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Impact of Cognitive-Affective-Motivational Factors on Trajectories of Risk Taking in Adolescence: Evidence from the CogBIAS Longitudinal Study

[redacted]

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Abstract

While many neurodevelopmental accounts of risk-taking behaviours in adolescence point to an adolescent brain-behavioural phenotype characterised by reward hypersensitivity, less research has focused on individual differences in the cognitive processing of emotional stimuli which may trigger approach motivated behaviours, predisposing some adolescents to take risks. This study sought to examine individual markers for age-related changes in risk propensity, measured on the Balloon Analogue Risk Task (BART), and real-world risk involvement by modelling both outcomes longitudinally in the same sample of adolescents. Behavioural inhibition, drive, reward responsivity, fun seeking, negative urgency and sensation seeking were assessed in a large normative sample (*N* = 504; mean ages: 13.4, 14.5, 15.7) across three waves as part of the CogBIAS Longitudinal Study. Age, NU, SS and Drive all uniquely predicted increased RI, while RR predicted lower prospective RI. In contrast, age was the only robust predictor of increased scores on the BART, with negative urgency inhibiting risk propensity. The strongest indicator of prospective risk involvement was negative urgency, with the greatest impact at ages 15–17. Interpretation bias, known to play a role in the triggering and maintenance of anxiety, was found to moderate the effect of negative urgency on risk involvement in later adolescence, offering a cognitive mechanism that may prove a useful focus for prevention or early intervention. This study adds to the differential susceptibility literature by examining the structure and function of an adolescent motivational drive and proposing key cognitive-affective markers for neurobiological reactivity predicting risk-taking in adolescence.

Keywords: *motivation, emotion, risk-taking, adolescence*

Declaration

This thesis contains no material which has been accepted for the award of any other degree of diploma in any University, and, to the best of my knowledge, this thesis contains no material previously published except where due reference is made. I give permission for the digital version of this thesis to be made available on the web, via the University of Adelaide's digital thesis repository, the Library Search and through web search engines, unless permission has been granted by the School to restrict access for a period of time.

Contributor Roles Table

ROLE	ROLE DESCRIPTION	STUDENT	SUPERVISOR 1	SUPERVISOR 2
CONCEPTUALIZATION	Ideas; formulation or evolution of overarching research goals and aims.	X	X	X
METHODOLOGY	Development or design of methodology; creation of models.	X		
PROJECT ADMINISTRATION	Management and coordination responsibility for the research activity planning and execution.		X	
SUPERVISION	Oversight and leadership responsibility for the research activity planning and execution, including mentorship external to the core team.		X	
RESOURCES	Provision of study materials, laboratory samples,		X	X

	instrumentation, computing resources, or other analysis tools.			
SOFTWARE	Programming, software development; designing computer programs; implementation of the computer code and supporting algorithms; testing of existing code.	X		
INVESTIGATION	Conducting research - specifically performing experiments, or data/evidence collection.			
VALIDATION	Verification of the overall replication/reproducibility of results/experiments.	X		X
DATA CURATION	Management activities to annotate (produce metadata), scrub data and maintain research data (including software code, where it is necessary for			X

	interpreting the data itself) for initial use and later re-use.			
FORMAL ANALYSIS	Application of statistical, mathematical, computational, or other formal techniques to analyze or synthesize study data.	X		
VISUALIZATION	Visualization/data presentation of the results.	X		
WRITING – ORIGINAL DRAFT	Specifically writing the initial draft.	X		
WRITING – REVIEW & EDITING	Critical review, commentary or revision of original draft		X	X

Author Statement

In the preparation of this thesis, I used Grammarly to help detect minor grammatical errors on the final draft of my thesis. I take full responsibility for the content of the thesis, having reviewed and edited the content, and verified all original sources relied upon.

Adolescence, defined as the life stage between puberty onset and the increasing independence of emerging adulthood (Blakemore & Choudhury, 2006), is a time of considerable social, cognitive, neural, and physiological development (Becht et al., 2018). It is also a time of increased risk, with many delinquent (e.g., illegal activity; substance abuse; traffic accidents) and health-harming behaviours (e.g., sexually transmitted infections; death by accident or suicide) increasing or peaking in adolescence (Centers for Disease Control and Prevention, 2021). While there is substantial inter-individual variability in the degree to which adolescents take risks (Crone & van Duijvenvoorde, 2021; Crone et al., 2016); Crone & van D, 2021) and the types of risks they take (Peeters et al., 2019), for those that do, these are relatively enduring patterns of behaviour (Booth et al., 2019) with life-changing consequences.

Motivation may be defined as the amount of effort or energy an individual is willing to exert to attain their goals (Knutson & Srirangarajan, 2019). While closely interrelated, emotion defines internal subjective states that influence the strength and direction of an individual's actions (Fox, 2018; Neta & Haas, 2019). Several lines of developmental research suggest that adolescence represents a critical window during which individual variations in neurobiological reactivity, driven by genetic differences, temperament, or personality (Belsky et al., 2009; Belsky & Pluess, 2013; Moore & Depue, 2016; van Ijzendoorn & Bakermans-Kranenburg, 2015) exert a heightened influence on otherwise normative affective-motivational changes via cascading neuromaturational processes (van Duijvenvoorde et al., 2014). As such, adolescence reflects a period of heightened risk for maladaptive psychosocial outcomes and heightened opportunity for flourishing and resilience (Booth et al., 2022; Booth et al., 2017). The central CogBIAS hypothesis proposes that cognitive processing biases, which are implicit and automatic, mediate the causal relationship between differential susceptibility to the environment, of which heightened emotional reactivity is a phenotypic expression, and pathological and resilient psychological functioning (Booth et al., 2017; Fox & Beevers, 2016; Fox et al., 2010). Cognitive biases (CBs) are thus key mechanisms by which emotional dispositions, reactions, and prevailing mood patterns are triggered and maintained (Fox & Beevers, 2016; Parsons et al., 2021). Figure 1, Appendix 1 presents the overarching framework for this study (Fox & Keers, 2019).

As a crucial period for goal-directed or motivated behaviour, such as exploration, forming new relationships, increasing intimacy, social learning and risk-taking (van Duijvenvoorde et al., 2016), understanding how these risk-reward neural substrates interact with subjective affective states to impact risk behaviours is a crucial empirical question. Despite this, relatively little research has examined compositional changes in affective-motivational variables which may predispose some adolescents to take risks. Furthermore, this longitudinal study is the first to examine how negative interpretation bias, a CB highly implicated in adolescent anxiety and risk outcomes (Brettschneider et al., 2015; Lau, 2013; Mathews & MacLeod, 2005; Miers et al., 2008), interacts with these affective-motivational factors to impact risk-taking trajectories across adolescence.

Definition, operationalisation and measurement of risk behaviours

The definition and operationalisation of risk and risk-taking in the behavioural sciences remains one of the critical challenges to research in the field (Trimpop, 1994). The constructs *risk*, *risk preference* or *propensity*, and *risk involvement* in real-world contexts, as highly contextualised behaviours, are not a unitary construct across populations or across development (Peeters et al., 2019). Two key paradigms in risk-taking research, behavioural economics, which defines risk-taking in terms of idiosyncratic preferences towards probabilities (Hertwig et al., 2019) and the developmental psychopathology approach, with its primary focus on risk-taking as behaviours increasing the potential for physical or psychosocial harm (Schonberg et al., 2011), have led to substantially different accounts of what risk-taking is, and to what extent, and why, adolescents engage in it.

Risk propensity is defined as a tendency to *discount probabilities* (Shead & Hodgins, 2009); either by overvaluing the (riskier) positive outcome or by undervaluing the (probabilistic) negative outcome (Reyna & Farley, 2006). Analogous active cognitions may be, “What’s the best that could happen?"; "It might not happen"; "What do I have to lose?" or even "Go for broke!". Though closely related, cognition research has consistently found a distinction between risk propensity and impulsivity (Holt et al., 2003), defined behaviourally as the tendency to overweight cues for near-term anticipated reward (*delay discounting*). A further distinction is made between *impulsive choice*, a tendency to prioritise immediate gains over delayed greater gains or losses (e.g., "I want it *now*" / "I'll worry about it later") and *impulsive action*, reflecting a failure to inhibit the stimulus-activated response (Isles et al., 2019), which are mediated by distinct neural circuitries (Bari & Robbins, 2013). However, while a discrete behavioural trait, impulsivity is likely to precede, mediate, or moderate some types of risk-taking, particularly in real-world settings (Defoe & Romer, 2022).

Attempts to reliably quantify adolescent trajectories of risk-taking attitudes in experimental paradigms have been mixed. A series of meta-analytic studies (Defoe et al., 2015; Defoe et al., 2019) found that risk-taking was highest in children and early adolescents (11–13-year-olds), decreasing in mid-late adolescence (ages 14–19) into adulthood. However, these effects were partially mediated by contextual and task demands. The recent *Developmental Neuro-Ecological Risk-taking Model* (DNERM) proposes that, rather than an adolescent risk-taking behavioural phenotype, increased risk-taking in adolescence is substantively attributable to differential environmental exposure at a vulnerable developmental stage (Defoe, 2021). Previous CogBIAS study findings found that mean risk involvement increased across adolescence, although the data best fit a random slopes model, reflecting substantial inter-individual variability in direction of change. Risk-taking on the Balloon Analogue Risk Task (Lejuez et al., 2007) also increased, though differential stability across waves was low, reflecting that baseline scores were not a reliable indicator of subsequent scores (Booth et al., 2019). Identifying compositional changes in individual difference predictors of risk outcomes across adolescence, to infer the processes underlying these behaviours, is thus an important empirical question. One way to measure these differences is to compare the relative impact of relatively stable personality traits (Roberts et al., 2012) known to influence risk-taking within the same population over time. To our knowledge, this is the first longitudinal study to compare the impact of affective-motivational factors on two risk outcomes: risk involvement, operationalised as real-world engagement in activities likely to cause physical or psychosocial harm, and risk propensity, operationalised as taking more chances to win earnings on the BART.

Adolescent development

Key to many neurodevelopmental theories of risk-taking in adolescence, such as dual processing (Steinberg, 2007; Strang et al., 2013), triadic processing (Ernst, 2014), and imbalance models (Casey et al., 2011; Casey, 2015) is the concept of heightened *reward-drive* tendencies (Galván, 2013; van Duijvenvoorde et al., 2016). This is an umbrella term for various reward-approach, reward-valuing, sensation-seeking and exploratory behaviours (Avila et al., 2008; Bjork & Pardini, 2015; Crowley et al., 2014) A wealth of neurobiological research points to changes in adolescent neural architecture and neurochemistries reflecting fine-tuning of motor functioning (Fuhrmann et al., 2015), higher order cognition (Choudhury et al., 2008; Van Leijenhorst et al., 2010) and cognitive control (Casey et al., 2011; Hayden, 2019), but also increased salience of rewarding environmental stimuli – particularly social stimuli (Blakemore, 2008; S.-J. Blakemore, 2018); heightened neural activation in response to rewards (Silverman et al., 2015), and heightened motivation to obtain rewards (Lamm et al., 2014; van Duijvenvoorde et al., 2014).

Multiple lines of research have thus been framed by this heuristic model of adolescent incentive/reward-approach motivation associated with dopaminergic systems and the encoding of positive emotional stimuli (Crowley et al., 2014; Galvan et al., 2007; Sherman et al., 2018; Sutton et al., 2022; Urošević et al., 2014). Positive affect was associated with increased likelihood of illicit substance use and risky sexual behaviours in young adults (Zapolski et al., 2009) with these behaviours predicted by hormonal changes and increased reward-related neural activation in Functional Magnetic Resonance Imaging (FMRI) studies (Peper et al., 2018; van Duijvenvoorde et al., 2016). Adolescents who selfdescribed as thrill-seeking took more risks in behavioural risk-taking tasks (Blankenstein et al., 2016) while Willmott & Ioannou (2017) differentiated a subset of thrill-seeking public rioters, who reported recreation, adventure seeking and fun as motivators for their behaviour.

Empirical findings, however, suggest a more complex view of the affective states and traits underlying some risk-taking behaviours (Gutnik et al., 2006; Kusev et al., 2017). Negative mood states were found to increase engagement in risky behaviours (Loewenstein et al., 2001). Elevated stress predicted increased likelihood of risky sexual, illicit substance-use, and health-harming behaviours in undergraduate and graduate students (Nelson et al., 2008). A recent meta-analysis of published and unpublished experimental studies (N = 91) found negative affect to increase appetitive risk behaviours (Ferrer et al., 2020). In the clinical literature, impulsivity and risky behaviours were found to be elevated among individuals with posttraumatic stress disorder (Tull et al., 2014). Strong positive associations were found between anxiety and impulsivity in binge eating among adolescents (Lim et al., 2019). Anxiety and impulsivity together predicted more severe risk behaviours (e.g. suicidality and mood disorders) in at-risk adolescents (Askénazy et al., 2003). Negative urgency, a dimension of impulsivity, is reliably predictive of self-harming and risky behaviours (Cyders et al., 2009; Dir et al., 2016; Dir et al., 2013; Ratner et al., 2022).

This complexity is modelled in biologically-based frameworks of motivation, self/identity and affect, such as Reinforcement Sensitivity Theory (RST; Gray 1990) where notably, the functional boundaries between the neural behavioural activation and behavioural inhibition systems (BAS/BIS) are not discrete, but overlapping; (e.g., BIS-mediated *defensive approach*; for a review see Corr & Perkins, 2006). Harmon-Jones et al. (2013) thus define approach motivation as the *impulse to move forward*, without specifying the *valence of stimuli* towards which the impulse is directed. In their review, they found that while positive affect was often a trigger for approach motivation, approach was occasionally evoked by negative stimuli, and may be *experienced* as a negative state. Reactive Approach Motivation

(RAM; McGregor, 2010) extends this hypothesis by proposing no other human motivation than the need to downregulate anxiety.

This study specifically assessed approach/avoidance motivation (BAS/BIS) and dimensions of impulsivity in relation to two hypothesised pathways to risk-taking: a positive/reward approach pathway, operationalised as higher levels of BAS Reward Responsivity (RR), BAS Drive, BAS Fun Seeking (FS) and Sensation Seeking, and a negative/reactive approach pathway, operationalised as higher levels of BIS and Negative Urgency. Considerable evidence supports a connection between distinct combinations of BIS and BAS activity and specific forms of psychopathology, and increasingly, risk behaviours (Kemp et al., 2019; Leota et al., 2023). Based on prior CogBIAS studies (Booth et al., 2019) we expected the impact of Drive on risk outcomes to increase over time. As evidence of SS trajectories in adolescence were mixed, with some studies showing mid-adolescent peaks (Peper et al., 2018) and others showing increases into young adulthood (Defoe et al., 2015; Defoe et al., 2019), we made no hypotheses as to the systematic effects of SS on risk outcomes across adolescence. Based on findings of increasing internalising symptoms in our sample (Songco et al., 2020), we expected the impact of NU and BIS on risk outcomes to be higher in later study waves. To assess the potential mediating effect of interpretation bias on risk-taking, we specifically assessed the impact of negative social interpretation bias (NSB) and negative non-social interpretation bias (NNonSB) combined with high BIS and high NU. Negative interpretation bias is defined as the tendency to interpret ambiguous situations in a hostile or threatening way (Schoth & Liossi, 2017). Meta-analytic findings support an association between negative interpretation bias and anxiety in children and adolescents (Stuijfzand et al., 2018) and social anxiety across populations (Chen et al., 2020). We expected higher levels of both negative interpretation bias to have an amplifying effect on risk-taking, in line with a RAM hypothesis. As gender and SES are known to influence psychosocial and risk outcomes (Barr et al., 2015; Daughters et al., 2013; Reniers et al., 2016) we controlled for these demographic variables in our analyses. Linear Mixed Effects Modelling (LMM) enabled us to test the relative strength of these predictors over time, while also modelling interand intra-individual variability in participants' scores (Meteyard & Davies, 2020). An advantage of LMM is that it is robust to many of the challenges faced using traditional analytic approaches for longitudinal data. For example, partially missing data, nonnormality, discrete measurement scales, non-linear trajectories and unequally-spaced time points.

Study Aims

The aims of this study were thus fourfold: Firstly, to examine individual markers for age-related changes in risk propensity (RP) and real-world risk involvement (RI) by assessing the relative strength of six known predictors of risk behaviours (age, RR, Drive, FS, BIS, NU, and SS) on both outcomes. Secondly, to assess the differential impact of each of these predictors across adolescence. Thirdly, to examine the relative impact of positive/reward-approach vs. negative/reactive-approach on both risk outcomes. Fourthly, to explore the moderating effect of negative interpretation biases on these relationships.

Our primary hypotheses were:

- Higher levels of BAS RR, BAS Drive, BAS FS, BIS, NU, and SS would independently predict increased RP and RI across waves, controlling for gender and SES. However, we made no predictions about the relative compositional strength of predictors for each outcome.

Our secondary hypotheses were:

- The positive impact of Drive on RP and RI would remain constant or increase across waves.
- The positive impact of NU and BIS on RP and RI would increase between Waves 2 and 3.
- The interactions between BIS and NSB, BIS and NNonSB, NU and NSB, and NU and NNonSB, (operationalised by the interaction terms BIS*NSB, BIS*NNonSB, NU*NSB and NU*NNonSB),

would increase RI across waves over and above their independent effects. However, we made no hypotheses as to their effects on RP.

These hypotheses and others were pre-registered on the Open Science Framework before accessing the data (https://osf.io/fsz4g).

Method

Participants

Data were selected from the three-wave CogBIAS Longitudinal Study (CogBIAS-L-S; Booth et al., 2017) which examined risk and protective factors to emotional and psychosocial functioning in adolescence. Data was collected over a four-year study period (2014 – 2018) in Southeast England, with assessment spaced 12 – 18 months apart to optimise identification of factors associated with developmental stability and change. Ethics approval was obtained from the United Kingdom (UK) National Research Ethics Service (NRES) Committee South Central, National Health Service (NHS, UK; Project ID: 141833; 14/SC/0128). Participant mean ages across each wave was 13.4 (SD = 0.7; N = 504; 55% female), 14.5 (SD = 0.6; N = 450; 56% female), and 15.7 (SD = 0.6; N = 411; 58% female). Participants were predominantly Caucasian (75.33%), with a median highest combined level of parental education of 4 ("Bachelor degree"; IQR = 2), a reliable indicator of socioeconomic status (Sohr-Preston et al., 2013). Exclusion criteria specified no diagnosis of a psychiatric or neurological disorder. Attrition was relatively low at 18.5%, compared with an average of 26.5% for similar studies reported in a meta-analysis (N=143; Teague et al., 2018). Independent samples t-tests revealed no effect of SES, cohort or ethnicity on attrition; however, more females than males were retained, t (502) = – 2.86, p = .004, d = .25. Full cohort profiles for each wave are detailed in Table 1 below.

Table 1. *Demographic information for the sample by cohort and wave*

Wave 1											
Cohort	Total	X1	X2	X3	X4	X5	X6	X7	X8	X9	X10
N	504	15	30	62	47	13	34	119	104	54	26
Mean Age (SD)	13.4 (.7)	12.6 (.4)	11.7 (.3)	13.4 (.3)	13.4 (.3)	12.2 (.4)	12.8 (.3)	14.0 (.4)	13.1 (.3)	14.3 (.3)	13.2 (.3)
Year group	7–9	7–8	7	8	8	7–8	8	9	8	9	8
Gender (% Female)	55%	40%	50%	100%	100%	100%	47%	0%	100%	0%	58%
Ethnicity (% Caucasian)	75%	60%	87%	68%	72%	69%	59%	86%	69%	76%	85%
SES (Median, IQR)	4 (2)	4 (2)	3 (2)	4 (2)	3 (2)	4 (2)	2 (2)	4 (1)	4 (2)	4 (2)	3 (2)
Wave 2											
Cohort	Total	X1	X2	X3	X4	X5	X6	X7	X8	X9	X10
N	450	9	25	60	40	6	26	109	101	50	24
Mean Age (SD)	14.5 (.6)	14.0 (.4)	13.3 (.3)	14.5 (.3)	14.8 (.3)	13.5 (.2)	14.0 (.3)	15.1 (.4)	14.1 (.3)	15.4 (.3)	14.3 (.3)
Year group	8–10	8–9	9	9	10	8–9	9	10	9	10	9
Gender (% Female)	56%	56%	52%	100%	100%	100%	42%	0%	100%	0%	58%
Ethnicity (% Caucasian)	75%	56%	84%	67%	73%	67%	65%	86%	69%	74%	42%
SES (Median, IQR)	4 (2)	4 (2)	3 (2)	4 (2)	3 (2)	4 (2)	2 (2)	4 (1)	4 (2)	4 (2)	3 (2)
Wave 3											
Cohort	Total	X1	X2	X3	X4	X5	X6	X7	X8	X9	X10
N	411	8	22	62	37	12	12	92	92	50	24
Mean Age (SD)	15.7 (.6)	15.3 (.4)	14.8 (.3)	15.9 (.3)	15.8 (.3)	14.5 (.4)	15.0 (.3)	16.0 (.4)	15.4 (.3)	16.1 (.3)	15.3 (.3)
Year group	9–11	10–11	10	11	11	9–10	11	11	11	11	10
Gender (% Female)	58%	50%	46%	100%	100%	100%	67%	0%	100%	0%	58%
Ethnicity (% Caucasian)	76%	63%	86%	68%	73%	75%	75%	85%	70%	74%	88%
SES (Median, IQR)	4 (2)	4 (2)	3 (2)	4 (2)	3 (2)	4 (2)	2 (2)	4 (1)	4 (2)	4 (2)	3 (2)

Note. Xn = Cohort Number; SES Socio-Economic Status; SD Standard Deviation; IQR Interquartile Range. Wave 2 attrition, 11%; Wave 3 attrition, 19%. Table reproduced from Booth et al. (2019).

Procedure

A two-phase school recruitment process was followed by the sending of Parental information sheets, disclosing the study purpose and confidentiality and anonymity of their child's data, consent forms, and a family demographic questionnaire in paper format or online version to entire student year groups. At each testing session, conducted during school hours either at the school's or at University of Oxford computer labs, participants were tested in groups (6–50 participants per session). Participants were instructed to complete the tasks on individual computer screens under exam conditions, (i.e., no talking or looking at their peers' screens). At least two trained research assistants and one teacher were present throughout testing. Measures were completed in fixed order of six behavioural tasks assessing attention, interpretation, and memory biases, and 13 self-report questionnaires assessing mood, psychosocial functioning, and trait features. Tests were completed in one two-hour session (with break), or two one-hour sessions on separate days. Participation was voluntary and participants were thanked, debriefed, and given a £10 (AU$20) Amazon voucher as compensation for their time. All data was anonymised and stored securely (password-protected server). Linkage codes and any personal identifying information were stored separately in a secure location.

Measures

Data from three self-report and two behavioural measures were selected for the current study. Continuous over categorical variables were selected to detect subtle differences in inter- and intra-individual stability and change (Altman & Royston, 2006).

Risk involvement in real-world settings was assessed with a modified version of the self-report *Risk Involvement and Perception Scale* (Lavery et al., 1993). As the target population was adolescents with healthy psychosocial development, 9 items reflecting extreme or antisocial behaviours (e.g., "taking cocaine") were removed from the original 23-item scale, developed for clinical adolescent samples. The remaining 14 items reflected a wide spread of "typical" behaviours broadly representative of mid-adolescent risk involvement, ranging from behaviours involving some measure of social, psychological and/or physical risk (e.g., “sunbathing”, “smoking cigarettes”, “skipping school”), those that are age-inappropriate (e.g., “having sex”, “drinking alcohol”), and behaviours that are societally illegal (e.g., “smoking marijuana”, “shoplifting”). Participants rated how frequently they undertook the risk behaviour over the past 12 months on a 9-point Likert scale ranging from 0 (“Never”) to 8 (“Daily or more”). The Risk Perception and Benefit Perception subscales were not used in the current analyses. RI was calculated by averaging the frequency of risk behaviours, with higher scores reflecting more regular endorsement of a given risk behaviour. The 23-item subscale demonstrated good criterion validity, internal consistency ($\alpha = .72$) and test-retest reliability (r =.72–.79; Lavery et al., 1993).

Risk-taking propensity was assessed with the BART-Y (Lejuez et al., 2007), a youth-adapted form of the BART (Lejuez et al., 2002). In this behavioural task, participants are instructed to pump a computer-animated red balloon by left-clicking on a button displayed on screen, and to save the points gained from each pump by left-clicking on a separate ‘bank’ button displayed on screen below a points meter. Each balloon ‘pump’ gains one point, and the aim is to bank as many points as possible. Participants are instructed that they should bank their points before they think the balloon will burst, as if the balloon bursts before banking, all gains are lost, and a new trial begins. Participants complete 20 balloon trials, which have an average bursting point of 60 pumps, ranging from 10 to 111. Participants are informed that “the explosion point varies across each of the 30 balloons, ranging from the first pump to enough pumps to make the balloon fill the entire computer screen” (Lejuez et al., 2007, p. 106). Consistent with previous uses of the BART (e.g., Lejuez et al., 2002), risk taking propensity was operationalised as the average number of pumps on balloons that did not burst. This adjusted value is optimal as the outcome is not constrained by the bursting point, which would limit between-subject variability (Lejuez et al., 2002). The BART demonstrates good split-half reliability (r = .70) and good incremental validity ($R^2\Delta$ = 4.5%), controlling for demographic and self-reported disinhibition variables (Lejuez et al., 2007). Within our study, the BART demonstrated adequate intrarater reliability/inter-wave variability ($ICC_{3,1}$ = .60, [95% CI = .51 – .68]).

Behavioural inhibition and behavioural activation were assessed using the 20-item self-report *BIS/BAS Scales for Children* (Muris et al., 2005), adapted from the 24-item *BIS/BAS Scale* (Carver & White, 1994). Respondents rate their agreement with each item on a 4-point Likert scale (0 = *Not true*, 1 = *Somewhat true*, 2 = *True*, 3 = *Very true*). The 5-item BIS Scale reflects sensitivity to ambiguous, aversive, or threatening environmental stimuli (e.g., non-reward, punishment, and novelty; "I feel pretty worried or upset when I think or know somebody is angry at me"). The BAS Reward Responsiveness subscale (e.g., "When I see an opportunity for something I like, I get excited right away"); BAS Drive subscale (e.g., "I go out of my way to get things I want"); and Fun Seeking subscale (e.g., "I crave excitement and new sensations") together represent dimensions of the behavioural activation system (BAS), characterised by sensitivity to reward, non-punishment, and evasion of punishment (Carver & White, 1994). The BIS/BAS Scales for Children show good criterion validity and internal consistency reliability for both BIS (α = 0.78) and BAS combined subscales (α = 0.81; Muris et al., 2005) with good measurement and structural invariance across adults and adolescents (Cooper et al., 2007; Ebesutani et al., 2012).

Two dimensions of impulsivity, Negative Urgency (NU) and Sensation Seeking (SS), were measured using the 8-item subscales of the 32-item self-report *UPPS Revised Child version* (Zapolski et al., 2010). Each factor-analytically derived subscale reflect four moderately-correlated dimensions of disposition to rash action (Whiteside & Lynam, 2001). We excluded lack of planning/premeditation and lack of perseverance from our analyses, as our focus was primarily affective-motivational rather than cognitive factors. SS and NU are proposed to represent discrete pathways to risky behaviour (Smith et al., 2016). NU refers to the tendency to act impulsively when experiencing negative emotional states (e.g., "When I feel bad, I often do things I later regret in order to feel better now"). SS refers to the preference for activities involving tension and/or exhilaration (e.g., "I like new, thrilling things, even if they are a little scary"). Participants are asked to rate how well each item describes them on a 4-point Likert scale (1 = *Not at all like me* to 4 =*Very much like me*). Total scores for the subscales were calculated by summing and averaging items. The measure demonstrated good internal consistency reliability (α = .87 and .90), interrater reliability (*ICC* = .99 and .98 for NU and SS respectively) good convergent, divergent, and predictive validity (Zapolski et al., 2010) and good criterion validity (Tomko et al., 2016).

Negative Non-social and Negative Social Interpretation Bias was measured with the *Adolescent Interpretation and Belief Questionnaire* (AIBQ; Miers et al., 2008). In this task, 10 ambiguous scenarios (5 social and 5 non-social) are presented to participants, who are then asked to indicate, for each scenario, how likely each of three potential interpretations (negative, neutral, and positive) are to pop into their mind using a 5-point Likert scale (1 = *does not pop in my mind*, 3 = *might pop in my mind*, 5 = *definitely pops in my mind*). A forced choice question follows these response inputs, asking which of the interpretations is most believable, however this question was not used for analysis. Scores were summed and averaged, where higher scores indicated greater interpretation bias, from 1 (no bias) to 5 (strong bias) for both positive and negative social and non-social interpretations. Internal consistency and differential stability across waves were high: ω = .78, .81 and .84; $ICC_{3,1}$ = 0.77, [95% CI = .73 – .81] for NSB and ω = .78, .81 and .84; $ICC_{3,1}$ = 0.77, [95% CI = .73 – .81] for NNonSB.

Data Analysis

All statistical analyses were performed in R (Version 4.3.1; R Core Team, 2019). Interaction variables were transformed using SPSS (Version 27; IBM Corp). All statistical output and data files are available in Supplemental Materials.

Missing data treatment. Missing data was analysed using the *naniar* package v.1.0.0 (Tierney & Cook, 2023). Percentage of all values missing was 9.8%. A Little's test showed that data were not missing completely at random (MCAR; $\chi^2 = 847.34$ (247); $p < .0001$). Missing values for each case and variable were identified by number and percentage missing, and missingness relationships were visualised using heatmaps and scatterplots. There were no differences on any study variable between complete and incomplete sets of observations, so data were concluded to be missing at random (MAR), thus missing data were estimated via full information maximum likelihood (FIML) expectation-maximization algorithms. This method of data imputation is preferable to either dropping missing cases or mean imputation procedures in reducing bias in population parameter estimates (Enders, 2010).

Removal of outliers. Post clerical review, it was decided to recode any scores of 0 on the BART to missing, as 0-scores may be indicative of indiscriminate button-hitting to pump the balloon until it burst or misunderstanding the task. Internal consistency for each measure was estimated using McDonald's omega (ω), which demonstrates superior estimates to Cronbach's alpha (Dunn et al., 2014). Inter-wave variability was examined by calculating intraclass correlation coefficients ($ICC_{3,1}$) for each variable. This estimates the correlation of measures across waves, where higher values indicate higher differential stability (Shrout & Fleiss, 1979). ICC estimates > .70 were considered highly stable (Koo & Li, 2016). Table 2, Appendix 2, presents correlations for all study variables across waves.

Linear mixed-effects modelling (LMMs; Raudenbush & Bryk, 2002) was conducted using the *lme4* (Version 1.1-34; Bates et al., 2015) and *TIDAL* (Version 0.1.0; Edmondson -Stait et al., 2023) packages in R. Prior to modelling, variables were visually explored for distributional shape. To check for multicollinearity, the Variance Inflation Factor was checked for all independent variables in our models.

All variables returned a VIF < 2.5, indicating low multicollinearity (Allison, 2012). All models were estimated using FIML estimation which accounts for nonnormality and nonindependence of data. Confidence intervals (CIs) for the parameter estimates were determined using 1000-fold bootstrap replicates, and *p*-values were obtained from the *z*-distributed Wald *t*-values. Alpha was set at 0.05. All variables bar gender were treated as continuous. Gender was coded 0 [male] and 1 [female]. The fixed effect intercept was shifted to the mean age of all assessments ($M = 14.45$), while a random effects structure allowed intercepts to vary by age for each individual. For all models, a quartic model with linear slopes best fit the data based on the lowest model deviance estimates. Post-modelling, error variance was visually inspected for normality using error plots.

Results

Descriptive statistics and internal consistency across waves for each variable are presented in Table 3 below.

Table 3. *Descriptive statistics and reliability estimates for study variables across waves.*

Measure (poss. range of scores)	**Wave 1**		**Wave 2**		**Wave 3**		**ICC$_{3,1}$** ***[LL,UL]***
BIS (0-15)	*N*=470		*N*=446		*N*=359		.64 [.41, .76]
Mean (*SD*)	1.51	(.54)	1.94	(.50)	2.00	(.50)	
ω	**.73**		**.73**		**.73**		
BAS – Drive (0-15)	*N*=470		*N*=446		*N*=359		**.72** [66, .77]
Mean (*SD*)	1.14	(.68)	1.23	(.67)	1.24	(.67)	
ω	**.78**		**.80**		**.81**		
BAS – Fun (0-15)	*N*=470		*N*=446		*N*=359		**.79** [.75, .83]
Mean (*SD*)	1.73	(.64)	1.68	(.66)	1.68	(.65)	
ω	**.70**		**.80**		**.72**		
BAS – Reward (0-15)	*N*=470		*N*=446		*N*=359		.68 [.61, .73]
Mean (*SD*)	2.17	(.55)	2.03	(.58)	2.03	(.53)	
ω	**.74**		**.71**		**.73**		
Negative Urgency (8-32)	*N*=497		*N*=446		*N*=359		**.79** [.75, .83]
Mean (*SD*)	2.47	(.61)	2.56	(.60)	2.57	(.64)	
ω	**.83**		**.84**		**.87**		
Sensation Seeking (8-32)	*N*=498		*N*=446		*N*=359		**.91** [.89, .93]
Mean (*SD*)	2.98	(.73)	2.93	(.73)	2.91	(.76)	
ω	**.88**		**.88**		**.90**		
Risk Involvement (0-14)	*N*=493		*N*=446		*N*=359		**.79** [.70, .85]
Mean (*SD*)	3.59	(2.20)	4.36	(2.39)	4.67	(2.44)	
ω	n/a		n/a		n/a		
BART (n/a)	*N*=483		*N*=447		*N*=405		.60 [.51, .68]
Mean (*SD*)	26.95	(12.30)	30.28	(14.13)	34.27	(16.15)	
ω	n/a		n/a		n/a		
NSB (5-25)	*N*=494		*N*=446		*N*=396		**.77** [.73, .81]
Mean (*SD*)	3.26	(.88)	3.13	(.94)	3.11	(.97)	
ω	**.78**		**.81**		**.84**		
NNonSB (5-25)	*N*=494		*N*=446		*N*=396		**.74** [69, .78]
Mean (*SD*)	2.58	(.63)	2.51	(.65)	2.55	(.67)	
ω	.56		.54		.57		

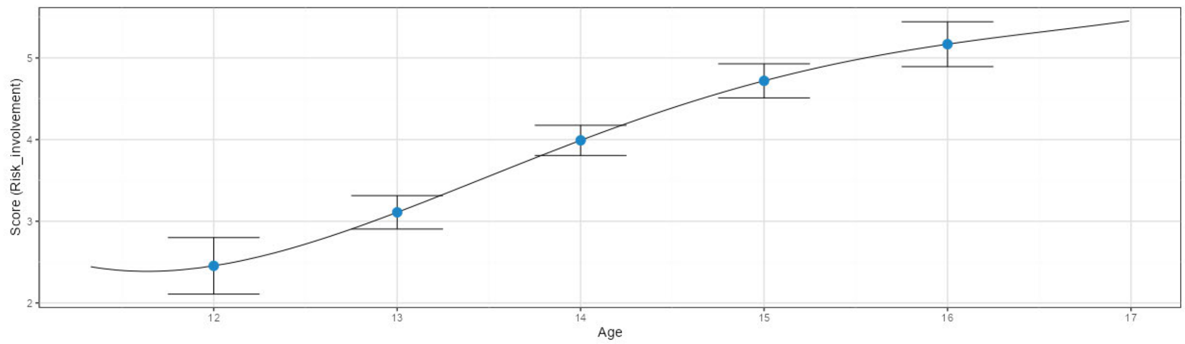
Note. BAS Reward = BAS Reward Responsivity. NSB = Negative Social Interpretation Bias. NNonSB = Negative Non-social Interpretation Bias. n/a = calculation was not appropriate for this data. Boldface indicates high reliability/stability. McDonald's ω) and differential stability (ICC) values copied with permission from Booth et al. (2019).

Model 1. To establish a baseline for our predictor model, we constructed the model:

RI / RP ~ Age + I(Age^2) + I(Age^3) + I(Age^4) + (1 + Age | id) + Gender + SES.

This revealed that RI increased by approximately 0.71 points per year, with the steepest increase occurring between ages 13 – 15 (with 0.88 and 0.73 points yearly increase respectively). Risk-taking on the BART also increased by 3.60-points per year, showing steeper increases post-15 years. As expected, even controlling for gender and SES, inter-individual variability was high for both outcomes, with scores at intercept (14.5 years) varying by 1.66 points for RI, and 8.55 balloon pumps on the BART. Full parameter estimates are presented in Table 4, Appendix 3. See Figures 1 – 4 below for both overall trajectories and random sample trajectories. Lower SES predicted increased RI (b = -0.150, SE = 0.065, pz = .021, 95% CI [-0.278 – -0.023]) though not RP. However further modelling was inconclusive (see Appendix B). Females scored significantly lower than males on RI, by over 1 point at age 14.45 (b = -1.305, SE = 0.166, pz <.0001, 95% CI [-1.631 – 0.979]). See Appendix C for further analysis.

Figure 2. *Overall levels of RI and rate of change over time.*



	12	13	14	15	16
Score (Risk_involvement) (95% CIs)	2.454 (2.108 - 2.8)	3.109 (2.905 - 3.314)	3.99 (3.805 - 4.175)	4.719 (4.511 - 4.928)	5.168 (4.893 - 5.443)

Figure 3. *RI trajectories from a random sample of 30 adolescents.*

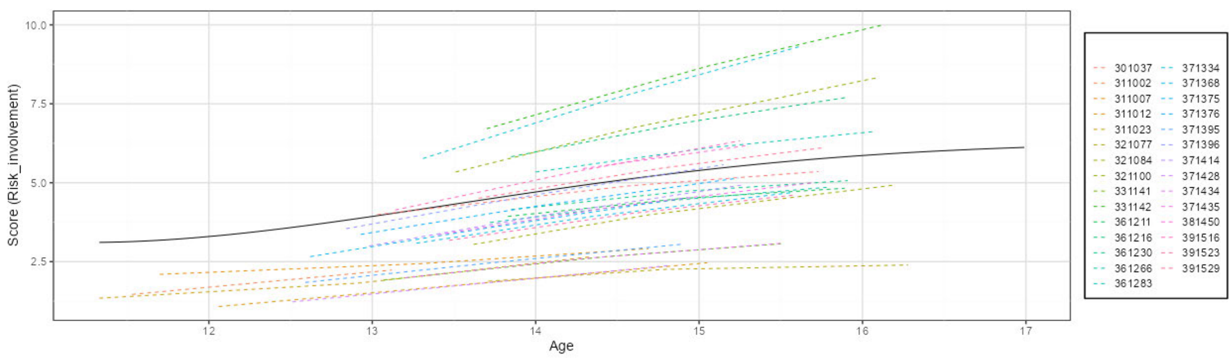
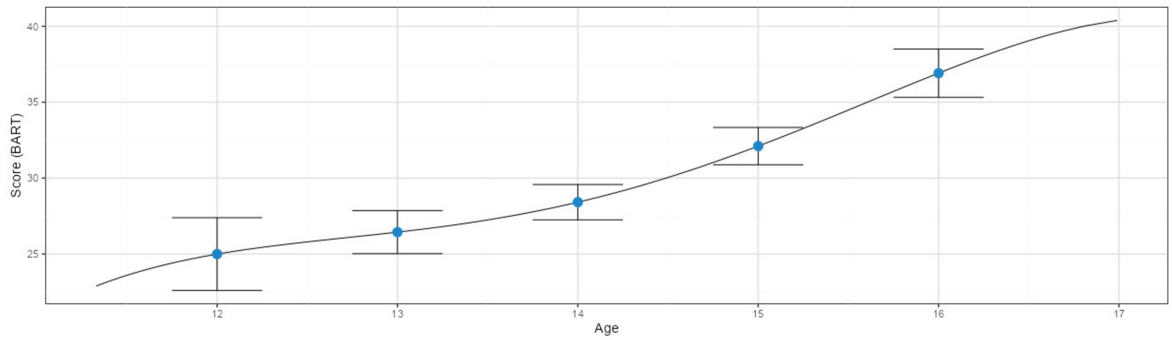
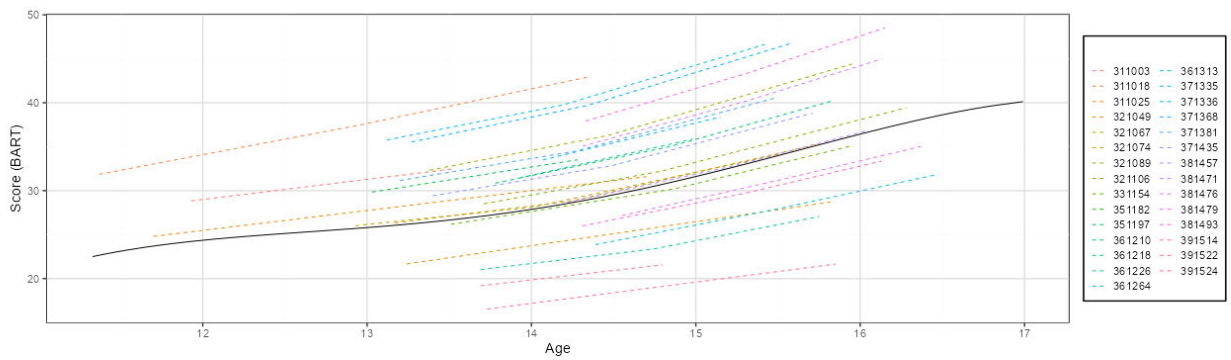


Figure 4. *Overall levels of RP and rate of change over time.*



	12	13	14	15	16
Score (BART) (95% CIs)	24.997 (22.6 - 27.393)	26.444 (25.024 - 27.863)	28.415 (27.251 - 29.579)	32.11 (30.882 - 33.338)	36.914 (35.325 - 38.503)

Figure 5. *RP trajectories from a random sample of 30 adolescents.*



Model 2. Our main hypothesis resulted in the following two models:

RI / RP ~ Age + I(Age^2) + I(Age^3) + I(Age^4) + (1 + Age | id) + Gender + SES + BIS + RR + FS + Drive + SS + NU

For both outcomes, Model 2 provided better fit to the data than Model 1, based on lower AIC, BIC and Deviance (for full parameter estimates and goodness-of-fit indices, see Table 5 below). The only variables that did not uniquely predict RI were FS (*b* = 0.156, SE = 0.120, *pz* = .192, 95% CI [-0.078 – 0.390]) and BIS (*b* = -0.196, SE = 0.114, *pz* = .086, 95% CI [-0.420 – 0.028.]). Following age, the strongest unique predictor of increased RI was NU (*b* = 0.591, SE = 0.100, *pz* <.0001, 95% CI [0.395 – 0.787]), followed by SS (*b* = 0.386, SE = 0.108, *pz* <.0001, 95% CI [0.175 – 0.598]) and Drive (*b* = 0.193, SE = 0.094, *pz* = .04, 95% CI [0.009 – 0.377]). RR was associated with significantly decreased risk involvement (*b* = -0.322, SE = 0.119, *pz* = .007, 95% CI [-0.556 – -0.088]). 42.18% of total model variance was attributable to individual variability at intercept (age 14.5), while 4.01% was attributable to between-individual variability for age. Intraindividual variability accounted for 33.85% of total model variance.

For RP, other than age, only NU uniquely impacted scores across adolescence, though it had an inhibiting effect (*b* = -1.817, SE = 0.705, *pz* = .01, 95% CI [-3.199 – -0.436]). 42.70% of total model variance was attributable to individual variability at age 14.5, while 2.74% was attributable to between-individual variability for age. Intraindividual variability accounted for 54.56% of total model variance.

Each significant unique predictor was interacted separately with age, controlling for all other model variables. Results are plotted in Figures 3 – 7 below. The two strongest unique predictors of RI, NU and SS, appear to show different functions depending on the level of the variable (+1 *SD* / -1 *SD* vs. population average), with both high NU and high SS showing a steeper positive impact on RI post-16 than low or average NU and SS, when their respective impacts appear to slow or decrease.

Table 5. *Parameter estimates for LMMs testing the effect of explanatory variables on RI and RP trajectories.*

	Risk involvement							Risk propensity (BART)						
	Fixed effects		97.5% CI			Random	effects	Fixed effects		97.5% CI			Random effects	
	b	SE	*LL*	*UL*	*p*=		*SD*	*b*	*SE*	*LL*	*UL*	*p*=		*SD*
(Intercept)	3.269	0.467	2.354	4.185	< .0001***			27.057	3.202	20.780	33.333	< .001***		
Age	0.744	0.085	0.578	0.910	< 0.001***			3.815	0.625	2.589	5.040	< 0.001***		
I(Age^2)	-0.051	0.085	-0.217	0.116	.55			0.548	0.634	-0.695	1.792	.387		
I(Age^3)	-0.040	0.031	-0.100	0.020	.19			-0.200	0.230	-0.651	0.252	.386		
I(Age^4)	-0.006	0.020	-0.046	0.035	.786			-0.056	0.152	-0.354	0.242	.712		
Female	-1.109	0.162	-1.427	-0.791	<.0001***			1.664	1.069	-0.431	3.759	.12		
SES	-0.131	0.062	-0.252	-0.010	.034			0.717	0.406	-0.078	1.512	.077		
Negative Urgency	0.591	0.100	0.395	0.787	<.0001***			-1.817	0.705	-3.199	-0.436	.01**		
Sensation Seeking	0.386	0.108	0.175	0.598	<.0001***			0.977	0.754	-0.501	2.455	.195		
BAS Reward	-0.322	0.119	-0.556	-0.088	.007**			-0.609	0.852	-2.278	1.060	.475		
BAS Drive	0.193	0.094	0.009	0.377	.04*			1.179	0.672	-0.138	2.496	.079		
BIS	-0.196	0.114	-0.420	0.028	.086			0.305	0.820	-1.303	1.913	.71		
BAS Fun	0.156	0.120	-0.078	0.390	.192			0.391	0.861	-1.296	2.077	.65		
Variance (Intercept)						2.113	1.454						74.929	8.656
Covariance (Intercept)Age						0.392	0.602						9.340	0.492
Variance (Age)						0.201	0.448						4.802	2.191
Residual variance						1.696	1.302						95.718	9.784
Model summary														
AIC		4922.4							9393.8					
BIC		5009.3							9480.2					
LogLik		-2444.2							-4679.9					
Deviance		4888.35							9359.82					
DF Residual		1212							1174					
n observations		1229							1191					
n groups		481							482					

Note. AIC = Akaike's Information Criterion. BIC = Bayesian Information Criterion (BIC). LogLik = Log-Likelihood. CI = confidence interval; LL = lower limit; UL = upper limit. $p < .05$*; $p < 0.01$**; $p < 0.001$***.

Figure 6. *Impact of negative urgency on risk involvement trajectories across adolescence.*

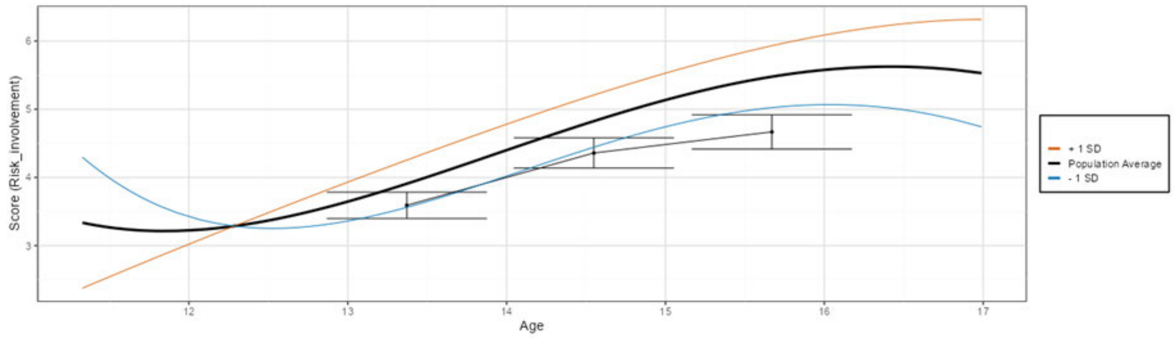


Figure 7. *Impact of sensation seeking on risk involvement trajectories across adolescence.*

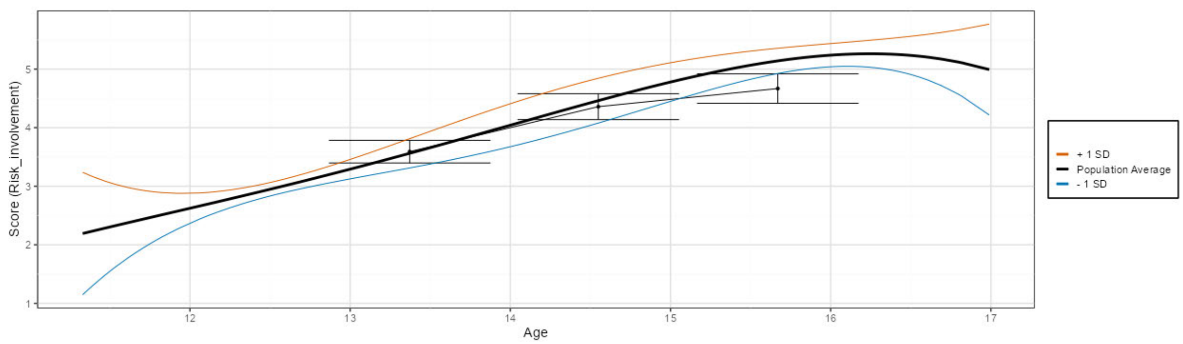


Figure 8. *Impact of reward responsivity on risk involvement trajectories across adolescence.*

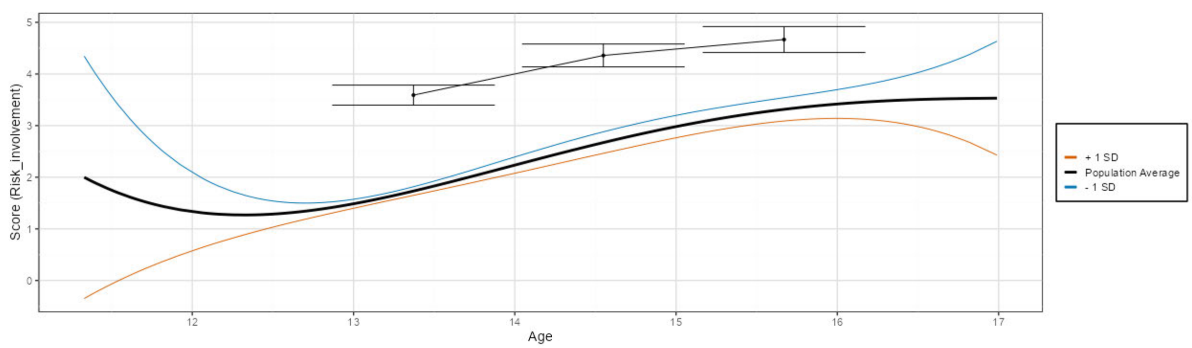


Figure 9. *Impact of drive on risk involvement trajectories across adolescence.*

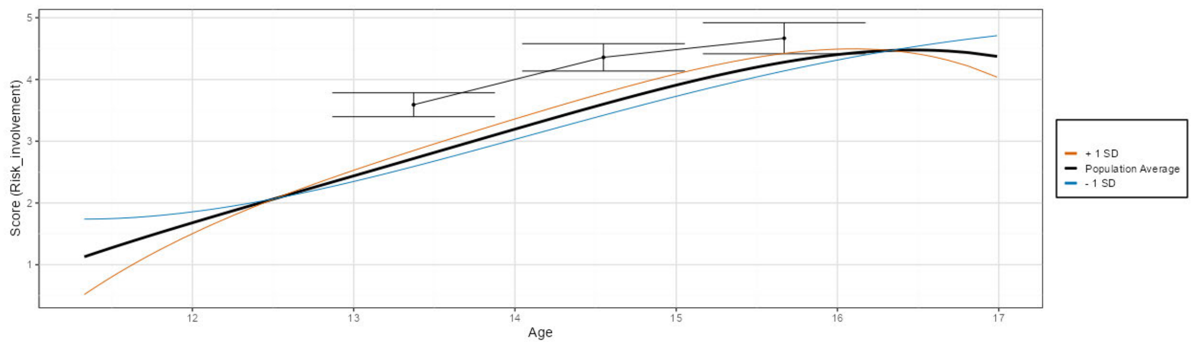
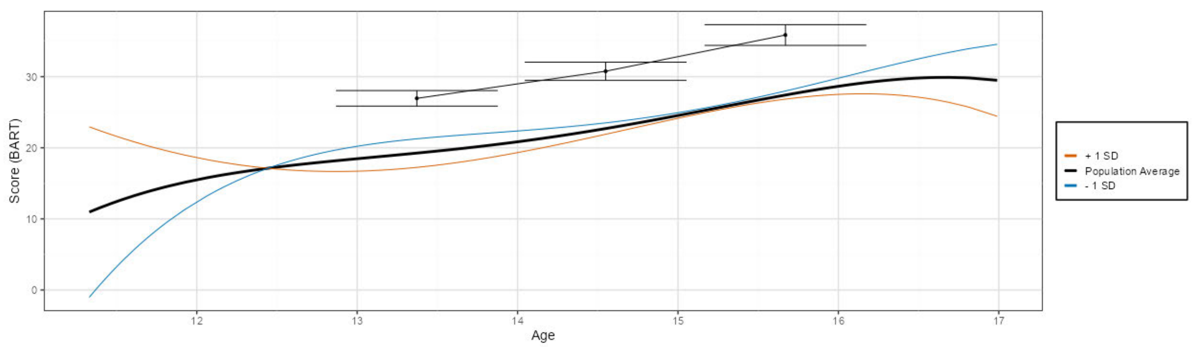


Figure 10. *Impact of negative urgency on risk propensity (BART) across adolescence.*



Note. For all figures, a descriptive overlay of overall mean trajectory for the model is shown with 95% confidence intervals.

Model 3. To test our secondary hypotheses that a) the impact of Drive on RI and RP would remain constant or increase across waves and b) the impact of higher NU and BIS on RI and RP would be higher at W2 and W3, we fitted a model where each predictor was grouped by wave, controlling for all other study variables. Differential impact was inferred by a significant *p*-value for the effect of one wave over another (or others). The impact of SS on increased RI was highest at Wave 1 (*M*age = 13.37; *b* = 0.498, SE = 0.210, *pz* = .017, 95% CI [0.088 – 0.909]), while for RP there was no significant difference between waves. Drive significantly increased RI at Wave 3 (*M*age = 15.67; *b* = 0.499, SE = 0.171, *pz* = .004, 95% CI [0.065 – 0.349]), appearing to exert a suppressing effect until this time point. Interestingly, Drive significantly decreased RP at Wave 3 (*b* = -3.419, SE = 1.159, *pz* = .003, 95% CI [-5.690 – -1.148]).

As hypothesised, the impact of NU on increased RI was highest at Wave 3 (*b* = 0.391, SE = 0.182, *pz* = 0.032, 95% CI [0.034 – 0.748]). There was no significant difference between waves for RP. The impact of BIS was significantly higher at Wave 3 although, contrary to expectations, it exerted an inhibiting effect on RI (*b* = -556, SE = 0.104, *pz* = 0.022, 95% CI [-1.033 – -0.079]). The impact of BIS on RP showed no significant effect of wave. Full parameter estimates are presented in Table 6, Appendix 6.

Interaction Models. For each of our interaction models, a base model was compared with an interaction model in a stepwise procedure. This was repeated for each wave. Each product term was also interacted with age. A significant *p*-value for the interaction term and change in independent effect value was considered evidence of moderation. For each term, this resulted in the model:

RI / RP ~ Age + (1 + Age | id) + Gender + SES + BIS + RR + FS + Drive + NU + SS + NSB + NNonSB + Product Term + Age * Product Term.

For RP, there were no significant moderation effects at any wave. For RI, the BIS*NSB and BIS*NNonSB interaction terms were not significant at any wave. There were no significant interactions at W1. At W2, however, NNonSB significantly suppressed the relationship between NU and RI (*b* = -

1.419, SE = 0.526, *pz* = .007, 95% CI [-2.451 – -0.387]). There was a significant positive moderating effect of age on NU*NSB and NU*NNonSB at W2 (*b* = 0.118, SE = 0.044, *pz* = .008, 95% CI [0.031 – 0.205] and *b* = 0.115, SE = 0.044, *pz* = .009, 95% CI [0.029 – 0.200] respectively); though this was likely driven by the main effect of NU in the model (*b* = 0.598, SE = 0.153, *pz* <.0001, 95% CI [0.298 – 0.899])). At W3, there was a significant suppression effect of NSB on NU (*b* = -0.935, SE = 0.048, *pz* = .037, 95% CI [-1.814 – -0.057]). Interestingly, NNonSB uniquely predicted increased RI at Wave 3 (*b* = 0.420, SE = 0.149, *pz* = .005, 95% CI [0.127 – 0.712]), along with NU (*b* = 0.682, SE = 0.145, *pz* < .0001, 95% CI [0.399 – 0.966]; though the interaction NU*NNonSB had a slight, though not significant, suppressing effect on RI (*b* = -1.010, SE = 0.530, *pz* < .057, 95% CI [-2.048 – 0.029]). Higher age was again associated with both increases in the impact of NU*NSB and NU*NNonSB on higher RI (*b* = 0.161, SE = 0.045, *pz* < .001, 95% CI [0.073 – 0.249] and *b* = 0.183, SE = 0.045, *pz* < .001, 95% CI [0.095 – 0.271] respectively). Full parameter estimates are presented in Table 7, Appendix 7.

Discussion

This study sought to examine individual markers for age-related changes in risk propensity and real-world risk-taking across adolescence. Our main hypothesis that age, RR, Drive, FS, BIS, NU and SS would independently predict increased RP and RI across waves, controlling for gender and SES, was partially supported. NU, SS and Drive all uniquely predicted increased RI, while RR predicted lower prospective RI. Age was the strongest predictor of prospective increases in risk-taking for both outcome measures. Gender was a robust predictor of RI, with boys involved in risk-taking earlier, and at higher levels than girls, an important maturational factor to consider. There was a slight impact of lower SES on increased RI, though this should be interpreted cautiously. This inconclusive result may be an artefact of the restricted range of SES scores for our sample, an acknowledged study limitation.

Individual variability. In line with prior findings (Booth et al., 2019; Crone & van Duijvenvoorde, 2021), there was substantial inter-individual variability in RI. However, differential stability was high, indicating that behavioural patterns of risk involvement were relatively enduring. While RP showed overall increases, differential stability was low, reflecting that an individual's scores at Wave 1 were not a reliable predictor of their scores at subsequent waves. There are several possible interpretations for this. The known or implied presence of peers is a known predictor of increased risk-taking in adolescents (Reniers et al., 2017). Thus, the group testing environment, though under examination conditions, may have biased how individuals scored, a key limitation to this study. However, as no record of individual cases was obtained at testing, this is speculative. There may also have been learning effects across waves. The BART may have been sensitive to state fluctuations associated with developmental changes; a person-centred approach such as growth mixture modelling (GMM) or latent class analysis would help to clarify patterns of intraindividual change in RP.

BART. Contrary to expectations, the same personality indicators were not predictive of both risk outcomes. Age and NU were the only reliable predictors of prospective BART scores, though NU in fact significantly predicted lower scoring. This was surprising given that the BART has been relatively more successful than other probabilistic risk tasks in predicting real-world risk-taking (Lejuez et al., 2007). Several factors may explain this. The BART's probability structure is *ambiguous*, with the reward structure learnt gradually through exploration (Blankenstein et al., 2016; Blankenstein et al., 2021). Interestingly, adolescents reliably outperform children and adults on ambiguous/exploratory learning tasks, including the BART (Peper et al., 2018; Peters & Crone, 2017). While tolerance to ambiguity, either through bias towards rewards or attenuated neural response to adverse outcomes, has often been linked to maladaptive outcomes (e.g., Blankenstein et al., 2016; Tymula et al., 2012), this may be an artefact of the prevailing focus of risk-taking research thus far.

The BART's task structure is also *dynamic* (Crone et al., 2021), where positive consequences (i.e., monetary gain) *beyond a certain point*, become outweighed by the possibility of negative consequences (i.e., the balloon exploding and all gains lost; Lejuez et al., 2007). Crucially, high scores on the BART reflect *successful* risk-taking: gaining the most money *without* bursting the balloons. Indeed, higher levels of BAS Drive, characterised by goal-directed, aim-focused behaviours, were the strongest personality indicator of increased RP on the BART across waves, though results were too variable to be significant. In real-world contexts, risk-taking may lead to positive or adaptive outcomes in certain circumstances (Nigg & Nagel, 2016), such as social benefits and achievement outcomes (Willoughby et al., 2014; Willoughby et al., 2021) often associated with self-efficacy and resilient functioning (Rutter, 2014).

Negative Urgency. The strongest indicator of prospective RI was NU. This finding has important implications for theory and practice. Impulsivity, particularly impulsive action, is a key dimension in many externalising and internalising psychopathologies (Ferrer et al., 2020). The impact of NU on RI was substantially higher for boys than girls (see Figure 11, Appendix 8). As hypothesised, the impact of NU on increased RI was highest at ages 15–17 approximately, in line with evidence of increased negative affectivity at these ages (Booth et al., 2019). Strengthening this connection, the impact of BIS – reflecting the degree of negative reactivity experienced to unpleasant, ambiguous or aversive circumstances – on RI was also highest at age 15–17; though contrary to expectations, BIS had a suppressing effect on RI. Furthermore, significant interactions were found between NU and negative interpretation biases, which increased significantly at around age 15 onward. This is line with findings by Renier et al. (2016) who identified direct and indirect pathways between social anxiety, impulsiveness, reward-sensitivity, behavioural inhibition (BIS), and risk behaviours in adolescents. Though interpretation bias had a suppressing effect on RI, given the robustness of the interaction in mid-late adolescence, this warrants further assessment against other psychosocial outcomes (e.g., mood disorders, aggression, bullying/victimisation, self-harm, binge eating) associated with this marker. This may be a focus for future interventions.

Reward-drive. Evidence for an adolescent reward-drive was mixed. SS and BAS Drive were robust predictors of increased prospective RI. While the impact of Drive on RI was highest at ages 15–17 (W3), the impact of SS on increased RI was highest at approximately ages 12–14 (W1). This is more consistent with findings by Peper et al. (2018) showing a mid-adolescent peak in SS, rather than a continued increase into adulthood (Defoe et al., 2015).

Taken together, findings reveal complex cumulative factors that interact to differentially impact risk behaviours in adolescence. Evidence of steep increases in risk involvement between ages 12 – 14 fits with a differential exposure hypothesis (Defoe, 2021) as teenagers start secondary school and are rapidly exposed to increased independence and a range of psychologically ambiguous and potentially anxiety inducing socially risky situations (S.-J. Blakemore, 2018). However, main effects findings reveal key differential susceptibility markers for risk-taking in adolescence, which offer important targets for intervention.

These findings extend current research proposing a biopsychosocial developmental framework of adolescent risk-taking (Blakemore, 2008; S. J. Blakemore, 2018; Choudhury et al., 2006; Tomova et al., 2021) by highlighting the key influence of cognitive, emotional and motivational factors on adolescent risk behaviours. Many of the key psychosocial challenges of adolescence: romantic, social, academic or achievement uncertainty, value-alignment, cognitive dissonance, moral dilemmas, and identity insecurity, may be framed as goal-conflicts shown to produce anxious uncertainty (for a review, see Jonas et al., 2014).

In line with prior findings, our study found an activation-congruency effect, but not a valence-congruency effect to risk involvement (see also Leota et al., 2023), suggesting evidence of a dual affective-motivational pathway to risky real-world behaviours. Affective circumplex models of emotion proposing two orthogonal dimensions of affective experience provide an integrative approach to cognitive-affective neuroscience, development and psychopathology (Posner et al., 2005; Russell, 1980). Thus, we suggest that a dual-continua model of approach motivated behaviour may better account for the range of positive to negatively-valenced emotions found to predict risk involvement in our study (see Figure 12, Appendix 9). Future research could test this circumplex hypothesis of risk-taking, whereby high activation mood states, regardless of valence, predict risk-taking, while person-centred approaches (e.g., GMM, latent class analysis) may be used to identify for whom these neurobiological reactivity markers are most salient.

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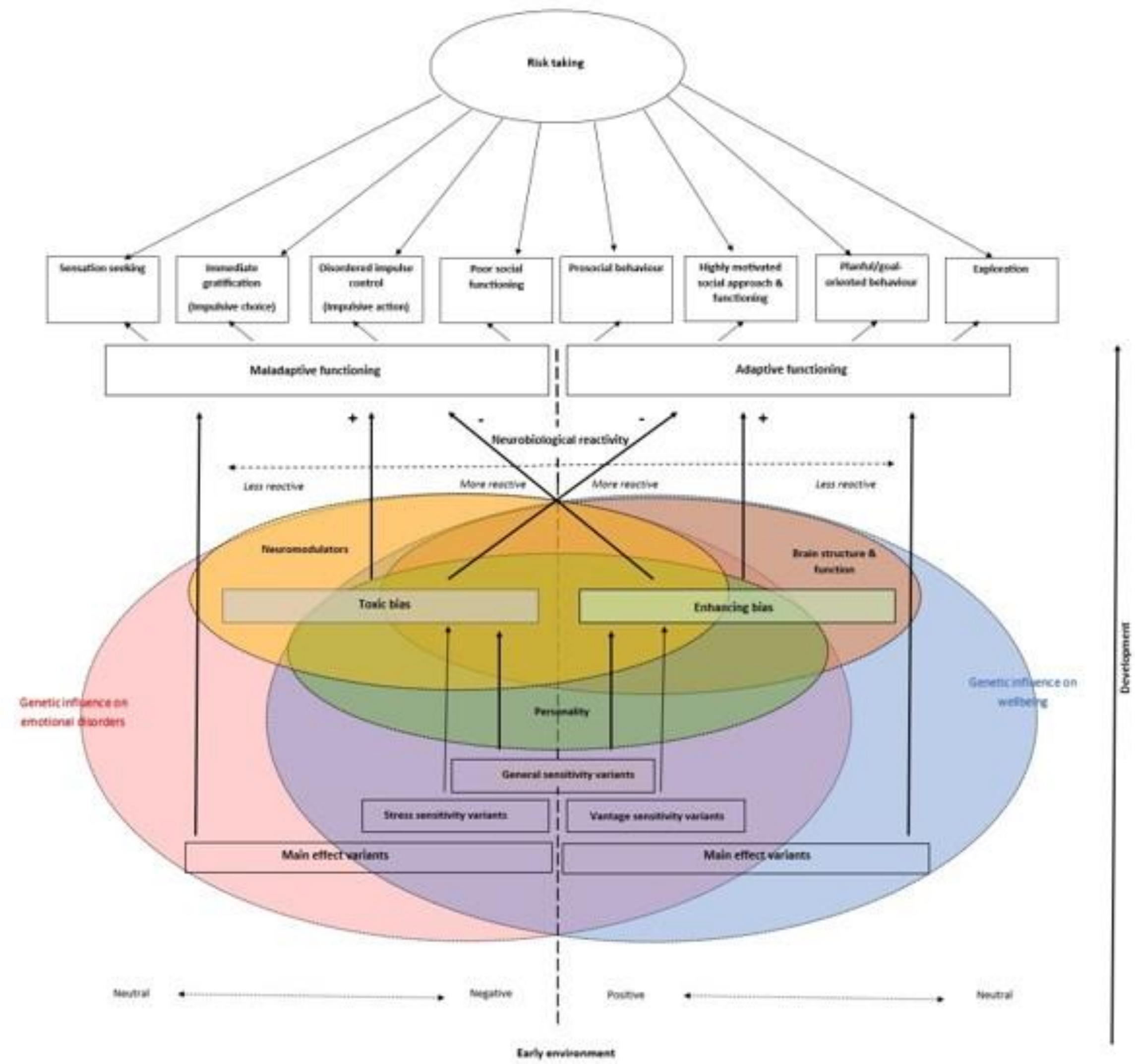
APPENDIX 1

Figure 1. *The central CogBIAS framework with proposed adaptive and maladaptive risk outcomes.*



Reproduced with modifications from Fox & Beevers (2016).

APPENDIX 2

Table 2. *Correlation tables for Waves 1, 2 and 3.*

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12	13.
1. SES	1												
2.Gender	-.127**	1											
3.Age	.170**	-.450**	1										
4.BART	.092*	-0.008	0.061	1									
5.Risk Involvement	0.005	-.437**	.436**	-0.008	1								
6.BIS	0.073	.144**	-0.066	0.011	-.105*	1							
7.BAS Drive	0.06	-.137**	0.054	0.079	.116*	.104*	1						
8.BAS Fun Seeking	0	-.161**	.121**	.119*	.260**	0.048	.453**	1					
9. BAS Reward	.117*	-0.039	0.026	.108*	0.047	.250**	.470**	.502**	1				
10.Neg Urgency	0.004	-0.028	0.053	0.003	.201**	.252**	.252**	.276**	.119*	1			
11.Sens. Seeking	0.048	-.227**	.140**	.093*	.335**	-.214**	.242**	.645**	.275**	.147**	1		
12.Neg. Soc. Bias	-0.013	.137**	0.043	-0.008	0.038	.443**	0.041	0.062	0.063	.393**	-.111*	1	
13.Pos. Soc. Bias	0.017	-.204**	0.002	-0.007	.134**	-.192**	.197**	.233**	.157**	-0.013	.194**	-.247**	1

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12	13.
1. SES	1												
2.Gender	-.127**	1											
3.Age	.139**	-.494**	1										
4.BART	0.067	0	0.017	1									
5.Risk Involvement	-0.069	-.358**	.382**	-0.017	1								
6.BIS	.100*	.274**	-0.015	0.013	-.196**	1							
7.BAS Drive	0.074	-.117*	.096*	-0.036	.191**	0.004	1						
8.BAS Fun Seeking	0.043	-0.009	0.062	0.026	.228**	-0.007	.448**	1					
9. BAS Reward	.161**	0.03	0.052	0.03	-0.06	.362**	.417**	.514**	1				
10.Neg Urgency	-0.019	.135**	-0.051	-.120*	.208**	.177**	.314**	.259**	.117*	1			
11.Sens. Seeking	0.048	-.115*	0.084	0.068	.246**	-.129**	.293**	.683**	.296**	.141**	1		
12.Neg. Soc. Bias	0.024	.208**	-0.058	-0.043	-0.018	.397**	-0.004	-0.037	0.062	.314**	-0.091	1	
13.Pos. Soc. Bias	.125**	-.202**	.184**	0.027	.130**	-.126**	.274**	.232**	.258**	0.01	.199**	-.206**	1

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12	13.
1. SES	1												
2.Gender	-.127**	1											
3.Age	.106*	-.350**	1										
4.BART	0.094	-.118*	.104*	1									
5.Risk Involvement	-0.102	-.142**	.158**	0.028	1								
6.BIS	0.049	.328**	0.032	-0.043	-.213**	1							
7.BAS Drive	0.056	-.198**	.170**	0.042	.242**	-.126*	1						
8.BAS Fun Seeking	0.074	0.007	.130*	0.041	.228**	-0.032	.484**	1					
9. BAS Reward	0.104	0.019	.166**	0.038	-0.09	.332**	.341**	.380**	1				
10.Neg Urgency	-.107*	0.088	-0.025	-0.007	.335**	0.092	.145**	.228**	-0.046	1			
11.Sens. Seeking	0.028	-.114*	0.067	0.076	.166**	-.216**	.325**	.641**	.284**	.147**	1		
12.Neg. Soc. Bias	-0.011	.183**	-0.017	-0.016	0.028	.366**	-0.04	0	0.075	.354**	-0.065	1	
13.Pos. Soc. Bias	0.093	-.253**	.185**	0.025	0.078	-.163**	.175**	.219**	.133*	-0.014	.259**	-.154**	1

*Note. ** = correlation is significant at the 0.01 level (2-tailed). * = correlation is significant at the 0.05 level (2-tailed).*

APPENDIX 3

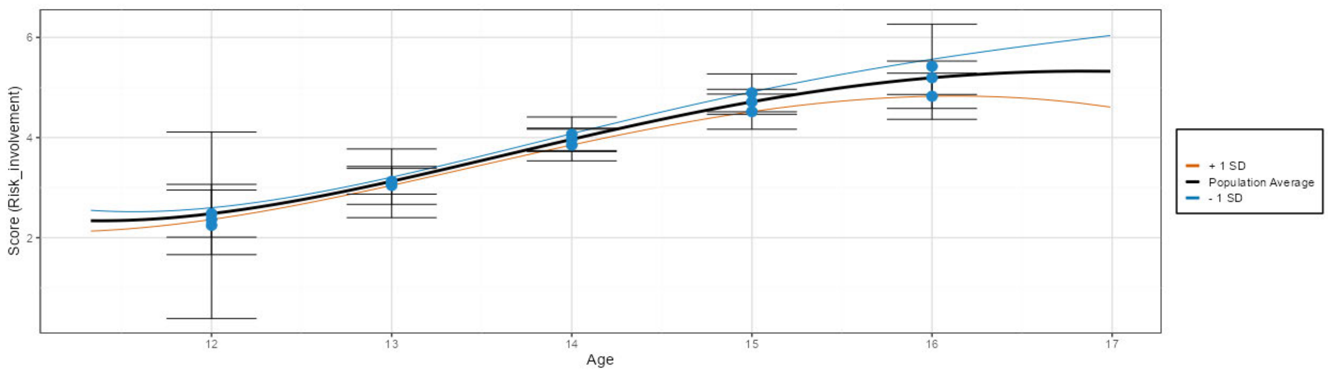
Table 4. *Parameter estimates for linear mixed models testing effect of SES, gender and age on risk involvement and risk propensity trajectories.*

	Risk involvement							**Risk propensity (BART)**						
	Fixed effects		97.5% CI			Random	effects	Fixed effects		97.5% CI			Random effects	
	b	SE	*LL*	*UL*	*pz*		*SD*	*b*	*SE*	*LL*	*UL*	*pz*		*SD*
(Intercept)	5.571	0.277	5.029	6.114	< .001***			26.719	1.690	23.408	30.030	< .001***		
Age	0.712	0.078	0.559	0.865	< .001***			3.602	0.571	2.483	4.722	< .0001***		
I(Age^2)	-0.065	0.069	-0.200	0.070	.345			0.759	0.516	-0.252	1.770	.141		
I(Age^3)	-0.028	0.029	-0.085	0.030	.343			-0.062	0.206	-0.466	0.342	.763		
I(Age^4)	0.000	0.014	-0.028	0.029	.991			-0.049	0.106	-0.257	0.159	.643		
Female	-1.305	0.166	-1.631	-0.979	< .0001***			0.947	0.997	-1.007	2.902	.342		
SES	-0.150	0.065	-0.278	-0.023	.021*			0.768	0.395	-0.006	1.542	.052		
Variance(Intercept)						2.741	1.656						73.128	8.551
Covariance (Intercept)Age						0.503	0.749						9.972	0.593
Variance(Age)						0.165	0.406						3.871	1.967
Residual variance						1.713	1.309						102.130	10.106
Model summary														
AIC		5073.7							10013.8					
BIC		5130.1							10070.4					
LogLik		-2525.8							-4995.9					
Deviance		5051.7							9991.85					
DF Residual		1242							1256					
n observations		1253							1267					
n groups		484							485					

Note. AIC = Akaike's Information Criterion. BIC = Bayesian Information Criterion (BIC). LogLik = Log-Likelihood. CI = confidence interval; LL = lower limit; UL = upper limit. p < .05; p < 0.01**; p < 0.001***.*

APPENDIX 4

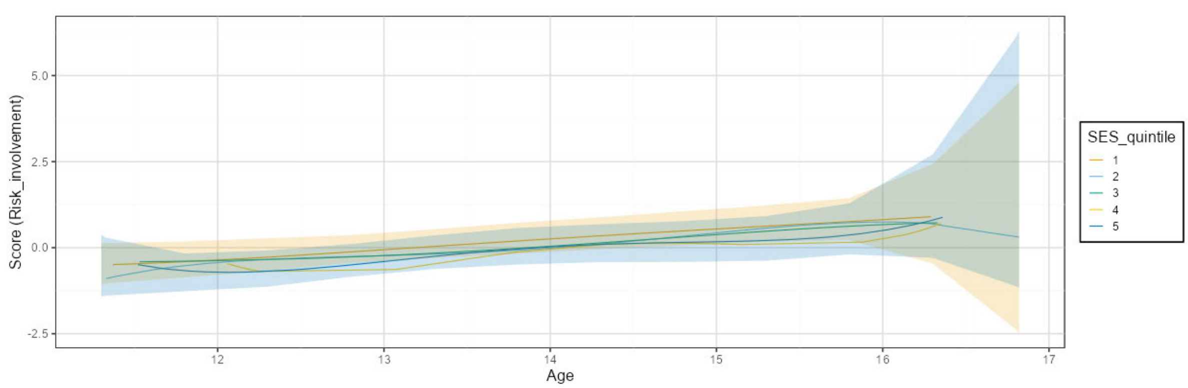
Figure. *Plot of RI stratified by SES (- 1SD/ + 1 SD vs. population average).*



Lower SES predicted increased RI (*b* = -0.150, *SE* = 0.065, *pz* = .021, 95% CI [-0.278 – -0.023])

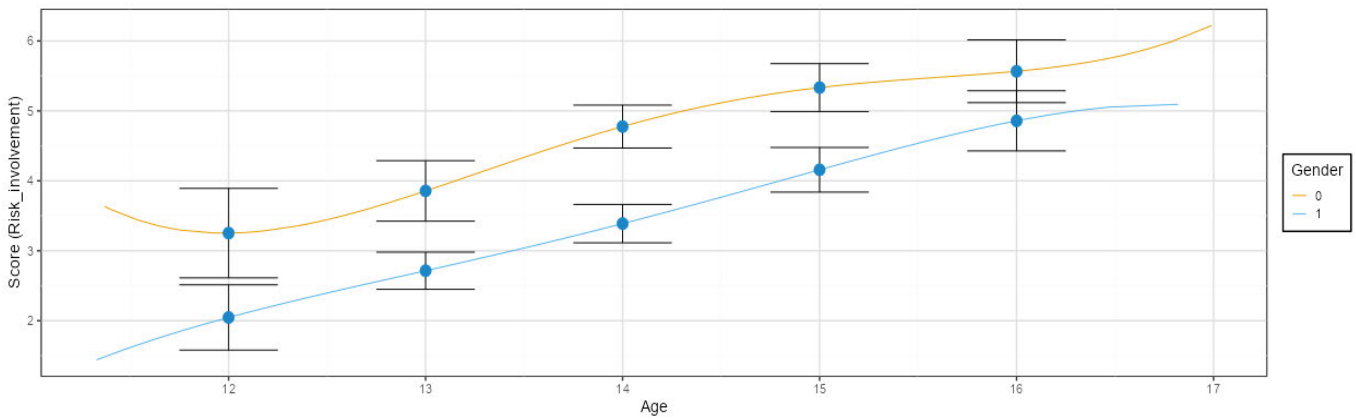
To further explore this effect, we modelled the interaction of SES, stratified by quintile, and age on the outcome risk involvement. However, when we further stratified SES by quintile, we found no meaningful differences in impact on risk involvement, with large, overlapping 95% confidence intervals for both top and bottom quintiles (see Figure below). Thus, results should be interpreted cautiously. There was a slight, though not significant, inhibiting effect of lower SES on scores on the BART (*b* = 0.768, *SE* = 0.395, *pz* = .052, 95% CI [-0.006 – 1.542]).

Figure. *Plot of RI stratified by SES quintile with 95% confidence intervals for lowest and highest quintiles.*



APPENDIX 5

Figure. *Plot of RI across adolescence stratified by gender.*



Gender differences in RI translated to a mean female score at age 12 of 2.046 (95% CI [1.579 – 2.513] versus 3.252 for males (95% CI [2.613 – 3.892). At age 16, average female risk involvement score was 4.858 (95% CI [4.428 – 5.289]) while for males it was 5.566 (95% CI [5.118 – 6.015). The steepest increase for males occurred between ages 13 and 14, with an average 0.92-point increase, while for females the steepest increase occurred later, between 14 – 15 (average 0.77-point increase). Around age 14 also marked the biggest difference in risk involvement between girls and boys. This may be an important maturational factor to note for potential interventions. There was no effect of gender on risk propensity (BART) trajectories (*b* = 0.947, *SE* = 0.997, *pz* = .342, 95% CI [-1.007 – 2.902]).

APPENDIX 6

Table 6. *Parameter estimates for LMM examining effects of explanatory variables by wave.*

	Risk involvement							Risk propensity (BART)						
	Fixed effects		97.5% CI			Random effects		Fixed effects		97.5% CI			Random effects	
	b	SE	*LL*	*UL*	*pz*		*SD*	*b*	*SE*	*LL*	*UL*	*pz*		*SD*
(Intercept)	3.049	0.758	1.563	4.535	0.000***			26.589	5.115	16.564	36.613	0.000***		
age	0.730	0.089	0.556	0.903	0.000***			3.713	0.660	2.419	5.006	0.000***		
I(age^2)	-0.019	0.095	-0.205	0.167	0.840			0.736	0.711	-0.657	2.130	0.300		
I(age^3)	-0.050	0.034	-0.116	0.016	0.134			-0.094	0.253	-0.590	0.402	0.710		
I(age^4)	-0.006	0.023	-0.051	0.040	0.813			-0.129	0.173	-0.468	0.210	0.456		
Female	-0.890	0.210	-1.302	-0.477	0.000***			1.957	1.429	-0.844	4.758	0.171		
SES	-0.106	0.074	-0.250	0.039	0.152			0.381	0.498	-0.594	1.357	0.444		
Neg. Urgency W0	0.129	0.179	-0.222	0.479	0.472			-1.774	1.207	-4.141	0.592	0.142		
Neg. Urgency W1	0.294	0.204	-0.107	0.694	0.150			-0.954	1.384	-3.666	1.758	0.491		
Neg. Urgency W2	0.391	0.182	0.034	0.748	0.032*			1.928	1.234	-0.490	4.345	0.118		
Sens. Seeking W0	0.498	0.210	0.088	0.909	0.017*			1.128	1.418	-1.652	3.908	0.426		
Sens. Seeking W1	-0.296	0.261	-0.807	0.216	0.257			-0.426	1.760	-3.875	3.023	0.809		
Sens. Seeking W2	-0.036	0.230	-0.487	0.414	0.874			0.814	1.554	-2.232	3.860	0.600		
BAS Reward Resp. W0	-0.035	0.210	-0.446	0.376	0.867			1.175	1.423	-1.615	3.965	0.409		
BAS Reward Resp. W1	-0.392	0.235	-0.852	0.067	0.094			1.494	1.580	-1.602	4.591	0.344		
BAS Reward Resp. W2	-0.212	0.227	-0.656	0.232	0.350			0.759	1.530	-2.239	3.756	0.620		
BAS Drive W0	-0.197	0.160	-0.510	0.116	0.218			0.729	1.085	-1.398	2.855	0.502		
BAS Drive W1	-0.062	0.189	-0.432	0.309	0.745			0.155	1.276	-2.346	2.656	0.903		
BAS Drive W2	0.499	0.171	0.163	0.835	0.004**			-3.419	1.159	-5.690	-1.148	0.003**		
BIS W0	-0.049	0.207	-0.454	0.356	0.812			-0.092	1.399	-2.835	2.651	0.947		
BIS W1	0.290	0.244	-0.187	0.768	0.234			1.940	1.645	-1.284	5.163	0.238		
BIS W2	-0.556	0.243	-1.033	-0.079	0.022*			-3.867	1.642	-7.085	-0.649	0.019*		
BAS Fun Seeking W0	0.116	0.214	-0.304	0.535	0.589			1.345	1.452	-1.501	4.192	0.354		
BAS Fun Seeking W1	0.434	0.234	-0.025	0.893	0.064			-3.460	1.585	-6.566	-0.354	0.029*		
BAS Fun Seeking W2	0.060	0.210	-0.351	0.471	0.774			1.262	1.423	-1.527	4.051	0.375		
Variance(Intercept)						1.637	1.279						67.196	8.197
Covariance (Intercept)Age						0.292	0.463						13.284	0.563
Variance(Age)						0.242	0.492						8.294	2.880
Residual variance						1.618	1.272						92.347	9.610
n observations		931							903					
n groups		312							312					

Note. CI = confidence interval; LL = lower limit; UL = upper limit. $p < .05$*; $p < .01$**; $p < .001$***

APPENDIX 7

Table 7. *Base model and interaction terms at each wave.*

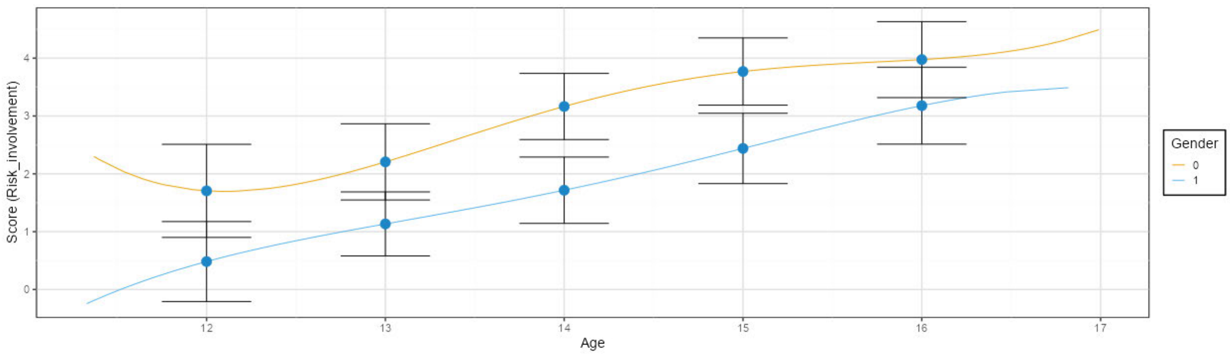
	Risk Involvement					Risk Propensity (BART)				
Variable	Fixed effects		97.5% CI			Fixed effects		97.5% CI		
Wave 1 – Base Model	*b*	SE	*LL*	*UL*	*pz*	*b*	*SE*	*LL*	*UL*	*pz*
(Intercept)	2.267	0.630	1.033	3.501	<.0001***	25.944	4.142	17.827	34.062	<.0001***
Age	0.676	0.047	0.583	0.769	< .001***	3.572	0.334	2.918	4.226	< .001***
Gender	-1.041	0.163	-1.360	-0.722	<.0001***	1.512	1.063	-0.572	3.597	.155
SES	-0.096	0.064	-0.223	0.030	.134	0.565	0.424	-0.265	1.395	.182
BIS W1	-0.414	0.172	-0.751	-0.076	.016*	0.179	1.128	-2.032	2.389	.874
BAS Drive W1	-0.080	0.131	-0.337	0.177	.540	-0.424	0.865	-2.120	1.271	.624
BAS Fun W1	0.334	0.181	-0.020	0.688	.064	1.369	1.184	-0.952	3.690	.248
BAS Reward W1	-0.218	0.176	-0.563	0.128	.217	1.679	1.158	-0.591	3.950	.147
Negative Urgency W1	0.584	0.144	0.302	0.865	<.0001***	-1.307	0.938	-3.146	0.531	.163
Sensation Seeking W1	0.489	0.144	0.207	0.770	.001***	0.521	0.940	-1.321	2.364	.579
Negative Social Bias W1	0.146	0.112	-0.074	0.367	.193	-0.671	0.735	-2.112	0.770	.361
Negative Nonsocial Bias W1	0.043	0.127	-0.207	0.293	.735	-0.035	0.835	-1.671	1.601	.967
Wave 1 – Interactions										
NU*NSB W1	0.053	0.433	-0.795	0.902	.902	-2.393	2.811	-7.903	3.117	.395
Age:NU*NegSocialBias W1	-0.003	0.046	-0.093	0.087	.952	0.017	0.324	-0.617	0.651	.958
NU*NegNonsocialBias W1	0.158	0.514	-0.849	1.165	.758	-5.843	3.326	-12.362	0.676	.079
Age:NU*NegNonsocialBias W1	0.031	0.046	-0.060	0.122	.501	-0.076	0.324	-0.711	0.558	.813
BIS*NSB W1	0.272	0.423	-0.558	1.102	.521	0.132	2.781	-5.317	5.582	.962
Age:BIS*NSB W1	-0.080	0.046	-0.170	0.009	.079	0.038	0.326	-0.601	0.676	.908
BIS*NegNonsocialBias W1	0.062	0.454	-0.829	0.952	.892	-0.639	2.984	-6.488	5.209	.830
Age:BIS*NegNonsocialBias W1	-0.059	0.047	-0.150	0.033	.209	0.080	0.327	-0.562	0.722	.806

	Risk Involvement					Risk Propensity (BART)				
Variable	Fixed effects		97.5% CI			Fixed effects		97.5% CI		
Wave 2 – Base Model	*b*	SE	*LL*	*UL*	*pz*	*b*	*SE*	*LL*	*UL*	*pz*
(Intercept)	3.671	0.627	2.443	4.899	<.0001***	29.746	3.920	22.064	37.429	<.0001***
Age	0.634	0.046	0.543	0.724	< .001***	3.452	0.320	2.823	4.080	< .001***
Gender	-1.371	0.176	-1.716	-1.025	<.0001***	2.411	1.102	0.251	4.570	.029*
SES	-0.126	0.066	-0.256	0.004	.057	0.363	0.415	-0.449	1.176	.381
BIS W2	-0.284	0.201	-0.678	0.109	.157	-0.182	1.253	-2.638	2.275	.885
BAS Drive W2	0.104	0.147	-0.184	0.392	.479	-0.167	0.916	-1.962	1.628	.856
BAS Fun W2	0.644	0.194	0.264	1.024	.001***	-1.323	1.206	-3.686	1.041	.273
BAS Reward W2	-0.577	0.191	-0.951	-0.203	.003***	1.145	1.190	-1.188	3.478	.336
Negative Urgency W2	0.598	0.153	0.298	0.899	<.0001***	-1.663	0.955	-3.535	0.209	.082
Sensation Seeking W2	0.130	0.149	-0.163	0.423	.386	1.618	0.932	-0.208	3.444	.082
Negative Social Bias W2	0.145	0.113	-0.077	0.366	.200	0.027	0.703	-1.350	1.405	.969
Negative Nonsocial Bias W2	-0.021	0.149	-0.314	0.271	.887	-0.548	0.929	-2.369	1.272	.555
Wave 2 – Interactions										
NU*NSB W2	-0.710	0.455	-1.601	0.181	.118	1.552	2.851	-4.036	7.139	.586
Age:NU*NegSocialBias W2	0.118	0.044	0.031	0.205	.008**	0.025	0.313	-0.589	0.639	.937
NU*NegNonsocialBias W2	-1.419	0.526	-2.451	-0.387	.007**	-2.021	3.329	-8.546	4.504	.544
Age:NU*NegNonsocialBias W2	0.115	0.044	0.029	0.200	.009**	0.007	0.308	-0.597	0.610	.983
BIS*NSB W2	0.199	0.478	-0.738	1.136	.677	2.229	2.974	-3.600	8.057	.454
Age:BIS*NSB W2	0.005	0.045	-0.085	0.094	.921	0.143	0.315	-0.473	0.760	.649
BIS*NegNonsocialBias W2	-0.590	0.514	-1.597	0.418	.251	3.565	3.207	-2.722	9.851	.266
Age:BIS*NegNonsocialBias W2	0.002	0.045	-0.086	0.090	.960	0.122	0.311	-0.489	0.732	.696

	Risk Involvement					Risk Propensity (BART)				
Variable	Fixed effects		97.5% CI			Fixed effects		97.5% CI		
Wave 3 – Base Model	*b*	SE	*LL*	*UL*	*pz*	*b*	*SE*	*LL*	*UL*	*pz*
(Intercept)	2.863	0.694	1.502	4.223	<.0001***	30.766	4.762	21.434	40.099	<.0001***
Age	0.611	0.047	0.519	0.702	< .001***	3.477	0.334	2.823	4.131	< .001***
Gender	-1.105	0.198	-1.493	-0.718	<.0001***	2.168	1.343	-0.465	4.801	.107
SES	-0.081	0.067	-0.213	0.052	.233	0.441	0.463	-0.467	1.348	.341
BIS W3	-0.505	0.208	-0.913	-0.096	.015*	-3.024	1.418	-5.803	-0.245	.033*
BAS Drive W3	0.310	0.150	0.017	0.603	.038*	-2.900	1.018	-4.895	-0.905	.004**
BAS Fun W3	0.346	0.186	-0.018	0.711	0.062	0.178	1.268	-2.308	2.663	.889
BAS Reward W3	-0.317	0.193	-0.696	0.062	.101	2.395	1.314	-0.180	4.970	.068
Negative Urgency W3	0.682	0.145	0.399	0.966	<.0001***	0.016	0.991	-1.926	1.958	.987
Sensation Seeking W3	0.135	0.149	-0.157	0.428	.365	0.916	1.018	-1.079	2.911	.368
Negative Social Bias W3	-0.185	0.111	-0.403	0.034	.097	0.308	0.763	-1.186	1.803	.686
Negative Nonsocial Bias W3	0.420	0.149	0.127	0.712	.005**	-0.679	1.019	-2.677	1.318	.505
Wave 3 – Interactions										
NU*NSB W3	-0.935	0.448	-1.814	-0.057	.037*	3.494	3.101	-2.584	9.573	.260
Age:NU*NegSocialBias W3	0.161	0.045	0.073	0.249	< .001***	0.220	0.327	-0.421	0.860	.502
NU*NegNonsocialBias W3	-1.010	0.530	-2.048	0.029	.057	-3.995	3.631	-11.112	3.122	.271
Age:NU*NegNonsocialBias W3	0.183	0.045	0.095	0.271	< .001***	-0.037	0.327	-0.678	0.604	.909
BIS*NSB W3	-0.289	0.453	-1.177	0.598	.523	0.495	3.080	-5.541	6.531	.872
Age:BIS*NSB W3	0.014	0.047	-0.077	0.105	.768	0.066	0.332	-0.586	0.717	.843
BIS*NegNonsocialBias W3	-0.682	0.549	-1.759	0.394	.214	2.163	3.751	-5.190	9.515	.564
Age:BIS*NegNonsocialBias W3	0.038	0.047	-0.054	0.130	.415	-0.146	0.334	-0.800	0.508	.662

APPENDIX 8

Figure 11. *Model plot of the impact of NU on RI stratified by gender.*



	12	13	14	15	16
Score (Risk_involvement) [Gender, level = 0] (95% CIs)	1.705 (0.9 - 2.51)	2.207 (1.548 - 2.865)	3.164 (2.593 - 3.736)	3.769 (3.187 - 4.35)	3.975 (3.318 - 4.632)
Score (Risk_involvement) [Gender, level = 1] (95% CIs)	0.483 (-0.208 - 1.175)	1.134 (0.58 - 1.687)	1.716 (1.143 - 2.29)	2.439 (1.831 - 3.047)	3.178 (2.513 - 3.844)

	12	13	14	15	16
Difference between 0 and 1 (95% CI)	-1.222 (-1.936 - -0.508)	-1.073 (-1.515 - -0.631)	-1.448 (-1.796 - -1.1)	-1.33 (-1.724 - -0.935)	-0.796 (-1.324 - -0.268)

APPENDIX 9

Figure 12. *Affective-motivational circumplex model proposing a dual-continua structure of approach motivation.*