items). The risky sports subscale was excluded from the analyses since the questions were potentially biased by availability and opportunity (e.g. skiing, rock-climbing). For each subscale, a likelihood score was calculated by averaging the responses. Very few participants reported any risky sexual activities (median score 1.0, IQR 1.0–1.3), illicit drug use (median score 1.0, IQR 1.0–1.2) or alcohol use (median score 1.2, IQR 1.0–2.0), which is in keeping with typical behaviour in the UK population in this age range (Hagell et al., 2015; Fitzsimons et al., 2018). Analyses were therefore performed on the combined score from the aggressive/illegal behaviours (median score 2.4, range 1.0–6.5, Cronbach's $\alpha = 0.83$) and academic risk-taking subscales (median score 3.4, range 1.0–7.0, Cronbach's $\alpha = 0.85$), which were highly correlated ($r = 0.57$, 95% C.I. 0.34, 0.74, $p < 0.001$). Details of questions of these two sub-scales are included in the Supplementary Materials. The combined score showed high reliability (Cronbach's $\alpha = 0.88$).

Participants also completed questionnaires assessing impulsivity (Barratt Impulsiveness Scale; (Patton et al., 1995)) and sensation seeking (modified Zuckerman Sensation-Seeking Scale: (Steinberg et al., 2008)) as part of a wider project. These were not included in the current analyses.

2.4. fMRI BART

During fMRI acquisition, participants completed a modified version of the BART (adapted from (Lejuez et al., 2002)), during which a series of drawings of balloons were projected on a screen. There were two, equally distributed, trial types (decision or control), with a maximum of two consecutive decision or control balloons. In **decision trials**, participants first saw a balloon worth £ 0.50 (Fig. 1A) and then chose by button press whether to inflate the balloon or not. If they decided to inflate the balloon (risky decision), it could increase in size and value (*expand outcome*) or it could pop and the money for that balloon would be lost (*pop outcome*). If the balloon successfully expanded, the participant could choose to inflate it further or not. With each successful inflation, the balloon's value increased by £ 0.50. If the participant chose to stop inflating the balloon (safe choice), they saw a tick on the screen, and the balloon's final value was saved and contributed to their total earnings (*stop outcome*) (Fig. 1A). In control trials, participants saw a different colour balloon, which was not worth any money, and pressed one of two buttons as indicated on the screen by a button labelled PRESS, and observed the outcome (*expand*, *pop* or *stop*) (Fig. 1B). Participants had no control over whether a risky or safe choice was made. The control condition was included to account for brain activation associated with the processing of the visual stimuli and execution of a motor response.

The decision and control balloons were identifiable by colour (yellow or green; counterbalanced between participants). The decision phase was self-paced (up to a maximum of 5 s). On trials in which a participant did not select an option within the time limit, the decision screen was presented again. If the participant chose to inflate the balloon, they were shown an 'inflation' screen followed by the outcome (an enlarged intact balloon, or a popped balloon). The inflation screen and outcome screens (stop balloon, pop balloon, enlarged balloon) were shown for between 1.0 and 1.5 s (jittered) (Fig. 1). Decision balloons were programmed to pop after 1–12 inflations (popping point) using a block randomisation method, such that within a block of 12 successive decision trials, there would be a trial programmed to pop at each of the 12 popping points. The control balloons were programmed to end (stop or pop) when they were the same size as the final size of the decision balloon two trials earlier in the run, to ensure that the decision and control balloons were approximately matched in trial duration and size of balloons seen. Control balloons were programmed so that equal numbers of pop and stop outcomes were seen over each run. Participants were trained on the task and performed eight practice trials outside the scanner and then completed four 6-min scanning runs of the BART.

2.5. Behavioural data analysis

Pearson's correlations were used to assess the correlation between self-reported risk-taking on the CARE questionnaire and risk-taking propensity on the BART, as indicated by the average point that participants chose to stop inflating decision trial balloons (*stopping point*) (Lejuez et al., 2007). The association between pubertal development and risk-taking behaviour on the BART, as well as self-reported risk-taking behaviour on the CARE questionnaire was assessed, using Pearson's partial correlations. All statistical analyses were performed using R (http://www.r-project.org/).

2.6. fMRI data acquisition and preprocessing

MRI scanning was performed using a 1.5 T Siemens Avanto head MRI scanner with a 32-channel head coil. Head movement within the MRI scanner and scanner noise were minimised using soft cushions to support the participant's head within the head coil. T2 * -weighted echo-planar (EPI) volumes with BOLD contrast were obtained during the BART task. Each run consisted of 127 volumes (TR=2.975 s; TE=50 ms; 35 axial slices; in plane resolution $3\times3\times3$ mm^3; sequential acquisition). A 3D T1-weighted anatomical scan was also collected for reference (TR=2.73 s, TE=3.57 ms, flip angle=7°, 176 slices, field of view $224\times256\times176$ mm^3). Two runs of diffusion weighted images were also acquired for a separate study (Menzies et al., 2015). Total scanning duration was approximately 50 min

The first four volumes were discarded to allow for T1 equilibration effects. Preprocessing was performed using SPM12 (Wellcome Trust Centre for Neuroimaging, London, UK; www.fil.ion.ucl.ac.uk/spm/) and included rigid-body transformation (realignment) to the first analysed volume, with second degree B-spline interpolation to correct for movement during the session, followed by slice timing correction. The bias-corrected structural image for each participant was co-registered to the mean realigned functional image and segmented using Montreal Neurological Institute (MNI) registered International Consortium for Brain Mapping tissue probability maps. Spatial normalisation parameters were applied to the realigned images to obtain normalised functional images with a $3\times3\times3$ mm voxel size, which were smoothed with a Gaussian filter of 8 mm full-width at half maximum. One participant was excluded from the analysis completely due to excessive movement, and scanning runs with excessive movement were excluded for 7 further participants (1 run for $n = 4$, 2 runs for $n = 3$; see Supplementary methods for full details of head motion processing).

2.7. fMRI data analysis

Statistical analyses were performed using SPM12. A general linear model (GLM) was created in which the four scanning sessions were treated as a single time series using the corresponding SPM12 option, which models session effects using regressors of no interest. Six event types were modelled: $\text{decision}_{\text{expand}}$, $\text{decision}_{\text{pop}}$, $\text{decision}_{\text{stop}}$, $\text{control}_{\text{expand}}$, $\text{control}_{\text{pop}}$, $\text{control}_{\text{stop}}$. Each event was modelled with the duration from the onset of the event to the end of the outcome screen for the event. As with many risky decision paradigms, the task is inherently asymmetrical. When a participant chooses to 'stop' inflating a balloon, the outcome of that is certain (see Fig. 1) and it is therefore not possible to separate the decision-making step from the outcome in stop events. In contrast, the 'expand' and 'pop' events start with an identical decision (in both cases the participant chooses to inflate the balloon) and the outcome is uncertain (the balloon may 'expand' or 'pop') until the outcome screen is shown. Additional parametric regressors modelling the number of inflations prior to each $\text{decision}_{\text{expand}}$ and $\text{control}_{\text{expand}}$ events were included. Trials in which participants did not make a response were modelled as events of no interest in the GLM. Time series

and the model were high-pass filtered at 128 s

GLM parameter estimates were used to create first-level linear contrast images of interest, namely: (i) the difference between decision and control balloons for each type of event ($decision_{expand}$ - $control_{expand}$, $decision_{pop}$ - $control_{pop}$, $decision_{stop}$ - $control_{stop}$); (ii) the difference between the decision and control balloons parametric regressors for expand trials; (iii) the difference between $decision_{expand}$ and $decision_{stop}$ events; and (iv) the difference between $decision_{expand}$ and $decision_{pop}$ events. These contrasts of interest were entered into three types of second-level analyses. First, one-sample t-tests were performed across the whole group. Then, to examine pubertal differences in neural activation, random effects regression models examined the association between the contrast of interest and average Tanner Stage and included chronological age as a regressor since Tanner stage and age are significantly correlated in our sample (results of regression models without the age covariate are included in the Supplementary Materials). Finally, random effects regression models were used to examine the association with testosterone and included testosterone level and chronological age as regressors.

SPMs were thresholded at $p < 0.001$ uncorrected at the voxel level and at family-wise error (FWE) corrected $p < 0.05$ at the cluster level (corresponding to a minimum cluster size of 70 voxels determined with SPM12). Activations that survived FWE whole-brain correction at $p < 0.05$ at the voxel level are indicated. All coordinates are given in MNI space. Significant effects were followed up by extracting the mean signal across all voxels of significant clusters with MarsBar (Brett et al., 2002) and running simple effects tests to establish what was driving the significant differences seen.

3. Results

3.1. Risk-taking behaviour

Both measures of puberty (Tanner stage and testosterone concentration) were significantly correlated with age and with each other (Table 1). There was no correlation between self-reported risk-taking on the CARE and Tanner stage. In contrast, testosterone was positively correlated with self-reported risk-taking, both before and after covarying for chronological age (see Table 1 and S1). Neither pubertal measure was correlated with IQ or any of the outcome indicators for the BART.

All participants completed at least three runs of the BART, and the results shown include BART task data from the runs used for the fMRI analyses. There was substantial variability in BART risk-taking behaviour, with the mean point at which participants chose to stop inflating varying from 1.6 to 6.8 (mean 4.5 inflations), and the within-participant standard deviation for number of inflations (indicating how variable an individual's responses were) varying from 0.1 to 3.6 inflations (mean 1.5). There was no significant correlation between measures of BART performance and chronological age (all $p > 0.2$), and no correlation between BART performance and either measure of puberty (Table 1). Furthermore, there was no correlation between self-reported risk-taking behaviour on the CARE and BART behaviour (r (Schonberg et al., 2012)$= 0.11$, $p = 0.5$).

3.2. FMRI results

3.2.1. Comparison of trials in the whole sample

Comparing $decision_{expand}$ trials to $control_{expand}$ trials showed widespread differences in BOLD signal (Fig. S1 and Table S1). Events in which participants successfully took a risk (ending up with a larger, more valuable balloon) showed greater activation in decision than control trials in a very large cluster including bilateral lateral prefrontal cortices, insula, striatum, ACC and occipital cortices, as well as the right supplementary motor area (SMA) (see Fig. S1 and Table S1).

Parametric analysis comparing $decision_{expand}$ to $control_{expand}$ trials for the whole group revealed that clusters in the dorsal ACC, bilateral insula and bilateral putamen (Fig. 2A and Table 2), as well as in bilateral parietal cortices, and right occipital and temporal cortices showed increasing activation with increasingly risky decisions (larger balloons worth more money) for decision trials while control trials did not show this modulation based on the size of the balloons (illustrated for three clusters in Fig. 2B). Note all risky decisions in this analysis resulted in a positive outcome (inflated balloon).

The ACC and bilateral anterior insula showed larger increases in BOLD signal during decision trials where participants chose to take a risk and there was a negative (pop) outcome, than during control trials (unsuccessful risky decision-making: $decision_{pop} > control_{pop}$) (Fig. 2C and Table 2b). This comparison showed partial overlap with the expand contrasts reported above.

Comparing decision trials in which participants chose to stop the trial and save the value towards their final earnings to control trials (safe decision-making: $decision_{stop} > control_{stop}$) revealed widespread differences in BOLD signal including in bilateral orbitofrontal cortex, inferior parietal and medial temporal lobes, as well as the left posterior insula, caudate, and occipital lobe (Fig. 2D and Table 2c).

Comparing $decision_{expand}$ vs $decision_{stop}$ trials, i.e. risky vs. safe decision-making, indicated that the bilateral NA, right caudate and left frontal operculum, as well as the left occipital cortex and right SMA (Fig. 3A and Table 3a) showed greater BOLD signal during the decision trials in which participants chose to inflate the balloon and the outcome was positive than when they chose to stop the trial and save the value towards their final earnings. The reverse contrast showed that in a large cluster including bilateral orbitofrontal cortices, medial prefrontal cortex, bilateral occipital cortex, left temporal cortex and right amygdala (Fig. 3A and Table 3b) there was increased BOLD signal when participants decided to bank the money rather than when they chose to inflate the balloon and the outcome was positive.

Finally, comparing $decision_{expand}$ vs $decision_{pop}$ trials, i.e. successful vs. unsuccessful risky decision-making trials, indicated the bilateral nucleus accumbens and right caudate, as well as the right middle frontal gyrus, bilateral postcentral lobules and left occipital lobe (Fig. 3B and Table 3c) showed greater BOLD signal during the decision trials when participants chose to inflate the balloon and the outcome was positive (expanded balloon) than when the outcome was negative (popped balloon). The reverse contrast indicated a number of regions including the bilateral insula, ACC, bilateral temporal lobes, supramarginal gyri, fusiform gyri and occipital cortices (Fig. 3B and Table 3d) showed greater BOLD signal when the balloon popped than when it inflated.

3.3. Interactions with pubertal measures

The second set of analyses investigated whether the comparisons above differed as a function of Tanner stage or testosterone concentration, controlling for chronological age (results with no age covariate are described in Table S2). There was no significant correlation between testosterone concentration and changes in BOLD signal in any contrast. There was also no significant correlation between Tanner stage and the $decision_{expand}$ vs $control_{expand}$, $decision_{stop}$ vs $control_{stop}$, or $decision_{expand}$ vs $decision_{stop}$ contrasts.

In the $decision_{pop}$ vs $control_{pop}$ contrast, there was a negative correlation between Tanner stage and brain activation in six different clusters covering parts of the left insula, dorsomedial prefrontal cortex (dmPFC), right putamen, right middle cingulate cortex (MCC) and right orbitofrontal cortex (OFC). In these clusters, males who reported being at a lower Tanner stage showed greater brain activation in trials where the balloon popped after they had made an active risky decision compared with trials where they observed the balloon popping after the risky decision was made by the computer. The difference in activation between trial types reduced or was reversed with increasing pubertal Tanner stage. The interaction was driven by changes in brain activation in both trial types (Fig. 4).

There was also a significant negative correlation between $decision_{pop}$

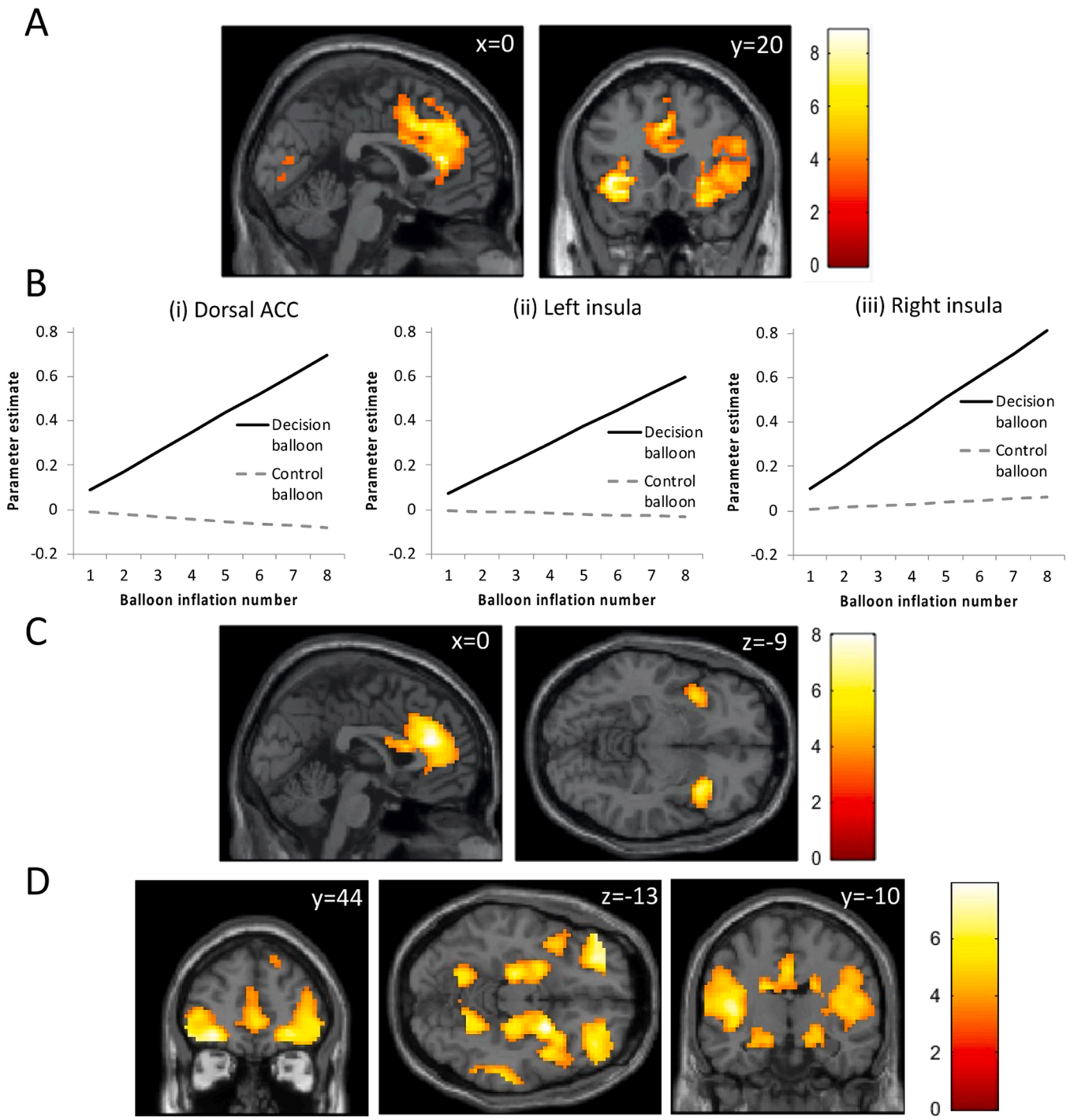

Fig. 2. Results of whole brain analyses of the whole group (N = 47) in the parametric analysis comparing the decision and control balloons for each trial type, thresholded at cluster corrected $p_{FWE} < 0.05$, voxel uncorrected $p < 0.001$. (A) Parametric $decision_{expand}$ - parametric $control_{expand}$ contrast, showing activation of the dorsal anterior cingulate cortex (ACC), and bilateral insula. (B) Line graphs where the slopes reflect the mean balloon inflation number parametric regressor estimate for $decision_{expand}$ (black line) and parametric $control_{expand}$ (grey line) trials for the (i) dorsal ACC, (ii) left insula and (iii) right insula clusters. (C) $Decision_{pop}$ - $control_{pop}$ contrast, with increased activation in the ACC and bilateral insula. (D) $Decision_{stop}$ - $control_{stop}$ contrast showing widespread activation of bilateral orbitofrontal cortex, inferior parietal and medial temporal lobes, as well as the left posterior insula and caudate. Colour bars indicate Z-score.

vs $decision_{expand}$ and Tanner stage in multiple clusters including the dmPFC, OFC, right temporal and frontal cortices, right hippocampus and occipital cortex (Table 4b). In these clusters, males who reported being at a lower Tanner stage showed greater brain activation in trials where the balloon popped after they had made an active risky decision compared with trials where the balloons successfully expanded. The difference in activation between trial types reduced with increasing pubertal Tanner stage. The interaction was driven by changes in brain activation in the unsuccessful outcome trials (Fig. 5).

(B) Line graphs illustrating the pattern of results in all clusters. The slopes reflect the mean parameter estimate of the linear association between Tanner stage and activation in $decision_{pop}$ (black line) and $control_{pop}$ (grey line) trials in the dmPFC cluster [peak voxel − 3 47 37] and the insula cluster [peak voxel − 31 11 − 14].

Table 2
Results of the comparison of decision and control expand (using the parametric regressor), pop and stop trials. Analyses were thresholded at cluster-level corrected $p_{FWE} < 0.05$, voxel-level uncorrected $p < 0.001$. * indicates peak voxel significant at voxel-level corrected $p_{FWE} < 0.05$.

Brain region	Size (voxels)	Z	MNI coordinates		
			x	y	z
(a) *parametric decision*$_{expand}$ vs *parametric control*$_{expand}$					
L anterior insula	479	6.73 *	-39	17	-5
L anterior insula		6.64 *	-33	17	-11
L putamen		3.95	-30	8	7
R anterior cingulate cortex	1169	6.58 *	3	32	13
R anterior cingulate cortex		6.24 *	3	17	37
L anterior cingulate cortex		5.63 *	-6	32	28
R inferior frontal cortex	1207	6.18 *	27	29	-2
R anterior insula		6.15 *	45	14	1
R amygdala		5.42 *	30	8	-14
R putamen		5.16	27	14	-2
R lingual gyrus	423	5.33 *	21	-76	-8
R lingual gyrus		4.98 *	12	-82	-8
R fusiform gyrus		4.36	30	-58	-14
R supramarginal gyrus	272	4.94 *	63	-37	31
R middle temporal gyrus		4.37	60	-46	10
R middle temporal gyrus		4.05	51	-40	1
R superior occipital gyrus	134	4.23	27	-79	22
R middle occipital gyrus		3.72	33	-79	10
L postcentral gyrus	98	4.15	-45	-31	49
L supramarginal gyrus		3.66	-57	-37	31
L superior parietal gyrus		3.52	-39	-46	61
***(b) decision*$_{pop}$ vs *control*$_{pop}$**					
R anterior cingulate cortex	907	6.29 *	3	38	22
Anterior cingulate cortex		4.58 *	0	17	16
R anterior cingulate cortex		4.56	12	29	13
R anterior insula	193	5.49 *	39	14	-5
R anterior insula		5.45 *	33	20	-11
L anterior insula	109	4.70 *	-39	11	-5
L anterior insula		4.47	-33	17	-11
***(c) decision*$_{stop}$ vs *control*$_{stop}$**					
R amygdala	7626	6.26 *	24	2	-14
L posterior insula		6.12 *	-42	-13	7
R hippocampus		5.67 *	27	-28	-8
R middle orbitofrontal gyrus		5.63 *	36	41	-11
L superior occipital gyrus		5.47 *	-9	-97	19
L caudate		5.46 *	-18	-13	25
L parahippocampal gyrus		5.45 *	-18	-22	-14
R angular gyrus		5.44 *	51	-58	46
R inferior parietal gyrus		5.33 *	54	-43	52
L middle orbitofrontal gyrus	430	6.18 *	-30	47	-11
L inferior orbitofrontal gyrus		5.73 *	-48	41	-14
L inferior frontal gyrus		4.74 *	-42	44	4
L inferior parietal gyrus	215	5.94 *	-51	-43	52
L inferior parietal gyrus		5.18 *	-48	-55	49
L angular gyrus		3.36	-60	-55	31

(B) Line graphs illustrating the pattern of results in all clusters. The slopes reflect the mean parameter estimate of the linear association between Tanner stage and activation in decision$_{pop}$ (grey line) and decision$_{expand}$ (black line) trials in the cerebellum/occipital lobe cluster [peak voxel-6 − 52 10] and the dmPFC cluster [peak voxel − 3 47 37].

4. Discussion

The current fMRI study investigated the relationship between brain activation during a risky decision-making task and pubertal status, as measured by self-reported Tanner staging or testosterone concentration, in 12.5–14.5-year-old males. Across the whole sample, brain regions involved in reward processing and cognitive control were activated in trials requiring active decision-making on the BART compared to trials where participants were not making decisions. Participants showed greater activation of the bilateral nucleus accumbens (NAcc) and right caudate on trials when they made a successful risky decision (expansion) compared to trials when they made a decision with a certain positive outcome or when their risky decision was unsuccessful (pop). The extent of NAcc activation was associated with neither the hormonal nor the physical measure of pubertal status. Physical Tanner stage was associated with activation in the left insula, dmPFC, right MCC, right putamen and right OFC in unsuccessful risky decision trials compared to unsuccessful control trials: males reporting lower Tanner stage scores showed greater activation of these regions during unsuccessful risky decision trials (compared to control unsuccessful trials) compared to males reporting higher Tanner stage scores. Tanner stage was also associated with brain activation in the comparison between unsuccessful risky decision-making trials and successful risky decision-making trials. Males reporting lower Tanner stage scores showed greater activation in the dmPFC, OFC, right temporal and frontal cortices, right hippocampus and occipital cortex in trials where the balloon popped after they had made an active risky decision than in trials where the balloons successfully expanded, compared to males with higher Tanner stages.

4.1. Activation in reward processing brain regions

The activation of multiple brain regions involved in reward processing and cognitive control, including the dorsal ACC, bilateral insula, bilateral parietal cortices, and the right occipital and temporal cortices (Fig. 2), with increasingly risky decisions on the BART task is in keeping with our prediction and other studies that have used the task (Granger et al., 1999; Fromme et al., 1997). Across the whole group, participants showed greater activation of the bilateral NAcc as well as the right putamen and left frontal operculum in trials in which they made successful risky decisions compared with those when they chose to stop inflating the balloon, making a safe choice with a guaranteed reward. These regions have been associated with voluntary risk-taking in previous studies using the BART task (Korucuoglu et al., 2020; Rao et al., 2008). While some studies have found puberty-related differences in NAcc activation during reward processing (Braams et al., 2015; Forbes et al., 2010; Op de Macks et al., 2011), we found no evidence of an association with puberty in this contrast in our sample, either using physical Tanner staging or sex steroid hormone concentration. However, the results from the previous literature are quite mixed, with different associations seen across studies (see (Vijayakumar et al., 2018; Goddings et al., 2019) for review). In one study, 11–13 year olds (males and females) in more advanced stages of puberty showed less caudate activation and greater rostral mPFC activation when processing reward outcome compared to their less mature, age-matched peers (Forbes et al., 2010). In addition, BOLD signal in the ventral striatum and testosterone were found to correlate positively during reward anticipation (in males), and negatively during reward outcome (in males and females; (Forbes et al., 2010). In contrast, Op de Macks and colleagues showed a positive correlation between testosterone levels and ventral striatum activation during reward outcome processing in males (Op de Macks et al., 2011). A more recent longitudinal study found no association between testosterone and NAcc activation during reward processing in participants aged 8–27 years after accounting for chronological age (Braams et al., 2015), but did find differences associated with a self-assessed pubertal score (the Petersen Pubertal Development Scale (PDS); (Petersen et al., 1988)), although this analysis did not take account of chronological age (Braams et al., 2015).

These inconsistent results might reflect differences in study design. First, since puberty and age are highly correlated in adolescence, our study design specifically focussed on a narrow age range in which males can be in any stage of pubertal development (Marshall and Tanner, 1970), and age was included as a covariate to minimise the possibility that any differences associated with pubertal development could be attributable to age. In our cross-sectional design, this means that our physical pubertal measure encompasses both self-reported pubertal stage and relative pubertal timing compared with peers. The volume of clusters reaching significance reduced in our data when age was not included as a covariate, which could indicate that the effects seen reflect pubertal timing i.e. when the physical changes associated with puberty

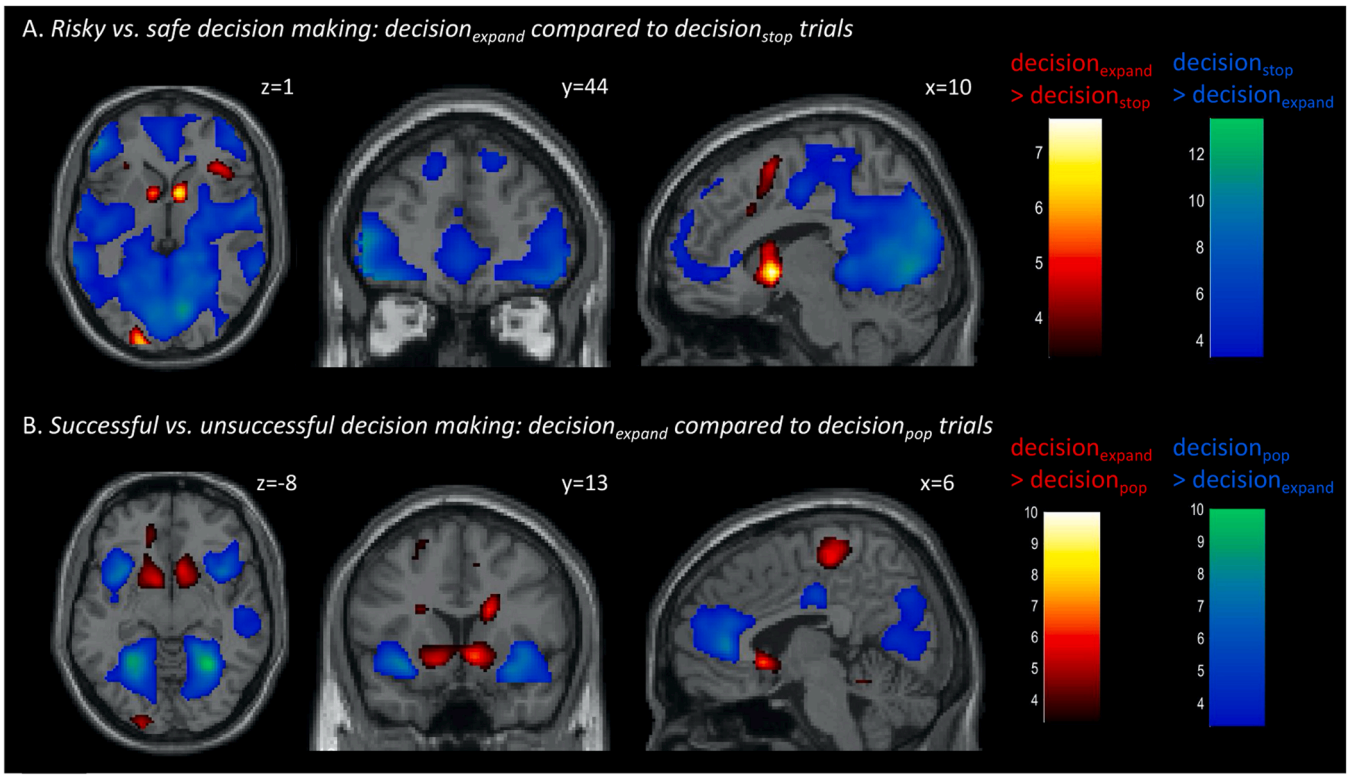

Fig. 3. Results of whole brain analyses of the whole group (N = 47), thresholded at cluster corrected $p_{FWE} < 0.05$, voxel uncorrected $p < 0.001$. Colour bars indicates Z score. (A) Risky vs. safe decision-making. $Decision_{expand}$ trials showed greater activation in bilateral nucleus accumbens, right caudate and left frontal operculum than $decision_{stop}$ trials (warm colours). Reversely, $decision_{stop}$ showed widespread greater activation than $decision_{expand}$ trials in bilateral orbitofrontal cortices, medial prefrontal cortex, bilateral occipital cortex, left temporal cortex and right amygdala (cool colours). (B) Successful vs. unsuccessful risky decision-making. $Decision_{expand}$ trials showed greater activation in bilateral nucleus accumbens, bilateral postcentral lobules and left occipital lobe than $decision_{pop}$ trials (warm colours). $Decision_{pop}$ trials showed greater activation of the bilateral insula, anterior cingulate cortex and fusiform gyri than $decision_{expand}$ trials (cool colours).

happen relative to an individual's peer group, but it is not possible to distinguish these associations from our data. Previous studies looking at pubertal associations with reward processing have incorporated wider age ranges, increasing the collinearity between puberty and age. For example, Op de Macks et al. (2011) tested 10–16 year olds but did not control for chronological age, making it more difficult to isolate puberty-specific associations (Op de Macks et al., 2011). Second, some studies have included both males and females (e.g (Braams et al., 2015; Forbes et al., 2010). We chose to limit our investigation to male participants as puberty differs significantly between the sexes in terms of the average age of onset, the predominant hormonal drivers for pubertal development (androgens vs. oestrogens) and the different secondary sexual characteristics that develop. The relationship between puberty and neurocognitive development may also differ between males and females (Shulman et al., 2015). Third, puberty is a complex bio-psychosocial developmental process, and different results may reflect differences in methods of pubertal measurement used (Goddings et al., 2019). In the current study, we found differences in brain activation with physical pubertal measures but not with salivary testosterone concentrations. The two measures reflect overlapping but distinct aspects of puberty. Physical pubertal measures provide an integrative indicator of the body's exposure to pubertal hormones, encompassing the length of exposure, levels of exposure and sensitive to hormones (Goddings et al., 2019). Self-assessment methods may also capture an individual's perception of their pubertal development, and the visible changes which may be perceived by wider society. In contrast, testosterone concentration provides a non-subjective measure of current circulating hormone levels that can be directly compared between participants. Both measures have their strength and limitations for interpretation and potential mechanisms underlying the associations seen, and understanding the differences between findings based on pubertal measure used remains an important challenge for the field.

4.2. Brain responses to unfavourable outcomes

When participants made an active risky decision (to inflate the balloon) and this had a negative outcome (the balloon popped and they lost their potential reward), there was activation of the bilateral insula and the dorsal ACC across the whole group compared with when the computer made a decision resulting in a negative outcome. The left insula and the dmPFC were also activated more by males reporting lower Tanner stages than those at higher tanner stages when the outcome of their risky decision-making trials was unsuccessful, compared to when it was successful. The dmPFC cluster overlapped with the dACC cluster reported in the main contrast of unsuccessful vs. successful risky decisions. Previous studies examining neural activation after unfavourable outcomes using different decision-making tasks have also reported insula and dorsal ACC activation in adolescent and adult samples, particularly in tasks where the probability of winning is not clear (Blankenstein et al., 2018), while younger children activated the insula but not the dorsal ACC region (Duijvenvoorde et al., 2015; Paulsen et al., 2015b). The insula has been identified in a meta-analysis as a brain region particularly active when individuals were facing losses as a result of their decisions, and has been postulated to be important for the representation of risk and the influence of emotion on risk processing (Mohr et al., 2010). Activation in the insula and dmPFC regions has previously been associated with lower levels of risk-taking in adolescents, particularly where exact levels of risk are unknown (ambiguous risks) (Blankenstein et al., 2018; Duijvenvoorde et al., 2015; Van Leijenhorst et al., 2010). Since the insula and dmPFC have been associated

Table 3
Results of analyses comparing decision expand trials to decision pop and decision stop trials. Contrasts were thresholded at cluster-level corrected $p_{FWE} < 0.05$, voxel uncorrected $p < 0.001$. * indicates peak voxel significant at voxel-level corrected $p_{FWE} < 0.05$.

Brain region	Size (no voxels)	Z	x	y	z
(a) decision$_{expand}$ vs decision$_{stop}$					
R nucleus accumbens	136	6.09 *	9	8	-2
R caudate		4.58 *	9	11	10
L middle occipital gyrus	73	5.98 *	-24	-97	7
L middle occipital gyrus		3.48	-18	-88	-8
L frontal operculum	137	5.25 *	45	20	7
L nucleus accumbens	72	4.99 *	-9	8	-2
R supplementary motor area	97	4.32	9	8	58
R supplementary motor area		3.95	15	5	64
R middle cingulate cortex		3.69	12	20	34
(b) decision$_{stop}$ vs decision$_{expand}$					
R fusiform gyrus	22,713	> 8.0 *	27	-52	-11
R fusiform gyrus		> 8.0 *	24	-46	-14
L fusiform gyrus		> 8.0 *	-24	-49	-11
L inferior orbitofrontal cortex		7.84 *	-36	38	-14
R lingual gyrus		7.72 *	12	-76	1
R amygdala		7.66 *	27	2	-14
L cuneus		7.63	-9	-91	-25
L inferior frontal gyrus		7.53 *	-45	41	7
L middle temporal gyrus		7.34 *	-57	-46	-8
L inferior temporal gyrus		7.31 *	-54	-49	-11
R calcarine fissure		7.27 *	21	-61	13
(c) decision$_{expand}$ vs decision$_{pop}$					
L postcentral gyrus	3556	> 8.0 *	-36	-22	55
R nucleus accumbens		6.09 *	9	17	-5
R paracentral lobule		5.93 *	12	-31	67
L paracentral lobule		5.77 *	-12	-31	70
R caudate		5.43 *	18	8	22
L nucleus accumbens		5.28 *	-9	8	-8
L calcarine gyrus	94	5.11 *	-12	-100	-2
R middle frontal gyrus	116	4.70 *	30	32	49
R middle frontal gyrus		4.29	36	41	34
(d) decision$_{pop}$ vs decision$_{expand}$					
R fusiform gyrus	2454	7.36 *	30	-55	-8
R fusiform gyrus		6.93 *	30	-40	-17
L fusiform gyrus		6.84 *	-27	-52	-8
R calcarine fissure		5.23 *	21	-58	10
R lingual gyrus		4.90 *	15	-73	-2
R cuneus		4.60 *	12	-88	31
L calcarine fissure		4.48	-18	-64	10
L cuneus		4.37	-9	-82	28
R parahippocampal gyrus		4.31	24	-28	1
L anterior insula	442	6.50 *	-33	17	-11
L anterior insula		6.13 *	-36	11	-8
L temporal pole		4.43	-30	5	-20
R anterior cingulate cortex	810	6.20 *	3	38	4
Anterior cingulate cortex		6.02 *	0	32	19
R medial superior frontal gyrus		4.77 *	3	53	19
R anterior insula	686	6.11 *	33	14	-14
R superior temporal pole		5.52 *	51	8	-17
R amygdala		4.93 *	30	2	-20
Middle cingulate cortex	135	5.61 *	0	-19	37
R superior temporal gyrus	802	5.40 *	57	-46	22
R supramarginal gyrus		5.33 *	60	-40	31
R middle temporal gyrus		5.21 *	54	-31	1
R middle occipital gyrus	125	4.90 *	42	-76	16
L supramarginal gyrus	177	4.46	-51	-49	28
L supramarginal gyrus		4.16	-63	-43	37

with the affective and cognitive components of uncertainty processing, increased activation may lead to risk aversion. Our findings suggests that changes in insula and dmPFC activation while processing risk between childhood and adolescence may in part be driven by pubertal changes between childhood and adolescence, providing a potential mechanism for the increased risky decision-making widely associated with adolescence.

Compared to participants reporting higher Tanner stages, males reporting lower Tanner stages showed greater activation of a number of other brain regions including the OFC, right temporal and frontal cortices, right hippocampus and occipital cortex when processing unsuccessful compared to successful risky decision trials (Fig. 5A). Since this contrast involved the same decision-making phase in both conditions, it reflects differences in brain activation during outcome processing, and across all regions the effect was primarily driven by changes in activation during decision$_{pop}$ trials (see Fig. 5B). Some regions showing association with pubertal stage overlapped with regions activated when comparing unsuccessful compared to successful risky decisions for the whole group. The variety of regions observed in these contrasts suggest that puberty may be associated with the development of a range of processes involved in processing and learning from the outcome of our decisions, and not only with the development of reward processing regions, which has been the focus of this research to date.

4.3. Variation in risk-taking

Self-reported levels of academic and aggressive/illegal risk-taking showed substantial variation in our sample and were positively correlated with testosterone levels, as well as with each other. In contrast, levels of high-risk health behaviours were low, with insufficient numbers reporting any alcohol use, drug use and sexual activity to include these subscales in the analyses. This is typical of the UK and other international populations at 12–14 years, when these behaviours still have relatively low prevalence (Hagell et al., 2015; Fitzsimons et al., 2018; Willoughby et al., 2013). A positive link between testosterone and risky behaviours in males has been shown in previous studies incorporating a wider age range including older adolescents when a greater range of typical health risk behaviours (for example, alcohol and drug use) would be expected (Vermeersch et al., 2008; Braams et al., 2016). The current data replicate the association between self-reported risk-taking behaviours and testosterone concentration in a group of young adolescents, focussing on low level risky academic and aggressive/illegal behaviours. These risks are known to increase in early adolescence (Fitzsimons et al., 2018), before the rise in drug use and sexual risk behaviours associated with mid-late adolescence (Willoughby et al., 2013).

In our sample, there were no pubertal-related differences in BART risk-taking. A longitudinal study examining an association between BART and testosterone in males aged 10–29 years also found no association between absolute testosterone concentration and task-based risk-taking, after accounting for age (Peper et al., 2018), although it did report an association between age-standardised testosterone levels and BART performance in participants aged 11–15 years (Peper et al., 2018). Future longitudinal studies should aim to further disentangle the developmental impact of chronological age and pubertal status, considering potential interactions between these two key developmental indices. Self-reported risk-taking was not correlated with BART performance in our young adolescent sample. BART represents decision-making in a lab setting on the day of collection, while the CARE used in our study is a self-report measure of expected future risky decision-making over a longer period and in real life settings. While a number of studies have correlated BART performance with real-life reported risk behaviours, these have tended to be in older adolescents or high-risk adolescent groups, and have involved health risks in which our participants were not engaging e.g. smoking, drug use (Lejuez et al., 2007; Galván et al., 2013). This does not necessarily indicate that the BART and the CARE questionnaire are not indicators of risk-taking, but that they may be measuring different aspects of this multi-faceted and complex behaviour e.g. positive vs negative risk-taking (Duell and Steinberg, 2019), and in different environments (Mamerow et al., 2016). An alternative possibility may be that performance on the BART task in younger adolescents might correlate with risk behaviours in later adolescence where there are more opportunities for risky behaviours e.g. easier access to alcohol. Further work is needed to assess whether BART performance is associated with variability in levels of risk-taking in younger adolescent and non-clinical, community-based samples who

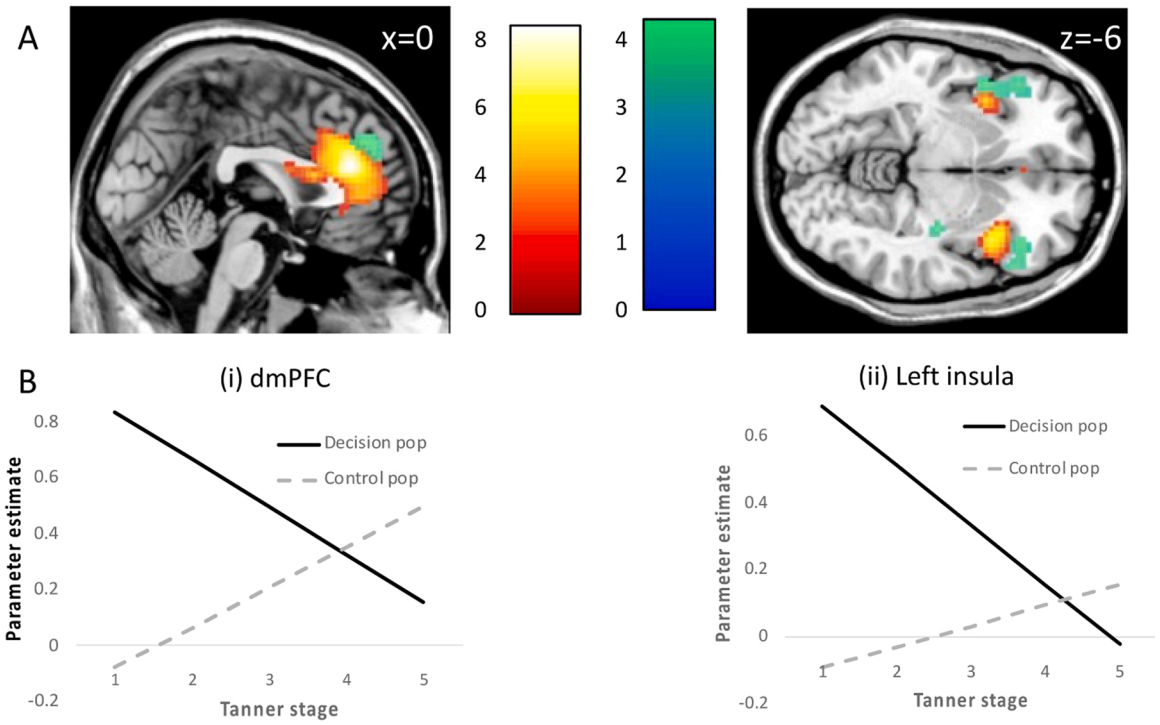

Fig. 4. Results of the correlation between decision$_{pop}$ vs control$_{pop}$ and Tanner stage, thresholded at cluster corrected $p_{FWE} < 0.05$, voxel uncorrected $p < 0.001$ including participant age as a covariate. Colour bars indicates Z score. (A) Decision$_{pop}$ vs. Control$_{pop}$ contrast showing greater activation in the dmPFC and bilateral insula/orbitofrontal cortex regions (cool colours) in those at lower Tanner stage than those at higher Tanner stage superimposed on the results of the decision$_{pop}$ vs. control$_{pop}$ contrast for the whole group (warm colours).

Table 4
Regions showing a reduction in the neural response to the negative outcome of a risky decision ((a) decision pop vs control pop trials, (b) decision pop vs decision expand trials) with increasing Tanner stage, including age as a co-variate. Contrasts were thresholded at cluster-level corrected $p_{FWE} < 0.05$, voxel uncorrected $p < 0.001$.

Brain region	Size (no voxels)	Z	x	y	z
(a) Negative correlation between Tanner stage and decision$_{pop}$ vs control$_{pop}$ contrast					
L insula	100	4.14	-33	11	-14
L superior temporal pole		3.66	-48	11	-2
L inferior orbitofrontal cortex		3.47	-48	29	-5
R middle cingulate cortex	155	4.03	9	17	43
R precentral cortex		3.77	45	5	40
R superior frontal cortex		3.75	21	11	40
R superior medial frontal cortex	90	3.96	3	47	34
L middle temporal cortex	91	3.92	-66	-34	1
R putamen	108	3.91	30	-13	10
R inferior orbitofrontal cortex	87	3.58	51	32	1
(b)Negative correlation between Tanner stage and decision$_{pop}$ vs decision$_{expand}$ contrast					
L cerebellum	1357	5.06	-6	-52	10
L lingual gyrus		4.31	-15	-79	-5
L middle occipital cortex		4.30	-33	-82	4
R precuneus		4.13	3	-61	28
L calcarine fissure		4.09	-12	-88	-5
R cuneus		3.92	15	-70	28
R superior occipital cortex		3.86	21	-94	16
L medial orbitofrontal cortex	256	4.42	-3	50	-11
L superior medial frontal cortex		3.66	0	56	10
L olfactory cortex		3.32	-3	17	-8
R hippocampus	91	4.31	30	-16	13
R superior temporal cortex	74	4.04	69	-19	7
L superior medial frontal cortex	81	4.03	-3	47	37
R inferior orbitofrontal cortex	92	3.99	48	44	-11
R middle frontal cortex	140	3.96	33	8	40
R superior frontal cortex		3.93	15	26	40

show overall low propensity to take risk, and longitudinal trajectories of risk-taking behaviours during adolescence.

4.4. Methodological considerations

While our findings add to an understanding of the role of puberty in functional development of the adolescent brain during decision-making, there are limitations to this study. Our sample was cross-sectional and the sample size was constrained by resources, limiting the power of the study to investigate small effect sizes or individual differences. Longitudinal studies that assess the relationship between pubertal changes and brain development within the same individuals are needed to explore different aspects of pubertal development, including status, tempo and timing. Salivary hormone levels were only tested at a single timepoint. While the timing of saliva collection was standardised between participants (early morning waking sample), this method does not fully capture differences between individuals in diurnal variation (Granger et al., 2003). Self-reported Tanner staging has been shown to be an adequate measure of pubertal stage during adolescence (Shirtcliff et al., 2009) and is currently the most appropriate tool available. It is nevertheless subject to potential inaccuracies as it relies on young people's understanding of their body's development. We opted to use the CARE questionnaire measure assessing future expected behaviour rather than past behaviour. This was to ensure that young people did not feel that they had to report behaviour that might be illegal or deemed socially inappropriate, but this may also have influenced the self-report and not be completely aligned with actual behaviour. Finally, we selected the BART as a measure of behavioural risk-taking as it has been widely used in adolescent populations, including in fMRI studies (Duell et al., 2018; Peper et al., 2018; Humphrey and Dumontheil, 2016; Qu et al., 2015; Rao et al., 2008; Schonberg et al., 2012). The asymmetry of the task, such that a 'stop' decision is always followed by a certain outcome, ensures that participants know that this is the 'safe' option, but means it is not possible to separate the decision-making step from seeing

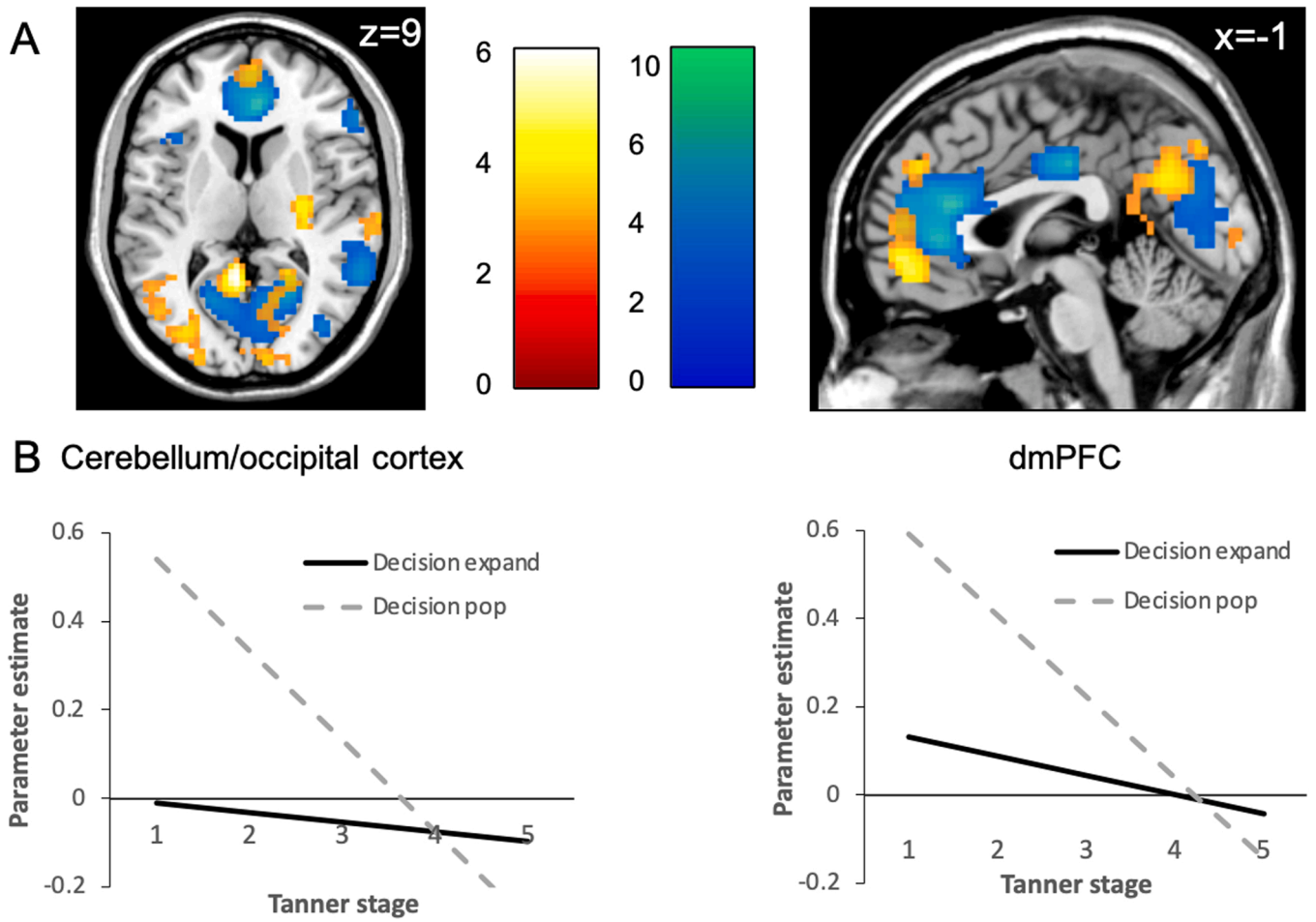

Fig. 5. Results of the correlation between decision$_{pop}$ vs decision$_{expand}$ and Tanner stage, thresholded at cluster corrected $p_{FWE} < 0.05$, voxel uncorrected $p < 0.001$ including participant age as a covariate. Colour bars indicates Z score. (A) Decision$_{pop}$ vs. decision$_{expand}$ contrast showing greater activation in the mPFC, cerebellum, occipital cortex, hippocampus and superior temporal lobe (warm colours) in those at lower Tanner stage than those at higher Tanner stage superimposed on the results of the decision$_{pop}$ vs decision$_{expand}$ contrast for the whole group (cool colours).

the outcome in the stop trials. It was also not possible using this task to explore potential differences in neural activation when anticipating the outcome of a risky decision compared to processing this outcome when seen. One previous study has identified different puberty-related brain activation in males when anticipating a reward compared to processing a reward (Forbes et al., 2010), and future studies using a variety of risk-taking tasks should explore these different phases further.

5. Conclusion

The current study investigated the role of puberty in the decision and outcome phases of a risky decision-making task in adolescent males aged 12.5–14.5 years. Including a sample of participants within a narrow age range allowed us to separate chronological and pubertal effects. Higher testosterone level was associated with more self-reported academic and aggressive/illegal risk-taking, but not experimental task-based risk-taking, in this young adolescent sample. There was also evidence of puberty-related differences in brain activation during trials where participants made unsuccessful risky decisions, with participants in earlier stages of puberty showing greater neural activation than those in later stages of puberty in the left insula, dmPFC and other brain regions. This may represent changes in puberty-related sensitivity to negative feedback during early to mid-adolescence in males.

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CRediT authorship contribution statement

ALG, RMV, SJB conceived and designed the study, ALG collected the data, ALG and ID analysed the data, ALG, ID, RMV and SJB wrote the manuscript. All authors gave final approval for publication.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.dcn.2023.101230.

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