Individual differences in risk preference: Insights from self-report, behavioral and neural measures, and their convergence

Inaugural Dissertation

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Approved by the Faculty of Psychology
at the request of
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Declaration

I, Loreen Tisdall (born November 7, 1984 in Bad Langensalza, Germany), hereby declare that I have written the submitted doctoral thesis "Individual differences in risk preference: Insights from self-report, behavioral and neural measures, and their convergence" without any assistance from third parties not indicated. I further declare the following:

(I) My cumulative dissertation is based on three first-author manuscripts, one published, one submitted and one to be submitted shortly. My cumulative dissertation furthermore includes a published manuscript on which I am the second author. I hereby certify that the articles in this dissertation reflect original work. Concerning the manuscripts in this dissertation on which I am listed as the first author, I contributed independently and substantially with respect to the ideas, experimental set up, analyses, interpretation of the results, and writing of manuscript 1. I contributed independently and substantially with respect to the data collection, analyses, interpretation of the results, and writing of manuscripts 3 and 4. Concerning the manuscript on which I am listed as the second author, manuscript 2, I contributed to the analysis plan, the analyses themselves, the interpretation of the results as well as the writing of the manuscript, including revisions requested by reviewers prior to publication.

(II) I only used the resources indicated.

(III) I marked all the citations.

Basel, _____

Loreen Tisdall _____

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Preface

In line with regulations of the Faculty of Psychology, University of Basel, this thesis is submitted as a cumulative (i.e. publication-based) dissertation, consisting of four manuscripts. The current framework is intended to (1) present the four manuscripts and highlight their individual contributions, and (2) based on the combined insights from the different manuscripts, derive broader conclusions for the topic of individual differences in risk taking.

I wish to highlight that the two published manuscripts appear under my maiden name Mamerow, whereas submitted manuscripts and those that are about to be submitted appear under my married name Tisdall.

This dissertation is based on the following four manuscripts:

(1) Mamerow, L., Frey, R., & Mata, R. (2016). Risk taking across the life span: A comparison of self-report and behavioral measures of risk taking. *Psychology and Aging*, *31*(7), 711.

(2) Yu, J., Mamerow, L., Lei, X., Fang, L., & Mata, R. (2016). Altered value coding in the ventromedial prefrontal cortex in healthy older adults. *Frontiers in Aging Neuroscience*, *8*, 210.

(3) Tisdall, L., Frey, R., Horn, A., Ostwald, D., Horvath, L., Blankenburg, F., Hertwig, R., & Mata, R. (2018). *Group versus individual differences in the neural representation of described and experienced risk.* Manuscript submitted for publication.

(4) Tisdall, L., Frey, R., Horn, A., Ostwald, D., Horvath, L., Pedroni, A., Blankenburg, F., Rieskamp, J., Hertwig, R., & Mata, R. (2018). *The risky brain: Local morphometry and degree centrality as neural markers of psychometrically derived risk preference factors.* Unpublished manuscript.

Abstract

From the time of conception until the time of death, the path of the human organism is created and shaped by decisions. Some decisions we make ourselves, some are made for us; some will make us, some will break us. What most decisions have in common, however, is that they are made under risk, that is, without complete information regarding the potential decision outcomes. One interesting feature about decisions under risk is variability: different individuals make different choices, and even the same individual may, given repeated occasions, make different choices. This doctoral thesis aims to address the issue of individual differences by looking at several specific variables which may impact inter- and intra-individual differences in risk taking, namely age, the measures used to assess risk-taking, neural function and neural structure.

In a set of four studies, the following questions were addressed: (1) To what extent do life span trajectories of risk taking change as a function of whether self-report or behavioral measures are used to assess risk taking? (2) Do younger and older individuals differ in the neural functional representation of risk and reward? (3) Do the neural representations of described and experienced risk converge, both at group and individual level? To what extent is neural function predictive of risky choice? (4) To what extent do individual differences in neural structure explain variance in psychometrically derived risk preference factors? The main findings are: (1) Self-report and behavioral measures of risk taking do not converge and lead to different life span trajectories. (2) The ventromedial prefrontal cortex is differentially activated in younger and older adults, with activation differences possessing differential explanatory power for choice in the two age groups. (3) Described and experienced risks show convergence at group level, divergence at the individual level, and are differentially predictive of risky choice. (4) Neural structural indices explain variance in the general risk preference factor, but not domain-specific risk preference factors.

Based on the findings from all four studies, this thesis provides corroborating evidence for the argument that not all risk-taking measures are created equal and that a taxonomy of risk-taking measures and their respective cognitive and affective demands is required to understand individual differences in risk taking.

Introduction

In 2017, Europe saw a 4-fold increase in measles cases on the previous year (World Health Organization, 2018), with the current outbreaks being traced back to vaccination scares following unsubstantiated claims of a connection between MMR immunization and autism (Flaherty, 2011). In 2008, an unforeseen global financial crisis burnt industries, economies, and ultimately countries, which it has been suggested was triggered by endemic 'wild risk taking' on Wall Street (Williams, 2010). In both cases, global effects are felt as the direct result of individuals making decisions in the face of risk, that is, in the absence of certain outcomes.

But one does not even have to consider global events to recognize the role of risk in human decision making. Whether it is choosing a restaurant, a partner, a political candidate, or a medical procedure, having to select between two or more alternatives that come with their very own list of pros and cons, and for which we do not know with certainty that the anticipated (dis)advantages will indeed materialize, is a situation which accompanies our daily lives. Risk, it seems, is everywhere: it contributes to our biggest successes as well as our steepest falls. Crucially, while some of our decisions remain comparatively inconsequential —unless a restaurant does not adhere to health and safety regulations and serves contaminated food, the worst outcome of trying somewhere new to eat is going home dissatisfied— others have far-reaching consequences. In the case of cancer screening, for example, not getting screened may result in the early stage of the disease being missed and left untreated. However, given the non-negligible rate of false positives and the potential for unnecessary treatment to be undertaken (Croswell, Baker, Marcus, Clapp, & Kramer, 2010; Elmore et al., 1998), what does one do with a test result that has come back positive?

One challenging aspect of decisions made under risk is understanding individual differences. Given the same choice set, one person opts for cancer screening, while another declines. Equally, given the same choice set, the same person may opt for screening on one occasion but may opt out at another occasion. The question which has spawned much interest

and which lies at the heart of this thesis is as follows: Why do individuals vary, both within themselves and between each other, with regards to decisions made under risk? Before I endeavor to provide some answers to this question, it is necessary to formally define risk as understood in this body of work, for how we define risk impacts on the chosen research methodology and consequently the interpretation of our results.

Definitions of risk

Formally, risk can be defined in the economic sense of outcome variance, which assumes uncertainty about the outcomes but is otherwise silent regarding the presence or absence of loss in the set of possible outcomes (Schonberg, Fox, & Poldrack, 2011; Weber, 2010). Under this definition, deciding between a 60% chance of 100 Francs and 10% chance of 600 Francs constitutes a risky decision. Alternatively, and perhaps more intuitively, risk has been defined as uncertainty about decision outcomes which entail the chance of incurring a form of loss, be that financial, physical, psychological, social, societal or otherwise (Schonberg et al., 2011; Slovic, 1987, 1998; Weber, 2010). It is interesting to note that some authors have gone even further and included the probability of loss as a criterion. For instance, Nigg defined risk taking as the "[a]daptive or maladaptive selection of rewarding behavioral option in the face of high probability of loss […]" (Nigg, 2017, p.4).

In the context of the studies presented within this doctoral thesis, whenever we manipulated risk, for instance in the context of using behavioral measures, risk was almost exclusively understood and operationalized as involving both gains and losses. To be precise, for all but one behavioral risk-taking measure in manuscript 1 and two behavioral risk-taking measures which informed the psychometric factors utilized in manuscript 4, were individuals presented with decision problems involving both rewards (i.e. gains) and losses, albeit of different magnitudes and probabilities. It could be argued that only by adopting a definition which incorporates both rewards and losses can we study individual differences in risk taking,

because what seems to drive individuals' understanding of risk appears to be loss, not simply variance (Slovic, 1987; Zeisberger, 2016).

I will now turn to the contribution this doctoral thesis makes to our understanding of individual differences in risk taking, starting with the role of two factors and their interplay: age and risk-taking measure.

Part I - Risk Taking Across the Life Span

Many factors that vary between (as well as partly within) individuals have become associated with risk taking. These include, but are not limited to, gender (Byrnes, Miller, & Schafer, 1999; Charness & Gneezy, 2012), economic status (Payne, Brown-Iannuzzi, & Hannay, 2017), reproductive cycle (Sylwester & Pawłowski, 2011), family background (Banducci, Felton, Dahne, Ninnemann, & Lejuez, 2015; Dohmen et al., 2011; Kennison, Wood, Byrd-Craven, & Downing, 2016), stress (Lighthall, Mather, & Gorlick, 2009), peer relationships (Telzer, Fuligni, Liebermann, Miernicki, & Galvan, 2014), quality of sleep (Telzer, Fuligni, Liebermann, & Galvan, 2013), affective state (Shao & Lee, 2014), as well as less intuitive factors such as simulated microgravity (L.-L. Rao et al., 2014).

One factor which has garnered substantial support for its impact on risk taking is age. Why would age capture the attention of researchers interested in risk taking? In a nutshell, factors such as improved living conditions, more efficient and effective medical treatment, as well as increased fertility, have contributed to a global population that is simultaneously expanding and aging. For example, between 1980 and 2017, the number of individuals globally over the age of 60 years has doubled from 382 million to 962 million, and is expected to more than double yet again by 2050 (Department of Economic and Social Affairs, 2017). Moreover, not only are there a greater number of older individuals, they are also living to increasingly advanced ages: In 2017, the number of individuals aged 80 and over was estimated to be 137 million, a number which is anticipated to triple by 2050 (Department of Economic and Social Affairs, 2017). To support an increasingly longer life span, even older individuals will need to stay active members of the workforce and society, and will inevitably face decisions regarding medical treatment, housing, pensions, and inheritance, to name but a few. Risk, it seems, is a constant companion, even to those of old(er) age.

To understand whether age influences how individuals deal with and take risks, and if so, through which pathways, research has started to address the life span trajectory of risk

taking. One prominent finding in this field is that age and risk taking are negatively correlated (Rolison, Hanoch, Wood, & Liu, 2013). Like many associations in psychological research, however, moderator variables play an important role and often qualify the conditions under which established bivariate associations hold. In the case of age and risk taking, studies based on panel data as well as meta-analytic approaches have consistently shown that life span trajectories of risk taking depend on the measures used to assess risk taking (Best & Charness, 2015; Byrnes et al., 1999; Josef et al., 2016; Mata, Josef, & Hertwig, 2016; Mata, Josef, Samanez-Larkin, & Hertwig, 2011). The pattern which emerges from these analyses is that self-reported risk-taking, regardless of domain, decreases across the life span, but that the association is less clear for behavioral measures of risk taking; for some measures, risk-taking declines across the life span, for others it increases, and yet for other measures no differences are observable. How can such divergent trajectories arise?

At this point, it is important to notice that a vast number of risk-taking measures exist (Appelt, Milch, Handgraaf, & Weber, 2011), leading to widespread diversity in the risk-taking measures adopted for research purposes. Diversity in the measures used to assess individual differences in risk taking is, in itself, unproblematic, for we may expect different measures to assess slightly different facets of a phenotype, and thereby yield a more complete picture of it. What creates a problem for the theory and measurement of risk taking is that the majority of measures do not converge, i.e. do not correlate or only weakly (Frey, Pedroni, Mata, Rieskamp, & Hertwig, 2017; Pedroni et al., 2017). This leads to a situation where, depending on the risk-taking measure used, we may come to very different conclusions about risk taking, for instance its change across the life span.

In manuscript one we directly address this issue by assessing risk taking across the life span using different risk-taking measures.

Manuscript One:

Convergence of risk-taking measures in a cross-sectional life span sample

Mamerow, L., Frey, R., & Mata, R. (2016). Risk taking across the life span: A comparison of self-report and behavioral measures of risk taking. *Psychology and Aging*, *31*(7), 711.

In front of the aforementioned backdrop of studies suggesting (a) low or no convergence between measures of risk taking, and (b) measure-dependent trajectories of risk taking across the life span, we conducted a correlational study investigating the convergence of risk-taking measures, and the extent to which convergence may change as a function of age.

In contrast to previous stand-alone studies, our research design included a large, cross-sectional, age-heterogeneous sample of participants, employed prominently used self-report and behavioral measures of risk taking, adopted a within-participants design, and compared the results obtained for the self-report item from the local sample against household panel data for the entire country (Switzerland). A further critical aspect of the study reported in manuscript one is that in addition to assessing self-reported general risk propensity (Dohmen et al., 2011; Josef et al., 2016; Mata et al., 2016), we employed two behavioral risk-taking measures: one experience-based risk-taking measure and one description-based risk-taking measure. The distinction between these two types of behavioral measures primarily arises from whether individuals are presented with choice-relevant information, or whether they have to learn this information over the course of the task, based on the experience of decision outcomes; the former describes decisions being made from description, whereas the latter describes decisions made from experience (Hertwig & Erev, 2009). We adopted the Balloon Analogue Risk Task (Lejuez et al., 2002) to assess risk taking in the context of decisions from experience, and used repeated choices between a certain and a risky monetary gamble (Rieskamp, 2008) to assess risk taking in the context of decisions from description.

Moreover, we used within-measure manipulations to gain further insights into why different measures (or generally, contexts) may foster different choice patterns. In the Balloon Analogue Risk Task, we employed a high-capacity and a low-capacity balloon to manipulate the level of perceived risk and increase learning demands within the same measure. For

decisions between a risky and a safe monetary gamble, we manipulated the expected value of the risky option: for half the trials, the risky option had the same expected value as the certain option; for the other half of the trials, the risky option had a lower expected value than the certain option. One specific aim of this particular within-measure manipulation was to test the 'certainty effect' reported for older adults (Mather et al., 2012), which suggests that age-related differences only emerge in the context of choosing between a certain and a risky option, but not between two risky options.

Overall, we observed patterns indicative of a reduction in risk taking with age for self-reported risk taking, but the evidence obtained from the two behavioral measures was mixed. The effect of a reduction in self-reported general risk taking across the life span observed for the local sample was replicated in the nationwide sample. As anticipated, absent or weak correlations between measures supported previous results suggesting convergence between measures at the level of the individual to be low. The within-measure manipulations for the description- and the experience-based behavioral measures yielded support for (a) the proposition that some conditions do not engender age differences in risk taking, (b) the certainty effect for older adults in the gain domain in equal expected variance trials but, interestingly, not in unequal expected variance trials, and (c) the possibility that task demands such as learning or computational capacity could contribute to diverging life span trajectories. Indeed, previous work indicates many neurological, cognitive, affective and motivational changes to manifest over the life span (Samanez-Larkin & Knutson, 2015; Shao & Lee, 2014), which may account for the differential effect of measures and within-measure manipulations on risk-taking trajectories.

The main conclusion from manuscript one is that to understand age differences in risk taking, research is required which systematically disentangles task demands from true age-related differences and life span changes. This also implies that instead of continually extending the pool of risk-taking measures, for example by developing ever more novel measures or

adding yet new variants to established tasks, what the field truly needs is a taxonomy of risk-taking measures, their cognitive, affective and motivational demands as well as corollaries.

Regrettably the experimental set up did not allow us to assess individual differences in cognitive functions, thus we were not able to test the mechanisms we proposed as underlying age-related differences in risk taking as a function of the measure used. In manuscript two we attempt to tackle this unresolved issue by shedding light on how the neurobiological basis of risk taking in the Balloon Analogue Risk Task is affected by age.

Manuscript 2:

Age-related differences in the neural representation of risk and reward in the Balloon Analogue Risk Task

Yu, J., Mamerow, L., Lei, X., Fang, L., & Mata, R. (2016). Altered value coding in the ventromedial prefrontal cortex in healthy older adults. *Frontiers in Aging Neuroscience*, *8*, 210.

The Balloon Analogue Risk Task (BART) has been widely adopted as a measure of risk taking and impulsivity (e.g., Lejuez et al., 2002, 2007; Lejuez, Aklin, Zvolensky, & Pedulla, 2003; Sharma, Markon, & Clark, 2014). In the BART, participants pump a virtual balloon ad libitum without knowing when the balloon will explode. Every pump increases the amount of money won but also the chance of explosion. When completing the BART, participants can stop pumping to save the money earned (cash out), or they can decide to continue pumping. However, if continued inflation results in an explosion of the balloon, the money accumulated up until that point is lost. Risk in the BART thus refers to the probability of a balloon explosion which will result in the loss of reward.

Presumably because the BART is purported to be an ecologically valid measure of risk taking, given its sequential decision-making process, experiential component and increasing tension as the balloon gets larger (Schonberg et al., 2011), it has also found widespread application in neuroimaging research (Congdon et al., 2013; Helfinstein et al., 2014; Kohno, Morales, Guttman, & London, 2017; Lighthall et al., 2012; H. Rao, Korczykowski, Pluta,

Hoang, & Detre, 2008; Schonberg et al., 2012). The results from these studies implicate a wide functional network as the neural correlates of risk taking in the BART, including the striatum, insular cortex, dorsolateral prefrontal cortex, (anterior) cingulate cortex, and ventromedial prefrontal cortex. Assigning function to neural structures, all of these regions have been implicated, albeit with more or less specificity, in the construction, representation and storage of subjective value (Bartra, McGuire, & Kable, 2013; Clithero & Rangel, 2014; Levy & Glimcher, 2012). Specifically, activation differences in striatum and frontal cortices have been associated with deliberative value-based judgments and decision-making, whereas the insular cortex activation has been implicated in primarily affective processing (Knutson & Huettel, 2015; Mohr, Biele, & Heekeren, 2010; Namkung, Kim, & Sawa, 2017; Paulus, Rogalsky, Simmons, Feinstein, & Stein, 2003; Platt & Huettel, 2008).

The research questions underlying manuscript two are as follows: How are activation differences in brain regions typically associated with risk taking in the BART affected by age? What insights do activation differences in circumscribed brain regions in the BART provide for our understanding of the mechanisms underlying age-related differences in risk taking? At the level of behavior, age-group comparisons of BART performance have yielded mixed findings (Cavanagh et al., 2012; Henninger, Madden, & Huettel, 2010; Mamerow, Frey, & Mata, 2016; Rolison, Hanoch, & Wood, 2012), with meta-analytic procedures suggesting risk taking in the BART to decline with age (Mata et al., 2011). At the level of the brain, however, we know comparatively little about the extent to which differences exist between younger and older adults. One potential mechanism for age-related differences to emerge in the BART is through activation differences in insular and ventromedial prefrontal cortex. Our ventromedial prefrontal cortex (vmPFC) hypothesis was based on previous findings suggesting that (a) the vmPFC represents a subjective value signal that is the outcome of a process integrating choice-relevant signals, including reward, risk and potentially affect (Bartra et al., 2013; Levy & Glimcher, 2012), (b) variance in vmPFC-related value signal increases with age (Halfmann,

Hedgcock, Kable, & Denburg, 2016), which may be the result of age-related differences in feedback-based learning rather than reward representation (Samanez-Larkin, Worthy, Mata, McClure, & Knutson, 2014), and (c) risk taking is associated with decreased vmPFC activation (Schonberg et al., 2012). Our insular cortex hypothesis was informed by previous findings which suggested affective changes over the human life span to impact on decision-making (Samanez-Larkin & Knutson, 2015; Shao & Lee, 2014). Within the framework of the mood maintenance hypothesis, for example, it has been suggested that older adults may engage in less risk taking to avoid negative consequences which may compromise a primarily positive status quo (Shao & Lee, 2014). We tested these hypotheses by comparing risk-taking behavior and neural activation in the BART for younger and older adults.

Our findings suggest that younger and older adults show similar risk-taking behavior in the BART. We observed no significant difference between younger and older adults in the mean number of pumps (adjusted for opportunity to pump), but the proportion of cash out trials was higher in older compared with younger adults. Regarding the neuroimaging results, group comparison revealed that younger and older adults' neural responses in the BART were not completely aligned. Specifically, when we compared pumps on risky with pumps on control balloons, we obtained no marked age differences for striatal and insular activation, or vmPFC deactivation. We obtained group differences in posterior parietal deactivation and cingulate cortex activation, which may have been indicative of less numerical integration in younger adults and more (conflict) monitoring in older adults for risky choices. When we investigated the parametrically modulated activation in response to increasing risk on a given balloon, we obtained no substantial differences between younger and older participants in the insular cortex and striatum. Interestingly, we however observed vmPFC deactivation to track risk in younger but not older adults, which point towards age-related differences in vmPFC-related integrative value signaling. When we investigated the explanatory power of neural activation differences for risky choice in the BART at the level of the whole brain, neural signal in striatum, insula

and vmPFC was predictive of the mean number of pumps for younger adults but not for older adults.

The results reported in manuscript two support and extend previous studies concerning both behavioral and neural age-related differences in risk taking. Behaviorally, the higher proportion of cash out trials for adults supports previous findings suggesting a decline in risk taking in older age (Henninger et al., 2010; Mamerow et al., 2016; Mata et al., 2011; Rolison et al., 2013). The neuroimaging results shed some light on the potential mechanisms underlying such age-related differences in risk taking. Overall the regions associated with risk taking in the BART in this study are in line with previous results implicating the striatum, insula, and frontal cortices, especially vmPFC (e.g., H. Rao et al., 2008; Schonberg et al., 2012). Regarding age differences, our results are interpreted as indicating the preserved neural tracking of risk and reward in the insula and striatum, respectively, in old age. This interpretation is supported by previous results suggesting reward representation in the striatum to remain intact in old age (Samanez-Larkin et al., 2014). However, the combination of intact insula and striatal signal in the presence of age-related vmPFC differences suggests that differences in risk taking in the BART may be rooted in age-related differences in the convergence and integration of information into a subjective value signal (Bartra et al., 2013; Clithero & Rangel, 2014; Halfmann et al., 2016). This line of reasoning is further supported by functional and anatomic changes in the vmPFC across the life span (Samanez-Larkin & Knutson, 2015).

To conclude, manuscript two suggests that rather than being the result of differences in the computation of a reward or risk signal per se, age-related differences in the BART (and perhaps in other risk-taking measures) may originate from older adults experiencing more difficulties with the integration of different sources of information (e.g. risk, reward) into a coherent, choice-preceding value signal. This may also explain why in manuscript one we obtained no differences for the low-capacity balloon as part of the within-measure manipulation in the BART. Low-capacity balloons may not engender age-differences because the earlier explosion

points may keep individuals from exploring and experiencing uncertainty, potentially even leading to strategy use. In other words, low-capacity balloons may not rely as much on the integration of choice-relevant signals because this type of balloon, regardless of the age of the participant, does not evoke the same (complex) decision-making processes compared with high-capacity balloons.

To summarize the contribution of this thesis to understanding risk-taking across the life span, the two studies (manuscript one and two) on the one hand provide further support for the assertion that age-related differences in risk taking arise from a complex interplay of biological, cognitive, motivational and affective changes taking place across the life span. On the other hand, and perhaps constituting the more important contribution, the two manuscripts (in particular, manuscript one) fuel the debate surrounding the convergence of risk-taking measures and by proxy the nature of individual differences in risk taking. How can we make progress understanding individual differences in risk taking, when the very existence of such differences seems to be dependent on our measures? In Part II of this dissertation, I turn to the idea that the biological underpinnings of risk taking, specifically brain function and structure, may yield some insights for this debate.

Part II - The Risky Brain: Convergence of Neural Indices of Risk

Consider the following starting point: A genome-wide association study with over one million individuals has identified 124 independent genetic loci associated with self-reported general risk taking (Karlsson Linnér et al., 2018). These genetic loci are highly expressed in brain tissue in the prefrontal cortex, striatum and midbrain. Furthermore, genetic correlations between general risk taking and different types of risky behaviors, including smoking, number of sexual partners, being self-employed, life time cannabis use, adventurousness, risky driving and alcohol consumption, are higher than phenotypic correlations, with many genetic loci being shared across risky behaviors and with general risk taking (Karlsson Linnér et al., 2018). In other words, a genetically-influenced, domain-general risk-taking factor seems to exist that influences individual differences in risk taking, via neural pathways implicated in value-based decision-making. Does this mean we can expect to find risk-related brain signals that are domain-general?

Manuscript Three:

Convergence of the neural functional correlates of described and experienced risk

Tisdall, L., Frey, R., Horn, A., Ostwald, D., Horvath, L., Blankenburg, F., Hertwig, R., & Mata, R. (2018). *Group versus individual differences in the neural representation of described and experienced risk.* Manuscript submitted for publication.

As previously discussed, risk is ubiquitous and risk taking as a phenotype has been associated with health, wealth, criminality, and general well-being (Moffitt et al., 2011; Steinberg, 2013). As a result, individual differences in risk taking and related phenotypes, such as self-control, have become promising entry points for intervention and ultimately prevention (Conrod et al., 2013). One approach to understanding individual differences in risk taking has been to study its neural correlates, including neural activation differences in response to risk. In fact, the interest in the neural correlates of risk has been sufficient to facilitate both qualitative (Knutson & Huettel, 2015; Platt & Huettel, 2008) and quantitative reviews (Bartra et al., 2013;

Mohr et al., 2010; Wu, Sacchet, & Knutson, 2012), converging on the importance of striatum, insula, anterior cingulate cortex, dorsolateral, and (ventro)medial prefrontal cortex.

Interestingly though, the role of risk-taking measures has so far received very little attention in the neuroimaging literature. Recall the distinction between experience-based and description-based measures of risk taking (Hertwig, Barron, Weber, & Erev, 2004; Hertwig & Erev, 2009): Contrary to standard economic theory, the same information encountered in different ways —either fully described or experienced— can lead to different choices. As prototypical examples of experience- and description-based measures of risk taking, the Balloon Analogue Risk Task (BART) and monetary gambles, respectively, have found widespread application for studying individual differences in risk taking, also in the field of neuroimaging (Barkley-Levenson, Van Leijenhorst, & Galván, 2013; Braams, van Duijvenvoorde, Peper, & Crone, 2015; Gilaie-Dotan et al., 2014; Helfinstein et al., 2014; Pletzer & Ortner, 2016). Can we assume that the regions identified by meta-analytical approaches as functional correlates of risk taking are shared by different types of measures, such as the BART and monetary gambles? Moreover, are these conjunction regions promising candidates for sources of brain-behavior associations?

Not necessarily. The crucial argument here is that most of our current knowledge regarding the shared neural correlates of risk taking is rooted in average activation patterns obtained from different studies (i.e. different individuals). Owing to the well-known but often neglected mismatch between group- and individual-level effects (Bornstein, Putnick, & Esposito, 2017), we cannot infer consistency (i.e. convergence) of neural function between measures from commonality. As suggested by the low convergence between risk-taking measures at the level of behavior (Frey et al., 2017), the same individual may respond very differently to different risk-taking measures. Thus, it is currently unclear to what extent repeated measures designs would support the suggested convergence of activation patterns, or,

alternatively, mirror the weak correlations observed between behavioral measures of risk taking.

In manuscript three, we directly address this question by comparing the neural functional correlates of an experience-based (BART) and a description-based (monetary gambles) risk-taking measure, which we assessed as part of a neuroimaging study conducted within the scope of the Basel-Berlin Risk Study. The Basel-Berlin Risk Study (BBRS) is a large-scale, multi-site, multi-method study which investigates individual differences, psychometric structure, and biological underpinnings of risk taking. Participants in the BBRS completed a one-day laboratory study involving an extensive test battery, including self-report, behavioral, frequency measures of risk-taking measures, as well as cognitive, personality, affective and personality assessments. An overview of all subsamples, measures, and further details on the BBRS is available from the Open Science Framework (https://osf.io/rce7g).

The analyses reported in manuscript three are based on a subsample of BBRS participants (N=116) who in addition to the laboratory session also completed a MRI session. Of relevance to the analyses reported in manuscript three are two measures which participants completed inside the MRI scanner, namely the BART and a monetary gambles paradigm. Our analyses focus on (1) the overlap of the average neural representation of risk in the BART and in monetary gambles, (2) whether individual activation differences correlate between the BART and monetary gambles, and (3) the explanatory power of neural indices from BART and monetary gambles for risky choice, both within and across the two measures.

The results from the comparison of activation differences in BART and monetary gambles suggest (1) joint activation increases for BART and monetary gambles in a part of the ventral striatum, the nucleus accumbens, but (2) inconsistent individual differences in nucleus accumbens, insula and anterior cingulate cortex activation across the two measures. With regards to (3) the explanatory power of neural indices for behavior, we observe significant within-measure brain-behavior associations only for monetary gambles, but not for BART.

Using whole-brain analyses, there was no link between neural activation in response to risk in the BART and risky choice in monetary gambles; ROI analyses suggest a link between anterior cingulate cortex activation in BART and the proportion of risky gambles accepted in monetary gambles.

Taken together, the results reported in manuscript three further help to clarify the biological basis of risk taking, specifically the commonalities and differences between experience-based and description-based measures. Importantly, our findings fall in line with the results obtained for behavioral measures of risk taking (Frey et al., 2017), suggesting that these two types of measures should not be used interchangeably if the aim is to capture risk preference. As such, these results have strong implications for developmental and longitudinal research designs which frequently target the links between individual differences in risk preference and neural indices (Braams et al., 2015; Büchel et al., 2017; Grubb, Tymula, Gilaie-Dotan, Glimcher, & Levy, 2016; Moffitt et al., 2011). Moreover, our results suggest that researchers should pay more attention to topics such as individual differences and convergence between measurements, and less attention to seductive but likely uninformative single indices of risk taking.

Going back to the starting point of a genetically-informed, domain-general factor of risk taking that is primarily expressed via neural pathways, we did not find evidence for this in neural function. In line with the argumentation provided in the context of manuscript one and partly manuscript two, highly contextualized measures such as the BART and monetary gambles may not capture much risk-preference signal amongst the noise created by measure-specific demands and corollaries. Put differently, there may exist a general risk-taking trait, but this is perhaps lost in single behavioral indices and state-dependent, on-task functional activation differences. In manuscript four we examine whether more trait-like behavioral and neural indices of risk taking shed some light on the biological underpinnings of individual differences in risk taking.

Manuscript Four:

The neural structural correlates of risk preference

Tisdall, L., Frey, R., Horn, A., Ostwald, D., Horvath, L., Pedroni, A., Blankenburg, F., Rieskamp, J., Hertwig, R., & Mata, R. (2018). *The risky brain: Local morphometry and degree centrality as neural markers of psychometrically derived risk preference factors.* Manuscript.

While the neural functional correlates of risk-related processes received considerable attention in the past, much less attention has been paid to the contribution of neuroanatomy to individual differences in risk taking. In contrast to on-task functional indices such as brain activation differences in response to pumping up balloons in the BART or making choices between two monetary gambles, however, brain structure is much less susceptible to the influence of contextual variables, hence may represent the biological dimension of a risk-taking trait.

In previous studies, grey matter volume in amygdala, insula, thalamus, orbitofrontal and posterior parietal regions was observed as differentially associated with various indices relevant to risk taking, including risk tolerance, mean number of pumps in the BART, loss aversion, cannabis use, alcohol intake and gaming pathology (Cai et al., 2015; Canessa et al., 2013; Gilaie-Dotan et al., 2014; Jung, Lee, Lerman, & Kable, 2018; Koehler, Hasselmann, Wüstenberg, Heinz, & Romanczuk-Seiferth, 2013; Nasiriavanaki et al., 2015). Considering that the brain is characterized better by a system of networks rather than a collection of independent regions, perhaps local volume per se is not a useful index of brain structure. After all, the size of a particular region may matter much less compared with how well this region is connected within the network. Indeed, in addition to volumetric measures, the degree of local structural connectivity and integrity of white matter tracts has become associated with various risk-taking indices (Jacobus et al., 2013; Jung et al., 2018; Kohno et al., 2017; Kwon, Vorobyev, Moe, Parkkola, & Ha, 2014; Squeglia et al., 2015).

One shortcoming of these previous studies is the use of a vast number of risk-taking measures with little to no systematic understanding of the mechanisms underlying the suggested brain-behavior associations. For example, the association between volume in the right posterior parietal cortex and risk tolerance has been replicated in independent samples and using model-free as well as model-based indices of risk tolerance (Gilaie-Dotan et al., 2014; Grubb et al., 2016). The right posterior parietal cortex however is mainly associated with numerical processing. Given that the task used to elicit the aforementioned association between grey matter volume and risk tolerance relies heavily on the integration of various numerically presented pieces of information, it is possible that the association is indeed one between numerical ability and grey matter volume, rather than risk tolerance. The same problem may arise using other measures, especially behavioral measures, which have a 'risk-taking' tag but first and foremost distinguish individuals based on risk-independent processes such as affect or numerical ability (Figner, Mackinlay, Wilkening, & Weber, 2009).

To overcome the specific problem of using a single neural measure, in manuscript four we report the results of a multi-modal imaging analysis that was based on grey matter volume and local degree centrality. The latter represents a quantitative indication of how connected a neural structure is to its first neighbors, and is computed as the number of direct connections to other regions inside the network under investigation (Rubinov & Sporns, 2010). For the structural analyses, we utilized a set of regions which we identified via meta-analytic approaches implemented in Neurosynth (neurosynth.org) as core correlates of the term 'risk'. This approach has the distinct advantage of being rather inclusive and based on previously established links between neural function and risk. As a result of this approach, we ran analyses initially for 18 regions, but reduced these to 10 regions after correlation analyses highlighted multicollinearity between predictors that was most strongly evident between hemispheres of the same structure. Thus, we generated a mean index for both volume and degree centrality across the two hemispheres of all bilaterally represented structures.

To address the issue of low convergence between risk-taking measures and overcome related shortcomings of previous studies, we combined the multi-modal imaging component of manuscript four with psychometrically derived risk preference factors from the Basel-Berlin Risk Study (BBRS) imaging subsample. One of the main contributions of the BBRS to date has been the extraction of psychometric factors of risk preference by Frey and colleagues (2017). Specifically, the authors implemented a bifactor model that gave rise to a general risk preference factor *R*, and seven orthogonal domain-specific risk preference factors. In contrast to single behavioral indices of risk taking, *R* was observed to account for 62% of the explained variance and showed high retest validity (.85). The general risk preference factor *R*, it is argued, captures the trait-dimension of risk taking (Frey et al., 2017). As a result, we worked with neural measures and risk preference indices which have been argued to represent the comparatively more stable, trait-like dimensions of risk taking.

The main research questions driving analyses reported in manuscript four are as follows: To what extent can neural structural indices account for variance in psychometrically derived risk preference factors? Here we are particularly interested in the individual contribution of the set of volumetric and connectivity indices, as well as how much additional variance would be explained if volumetric and connectivity indices were combined to account for risk preference. Moreover, given that the 10-region risk network we built using Neurosynth covered all regions which previous studies had indicated to be linked to risk taking via grey matter volume and degree centrality, we attempt to provide a conceptual replication of these established brain-behavior associations using the BBRS risk preference factors. To quantify the robustness of the contribution of individual neural indices, we supplement traditional multiple regression analyses with Bayesian model comparison and selection.

The results from our multiple regression analyses suggest that structural indices can account for variance in the general risk preference factor R, but that they are not predictive of domain-specific risk preferences. The latter finding is particularly interesting given that one of

the psychometric factors (F7) comprises behavioral measures of risk taking (e.g. monetary lotteries). Thus, if not for *R*, it was reasonable to expect associations of previously identified structural indices with F7. Volumetric indices explained close to 9% of variance in R, connectivity indices only around 5%, and the combination of volumetric and connectivity indices explained over 15% of variance. At the level of individual predictors, grey matter volume in the ventral striatum was identified by Bayesian model comparison as the most influential predictor of general risk preference, followed by grey matter volume in frontal regions and degree centrality of the amygdala. The finding for ventral striatal volume had previously only been observed for pathology (Cai et al., 2015; Koehler et al., 2013). The striatum was already firmly on the map based on its functional role for reward-related processes of risky choice. The finding of increased volume in the striatum to be linked to increased general risk preference is therefore very informative for understanding the mapping of structure to function and their respective contributions to risk taking. Equally, frontal cortices and amygdala contributions are in line with functional and structural links to risk-related indices. As such, our results support and extend the importance of certain neural region for risk taking to the structural domain.

Interestingly —and contrary to expectations— some of the regions which had previously been linked via grey matter volume to individual differences in risk taking were not observed to be linked to the general risk preference factor, including insula and posterior parietal cortex. We suggest that these results, which we take to constitute a failed conceptual replication of earlier findings, are indicative of the fragility of brain-behavior associations and arise because individual differences in some risk-taking measures are primarily driven by specific task demands, such as numerical ability.

The results reported in manuscript four imply that neural structure may indeed provide independent contributions to general risk preference, and as such map onto the suggested genetically-influenced domain-general factor of risk taking called into existence by a genome-

wide association study (Karlsson Linnér et al., 2018). Of course, much variance is left to be explained, but given that we utilized decontextualized risk preference factors, the fact that 15% of variance in general risk preference is explained by very simple indices of neural architecture provides new hope for a general risk-taking trait. As a major upshot of this study, we recommend that other researchers follow suit and build their research studies around robust, psychometrically informed phenotypes.

General Discussion

This thesis set out to address the fascinating question of why individuals, sometimes between each other, sometimes within themselves, differ. Differ with regards to the choices they make when faced with uncertainty, specifically, when faced with risk. Many factors have been found to be associated with and potentially even drive individual differences in risk taking, ranging from factors that arise from within the person, to external factors, such as how risk taking is assessed. In this body of work, my co-authors and I looked specifically at the contribution of age, risk-taking measures, and their interplay, as well as the extent to which biological substrates, in this case neural function and structure, vary with risk taking.

The results from the four manuscripts cultivate the following four conclusions. (1) Life span trajectories of risk taking may arise not simply from age-related differences in risk perception, but from the (compromised) integration of complex information. Thus, we can relatively easily help the aging decision-maker deal with risk by communicating information in ways that it can be easily integrated. (2) Individuals matter! If we want to understand individual differences in risk taking, group-level (i.e. aggregate patterns) can at best suggest hypotheses for individual differences, but these most certainly need to be tested. (3) Biology, particularly the brain, still holds many promises for discovering the pathways underlying individual differences in risk taking. If the neural correlates can be mapped onto cognitive, affective, and motivational processes, we may find an entry point to causative inference. (4) Across the manuscripts contained in this dissertation, a unifying conclusion speaks to the importance of knowing our measures better. The field of risk-taking research, particularly when geared towards understanding developmental patterns or pathology, suffers from many piecemeal approaches, due to the diversity of risk-taking measures available and utilized. For now, the field does not need more measures, or even better measures, but first a taxonomy of the measures that are in use. This taxonomy needs to spell out, perhaps even classify, the cognitive, affective and motivational demands, corollaries, and other contextual factors that need to be

distinguished from the risk signal captured by a particular measure. Otherwise, when we try to see the wood for the trees by synthesizing the evidence for/against certain pathways, we come to an early fork in the road. The story of this thesis is a story of choice, not just risky choice, but also choice between measures.

Two roads diverged in a yellow wood,
And sorry I could not travel both
And be one traveler, long I stood
And looked down one as far as I could
To where it bent in the undergrowth;

Then took the other, as just as fair,
And having perhaps the better claim,
Because it was grassy and wanted wear;
Though as for that the passing there
Had worn them really about the same,

And both that morning equally lay
In leaves no step had trodden black.
Oh, I kept the first for another day!
Yet knowing how way leads on to way,
I doubted if I should ever come back.

I shall be telling this with a sigh
Somewhere ages and ages hence:
Two roads diverged in a wood, and I—
I took the one less traveled by,
And that has made all the difference.

Robert Frost (1916)

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List of Appendices

APPENDIX A: Manuscript One

Risk taking across the life span:

A comparison of self-report and behavioral measures of risk taking

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Abstract

Aging has long been thought to be associated with changes in risk-taking propensity. But do different measures converge in showing similar age-related patterns? We conducted a study to investigate the convergent validity of different self-report and behavioral assessments of risk taking across adulthood ($N = 902$). Individuals between 18 and 90 years of age answered a self-report item and completed two incentivized behavioral tasks, a gambles task and the Balloon Analogue Risk Task. Our results indicate that although all measures show some patterns indicative of an age reduction in risk taking, the correlations between measures are small. Moreover, age differences in behavioral paradigms seem to emerge as a function of specific task characteristics, such as learning and computational demands. We discuss the importance of understanding how specific task characteristics engender age differences in risk taking and the need for future work that disentangles task demands from true age-related changes in risk-taking propensity.

Keywords: aging; risk taking; BART; gambles; self-report

Word count: 8177

Risk taking across the life span:
A comparison of self-report and behavioral measures of risk taking

Aging is associated with changes in cognitive abilities, motivation, and affect that may have important implications for decision-making preferences and outcomes (Figner & Weber, 2011; Samanez-Larkin & Knutson, 2015). For example, some researchers have concluded that just "as elders show profound declines in cognitive function, they also show profound declines in choice rationality compared with their younger peers" (Tymula, Rosenberg Belmaker, Ruderman, Glimcher, & Levy, 2013; p. 17143). One prominent feature of many decision situations encountered across the life span is risk. Whether to party or study for an exam, start a family or pursue a career, spend or invest the money earned, which way to vote, take up treatment or not: all of these decisions involve some level of uncertainty regarding the nature and probability of future outcomes. Individual differences in the tolerance of and appetite for risk can lead to substantially different choices given the same set of options, yet comparatively little is known about the trajectory of risk-taking propensity across the life span. Previous research has been inconclusive, showing different and sometimes even opposite age-risk trajectories. Although many studies suggested a decline in risk taking with age, some have suggested an increase in risk taking with age, and still others found no differences between risk-taking propensity of younger and older individuals (cf. Best & Charness, 2015; Henninger, Madden, & Huettel, 2010; Mata, Josef, Samanez-Larkin, & Hertwig, 2011; Shao & Lee, 2014).

One potential reason for the observed divergence of trajectories is the adoption of different approaches to measuring risk-taking propensity, such as self-report measures or diverse behavioral measures. Beyond choosing between risky options, some of the latter also tap into additional cognitive demands, for example, learning. How these different approaches relate conceptually and psychometrically still remains poorly understood (Appelt, Milch,

Handgraaf, & Weber, 2011). Moreover, our current understanding regarding the convergent validity of different risk taking measures is based primarily on adoption of multiple measures within a particular age group. Consequently, little is known about the convergent validity of risk-taking measures across the adult human life span.

Using a large-scale cross-sectional study that allows for the treatment of age as a continuous variable, we report independent age-risk trajectories for three measures, each of which is representative of the main approaches to capturing risk-taking propensity: self-reports, description-based, and experience-based behavioral measures. Further, we examine the convergent validity of these three measures as a function of age in order to contribute to the conceptual debate regarding the measurement of risk-taking propensity.

Risk Taking Across the Life Span

The assessment of whether and how core factors, such as cognitive and motivational variables, affect age-related changes in decision-making requires a good understanding of decision measures and their interrelations. Yet the empirical and conceptual diversity associated with risk taking make such an enterprise difficult (Aven, 2012; Schonberg, Fox, & Poldrack, 2011). Different measurement approaches have been adopted to approximate individuals' risk taking in real life, namely self-report and behavioral measures (Josef et al., 2016); the latter category of behavioral measures can be further divided into description and experience-based tasks (Mata et al., 2011).

Age-risk trajectories for self-report measures. Mirroring the complexity of the risk-taking construct, self-report measures of risk taking are inherently heterogeneous: while some items and instruments assess general propensity (e.g. Benjamin et al., 2012; Dohmen et al., 2011; Drobetz, Maercker, Spiess, Wagner, & Forstmeier, 2012; Josef et al., 2016; Mata, Josef, & Hertwig, 2016; Vieider et al., 2013) others tap into more domain specific aspects (e.g. Bonsang & Dohmen, 2015; Dohmen et al., 2011; Lönnqvist, Verkasalo, Walkowitz, &

Wichardt, 2014; Rolison, Hanoch, Wood, & Liu, 2013; Vieider et al., 2013). Regardless of specificity, both general and domain-specific items used in cross-sectional and longitudinal studies coincide in the suggestion that aging is associated with reductions in risk-taking propensity across the life span (Dohmen et al., 2011; Josef et al., 2016; Mata et al., 2016; Rolison, et al., 2013; Vieider et al., 2013). This trend also holds for many countries around the world, albeit with systematic variation between countries as a function of the utility and necessity of risk-taking behavior in the (local) ecological context (Mata, et al., 2016). For general and domain-specific self-report items, there is an overall age-related decline in risk taking for financial, recreational, ethical, social and health-related activities, yet form and magnitude of this change may be domain-specific (Josef et al., 2016; Rolison et al., 2013). Critically, research by Dohmen and colleagues (2011; also see Vieider et al., 2013 and Josef et al., 2016 for comparable findings) indicated that self-reported general and domain-specific risk taking were strongly correlated. Thus, although self-reported general and domain-specific risk taking follow slightly different developmental trajectories, using a general risk-taking index is an efficient way to capture individual differences in risk-taking propensity (Dohmen et al., 2011). Speaking to the test-theoretic utility of a general risk-taking item, self-reported general risk taking has been found to relate with real-world behavior (Dohmen et al., 2011), to evidence high test-retest reliability and moderately correlate with genetic factors (Benjamin et al., 2012), and to describe a phenotype with moderate stability across the adult human life span (Josef et al., 2016).

Age-risk trajectories for description-based behavioral measures. In contrast to the overall homogenous pattern of a negative association between self-reported risk taking and age, trajectories derived from behavioral measurements are less consistent, even making opposite predictions for the association between age and risk taking (Best & Charness, 2015; Defoe, Dubas, FIgner, & van Aken, 2015; Mata, et al., 2011; Shao & Lee, 2014). A

commonly used way of eliciting propensity for risk taking via tasks is to present individuals with two or more lotteries (gambles) within a forced-choice framework (Holt & Laury, 2002; Rieskamp, 2008; Weller, Levin, & Denburg, 2011). Tasks of this kind provide the decision maker with all decision-relevant information, such as outcome magnitude and probability, and do not require any learning; they are therefore sometimes referred to as 'decisions from description' (Hertwig, Barron, Weber, & Erev, 2004). For example, the realistic scenario of selecting between a pension fund with a stable return and investment schemes with probabilistic returns can, in principle, be represented by a lottery with a fixed outcome A and a risky outcome B (assuming that the variance of the investment scheme is known from historical data and presented accordingly).

Age-risk trajectories derived from description-based gambles suggest common as well as unique trajectories, depending on various aspects of the task. Across studies, trajectories for description-based decisions between options associated with gains indicate increased risk aversion in older age (Mather et al., 2012; Tymula et al., 2013; Weller et al., 2011), a pattern matching that observed for self-report measures. When it comes to decisions involving losses, however, findings become more differentiated, for older adults were found to make similar (Weller at al., 2011) or more risk-seeking choices compared with younger adults (Mather et al., 2012, Tymula et al., 2013). A recent meta-analysis confirmed the influence of outcome domain on decision-making in younger and older adults (Best & Charness, 2015), concluding positively (negatively) framed items to be associated with more (no differences in) risk-averse choices in older relative to younger adults. Apart from the domain, whether the choice set includes a sure outcome seems to impact on age-risk trajectories. Decisions between a risky and a sure option for instance have been found to yield similar (Mata et al., 2011) or divergent (Mather et al., 2012) choice patterns for older and younger adults. Interestingly, whilst Mather and colleagues (2012) found no age differences for gambles involving two risky options, age-

differences were observed in another study (Weller et al., 2011). These patterns are further qualified by the observation of a non-linear development of risk-taking propensity across the life span, accompanied by increased choice inconsistency and decreased choice rationality by older individuals (Tymula et al., 2013; Weller et al., 2011).

These results exemplify the notion that description-based decisions are not impacted equivocally by age, but instead that task-specific attributes contribute to diverging age-risk trajectories. Some of the observed age differences may result from the complexity of the task, for it has been shown that the integration of several described cues (e.g., outcome magnitudes, probabilities) poses more of a challenge to aging decision makers (Bruine de Bruin, Parker, & Fischhoff, 2012; Tymula et al., 2013). Additional task demands that tap into wide-ranging cognitive, motivational, affective and perceptual differences - many of which have been linked to developmental changes in neural correlates (Samanez-Larkin & Knutson, 2015) - may further contribute to age-related certainty and framing effects, such as developmental affective changes leading to a shift in the weighting of gains and losses (Shao & Lee, 2014).

Age-risk trajectories for experience-based behavioral measures. In stark contrast to description-based measures are paradigms that entail (some level of) exploration and feedback-based learning. In real life, decisions often have to be made based on information that cannot be condensed into neat outcome magnitudes and probabilities. Instead, individuals often have to repeatedly extract and update their beliefs about the environmental contingencies – that is, to learn about the underlying option attributes - through exploration, choice, and feedback. Whether to study for an exam or enjoy one's free time, only repeated experience will furnish the individual with option-associated values. If the individual does not learn from previous experience or does not calibrate his/her behavior accordingly, sub-optimal choices may result.

Several behavioral measures that simulate decision-making under *experienced* risk exist, including the Balloon Analogue Risk Task (BART, Lejuez et al., 2002). The BART has become one of the go-to risk-taking measures because it holds predictive qualities for individual differences in actual risk taking (Lauriola, Panno, Levin, & Lejuez, 2014), including substance use, delinquency, rebellious and risky sexual behaviors (Aklin, Lejuez, Zvolensky, Kahler & Gwadz, 2005; Lejuez, Aklin, Zvolensky, & Pedulla, 2003; Lejuez, Simmons, Aklin, Daughters & Dvir, 2004; Skeel, Neudecker, Pilarski, & Pytlak, 2007), as well as a composite score of risky behaviors in adolescents (Lejuez et al., 2007) and psychopathy (Hunt, Hopko, Bare, Lejuez, & Robinson, 2005). Importantly, the BART has been shown to yield moderate to high test-retest reliability across different temporal intervals (Lejuez et al., 2007; White, Lejuez, & de Wit, 2008; Weafer, Baggott, & de Wit, 2013). Age-risk trajectories for the BART, like self-report and some description-based behavioral measures, delineate a negative association between risk taking and age (Mata et al., 2011), however different versions of the BART have also led to trajectories suggestive of increased risk taking in older adults (Cavanagh et al., 2012). The heterogeneity of trajectories is not restricted to versions of the BART, however, but applies to other experience-based behavioral measures, including the Iowa Gambling Task (cf. Mata et al., 2011), Columbia Card Task (Figner, Mackinlay, Wilkening, & Weber, 2009), as well as classical sampling paradigms (Frey, Mata, & Hertwig, 2015; Spaniol & Wegier, 2012). Interestingly, age-risk trajectories differed not only between tasks, but also between different versions of the same task (Frey et al., 2015; Figner et al., 2009; Mata et al., 2011).

One potential reason for the observed disagreement between experience-based measures is the extent to which different measurements tap into different cognitive processes that are subject to age-related changes. For example, we have argued that learning and memory demands may contribute substantially to age differences found in experience-based risky-

choice paradigms, with differences reflecting effects of age differences in cognitive abilities rather than risk propensity per se (Mata et al., 2011; Frey et al., 2015). Specifically, tasks that require the extraction of information from samples of experienced choice outcomes – such as BART, Iowa Gambling Task, Columbia Card Task - yield more or less risk-seeking behavior for older adults, depending on whether the underlying contingencies favor more or less risk taking to achieve a higher payoff (Mata et al., 2011; Shao & Lee, 2014). If the contingencies are not sufficiently extracted from choice and feedback, behavior on subsequent trials may not be appropriately calibrated. Importantly, even within risky-choice paradigms with learning demands, the extent to which age-differences emerge has been found to depend substantially on task demands, with age differences only arising as a function of increasing task complexity (e.g., number of choice options to learn about, Frey et al., 2015).

Returning to the convergence of trajectories derived from self-report, description and experience-based behavioral measures, under some circumstances, all three measurement approaches suggest risk taking to decline with age. However, especially for behavioral measures, age-risk trajectories vary, possibly as a function of different measures tapping into different cognitive processes that are impacted by age-related changes (Mata et al., 2011). Simply put, just as the (cognitive) demand placed on the decision maker increases from self-report to description-based to experience-based behavioral measures, so does the potential for trajectory divergence, especially when task demands are differently or insufficiently met by a cognitive system subject to age-related change. Task characteristics may therefore not only play an important role in engendering differences between measures within a specific age group (e.g., decisions from description vs. from experience in younger adults, Hertwig et al., 2004), but particularly *across* age groups (Mata et al., 2011).

The Current Study

Our main goal was to examine the cross-sectional age-risk trajectories obtained from three different measures, each representative for the reviewed measurement approaches, and to assess their convergent validity as a function of age. Our battery comprised the self-report item of the German Socio-Economic Panel (SOEP) assessing general risk-taking (Dohmen et al., 2011; Josef et al., 2016), a gambles task involving described monetary lotteries (e.g., Rieskamp, 2008; Tymula et al., 2013) as an example measure *without* a learning component, and the Balloon Analogue Risk Task (BART; Lejuez et al., 2002) as an example of a behavioral measure *with* a learning component. The three specific measures a) are arguably the most prominently used ones in each of the categories and b) are therefore among the best-studied measures regarding their test-theoretic properties, including test-retest reliability and external validity (cf. Benjamin et al., 2012; Dohmen et al., 2011; Josef et al., 2016; Lejuez et al., 2007; Lauriola et al., 2014). Evidence pertaining to the test-retest reliability of common indices derived from description-based behavioral measures is mixed, but was suggested to be better for less complex measurements (Chuang & Schechter, 2015).

To examine the role of task characteristics and their interplay with age, we used the following specific within-task manipulations. In the gambles task, we included both trials in which the risky option featured the same or a lower expected value relative to the safe (certain) option (cf. risk-disadvantageous options in Weller et al., 2011). The two task conditions were introduced to vary the utility of taking a risk: in equal expected value trials, selecting either option confers no benefit over the alternative other than deciding for or against certainty. In trials for which expected value was lower for the risky option, however, a safe choice is the better strategy. One focus of our analysis was thus to examine the certainty effect in the domain of gains reported for older adults (Mather et al., 2012), specifically whether this is influenced by the utility of a safe or risky choice (Weller et al., 2011). In

general, we expected such within-task manipulations to help us examine whether individuals adjust their risk taking as a function of task characteristics and assess their role in engendering age differences. In the BART, we varied balloon capacity across two types of trials, thus increasing learning demands and allowing us to examine whether age differences in learning can account for potential age differences in risk-taking propensity.

Finally, many previous studies on age-differences in risk taking relied on extreme age comparisons (e.g., young vs. older adults) and/or collected data from comparatively small and non-representative samples (cf. Best & Charness, 2015; Mata et al., 2011; Tymula et al., 2013). An additional aim of this study was therefore to collect and analyze data from a fairly large sample, characteristic of the local adult population, to better estimate cross-sectional trajectories of risk-taking across the entire adult life span using both self-report and behavioral measures.

Method

Participants

973 individuals between 18 and 90 years of age participated in a survey of the population of Basel, Switzerland. Participants were recruited from the street in the city center and in a large shopping mall and asked to complete a computer-assisted personal interview (CAPI). Data from 71 individuals were excluded from all analyses due to incomplete data or repeated runs of the survey, which arose from technical problems in data collection due to temporarily poor quality of Internet access at the study locations. The final sample included 902 individuals (M_{age} = 47.4, *SD* = 17.4, *range* = 18–90; 492 females, 55%). Individuals could earn money depending on their performance on the two behavioral tasks and earned on average 10.1 CHF (Swiss francs, 1 CHF ~ 0.98 USD) (SD = 6.18). Informed consent was obtained from all individuals prior to participation. The Institutional Review Board of the Faculty of Psychology, University of Basel, reviewed and gave ethical approval for the study.

In addition, we used cross-sectional data from 13,699 participants of the Swiss Household Panel (swisspanel.ch; Zimmermann et al., 2003) to perform a qualitative comparison of the developmental trajectories for self-reported risk taking between our local sample and the Swiss population at large (see Supplementary Materials for additional sample details).

Materials

Self-reported risk taking. Participants were presented with the following item (cf. Dohmen et al., 2011): "How do you rate yourself: Are you in general a risk-taking person or do you usually try to avoid taking risks? Please provide your answer with reference to a scale of 0 to 10. The value 0 indicates absolutely not risk taking, and the value 10 indicates very risk taking. You can adjust your response by selecting any value in between." Participants were presented with an integer scale ranging from 0 to 10, and indicated their response accordingly.

Description-based gambles task. All participants completed two sets of eight description-based gambles (Supplements, Table S1; cf. Mather et al., 2012; Rieskamp, 2008). On each trial, individuals were required to choose between a certain and a risky option presented as two boxes on the screen (Figure 1). The certain option either offered 4 or 8 CHF (Swiss francs, 1 CHF ~ 0.98 USD) with a probability of 1, whereas the risky option offered between 6 and 40 CHF with a specific probability and 0 otherwise. Half of all trials had a 50/50 chance associated with the risky option, and the other half of trials were associated with a 20/80 chance of winning money or not, respectively. The 50/50 and 20/80 trials were further divided into trials that yielded the same expected value (calculated as option outcome value multiplied by its probability, hereafter referred to as "equal EV trials") for the risky and the safe option, whereas the other half of trials yielded a higher expected value for the safe option (hereafter referred to as "unequal EV trials"). The two task conditions were introduced to

assess whether differential utility of risk taking as a function of EV (in)equality engenders age-related differences in risk-taking behavior. The order in which the eight gambles were presented was randomized across participants. Participants selected their preferred option by clicking on the respective box; option selection was self-paced. Each choice was then played out and the outcome saved on a temporary account (but not presented to participants, in order to avoid any sequence/learning effects). Participants obtained written instructions for the task and were provided with an independent (i.e., not included in the test set) example trial in order to become familiar with the task objective and response modus. Inspection of the data revealed that two participants did not complete two sets of eight unique gambles but instead one gamble was presented more than twice across the two sets. These two participants were excluded from analysis of the gambles task to ensure all participants had seen the same full set of gambles and had made decisions on the same eight unique lotteries.

Balloon Analogue Risk Task (BART). We employed a variant of the Balloon Analogue Risk Task (BART; Lejuez et al., 2002) in which participants could pump up balloons to gain monetary rewards. In each trial, participants could administer sequential pumps up to the point where the person decides to stop inflation or the balloon bursts (Figure 2). Each successful pump resulted in an increase of .02 CHF on a temporary account, and the total score of the current trial was saved and added to the final payoff if a person decided to stop pumping before an explosion. Otherwise, the temporary account was reset to 0. Participants completed 20 trials of two types of balloons differing in their color (red, blue) and pumping capacity (low vs. high; 32 vs. 16 maximum pumps), with assignment of balloon type and color being counterbalanced across participants. The two balloon conditions were introduced to vary learning demands. On inspection of the trial-by-trial data we discovered that a small number of participants completed more than 20 trials per balloon type (likely due to a lag in the Internet connection resulting in a few repeated trials; $n = 40$). The findings

below are based on analyses for which the maximum number of trials was restricted to the first 20 trials per balloon type; however, control analyses yielded the same results including the extra trials.

Demographics. Beyond the self-report item and behavioral tasks related to risk taking, we collected information on individuals' age, gender, number of children, marital status, work status, highest level of completed education, area of residence (postal code), as well as financial information regarding personal income, assets and debts. For the main analysis, we used age as a continuous independent variable (IV), participants' gender (male = 0, female = 1), whether they have children (0 = no, 1 = yes), their work status (0 = not employed, 1 = employed) as binary categorical IVs, and education (nine levels, reference = primary school) and marital status (eight levels, reference = married and living together) as categorical IVs on a nominal scale. Area of residence and financial information were not included in the current set of analyses because the former only served to warrant a sample of participants that characterizes the local population, and only a minority of participants provided responses to the latter.

Procedure

We employed an independent market research company to recruit and collect data from a quota-driven sample of participants in Basel, Switzerland. Six interviewers were trained on the study materials and provided with detailed instructions and study protocols. Recruitment of participants took place on the street in the city center and in a shopping mall, and included a brief introduction of the research as well as an initial screening consisting of individuals' postal code of their main residence, gender, and age, to confirm eligibility for participation with respect to the quota required for a fairly representative sample concerning residence, gender, and age. Successful recruitment on the street resulted in immediate data collection in a nearby hotel or coffee shop. Individuals recruited in the shopping center

completed the study in the shopping center. The setup was kept as similar as possible across the three test locations, with each location providing separate, closed off workstations with laptop computers and a mouse for participants to complete the study. Participation involved the same set of questions and tasks for all participants, with interviewers guiding individuals through the study on a one-to-one basis. The latter was thought to facilitate participation by older adults and improve data quality (i.e., reduce the number of missing items or incorrect data entry). On entering the test setting, participants' responses to the three screening questions were confirmed and recorded, followed by the provision of detailed information about the study's aims and content, and consent procedures. Subsequently, participants were presented with the self-report item assessing general risk taking, the two behavioral risk-taking tasks, and several questions concerning their demographics. The order of measurements was fixed (self-report, BART, gambles). All oral instructions and written materials were given in German. Data were recorded electronically using CAPI and entered either by the interviewer or the participants themselves (e.g., sensitive questions, responses during the two behavioral tasks). All earnings were paid to participants immediately after study completion.

Results

Overview of Statistical Analysis

Our goal was to examine the effect of age on risk taking across a variety of measures whilst controlling for demographic variables shown to be associated with risk taking, including gender, educational attainment, work status, marital status and parenthood (e.g., Baker & Maner, 2009; Byrnes, Miller, & Schafer, 1999; Dohmen et al., 2011; Rolison et al., 2013; Schurer, 2015; Wang, Kruger, & Wilke, 2009). Given the correlational nature of this research, multiple regression analyses were used to discern the relation between the continuous variable age and the three risk-taking measures (i.e., self-reported risk taking,

choices in gambles task, performance in BART). Sample-based descriptive statistics for all risk-taking measures used are given in Table 1.

Age-risk trajectories for self-reports were analyzed using a fixed effects regression model. We applied mixed-effects modeling to individuals' trial-by-trial performance in the BART (number of pumps) and the gambles task (choice of risky or safe option) in order to account for variance in the outcome measure that is not systematic across the group but instead originates from random (i.e., individual level) effects, such as effects of trial or condition, which may vary significantly across participants. For the gambles task, the binary choice outcome (risky or safe option) was regressed on age, gender, trial type (i.e., whether the trial was an equal or unequal EV trial), and two interaction terms (age and sex, age and trial type). The model also allowed for random effects of trial type, clustered within participants. For the BART, mixed-effects modeling included the following fixed effects: age, gender, balloon capacity (high or low), whether the previous trial resulted in an explosion (yes or no), the trial number (scaled separately for high and low-capacity balloons), and interaction terms for age and sex, age and balloon capacity, age and previous explosion trial, age and trial number, balloon capacity and trial number as well as a three-way interaction term between age, balloon capacity and trial number. We allowed for random effects for trial number, balloon capacity, previous explosions, and a trial number by capacity interaction, clustered within participants.

Analyses were carried out in the software R (R Core Team, 2014) using the packages lme4 (Bates, Maechler, Bolker, & Walker, 2015) and lmerTest (Kuznetsova, Brockhoff, & Christensen, 2015**)** for mixed-effects modeling of continuous (lmer) and binary (glmer) outcome variables. Calculation of the denominator degrees of freedom required to derive p-values for the fixed effects test statistics in lmerTest (Kuznetsova et al., 2015) uses Satterthwaite's approximation (based on SAS proc mixed theory). For all analyses reported,

continuous outcome variables were normalized prior to analysis; self-report scores were normalized across the entire sample, while individuals' number of pumps per trial in the BART were normalized separately for each balloon type. Continuous predictor variables (e.g. age) were normalized, and categorical variables dummy-coded. Reflecting previous findings that indicated the effect of age to take non-linear forms and follow different trajectories as a function of domain and/or gender (Josef et al., 2016; Rolison et al., 2013; Tymula et al., 2013), all initial analyses included linear, quadratic and cubic effects of age and gender as well as their respective interactions. Initial analyses yielded no support for the inclusion of quadratic or cubic terms in the mixed-effects models of individuals' trial-by-trial performance on the BART and the gambles, hence were dropped and only a linear term for age was included. Parameter estimates for the set of demographic variables (besides gender) entered into the modeling process in order to control for potentially confounding effects were not the focus of the current research, and are not discussed here.

All analyses were conducted using the entire age range (18 to 90) and compared with analyses including only individuals between 18 and 78 years of age to control for a marked reduction in data points from individuals between 79 and 90 years of age (i.e., fewer than five individuals for every year). The general pattern of results was robust against in/exclusion of individuals between 79 and 90 years of age (n=15); only the results obtained from analyses covering the entire age range are reported below.

Self-reported risk taking

Self-reported risk taking was negatively associated with age (β =-0.27, SE=0.1, p=.01) and gender (*b*=-0.34, SE=0.1, p<.001), where increasing age and being female were attributes associated with lower self-reported risk taking (Table 2; Figure 3, Panel A). In addition, a significant interaction between age (linear term) and gender emerged (*b*=0.29, SE=0.14, p=.04). Quadratic and cubic age terms as well as their respective interactions with

gender were not significant. Main effects for age and sex remained after removal of non-linear terms, and both main effects and the interaction effect remained significant after controlling for educational attainment, work status, marital status, and children; marital status emerged as significantly predictive of self-reported risk taking (Supplements Table S2). The results from the local survey are in line with previous research, which has indicated self-reported risk taking to decrease with age and to be lower for females than for males. Moreover, a very similar pattern—negative association between age and self-reported risk taking and a main effect of gender—was observed for fixed effects modeling using Swiss Household Panel data (see Supplements for further details), albeit suggesting a non-linear association between age and risk taking. Overall, the analysis suggests that our local sample captures the common findings regarding the patterns of age and sex on self-reported risk-taking propensity that generalize across the Swiss population.

Gambles

Sex and age were not significantly predictive of choosing the riskier option, but a main effect of trial type emerged (b=-0.52, SE=0.05, p<.001; Table 3). For an individual of mean age this effect translates into a 12% decrease in the probability of selecting the risky option, from 46% on equal EV trials to 34% on trials in which the higher expected value was associated with the safe option. Moreover, relative to younger participants, older participants selected the risky over the safe option more often in trials in which the safe option had a higher expected value than the risky option, as indicated by a significant age by trial type interaction (b=0.24, SE=0.04, p<.001; Table 3; Figure 3, Panels B and C). To put these estimates into context, whereas the probability of selecting the risky option in unequal EV trials (31%) was lower than the probability of selecting the risky option in equal EV (49%) trials for a younger individual (mean age minus 1 SD), this reduction was comparatively smaller for an older person (mean age plus 1 SD), from 43% in equal EV trials to 36% in

unequal EV trials. Note that Panels B and C in Figure 3 depict trial type-specific densities and coefficients, which were standardized separately for equal and unequal EV trials; consequently, the zero point representing the mean proportion of risky choice in equal and unequal EV trials is not the same in the two conditions (0.48 and 0.39, respectively). The results were robust against removal of interaction terms and inclusion of control variables. For the model containing demographic control variables, marital status emerged as an additional significant predictor of choices on the gambles task (Supplements Table S6). Compared with married respondents' choices in equal EV trials, individuals living in a civil partnership were more likely to choose the riskier option (b=1.50, SE=0.75, p<.05), whereas individuals in a civil partnership but living apart were less likely to select the risky option (b=-2.53, SE=1.28, p=.05).

In summary, patterns obtained from the gambles task highlight the importance of task characteristics for risk taking in the absence of main effects of age and gender. The interaction between trial type and age suggests that task demands differentially affect younger and older respondents and thereby influence their respective choices. In unequal EV trials the safe option offers a higher EV compared with the risky option. If individuals base their decision making on EV, it stands to reason that the safe option is the more attractive one, and therefore risk taking should decrease on unequal EV trials. The likelihood of selecting the risky option on unequal EV trials increased with age, however, suggesting that older adults may be more risk taking because they cannot (or prefer not to) adhere to EV calculation.

Balloon Analogue Risk Task

We used the average number of pumps in the BART (the same results hold for the "adjusted number of pumps", cf. Lejuez et al., 2002) as a measure of risk taking. The regression analysis revealed significant main effects for balloon capacity (b=-0.22, SE=0.03, p<.001), whether the previous trial was an explosion trial (b=-0.19, SE=0.01, p<.001), and

trial number (*b*=-0.11, SE=0.02, p<.001) on risk taking (Table 4). Moreover, several interaction effects were observed (Table 4), including interactions between age and balloon capacity (*b*=-0.1, SE=0.03, p<.01), age and previous explosion (*b*=0.03, SE=0.01, p<.01), as well as between balloon capacity and trial number (*b*=0.25, SE=0.03, p<.001). Specifically, high-capacity balloons were associated with more pumping compared with low-capacity balloons, whereas having experienced an explosion on the previous trial was associated with less pumping. Higher age was associated with significantly less pumping on high-capacity balloons (Figure 3, Panels D and E), but significantly more pumping on trials following explosion trials.

Across individuals, a learning effect was inferred from the main effect of trial number and an interaction between trial number and balloon capacity: contrary to low-capacity balloons for which pumping decreased over time, on high-capacity balloons the number of pumps administered increased with increasing number of trials (Supplements Figure S1). The results obtained from the mixed modeling of pumps on the BART were robust against controlling for demographic confounds (Supplements Table S7). Moreover, removal of the interaction terms preserved the main effect of balloon capacity, explosion trial and trial number, whilst age and sex remained not significantly predictive of pumping.

Overall, results from the BART support the idea that aging is associated with a reduction in risk taking. Notably, age differences were only visible in low risk (high capacity) trials, presumably because these trials facilitated elicitation of individual differences with respect to risk taking and cognitive capacity, and consequently increased variance in behavioral outcomes. Whilst learning was not directly assessed, differences in learning may account for the differences in risk taking between young and older adults in the low risk BART trials.

Convergent validity of risk measures across the life span

To address the question of whether different measurements paint the same picture regarding an individual's risk-taking profile, we computed zero-order correlations between self-report and task-derived risk-taking indices. Correlations were also obtained for scores derived from different conditions within a task (e.g., between number of pumps on high and low-capacity balloons in the BART) in order to ascertain whether different task components are equally sensitive and informative with respect to risk taking or whether, in fact, different task conditions yield different response patterns (for instance, due to adaptation to task demands). In order to examine whether the relation between measurements changes as a function of age, zero-order correlations between measures were also computed after splitting the data set into three age groups, comprising individuals aged between 18 and 39 (n=308), 40 and 59 (n=353), and 60 and 90 years (n=241).

The association between risk-taking scores stemming from different measurements across the whole sample were low (i.e., irrespective of age), and self-report was only weakly associated with behavioral measures, albeit more strongly with risk taking in the gambles task, $r \approx .2$, than in the BART, $r \approx .1$ (Figure 4a).

Turning to risk-taking propensity as measured with the two different behavioral measures, performance on the BART showed weak or no correlation with the proportion of risky choices on the gambles task. This pattern suggests that even within the same modality (i.e., behavioral paradigm with financial incentive structure), tasks are likely to tap into different aspects of risk taking and therefore cannot be taken as exchangeable indicators of risk-taking propensity (cf. Henninger et al., 2010). Correlations between pumping on high and low-capacity balloons on the BART were markedly lower than those observed for risky choices in the two gambles conditions (r=0.37, r=0.71, respectively), perhaps reflecting commonly observed floor effects on pumping behavior in the BART and specifically the low-

capacity balloon (e.g., Cavanagh et al., 2012; Henninger et al., 2010; Rolison et al., 2012; Schonberg et al., 2012).

Do the associations between measurements change as a function of age? We did not observe substantially different directions and magnitudes of associations between measurements for different age groups (Figures 4b-d) compared with those obtained for the whole sample. However, one interesting pattern emerged for the association between measures obtained from the same task. For the BART, correlations between pumping on the low and high-risk balloons increased from the youngest (r=0.18) to the middle-aged (r=0.43) to the oldest group (r=0.5), with the difference between younger and older adults' correlation coefficients being significant (z=3.95, p<.0001), suggesting that older individuals' pumping behavior was not as context-dependent as that of younger participants. A similar pattern was observed for proportion of risky choices in the two gambles conditions, where correlations increased from the youngest (r=0.49), to the middle-aged (r=0.69), and again to the oldest group (r=0.8); as for the BART, the difference between younger and older adults' correlation coefficients was significant (z=6.6, p<.0001). These results suggest that older adults were overall less adaptive in their risk-taking behavior as a function of task manipulations, perhaps signaling reduced cognitive plasticity.

Discussion

We investigated age-risk trajectories of three different measures of risk taking, namely a self-report and two different behavioral risk-taking measures using a large cross-sectional adult sample, and their convergent validity. The present findings paint a differentiated picture concerning the effect of age on risk taking. All three measures yielded *some* evidence for reduced risk taking with increased age: Relative to younger adults, older participants reported lower propensity for risk, pumped less in the BART in the low risk condition, and selected the risky gamble less often when the risky and safe option had the same expected value.

The pattern of declining self-reported risk taking across the adult human life span identified in our local and national sample falls in line with previous results (Dohmen et al., 2011; Josef et al., 2016; Mata et al., 2016; Rolison et al., 2013; Shao & Lee, 2014). In line with our hypothesis, manipulations within behavioral measures led to divergent age-risk trajectories, which suggest specific mechanisms are at play in engendering specific age differences in risky choice, such as the learning and information integration demands required to complete each task (Frey et al., 2015; Mata et al., 2011). In the BART, age differences presumably emerged as a function of learning demands, because older adults only seem to have pumped less relative to younger adults in trials for which a higher maximum balloon capacity may have facilitated the elicitation of individual differences. Previous work by Henninger and colleagues (2010) supports this notion, suggesting processing speed to mediate age-related deficiencies in decision quality in the BART (Henniger et al., 2010; Mata et al., 2011).

In the gambles task, we expected individuals overall to select the safe option more often than the risky option in equal EV trials. Although in the long run selection of one option confers no benefit over selection of the other in equal EV trials, for a limited number of trials selecting the safe option arguably leads to more advantageous outcomes and risky choices may be motivated by the utility of gambling itself. Our estimates confirm this expectation. Both younger and older adults selected the risky option less often in equal EV trials, yet older adults tipped the balance in favor of the safe option. These patterns are in line with previous results of increased risk aversion for risky versus sure gain gambles in older adults (cf. certainty effect, Mather et al., 2012). Interestingly, we observed the opposite pattern in unequal EV trials (i.e., when the safe option had a higher expected value compared to the riskier option). Here, increasing age was associated with increased risk taking, despite the relative disadvantage of choosing the risky over the safe option. We do not propose that

unequal EV trials triggered more risk-seeking in older adults, but instead we suggest that – comparable to age-risk trajectories for different conditions in the BART – older adults were less able to adapt to within-task demands and consequently experienced challenges to EV calculation and maximization.

Our results of an interaction between age and trial type in the gambles task are comparable to the results for risk-disadvantageous gain trials in the study by Weller and colleagues (2011), where a slight increase in disadvantageous risk taking by older adults was observed (cf. study by Tymula and colleagues (2013) for similar patterns of lower choice rationality in older adults). Moreover, the proportion of risky choices in unequal EV trials (current study) and risk-disadvantageous trials (Weller et al., 2011) made by younger and older adults was comparable (in both studies around 35% and 40%, respectively), despite the markedly lower number of trials completed by individuals in the current study. This suggests that age-related differences in individuals' adaptation to the dynamic utility of risk taking describe a replicable, if not robust pattern. Crucially, in the study by Weller and colleagues (2011) as well as the current study, younger and older adults appeared to adapt their choice of the risky option to the task condition, namely by evidencing a decreased proportion of risky choices for unequal EV / risk-disadvantageous gain trials compared with equal EV / risk-advantageous gain trials. Thus, whilst both younger and older adults in this study appear to reconcile changes in EV with lower risk taking, the slope was less negative for older compared with younger adults.

In sum, the patterns of age differences in behavioral tasks seem to suggest a decline in propensity for risk across the life span, albeit with strong dependence on specific task characteristics. To note, although in the BART aging was associated with overly risk-averse behavior that was not optimal in the task, in the gambles task, aging was associated with increased risk taking when this was disadvantageous (i.e., the risky option had a lower

expected value). The blatant difference in the ecological rationality of risk taking in these two task conditions illustrates the need to understand when age differences in decision-making preferences and strategies lead to good or poor choices.

Of further interest to the current study was the convergence between different measures adopted both within and across measurement traditions, specifically the extent to which they capture similar or dissimilar trajectories in risk taking. The current findings mirror significant yet weak correlations between self-report and behavioral measures obtained with cross-sectional life span (Josef et al., 2016), young adult (Mishra & Lalumière, 2011), case-control (Szrek, Chao, Ramlagan, & Peltzer, 2012) and cross-cultural (Vieider et al., 2013) samples, and are suggestive of risk-related attitudes and propensities as measured via self-report and behavioral paradigms to assess distinct facets of a complex construct. Questions pertaining to the mechanisms underlying convergence or divergence of risk-taking measures however remain open to investigation.

Previous findings speaking to the self-report item's heritability and test-retest reliability (Benjamin et al., 2012), stability (Josef et al., 2016) and correlation with real life behaviors (Dohmen et al., 2012) as well as more domain-specific risk taking measures (Dohmen et al., 2011; Vieider et al., 2015; Josef et al., 2016) suggest that this item does capture the phenotype of interest and renders the explanation of low correlations being driven by an excessively noisy self-report measure unlikely. Alternatively, we agree with the suggestion by Chuang and Schechter (2015) that self-report measures may simply be easier to understand than behavioral measures, which could account for the observed instability of risk-taking propensity elicited via experimental paradigms as opposed to the relative stability of attitudes assessed via surveys (Chuang & Schechter, 2015). One limitation of the current study is the absence of cognitive control variables (e.g., processing speed, working memory, comprehension, numeracy) required for pinpointing potential drivers of age-related

differences in risk taking for the measures utilized. Given the scope and logistics of our approach it was not feasible to include further measures, yet we strongly recommend prospective studies to incorporate cognitive capacity measures to exclude alternative interpretations of age-related differences for different risk taking measures.

Unfortunately, we were unable to extend our described gambles structure by losses and gamble pairs for which the risky option had the higher EV. Whilst losses seem to loom equally large for older adults, Weller and colleagues (2011) observed the proportion of disadvantageous risky choices in the loss domain to follow a similar trend to the gain domain, suggesting overall more disadvantageous risk taking by older than younger adults. Especially with respect to the convergent validity of risk propensity when losses are present (e.g. the BART or the possible loss-based interpretation of the self-reported risk taking item), correlations between measures may have been higher had we included mixed or loss only gambles.

On a related note, we cannot completely rule out the possibility of priming effects of responding to a self-report item on behavior due to the fixed order of measures presented to our participants. However, we would argue that the existence of a priming effect would artificially inflate the correlations observed, perhaps due to an anchoring bias; if this was the case, the *actual* correlations between self-report and behavioral measures would be even lower than observed here, yielding further support for our main results, namely low convergent validity between risk-taking measures. Moreover, if individuals established a 'risk' anchor from their self-report, the correlations between the two subsequent behavioral measures could expectedly be higher because the self-report primer should affect both tasks. Whilst we believe that a potential priming effect does not invalidate our conclusions, only a counterbalanced design can convincingly discount this possibility and further studies are required that directly examine the impact of measurement order on risk-taking indices. In

general, our results are in line with previous estimates of comparatively low convergence between self-report and behavioral measures (e.g., Josef et al., 2016), thus we submit that any biases due to priming do not substantially alter the main findings.

There are three novel aspects of our results that deserve consideration in future work. One aspect concerns younger and older individuals' adaptation to task characteristics on a trial-by-trial basis. Several motivational and affective frameworks have been formulated, according to which older individuals' differential use of emotion regulation strategies dampen the impact of negative affect (Mata & Hertwig, 2011; Mata et al., 2011; Samanez-Larkin et al., 2007). In our sample, increasing age was associated with the administration of fewer pumps on the high-capacity (i.e., low risk) balloon as well as lower sensitivity to explosion trials. It would be interesting to also further examine whether older adults are less sensitive to losses than younger individuals, or, alternatively, do not extract the same meaning from loss cues in the BART. Second, in the described gambles task, increasing age was associated with selecting the risky option more often in trials where the certain option had the higher expected value compared with equal expected value trials. Future work should try to capture the specific strategies that can account for such changes (Mata, Schooler, & Rieskamp, 2007). Finally, we explored the age differences in within-task correlations and found that older adults' risk-taking behavior was less sensitive to within-task manipulations. Future work could try to assess whether this lack of adaptivity is a marker for aging decision makers' overall reduced cognitive plasticity (Lövdén, Bäckman, Lindenberger, Schaefer, & Schmiedek, 2010).

In conclusion, different measures do not entirely succeed in capturing the same trajectories of risk-taking propensity across the life span, with the patterns of age reductions in risk taking being suggested to emerge as a function of learning and integration demands of specific measures. Our results show that in order to truly understand the life span

development of risk-taking propensity, research must take into account the convergent and predictive validity of self-report and behavioral measures of risk taking.

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Tables

Table 1. Descriptive statistics for self-reported and performance-based risk-taking measures (N=902).

Risk-taking measure	Range	Mean (SD)	Median	Mode
Self-reported risk taking	0 -10	6.08 (1.81)	6.00	5.00
Gambles: Proportion of risky choices (mean)	0.00 – 1.00	0.43 (0.28)	0.44	0.00
Gambles: Proportion of risky choices (equal EV trials)	0.00 – 1.00	0.48 (0.30)	0.50	0.50
Gambles: Proportion of risky choices (unequal EV trials)	0.00 – 1.00	0.39 (0.30)	0.38	0.00
BART: Number of pumps (mean)	3.12 – 12.70	8.70 (1.57)	8.95	9.70
BART: Number of pumps (low-capacity balloons)	3.10 – 8.05	6.50 (0.71)	6.55	6.40
BART: Number of pumps (high-capacity balloons)	3.15 – 17.50	10.90 (2.81)	11.20	13.45

Table 2. Model parameters for fixed effects modeling of self-reported risk taking (N=902). Self-report scores were normalized prior to analysis. Age (normalized) was entered as a continuous variable, while sex was entered as categorical (dummy coded) variable. Brackets refer to the level of the categorical variable for which the parameters were estimated, relative to the reference level. Reference levels are given below the table.

	b	***SE***	***t***	***p***
Intercept	0.16	0.07	2.24	**0.03**
Age	-0.27*	0.10	-2.64	**0.01**
Age^2	-0.03*	0.05	-0.56	0.57
Age^3	0.06*	0.04	1.47	0.14
Sex (female)[1]	-0.34	0.10	-3.58	**<0.001**
Age x Sex	0.29	0.14	2.07	**0.04**
Age^2 x Sex	0.09	0.07	1.32	0.19
Age^3 x Sex	-0.09	0.06	-1.47	0.14

Note: *b*=regression coefficient; *SE*=standard error; *t*=test statistic; *p*=significance level; *=standardized regression coefficient; [1]reference=male

Table 3. Model parameters for mixed-effects modeling of risky choices on a trial-by-trial basis on the gambles task (N=900). Shown are parameters for fixed effects only. Risky choice was modeled as a binary variable. Age (normalized) was entered as a continuous variable, while all other predictor variables were entered as categorical (dummy coded) variables. Brackets refer to the level of the categorical variable for which the parameters were estimated, relative to the reference level. Reference levels are given below the table.

	b	***SE***	***z***	***p***
Intercept	-0.22	0.09	-2.53	**0.01**
Age	-0.12	0.08	-1.50	0.13
Sex (female) [1]	0.11	0.11	1.02	0.31
Trial type (unequal EV) [2]	-0.52	0.05	-11.45	**<0.001**
Age x Sex	0.02	0.11	0.19	0.85
Age x Trial type	0.24	0.04	5.33	**<0.001**

Note: *b*=regression coefficient; *SE*=standard error; z=test statistic; *p*=significance

Table 4. Model parameters for mixed-effects modeling of the number of pumps administered on a trial-by-trial basis on the BART (N=902). Shown are parameters for fixed effects only. Prior to analysis, number of pumps was normalized (separately for the two balloon types). Age (normalized) and trial number (centered by balloon type) were entered as continuous variables; all other predictor variables were entered as categorical (dummy coded) variables. Brackets refer to the level of the categorical variable for which the parameters were estimated, relative to the reference level. Reference levels are given below the table.

	b	***SE***	***t***	***p***
Intercept	0.16	0.03	5.68	**<0.001**
Age	-0.002*	0.03	-0.07	0.95
Sex (female)[1]	-0.02	0.03	-0.84	0.4
Balloon capacity (high)[2]	-0.22	0.03	-6.72	**<0.001**
Explosion trial (yes)[3]	-0.19	0.01	-17.81	**<0.001**
Trial number	-0.11	0.02	-4.98	**<0.001**
Age x Sex	-0.001	0.03	-0.03	0.98
Age x Balloon capacity	-0.1	0.03	-3.08	**0.002**
Age x Explosion trial	0.03	0.01	2.82	**0.005**
Age x Trial number	-0.02	0.02	-1.06	0.29
Balloon capacity x Trial number	0.25	0.03	9.12	**<0.001**
Age x Balloon capacity x Trial number	-0.002	0.03	-0.08	0.93

Note: *b*=regression coefficient; *SE*=standard error; *t*=test statistic; *p*=significance level; *=standardized regression coefficient; [1]reference=male; [2]reference=low-capacity balloon; [3]reference=previous trial did not end in explosion.

Figures

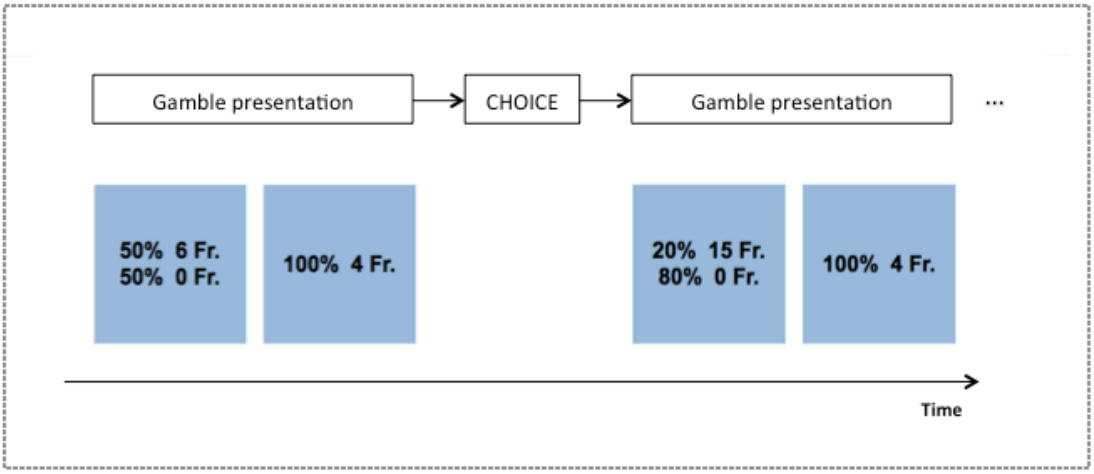

Figure 1. Example layout of the gambles task. The side of the screen on which the certain outcome was presented was counterbalanced between trials. Participants selected their preferred option by clicking on the associated box. Between trials individuals were informed that their choice was logged and that the preferred option would be entered into the set from which their payoff was to be drawn. A button click took participants to the trial.

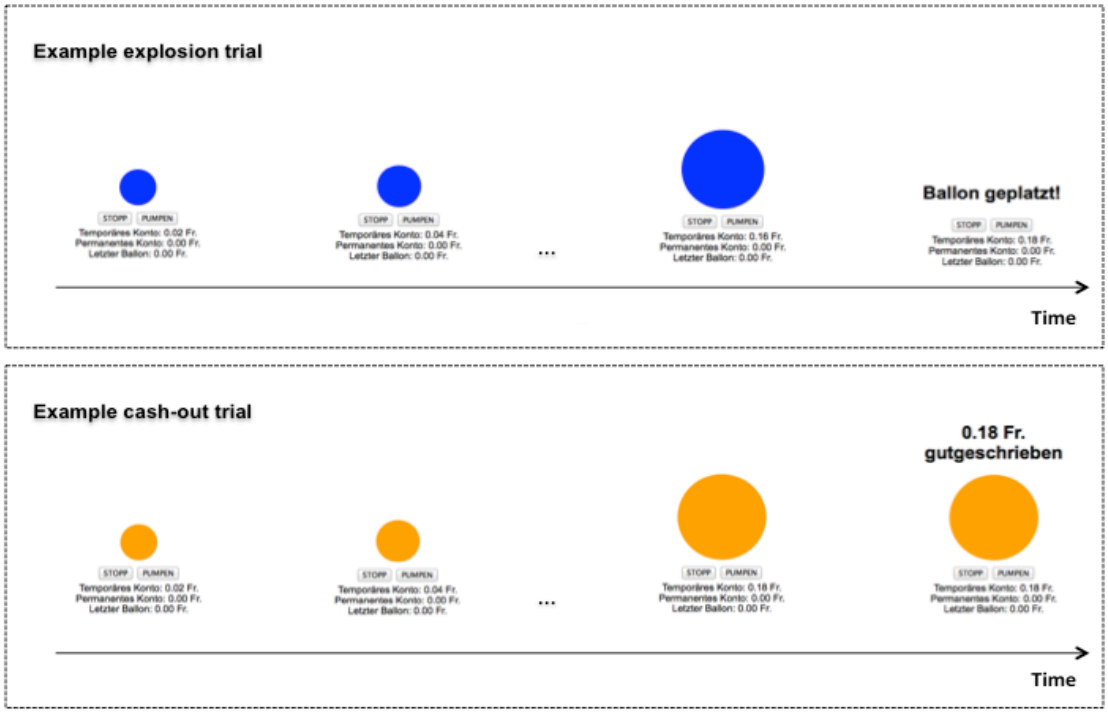

Figure 2. Example trials in the BART for an explosion trial, and a trial in which the participant cashed out. Underneath the balloon, the temporary account shows money earned on the current trial, the permanent account indicates how much had already been earned prior to the current trial. The last line indicates how much money was earned on the previous trial. For purposes of illustration, intermediate balloon inflations are not shown but would otherwise reflect a gradual increase of 0.02 CHF (Swiss Francs, 1 CHF ~ 0.98 USD) per additional pump. Color-capacity assignment was counterbalanced.

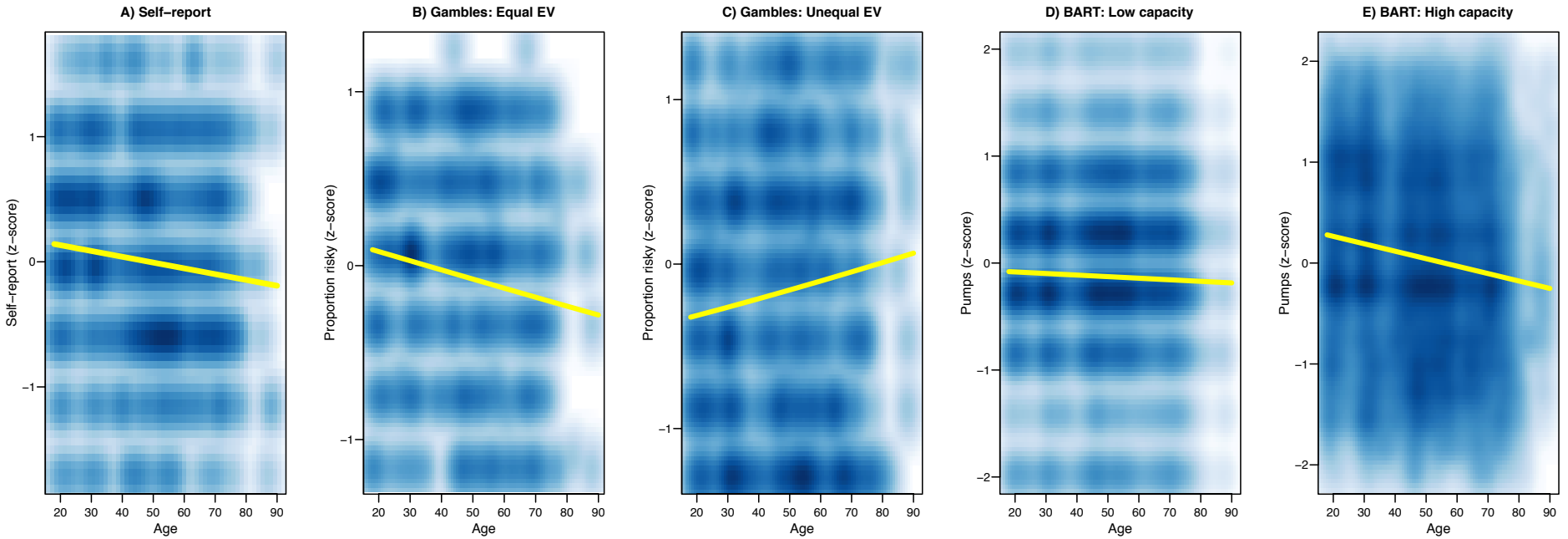

Figure 3. Cross-sectional age trajectories (18–90 years) for three risk-taking measures. Trajectories are plotted against kernel density maps of the distribution of risk-taking indices as a function of age. Darker (lighter) areas represent higher (lower) density of observed scores. For the purpose of illustration, outcome variables were standardized and plotted against raw age. Indices and distributions for the behavioral measures were normalized by condition; zero points represent condition rather than task-specific means (thus are not the same). Panel A) Trajectory for the self-report measure based on coefficients obtained from a model that includes linear terms for age, gender and their interaction (model R^2=0.03, p<.0001; N=902). B and C) Trajectories for description-based risky choices in (B) equal expected value (EV safe = EV risky), and (C) unequal expected value (EV safe > EV risky) trials; trajectories are based on model coefficients obtained from the full model (N=900; Table 3). D and E) Trajectories for experience-based pumping behavior in the BART for (D) low and (E) high capacity trials; trajectories are based on model coefficients obtained from the full model (N=902; Table 4). Accounting for the learning effect, pump trajectories refer to the last trial.

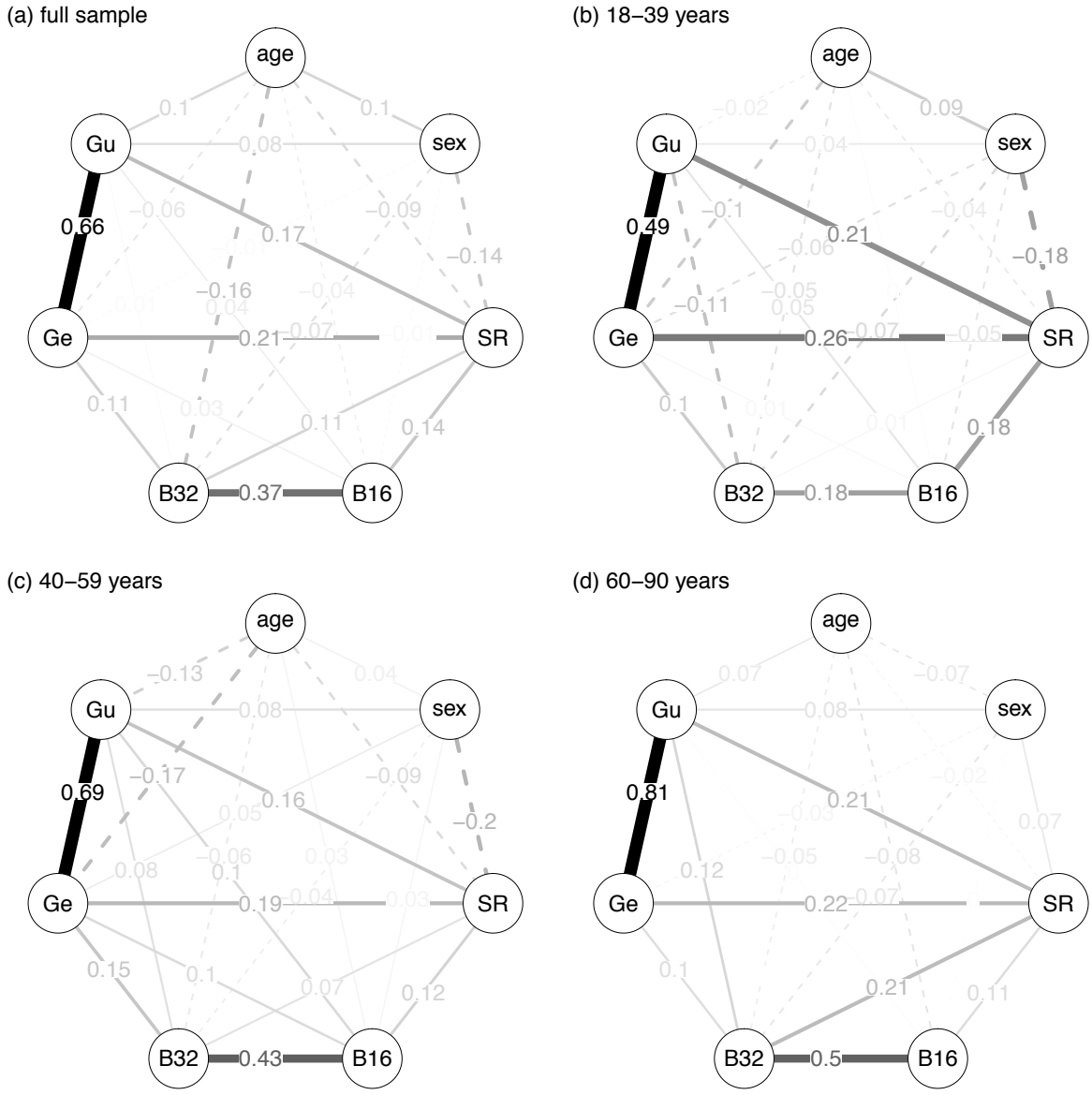

Figure 4. Zero-order correlation plots for risk-taking measures used in this study. Line thickness represents the strength of the correlation coefficients. Straight (dotted) lines represent positive (negative) correlations. Plots present correlations for risk-taking measures for (a) all participants in the study (18–90 years, N=902), (b) individuals between 18 and 39 years of age (N=308), (c) individuals between 40 and 59 years of age (N=353), and (d) individuals between 60 and 90 years of age (N=241). The position and equal spacing between factors is for visual purposes only and not indicative of correlation strength. Associations are plotted for self-reported risk taking propensity (SR), pumping on low (B16) and high-capacity (B32) balloons in the BART, and proportion of risky choices in the gambles task for equal (Ge) and unequal expected value trials (Gu).

APPENDIX B: Manuscript Two

frontiers in Aging Neuroscience

ORIGINAL RESEARCH
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Altered Value Coding in the Ventromedial Prefrontal Cortex in Healthy Older Adults

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Previous work suggests that aging is associated with changes in risk taking but less is known about their underlying neural basis, such as the potential age differences in the neural processing of value and risk. The goal of the present study was to investigate adult age differences in functional neural responses in a naturalistic risk-taking task. Twenty-six young adults and 27 healthy older adults completed the Balloon Analogue Risk Task while undergoing functional magnetic resonance imaging. Young and older adults showed similar overt risk-taking behavior. Group comparison of neural activity in response to risky vs. control stimuli revealed similar patterns of activation in the bilateral striatum, anterior insula (AI) and ventromedial prefrontal cortex (vmPFC). Group comparison of parametrically modulated activity in response to continued pumping similarly revealed comparable results for both age groups in the AI and, potentially, the striatum, yet differences emerged for regional activity in the vmPFC. At whole brain level, insular, striatal and vmPFC activation was predictive of behavioral risk taking for young but not older adults. The current results are interpreted and discussed as preserved neural tracking of risk and reward in the AI and striatum, respectively, but altered value coding in the vmPFC in the two age groups. The latter finding points toward older adults exhibiting differential vmPFC-related integration and value coding. Furthermore, neural activation holds differential predictive validity for behavioral risk taking in young and older adults.

Keywords: aging, decision making, ventromedial prefrontal cortex, anterior insula, Balloon Analogue Risk Task

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INTRODUCTION

Aging is associated with changes in cognition, emotion, and motivation that have important consequences for decision making (Tymula et al., 2013; Samanez-Larkin and Knutson, 2015; Schiebener and Brand, 2015). For example, recent meta-analyses suggest that aging is associated with changes in a variety of risky choice tasks (Mata et al., 2011; Best and Charness, 2015). But what are the potential mechanisms underlying age-related changes in dealing with risk and uncertainty? In our work, we aim to contribute to the understanding of possible mechanisms underlying age differences in risk taking by investigating young and older adults' neural activations associated with a well-known risk-taking task, the Balloon Analogue Risk Task (BART; Lejuez et al., 2002). The

BART is a popular and useful tool for measuring cognitive and affective mechanisms underlying risk-taking behavior (Lejuez et al., 2002; Schonberg et al., 2012), thus also representing a promising tool to uncover sources of age differences in cognitive and motivational components on decision making.

Participants in the BART are asked to pump up a balloon as much as they like, which, although leading to increased accumulation of (monetary) gains with each pump, simultaneously increases the probability of the balloon exploding (Lejuez et al., 2002). Thus, risk on the BART refers to the probability of an explosion resulting in the loss of all accumulated gains in a trial. The structure of the task captures not only participants' valuation of possible gains and losses simultaneously but also affective processes that could arise as a consequence of the increasing tension and uncertainty associated with additional pumps on a given balloon. As such, the BART mimics the risk–reward trade-off as well as the sequential process that characterizes decisions in many natural environments (Schonberg et al., 2011; Pleskac and Hertwig, 2014). Importantly, the task may have some predictive validity for real-life impulsive or risk-taking behavior, such as drug use, delinquency, gambling, and risky sexual behaviors (Lejuez et al., 2003, 2004; Aklin et al., 2005; Hunt et al., 2005).

The BART has found wide application in the field of behavioral as well as neural research, yielding a backdrop of findings for the current work. Specifically, previous neuroimaging studies have identified a set of key brain regions as being differentially involved in this task, including the ventromedial prefrontal cortex (vmPFC), dorsal lateral prefrontal cortex (dlPFC), anterior cingulate cortex (ACC), anterior insula (AI), striatum, and the midbrain (Rao et al., 2008, 2014; Chiu et al., 2012; Lighthall et al., 2012; Schonberg et al., 2012; Kohno et al., 2013; Telzer et al., 2013; Helfinstein et al., 2014). All of these areas have been implicated—in some form or another and with more or less specificity—in the construction, representation and storage of subjective value (for reviews, see Glimcher, 2010; Levy and Glimcher, 2012; Bartra et al., 2013; Clithero and Rangel, 2014). Specifically, although striatal and frontal activation patterns are widely recognized as key regions for value-based judgment and decision making, insular activation appears to be more prevalent for paradigms in which decision making extends beyond purely deliberative and into affective processing, including loss anticipation and harm avoidance (Paulus et al., 2003; Knutson and Bossaerts, 2007; Preuschoff et al., 2008; Mohr et al., 2010a; Bartra et al., 2013). Further work relevant to risk taking on the BART pertains to the neural correlates of uncertainty, with previous work implicating the right AI in the tracking of uncertainty (e.g., Volz et al., 2003). However, considering that uncertainty often implies the possibility of loss or harm, it is somewhat unclear whether the covariation between insular activity and uncertainty reflects the tracking of the abstract (mathematical) or affective component of uncertainty.

Of particular interest to this study are previous results obtained with the BART that identified decreasing vmPFC activation as a neural correlate of risk taking (Schonberg et al., 2012; Rao et al., 2014). For several decision-making tasks, vmPFC activity has been implicated in the representation of subjective value; that is, representing a signal that reflects the outcome of an integration of reward, risk (uncertainty), and potentially also affective evaluation (Kim et al., 2010; Rangel and Hare, 2010; Rushworth et al., 2011; Levy and Glimcher, 2012; Bartra et al., 2013). Some have proposed that the vmPFC is a critical substrate for information integration which triggers secondary emotional responses that help guide advantageous decision-making (Bechara and Damasio, 2005; Levin et al., 2012). Considering these previous studies and theoretical models of decision making, vmPFC-related activation in the BART could be representative of an integrative function of the vmPFC, coding the decreasing subjective value of additional pumping over time by integrating the potential gains with the increasing probability of loss (i.e., explosion). Taken together, the properties of the BART that make it a comparatively valid behavioral measure of risk taking—where risk is understood not only as outcome variability but also as exposure to potential loss—are mirrored in neural activity patterns. Previous work that adopted the BART in conjunction with findings from other paradigms provide some insight into the possible functional roles of different neural regions on the BART, including the coding of loss, reward, uncertainty, and integrated (subjective) value, each of which could be affected by cognitive and neural changes due to aging.

With regards to the computational drivers of age-related behavioral and neural differences in risk taking, it has been proposed that aging may be associated with difficulties in learning or representing the subjective (integrated) value of options, which can conceptually be thought of as arising from noisy representations due to low signal-to-noise ratio of information processing (Li and Rieckmann, 2014). For example, older adults typically show difficulties in learning the utility of options from probabilistic feedback, possibly due to age-related declines in neuromodulator systems that help form value representations (Li et al., 2007; Mohr et al., 2010b; Eppinger et al., 2011; Chowdhury et al., 2013). In one study, Samanez-Larkin et al. (2014) showed age-related reduction in the frontal representation of reward prediction error for paradigms involving feedback-based learning, but no such differences for the representation of reward magnitude. Moreover, several studies have shown differences in vmPFC-related reward and value signals as a function of age (Baena et al., 2010; Mohr et al., 2010b; Eppinger et al., 2013; Halfmann et al., 2016), leading to the suggestion that increasing variability in vmPFC signaling accounts for differences in performance (Rogalsky et al., 2012; Halfmann et al., 2016). The notion of increasingly varied neural responses, both with regards to inter-individual and intra-individual variability, is not limited to the vmPFC and related functions, but has already been found in other neural areas implicated in decision-making processes aversively affected by age (Li et al., 2007; Samanez-Larkin et al., 2010). Moreover, affective changes over the human lifespan may impact on decisions under risk, both behaviorally (Huang et al., 2013; Shao and Lee, 2014) and neurally (Shao and Lee, 2014). Taken together, multiple pathways are implicated in accounting for age-related changes in decision making (under risk), and several—such as altered information integration, feedback-based

learning, or changes in affective responses to stimuli, choices and their outcomes—could play a role in leading to age differences in the BART. A few behavioral studies have used the BART to investigate adult age differences in risk taking. However, the results of extant comparisons of young and older adults using the BART are inconsistent; although two found that older adults were somewhat less risk-seeking relative to young adults (Henninger et al., 2010; Rolison et al., 2012), another found the opposite (Cavanagh et al., 2012). Gaining a better understanding of the different neural components underlying age differences in the BART could be helpful in predicting when young and older adults differ in risk taking.

The goal of the present study was to investigate adult age differences in neural signals of risky decision making on the BART, a paradigm that captures the perceptible escalating tension between risk and reward not evident in other paradigms (e.g., described lotteries). Thus, we were interested in using the BART to compare young and older adults' neural signatures of risky decision making and establish whether differences arise in areas previously implicated in processes subsumed in the concept of risk taking, specifically the notion of harm avoidance and tracking of potential losses in the insula and the representation of utility (i.e., value) in the vmPFC. Moreover, we were particularly interested in assessing whether signals originating in the insular cortex or the vmPFC are similarly predictive of individual differences in behavioral outcomes (i.e., risky choices). We thus hoped to contribute to the challenge of uncovering possible age differences in decision making under risk, and eventually the dissociation of drivers of age-related differences such as the processing of reward, risk and subjective value.

MATERIALS AND METHODS

Participants

Twenty-six young and 27 older adults were recruited for the present study. Young adults were students of Southwest University, China, and older adults were recruited from communities in or near Southwest University. One young and three older adults were excluded due to excessive head movement during scanning (see below for exclusion procedure). In addition, one older adult was excluded for cashing out all reward balloons after just one pump. Forty-eight healthy right-handed participants were included in the final analyses, 25 young adults (11 male, mean age: 21.0 $\pm$ 1.6 years, age range: 18–24 years) and 23 older adults (eight male, mean age: 65.3 $\pm$ 5.3 years, age range: 60–79 years). Participants had no prior history of stroke, neurological or psychiatric disorder, and all older participants were independent community-dwelling adults whose Mini-Mental State Examination (MMSE; Folstein et al., 1975) scores were above 26 (mean score: 29.2 $\pm$ 1.2). Participants received 60 CNY (ca. 10 USD) for participation in the study, with the opportunity to earn up to an additional 15 CNY (ca. 2.5 USD) based on performance in the decision task. All participants provided written informed consent and the study was approved by the Institutional Review Board of the Brain Imaging Center, Southwest University, China.

Materials and Procedures

Participants completed a variant of the BART inside the MRI scanner (for further information on previous uses of the BART, see Lejuez et al., 2002; Schonberg et al., 2012). Prior to entering the scanner, participants were given instructions and completed a short practice trial. They were told that their goal was to maximize their scores in the task to increase their final payment. Participants could inflate a balloon on each of a number of trials by pressing a "pump" button. Each pump could earn participants 0.1 CNY (ca.0.02 USD); however, if the balloon exploded, they would lose the money accumulated in that trial. In order to avoid the explosion, participants could "cashout" the money at any point and secure their money by adding it to the "bank." There were three balloon types in the task, two reward balloons and one control balloon. Control balloons were gray balloons, which did not explode but also had no monetary value. Participants were simply asked to pump up the gray balloons until they disappeared from the screen. The two reward balloons could lead to monetary gains but differed in the maximum number of pumps that they could receive, thus creating a distinction between high- and low-capacity balloons. We used the two balloons as proxies for low- and high-risk conditions in contrast to no risk for the control balloon in order to examine whether behavioral and/or neural differences would emerge as a function of risk level and also whether this effect would be subject to age differences. Participants were not provided with any information about the differences between high- and low-capacity balloons but could in principle keep track of the two different types because they were assigned a different color, red or blue, with color assigned to each balloon type being counterbalanced between participants. The probability of the balloons exploding (or disappearing from the screen, in the case of the control balloons) was $p(\text{explosion}) = 1/(\textit{maximum-pumps})$, with a maximum of 12, 20, 16 for the low-capacity, high-capacity, and control balloons, respectively. The order of presentation of the balloons was randomized. The task was self-paced, therefore the number of balloons varied between participants in the fixed-duration 10-min scanning run. The interval between pumps varied randomly between 1 and 2 s, and the interval between trials (balloons) varied between 1 and 12 s, with a mean of 4.5 s.

Behavioral Analysis

We calculated the average number of pumps for cashout balloons (i.e., adjusted pumps), as is typically done in the BART literature in order to limit analyses to balloons for which the final number of pumps was not capped by an explosion (Lejuez et al., 2002). We also calculated the average number of reward trials, proportion of cashout trials, and average reaction time for each pump. We performed a 2 (age: young vs. older) $\times$ 2 (balloon: high- vs. low-capacity) repeated measures ANOVA on adjusted pumps, and conducted one-way ANOVAs on the number of reward balloons, proportion of cashout trials, and mean reaction time to estimate age differences. Statistical analyses of behavioral data were performed using SPSS 20.0 (IBM Corporation, Somers, NY, USA).

Image Acquisition

Participants were scanned at the Brain Imaging Center in Southwest University using a 3.0 T Siemens Tim Trio MRI system (Erlangen, Germany). For each participant, functional echo planar image data were collected using the following parameters: time repetition (TR) = 2000 ms, time echo (TE) = 30 ms, flip angle = 90°, field of view (FOV) = 200 mm × 200 mm, 33 axial slices, slice thickness = 3.0 mm, gap = 0.6 mm, acquisition matrix = 64 × 64, in-plane resolution = 3.125 × 3.125, and 200 volumes. High-resolution, three-dimensional T1-weighted structural images were acquired for each participant, with the following parameters: 176 slices, acquisition matrix = 256 × 256, voxel size = 1 mm × 1 mm × 1 mm, TR = 1900 ms, TE = 2.2 ms, and flip angle = 9°.

Image Preprocessing

Data preprocessing was performed using the Statistical Parametric Mapping program[1] (SPM8). First, the difference in acquisition time between slices was corrected, followed by a rigid-body correction for head motion. Participants included in the present study had less than 3.0 mm maximum translation and 3.0° rotation head motion throughout the scan. For normalization, we used a study-specific template created using unified segmentation and diffeomorphic image registration (DARTEL, Diffeomorphic Anatomical Registration using Exponential Lie Algebra; Ashburner, 2007). First, each subject's image was segmented into gray matter, white matter, and cerebral spinal fluid probabilistic images. The segmented gray-matter images were then normalized to Montreal Neurological Institute (MNI) space as defined by SPM8. DARTEL represents better localization of functional magnetic resonance imaging (fMRI) activity than does the optimized normalization procedure, by treating the brain template as a deformable probability density map, comparing the signal intensities of each voxel for every brain (Leshikar and Duarte, 2014). The resulting normalized images were then spatially smoothed using a 6 mm full-width half-maximum (FWHM) kernel to decrease spatial noise.

fMRI Analysis

Analysis of the functional MRI data was carried out in three steps. First, neural activity was modeled using the general linear model in a similar fashion to previous studies (Schonberg et al., 2012) with a high-pass filter of 1/128 Hz. In the general linear model analysis, two regressors for pumps were included: (1) $\text{Pumps}_{\text{Average}}$, capturing average activity across all pumps, and (2) $\text{Pumps}_{\text{Parametric}}$, capturing parametrically modulated activity by sequentially increasing the number of pumps within each trial. These two regressors were also implemented for the control balloons ($\text{Control}_{\text{Average}}$ and $\text{Control}_{\text{Parametric}}$). Because we found no significant differences between the activities elicited by the low- and high-capacity balloons, the two experimental balloon types were collapsed and a single regressor was used to model both types of trials. In order to remove visual and motor effects unrelated to risk and reward processing, we contrasted the reward pumps to those in the control condition ($\text{Pumps}_{\text{Average}}$ vs. $\text{Control}_{\text{Average}}$ and $\text{Pumps}_{\text{Parametric}}$ vs. $\text{Control}_{\text{Parametric}}$). To control for the potential confounding effects of head movement, six motion parameters (three translation and three rotation parameters) were entered into the GLM as regressors of no interest. The resulting activation patterns were labeled positive effects for a BOLD signal that was higher for reward than for control balloons when contrasted, whereas higher BOLD for control vs. reward balloons was taken to indicate a negative effect. Two-sample *t*-tests were computed to determine age group differences, specifically to examine BOLD signal differences between groups in each contrast to observe the influence of age on neural activity related to risk taking. Moreover, in order to illustrate the age differences on "$\text{Pumps}_{\text{Average}}$ vs. $\text{Control}_{\text{Average}}$" contrast more clearly, we conducted the two-sample *t*-tests masked by a positive effect map and a negative effect map, respectively, to observe the age differences on the positive effect regions and negative effect regions separately. The positive effect mask is a binary mask, which was generated from the combination of young and older age groups' positive effect map on "$\text{Pumps}_{\text{Average}}$ vs. $\text{Control}_{\text{Average}}$" contrast after correction, and the negative effect mask was generated likewise.

Whole-brain regression analyses were performed in order to identify brain regions that correlated with participants' risk-taking behavior. We examined the correlation between each individual's neural activity during $\text{Pumps}_{\text{Parametric}}$ vs. $\text{Control}_{\text{Parametric}}$ and his/her mean number of adjusted pumps. The individual difference analysis in the form of whole brain regression was conducted both across groups and by age group.

In addition to the whole-brain regression analysis, region of interest (ROI) analyses were adopted; these allowed us to test for the neural–behavioral association in specific brain regions that might not have been captured after correcting for multiple comparisons at whole-brain level. ROIs were created as 4 mm radius spherical regions covering the bilateral AI and striatum, respectively, and an 8 mm radius spherical region in the vmPFC. The center coordinates for the ROI masks (vmPFC [2 46 −8]; left AI [−36 20 −6]; right AI [40 22 −6]; left striatum [−12 4 2]; right striatum [12 10 −2]) were defined based on a recent meta-analysis examining neural correlates of subjective value (Bartra et al., 2013). In addition to using published coordinates to build ROI masks, center coordinates derived from the current sample (peak coordinate from $\text{Pumps}_{\text{Parametric}}$ vs. $\text{Control}_{\text{Parametric}}$ across age groups, vmPFC [−15 39 −12]; left AI [−33 24 3]; right AI [39 21 6]; left caudate [−12 6 9]; right caudate [9 3 9]) were used in secondary analyses aimed at testing the reliability of the results. These supplementary analyses also included spheres of different sizes, with sphere radii ranging from 3 to 10 mm, covering 1-mm increments between the lower and upper bound. Pearson's correlation analysis was performed to evaluate the relation between activation in the bilateral AI and the vmPFC (activation from parametric contrast on increasing number of pumps) and an individual's behavioral performance (i.e., mean adjusted pumps).

Functional magnetic resonance imaging analyses were examined at a threshold corrected for multiple comparisons (corrected by the false discovery rate, FDR, $p < 0.05$). All coordinates are reported in MNI format. Anatomical labels

[1] http://www.fil.ion.ucl.ac.uk/spm

of neural regions were obtained by importing the resulting statistical parametric maps into xjview[2].

RESULTS

Behavioral Results

Table 1 presents the average adjusted pumps, proportion of cashout trials, and other BART variables separately for young and older adults. We also plotted the performance as a function of adjusted number of pumps for each participant in the two reward balloons (**Figure 1**). For individuals' distribution of pumps for low- and high-risk balloons, see the (Supplementary Figure S1). As expected, participants behaved adaptively by pumping more in the high-capacity relative to the low-capacity balloon but most participants showed risk-averse behavior in the sense of pumping less than the expected value maximizing amount. Concerning age differences, as can be seen in **Table 1** and **Figure 1**, older adults were more likely to cash their earnings relative to young adults, yet this tendency did not translate into a significantly lower number of pumps or earnings for either balloon type. A 2 (age: young vs. older) × 2 (balloon: high-capacity vs. low-capacity) mixed-model ANOVA on adjusted pumps did not find age differences [$F(1,46) = 0.82$, $p = 0.371$] but a significant effect of balloon [$F(1,46) = 8.17$, $p < 0.01$] with more pumps being observed for the high-capacity relative to the low-capacity balloon. The interaction between age and balloon type was also not significant [$F(1,46) = 0.01$, $p = 0.944$]. These results suggest that both young and older participants learned to differentiate between the two balloons despite not having been explicitly informed about the differences. Moreover, average reaction times were larger for older adults (**Table 1**).

In sum, although young and older adults did not differ in average adjusted pumps, older adults had more cashout trials than young adults, possibly indicating more risk-averse behavior in older relative to young participants. We now turn to the issue of potential age differences in neural activations in the BART.

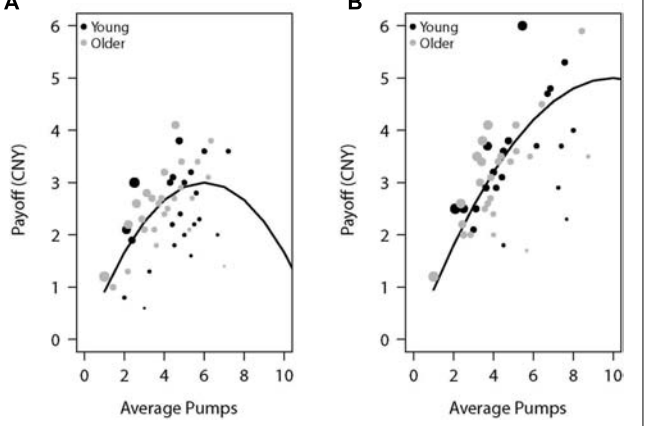

FIGURE 1 | Payoff as a function of average pumps for the (A) low-capacity and (B) high-capacity reward balloons. The lines represent the expected value of the specific average pumps across 10 trials of each balloon type (the average number of trials experienced by participants). Each dot represents a participant, with its diameter being a function of the proportion of cashout trials.

fMRI Results

In what follows, we present three sets of fMRI analyses. First, we report comparisons between average neural activity associated with pumping on experimental (i.e., balloons that were associated with monetary gains/losses) relative to control balloons (i.e., balloons that were not associated with any monetary gains/losses) for young and older adults, as well as any differences between the two groups. This comparison allowed us to capture reward/loss processes and age differences therein while subtracting activation due to attentional or motor processes that were of no interest to the current research. Second, we report parametric analyses of the neural activity of experimental relative to control balloons as a function of the number of pumps administered on a given trial. The rationale for this second set of analyses is similar to the one above but the pump-by-pump analysis provides a window into the processing of risk and reward as it unfolds over the course of a single trial. Finally, we report individual difference analyses that link neural activation of specific regions of interest to behavioral levels of risk taking. These latter analyses clarify the functional role of specific neural activations and whether these are differentially informative regarding individual and age differences in risk-taking behavior.

Neural Activity: Average Effects

A whole-brain contrast revealed widespread neural activity for the reward vs. control pumps contrast. Specifically, both young and older adults displayed positive effects (i.e., $\text{Pumps}_{\text{Average}} > \text{Control}_{\text{Average}}$) in the bilateral AI, striatum (caudate and putamen), dorsal ACC, superior frontal cortex and the visual cortex (**Figure 2A**, Red; **Tables 2** and **3**, Average: $\text{Pumps}_{\text{Average}} > \text{Control}_{\text{Average}}$). These areas have been identified in previous studies of the BART (Rao et al., 2008; Schonberg et al., 2012) and similar decision tasks (Mohr et al., 2010a; Wu

[2]http://www.alivelearn.net/xjview8

TABLE 1 | Behavioral results in young and older adults Groups (M ± SD).

Outcome	Young adults	Older adults	*F*	*p*
Mean adjusted pumps	4.82 ± 1.55	4.43 ± 1.56	0.76	0.388
Number of reward balloons experienced	20.92 ± 2.41	19.65 ± 3.24	2.39	0.129
Proportion of cashout trials	0.61 ± 0.12	0.70 ± 0.13	7.38	<0.010
Mean pump RT (ms)	521.10 ± 88.70	815.13 ± 341.18	17.33	<0.001

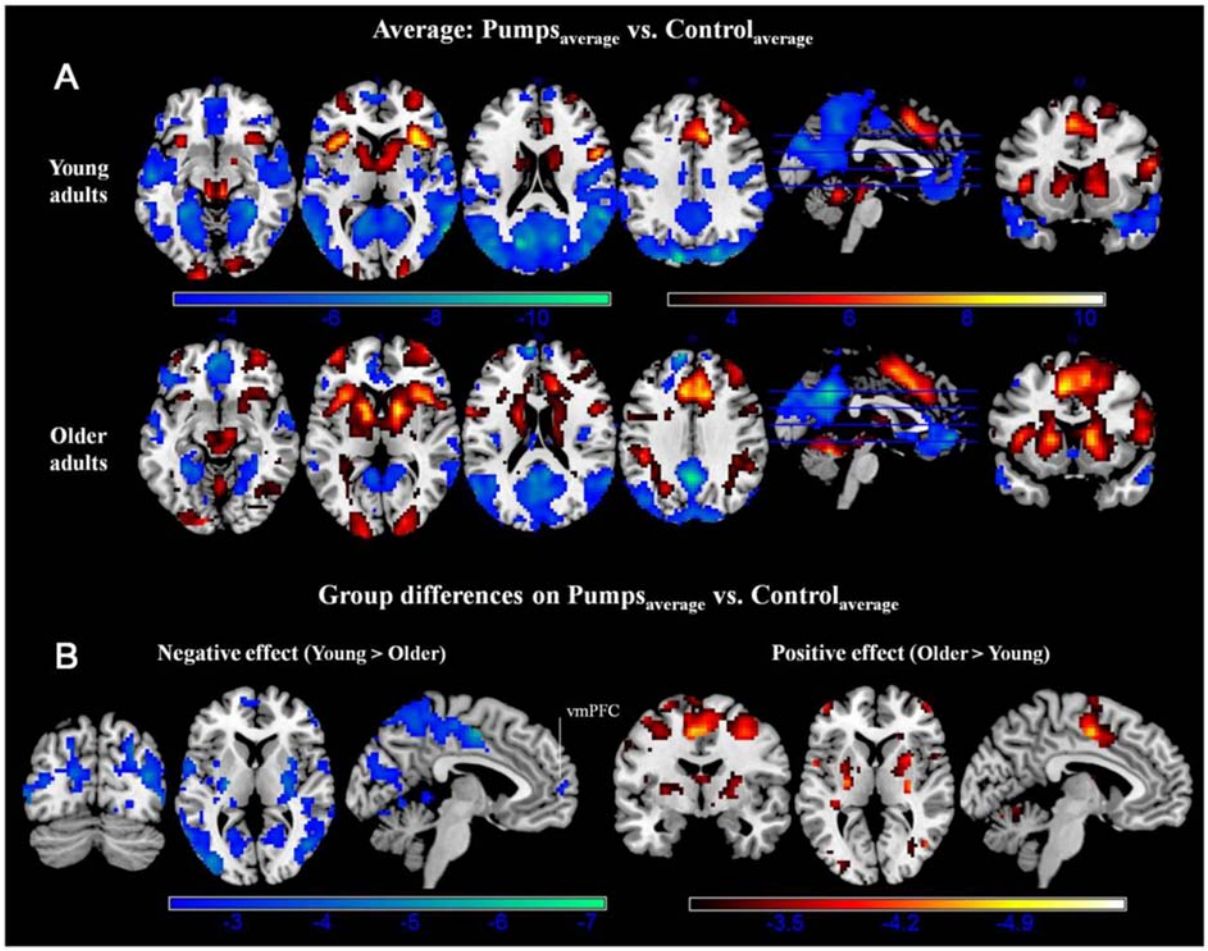

FIGURE 2 | (A) Average activity during pumps in the young and older age group. The red scale represents $Pumps_{Average}$ > $Control_{Average}$ and the blue scale represents $Control_{Average}$ > $Pumps_{Average}$. **(B)** Age group differences for average neural activity during pumps. Blue patterns represent neural regions in which negative effects were larger for the young compared with the older age group. Red patterns represent neural regions in which the positive effect was larger for older than for young adults, $p < 0.05$, corrected (scale represents the range of *t*-values). No brain regions were discovered for which (1) young adults had larger positive effects relative to older adults or (2) older adults had larger negative effects than young adults.

et al., 2012; Bartra et al., 2013) as being related to reward and risk processing. Moreover, both age groups displayed negative effects (i.e., $Control_{Average}$ > $Pumps_{Average}$) in the inferior frontal gyrus, middle temporal gyrus, precuneus, and the vmPFC (**Figure 2A**, Blue; **Tables 2** and **3,** Average: $Control_{Average}$ > $Pumps_{Average}$). In particular, activity in the vmPFC has been shown to correlate with valuation in various decision-making tasks (Levy and Glimcher, 2012; Bartra et al., 2013), including the BART (Schonberg et al., 2012; Rao et al., 2014).

Age group difference analyses showed that young adults had more activation than older adults in the postcentral gyrus, superior temporal gyrus, middle frontal gyrus, and medial frontal gyrus, whereas no regions were obtained for which older adults had more activation. To further distinguish these age differences, we performed group difference analyses masked separately by positive and negative effect maps. Young adults showed more negative effects (i.e., $Control_{Average}$ > $Pumps_{Average}$) than older adults in the fusiform, bilateral middle occipital lobe, precentral/postcentral gyrus, and a minor positive difference in vmPFC (**Figure 2B**, Blue); no regions were obtained for which older adults had more negative effects than young adults. For positive effects (i.e., $Pumps_{Average}$ > $Control_{Average}$), we found that older adults showed higher activation in the middle frontal gyrus, inferior parietal lobule, middle temporal gyrus, putamen, middle occipital gyrus, and supplementary motor area (SMA) (**Figure 2B**, Red); no regions were observed which evidenced higher activation in young compared with older adults.

Neural Activity: Parametric Effects

We aimed to capture the dynamic nature of risk processing in the BART by estimating the parametric modulation of BOLD responses as a function of the sequentially increasing pumps on reward vs. control balloons (see Schonberg et al., 2012, for a similar analysis). The parametric analysis yielded less widespread neural activity compared to the average pumps

TABLE 2 | Significant clusters of activation in young adults.

Region	L/R/B	*X*	*Y*	*Z*	*t*-value	Cluster size
Average						
$Pumps_{Average}$ > $Control_{Average}$						
Insula	R	39	18	3	10.28	109
Insula	L	−33	21	3	8.66	142
Superior frontal gyrus	R	30	57	15	7.12	143
Middle frontal gyrus	L	−33	54	9	5.05	92
Cingulate	B	9	27	30	9.94	181
Calcarine	R	18	−96	−3	6.82	55
Middle occipital gyrus	L	−18	−96	−3	7.06	67
$Control_{Average}$ > $Pumps_{Average}$						
Temporal lobe, Parietal lobe, Precuneus	B	21	−78	39	11.40	15251
vmPFC	B	36	42	−18	7.27	478
Parametric						
$Pumps_{Parametric}$ > $Control_{Parametric}$						
Insula	R	39	21	6	6.08	73
Insula	L	−39	15	0	5.29	59
Thalamus	R	6	−24	9	5.53	10
Cingulate	R	9	30	30	6.28	36
$Control_{Parametric}$ > $Pumps_{Parametric}$						
Postcentral	L	−66	−18	27	7.30	495
Fusiform	R	39	−6	−33	4.89	15
Middle frontal gyrus	L	−21	18	48	4.45	107
vmPFC	L	−12	33	−15	5.51	58
Correlation[a]						
Negative correlation						
Insula	R	33	21	0	−4.86	64
Insula	L	−27	21	−3	−5.57	132
Caudate	R	9	6	9	−5.22	65
Caudate	L	−12	6	12	−5.54	63
Anterior cingulate	R	6	39	9	−3.88	45
Positive correlation						
Middle temporal gyrus	L	−45	−60	3	7.59	293
Middle temporal gyrus	R	54	0	−24	5.07	64
Medial frontal gyrus	L	−18	39	−12	5.15	111
Culmen	R	15	−36	−24	5.14	58

R, Right; L, Left; B, Bilateral. [a]*Correlation with mean number of adjusted pumps.*

contrast described above. Young adults displayed positive effects (i.e., $Pumps_{Parametric}$ > $Control_{Parametric}$) in the bilateral AI, thalamus, and dorsal ACC, and negative effects (i.e., $Control_{Parametric}$ > $Pumps_{Parametric}$) in the fusiform, postcentral gyrus, and vmPFC. Older adults showed positive effects in the bilateral AI, caudate, and SMA, and displayed negative effects in some occipital-parietal regions, but, crucially, no vmPFC areas survived correction (**Figure 3A**; **Tables 2** and **3**, Parametric).

Further, although results from the between-group *t*-tests did not survive whole brain correction, there were voxels in the vmPFC that showed age group differences at $p < 0.005$ uncorrected (**Figure 3B**). The decreasing activity of the vmPFC obtained from the parametric contrast has been suggested to capture value integration in the BART (Schonberg et al., 2012) and the differential pattern of vmPFC parametric activation for young but not older adults suggests that the value integration processes during sequentially increasing pumps is less distinct in older adults compared with young adults. We explored young and older adults' activation maps at $p < 0.005$ uncorrected to check for differences which may have arisen due to factors such as signal heterogeneity or the small number of subjects in each group. At $p < 0.005$ uncorrected, we observed minor striatal activation in both young and older adults (Supplementary Figure S2), which might be suggestive of some form of reward tracking in the striatum as a function of increasing number of pumps. Interestingly, even at uncorrected level, older adults did not show any vmPFC-related activity, pointing toward genuine age-related differences in vmPFC-related integrative signaling.

TABLE 3 | Significant clusters of activation in older adults.

Region	L/R/B	*X*	*Y*	*Z*	*t*-value	Cluster size
Average						
$Pumps_{Average}$ > $Control_{Average}$						
Supplementary motor area, superior frontal gyrus, insula, caudate, putamen	B	−6	0	51	11.20	10387
Lingual gyrus	B	18	−90	−3	11.25	142
Middle temporal gyrus	R	57	−27	−12	3.99	24
Inferior parietal lobule, Precuneus, Middle occipital gyrus	B	48	−45	51	7.42	976
$Control_{Average}$ > $Pumps_{Average}$						
Precuneus	B	−3	−54	33	9.22	3330
Middle temporal gyrus	L	−36	24	−27	5.38	409
Middle temporal gyrus	R	60	6	−15	8.32	201
Superior temporal gyrus	R	63	−54	21	5.95	490
Inferior frontal gyrus	L	−45	30	−6	5.27	170
vmPFC	B	−9	57	36	7.89	326
Parametric						
$Pumps_{Parametric}$ > $Control_{Parametric}$						
Insula	R	33	21	−3	6.51	44
Insula	L	−33	21	−9	7.35	56
Caudate	R	15	6	3	5.08	13
Supplementary motor area, cingulate	B	6	18	51	6.28	30
Lingual gyrus	B	−9	−84	−3	5.09	10
$Control_{Parametric}$ > $Pumps_{Parametric}$						
Inferior occipital gyrus	L	−42	−69	−6	6.14	93
Lingual	R	24	−90	−3	5.75	4
Superior parietal lobule	L	−21	−81	45	5.29	25
Precentral gyrus	R	51	−12	54	4.61	7

R, Right; L, Left; B, Bilateral.

Regions Correlated with Behavioral Performance

We conducted a whole-brain regression analysis linking a measure of risk taking, mean adjusted pumps in the BART, and neural activity obtained from the "$Pumps_{Parametric}$ vs. $Control_{Parametric}$" contrast. We thus hoped to assess how individual differences in behavioral risk taking were associated with average neural activation patterns. Across age groups, the regression analysis revealed significant negative correlations between participants' risk taking and activity in the bilateral AI and caudate (**Figure 4A**; **Table 4**). In turn, positive correlations were found between adjusted pumps and activity in the bilateral middle occipital cortex, inferior parietal lobule, and vmPFC. The positive association between behavior and vmPFC activation is reflective of individual differences in the steepness of the predominantly negative slopes observed in the vmPFC: individuals with flatter (i.e., smaller negative effect) slopes on average administered more pumps on cashout balloons compared with individuals with steeper (i.e., greater negative effect) slopes. It is therefore postulated that individuals take more risks on average (i.e., administer more pumps) if the decrease in vmPFC activity is more gradual. Regarding age-related differences, young adults' regression results were similar to the findings obtained across all individuals, albeit stronger in several regions (**Figure 4B**; **Table 2**, Correlation). However, regression of whole brain activation on mean adjusted pumps for older adults yielded no significant voxels at the correction threshold of $p < 0.05$ and only very sparse association patterns at $p < 0.005$ uncorrected (Supplementary Figure S3).

To check that the whole brain regression results were not influenced by outliers and visualize the results with respect to individual differences, additional ROI analyses were conducted on the bilateral AI, bilateral striatum and vmPFC. In particular, mean beta weights were extracted from spheres based on the relevant center coordinates provided by Bartra et al. (2013) to achieve an independent definition of the structures of interest. To note, these analyses are merely for visualizing the relationship between neural activity and performance in the two age groups; the authors acknowledge a degree of circularity when extracting activation from regions identified by whole brain

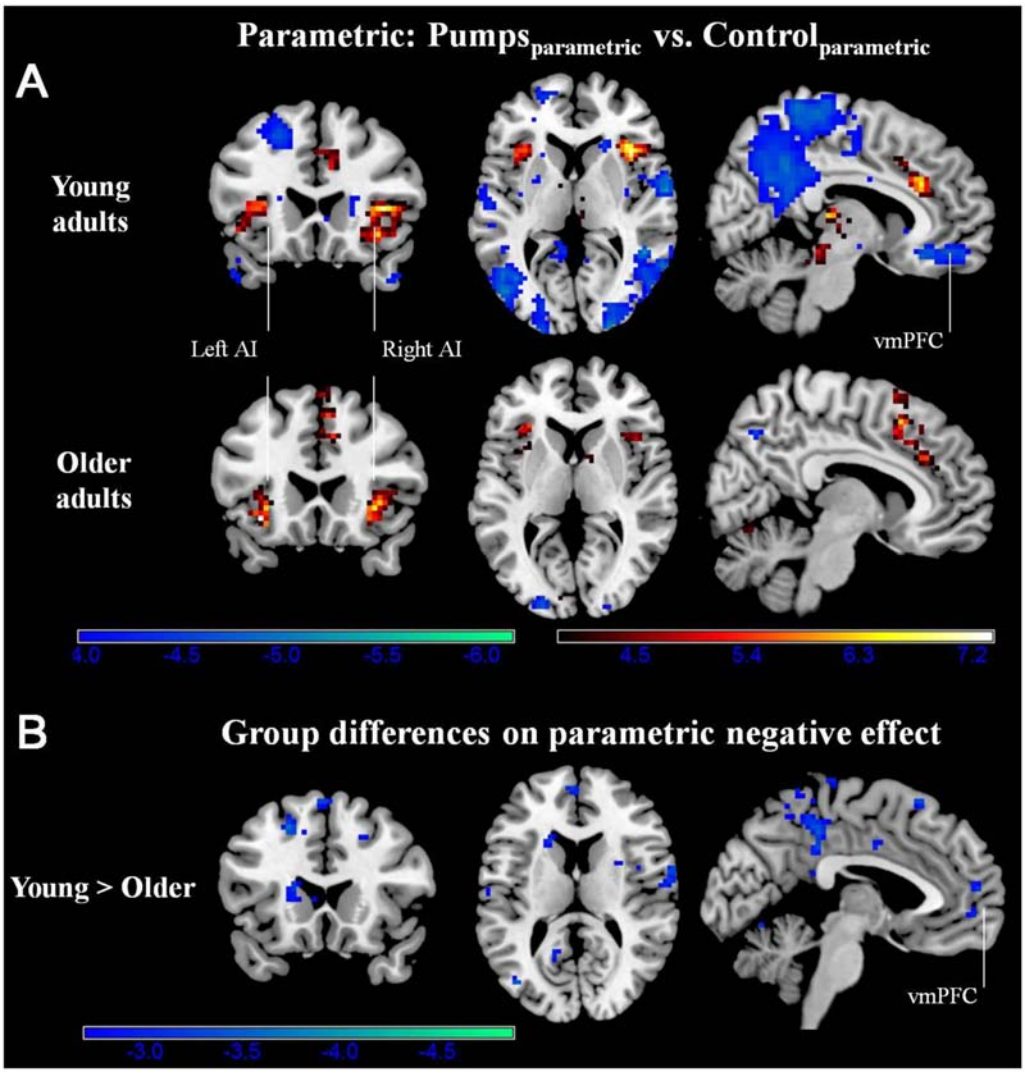

FIGURE 3 | (A) Parametric modulation of increasing number of pumps in the young and older age group. The red scale represents $Pumps_{Parametric}$ > $Control_{Parametric}$ and the blue scale represents $Control_{Parametric}$ > $Pumps_{Parametric}$. $p < 0.05$, corrected. **(B)** Age group differences on parametric contrast. The blue scale represents neural regions in which young adults had more negative effect than older adults. $p < 0.005$, uncorrected (scale represents the range of *t*-values). No brain regions were discovered for (1) which older adults had larger negative effects relative to young adults or (2) age group differences on parametric positive effect at this threshold.

analyses as being associated with performance. However, given that no significant association was obtained from the whole brain analyses for older adults, we were interested in visualizing the distribution of performance against activity in both age groups.

Activity in the seed region of the left AI during $Pumps_{Parametric}$ vs. $Control_{Parametric}$ was negatively correlated with adjusted pumps in young ($r = -0.60$, $p < 0.01$), and older adults ($r = -0.51$, $p < 0.05$; $Z = 0.42$, $p > 0.05$; **Figure 5A**). A comparable pattern was found in the right AI, with older adults showing a correlation between adjusted pumps and brain activity that was similar to that found for young adults ($r = -0.44$, $p < 0.05$, $r = -0.58$, $p < 0.01$, respectively; $Z = 0.62$, $p > 0.05$; **Figure 5B**). These findings merely visualize the whole brain regression results, suggestive of comparable insular tracking of potential loss (uncertainty) in older and young adults. In addition, extracted beta weights from the left (but not right) striatum correlated negatively with mean number of adjusted pumps in young ($r = -0.68$, $p < 0.001$) but not older adults ($r = 0.15$, $p = 0.50$; **Figure 5C**); the difference between these two correlations was significant ($Z = 3.17$, $p < 0.01$). As expected from the whole brain analyses, activation in the vmPFC was positively correlated with adjusted pumps in young adults ($r = 0.48$, $p < 0.05$), but not in older adults ($r = -0.22$, $p = 0.31$; **Figure 5D**); the difference between these two correlations was significant ($Z = 2.42$, $p < 0.05$). We obtained comparable results when using masks derived from peak contrast coordinates and varying radii. To note, although occupying a

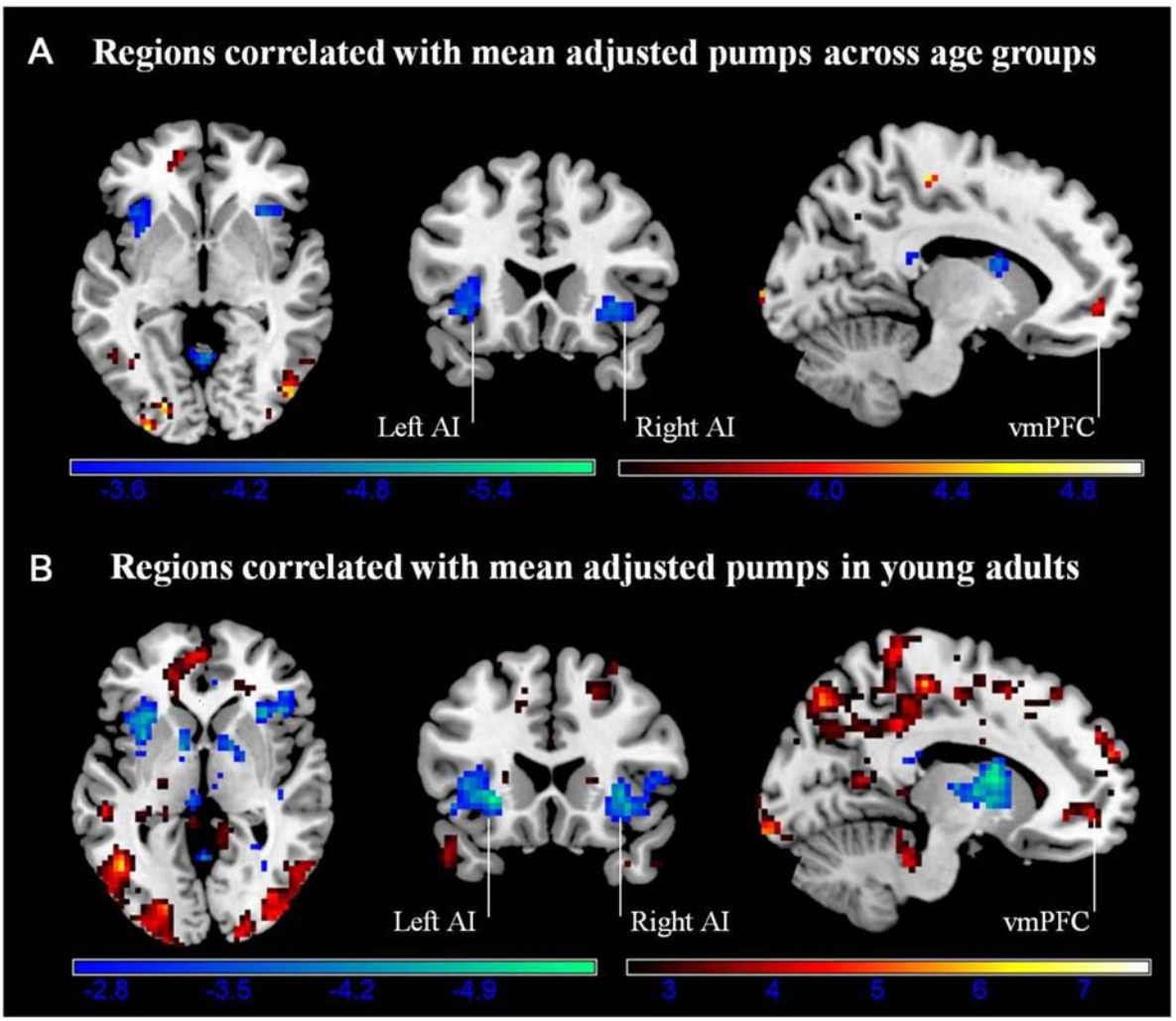

FIGURE 4 | (A) Regions correlated with mean adjusted pumps (whole sample). **(B)** Regions correlated with mean adjusted pumps in young adults. Activity obtained from parametric modulation of increasing number of pumps ($Pumps_{Parametric}$ vs. $Control_{Parametric}$) in the bilateral anterior insula (AI) is negatively related to and ventromedial prefrontal cortex (vmPFC) is positively related to participants' mean adjusted pumps in the young adults. The red scale represents a positive correlation, whereas the blue scale represents a negative correlation, $p < 0.05$, corrected (scale represents the range of t-values).

similar range, the distribution of mean beta weights extracted from the parametric modulation of vmPFC activity in older adults appears positively skewed compared with a relatively more normal distribution for young adults (**Figure 5D**). In contrast, the distribution of extracted mean activation slopes for the insula and striatum is relatively more similar in older and young adults.

Taken together, these results suggest that although neural representations of reward and risk as well as the tracking thereof remain relatively stable across age groups, their predictive validity for behavior may be different for young and older adults. Moreover, older adults' tracking of value in the vmPFC was different from that of young adults, also manifested by the differential vmPFC activation profiles and predictive validity of vmPFC activation for mean pumping (i.e., risk taking) behavior in the BART.

DISCUSSION

The present study investigated adult age differences in behavior and neural activations associated with the BART, a widely used naturalistic risk-taking task (Lejuez et al., 2002). Specifically, we asked young and older adults to undergo fMRI while completing a version of the BART consisting of different types of balloons, which either did (experimental) or did not (control) involve monetary risks and rewards. The different balloon types were leveraged to build contrasts that captured the neural signatures associated with young and older adults' risky decision-making processes (Rao et al., 2008; Schonberg et al., 2012; Helfinstein et al., 2014).

Our results indicate considerable similarity between young and older adults in the behavioral outcomes of the BART, including similar average number of pumps per balloon for

TABLE 4 | Clusters correlated with mean number of adjusted pumps in across age groups.

Region	L/R/B	*X*	*Y*	*Z*	*t*-value	Cluster size
Negative correlation						
Insula	L	−36	21	−6	−4.81	83
Insula	R	33	21	−3	−4.52	33
Caudate	B	−12	6	18	−4.62	36
Culmen	B	3	−57	0	−5.89	13
Positive correlation						
Inferior temporal gyrus	L	−48	−57	−9	4.58	38
Middle occipital gyrus	R	42	−69	−12	4.75	23
Middle occipital gyrus	L	−21	−84	−3	4.73	62
Inferior parietal lobule	L	−45	−45	45	4.59	36
Medial frontal gyrus	L	−18	45	3	4.19	12
Medial frontal gyrus	L	−12	−27	57	4.60	12

R, Right; L, Left; B, Bilateral.

the two age groups. Older adults were, however, more likely to cashout their temporary wins relative to young adults, potentially indicating higher levels of risk-aversion with increased age (Mata et al., 2011; Best and Charness, 2015). Overall, these behavioral outcomes contribute to the heterogeneity of findings concerning age differences in the BART (Henninger et al., 2010; Cavanagh et al., 2012; Rolison et al., 2012).

Concerning our neuroimaging results, we replicated past findings with young adult samples suggesting a link between neural activation and the processing of risk and reward. Specifically, using contrasts between neural activation while pumping in experimental relative to control balloons in the BART, we found significant neural activations in the caudate, bilateral insula, and parietal regions, as well as in the vmPFC, which are comparable with previous findings (Rao et al., 2008, 2014; Schonberg et al., 2012). Also consistent with a previous study that analyzed parametric neural activation as a function of increased exposure to risk and rewards, we found that vmPFC activity decreased whereas bilateral AI activity increased as participants pumped up each balloon (Schonberg et al., 2012). Concerning age differences, group average comparisons identified similar patterns of activations in the striatum and AI as well as deactivation in the vmPFC in both age groups. Our findings are in line with previous studies showing intact representation of reward (Samanez-Larkin et al., 2007, 2014) and loss anticipation (Samanez-Larkin et al., 2008; but see Samanez-Larkin et al. (2007) for altered insular sensitivity during loss anticipation). The lack of differences between young and older adults in ventral striatal activation during gain anticipation may imply that the ventral striatal regions may not be as compromised by age as are the neural substrates recruited in reward reversal learning tasks, such as the PFC regions (Marschner et al., 2005; Samanez-Larkin et al., 2007). Some differences between young and older individuals were observed for the average contrasts: the comparatively lower deactivation/higher activation for risky vs. control balloons in older adults may suggest systematic differences in the neural representation of value-related processes, for instance slightly higher sensitivity to gains (higher striatal activation) or weaker integration (less vmPFC deactivation). It is noteworthy that some of the regions for which age differences were observed in the average contrast analysis overlap with regions engaged in the default mode network (Raichle et al., 2001) and brain networks identified for working memory tasks (Tomasi et al., 2006). Consequently, it is also possible that the few differences observed for average contrasts stem from older adults dealing differently with the process of being engaged in and completing a task with some memory demands.

In contrast, parametric analyses at group level found that young and older adults evidenced similar tracking of pumps in the AI, but only young adults showed parametrically decreasing activity in the vmPFC. Interestingly, strong striatal activation might be expected as a function of parametric pumps, given that the striatal coding of gains (cf. Tom et al., 2007) ought to be reflected in the parametric tracking of pumps, the latter being a potential proxy for increasing gain on a given trial in the BART. The absence of a strong striatal signal in this study as well as in the study by Schonberg et al. (2012) is likely to be reflective of increasing pumps being processed not as increasing gain, but as increased risk of loss. Against a backdrop of work that has assigned the processing of risk to the insula (Volz et al., 2003; Kuhnen and Knutson, 2005; Preuschoff et al., 2008; Samanez-Larkin et al., 2008), our parametric results further support findings from the average contrasts, speaking to unaltered insula-based tracking of increasing risk in old age.

The combination of relatively preserved insula signaling and age-related differences in vmPFC signaling in response to increasing risk observed from the parametric analyses support the notion of the vmPFC as a platform for integration and convergence of information (Schonberg et al., 2012; Bartra et al., 2013; Clithero and Rangel, 2014; Halfmann et al., 2014, 2016). Specifically, we propose that with age, individuals may attach different weights to different aspects of a decision context, or alternatively, are less consistent across time in the weights attached to particular options. Put differently, although older and young individuals in the current study responded with comparable risk and reward signals, the two groups differed with respect to the integration of risk and reward into a

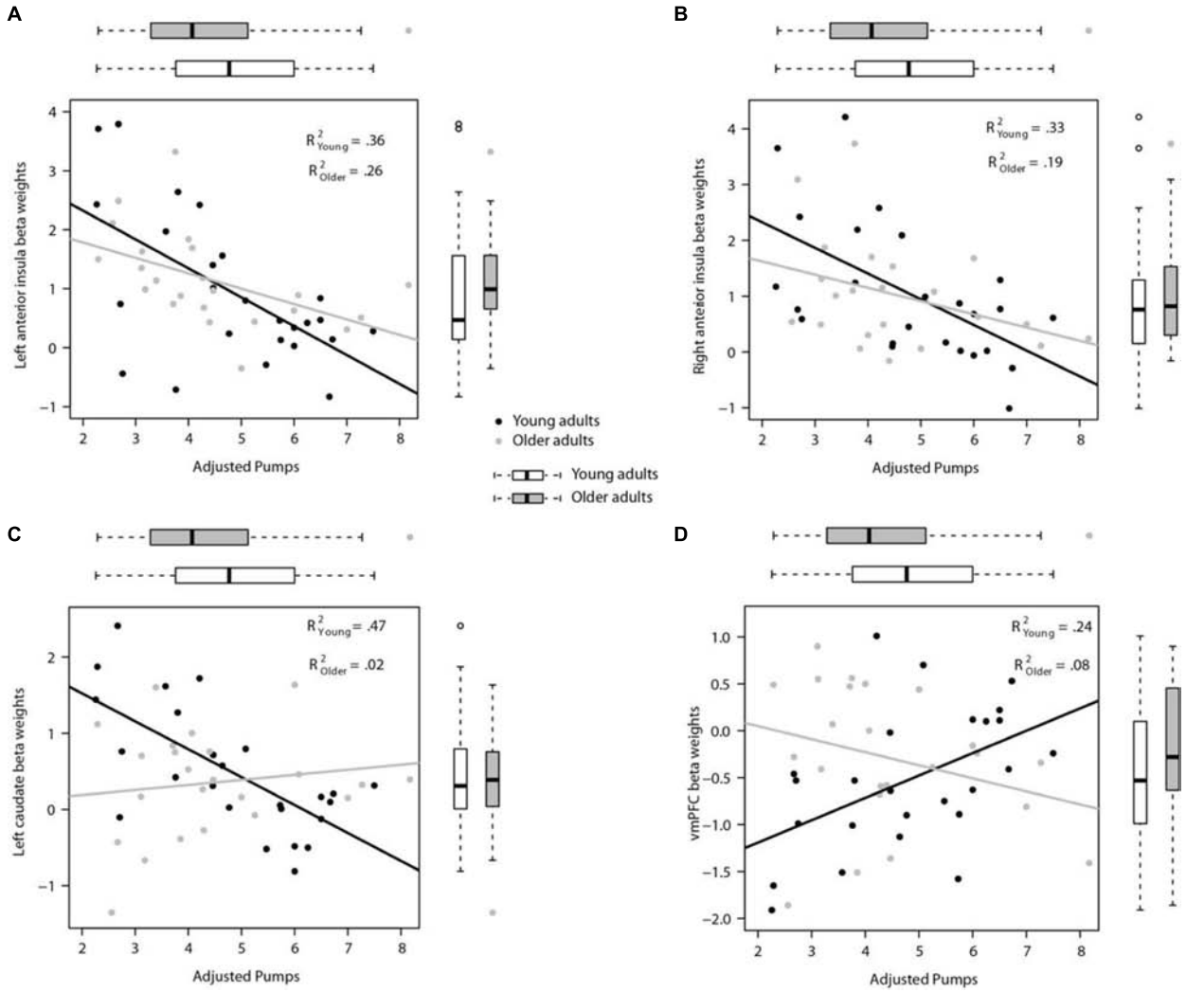

FIGURE 5 | Region of interest (ROI) analyses for links between individual neural and behavioral differences. (A) Participants' mean adjusted pumps negatively correlated with their BOLD signals in the left AI and **(B)** the right AI for both young and older adults. **(C)** Activity in the left striatum was significantly correlated with mean adjusted pumps in young adults, but not in older adults. **(D)** Activity in the vmPFC was positively correlated with mean adjusted pumps in young adults, but not in older adults. The boxplots on top of the plot show the distribution of mean adjusted pumps in the young and older age group, respectively, whereas boxplots to the left of the plot show the distribution of the signal changes in the left/right AI, vmPFC and left striatum, respectively. ROIs were created as 4 mm radius spherical regions covering bilateral anterior insula and left striatum, and an 8 mm radius spherical region in the vmPFC (center coordinates based on meta-analysis by Bartra et al., 2013).

subjective value signal. In support of this line of argument, past theoretical and empirical work converges on the idea that value representations are affected by age-related anatomical and/or functional differences. Anatomically, there is a global declining of gray matter volume in the prefrontal cortex (PFC) with age (Raz et al., 1997), a thinner cortical thickness of left vmPFC (Cassidy and Gutchess, 2012), and a decreasing white matter integrity in thalamocorticostriatal paths, which run from the thalamus to the medial PFC and from the medial PFC to the ventral striatum (Samanez-Larkin et al., 2012). Functionally, impaired integration processes from the vmPFC may arise from less effective coding by single systems or degrading glutamatergic projections from the medial PFC to the striatum (Samanez-Larkin and Knutson, 2015). Recent work by Halfmann et al. (2014, 2016) linked reduced vmPFC signaling to disadvantageous decision patterns in the Iowa Gambling Task, which the authors interpreted as support for the notion of noisier value representation in older adults (Li et al., 2007; Samanez-Larkin et al., 2010). This view is also consistent with previous studies showing age-related reductions in activity during learning from rewards in the vmPFC but not during learning from monetary losses in the insula and striatum (Eppinger et al., 2013). Bridging the gap between the neural and the behavioral level, it is conceivable that a decreased signal-to-noise ratio in older adults may in part be underlying the mixed behavioral patterns obtained in past work using the BART (Henninger et al., 2010; Cavanagh et al., 2012;

Rolison et al., 2012): different experimental implementations of the BART may rely on more (or less) efficient integration of information, hence decision outcomes are perhaps affected differently by an integration process that is subject to age-related changes. Although the current study cannot offer direct evidence supporting this suggestion, the notion of heterogeneity in study results being linked to brain signal heterogeneity offers a potential avenue for research aimed at connecting age-related neural and behavioral differences in decision-making tasks.

Current theories emphasize the contribution of both cognitive and affective processes to age differences in decision making (Samanez-Larkin and Knutson, 2015; Schiebener and Brand, 2015). Our results, however, indicate that what could be potentially considered affective components, such as neural coding of risk in the AI and reward in the striatum, are relatively preserved with aging. In turn, value coding and integration in the vmPFC seems less robust. Whether such changes can be deemed the result of cognitive or affective components is unclear. The absence of both a consistent group level value signal and a correlation with behavior in the vmPFC in our sample of older adults for instance may result from older adults exhibiting potentially noisier intra-individual (e.g., Samanez-Larkin et al., 2010) or more heterogeneous inter-individual coding of value in this region, suggesting a more cognitive explanation. Alternatively, given that we find older adults' risk-taking behavior to be linked with insula more so than with vmPFC signaling in response to increasing risk, there may also be motivational components associated with the relative importance or attention devoted to gains and losses (Mata and Hertwig, 2011).

The exact mechanisms underlying age differences in value coding and integration in the vmPFC are still to be identified. Future work using the BART could contribute to clarifying these issues by manipulating task characteristics, such as reward structure and loss probability, to better tease apart the contribution of neural risk and reward signals in young and older adults to an overall utility signal coded in the vmPFC. Future work may also want to directly test the role of anatomical and functional deficits in and between medial prefrontal and other brain regions by using behavioral performance indices in voxel-based morphometry (e.g., Strenziok et al., 2011; Peper et al., 2013; Gilaie-Dotan et al., 2014), diffusion tensor imaging (e.g., Kwon et al., 2014; Van den Bos et al., 2014; Leong et al., 2016), or effective connectivity analysis (e.g., Hare et al., 2014).

With respect to limitations, risk and reward were directly correlated in the current BART version hence it was not possible to dissociate risk from reward through parametrically altering each decision component. However, given the comparatively rich pool of studies that have investigated risky decision making as well as the impact of aging thereon, the advantage of using a task that offers external validity outweighs many of its shortcomings. As alluded to above, future work is required which tries to dissociate reward from risk as well as reward and risk from subjective value. We are currently in the process of answering this call to uncover differential sensitivity to risk or rewards as a function of age. Further, future studies should strive to collect data from lifespan samples to account for intra- as well as inter-individual change to derive neural and behavioral trajectories of risk taking across the full range of the adult lifespan.

CONCLUSION

To conclude, our comparison of young and older adults' neural activation during decision making in the BART suggests that the two age groups show similar patterns of activation in the AI, possibly coding for the probability of loss, yet differ in the recruitment of the vmPFC, which is thought to subserve value integration and representation. Our results suggest that the integration of risk and reward resulting in overall utility representations may be affected by aging. Our results show the need for distinguishing different neural components underlying risk taking, including the processing of risk, rewards, and the integration of the two, to uncover possible differences in risk taking across the lifespan.

AUTHOR CONTRIBUTIONS

JY conceived the idea, designed the study, analyzed and interpreted data, drafted the manuscript. RM and LM interpreted data, and participated in writing up and revising the manuscript. XL and LF assisted to analyze and interpret data.

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SUPPLEMENTARY MATERIAL

The Supplementary Material for this article can be found online at: http://journal.frontiersin.org/article/10.3389/fnagi.2016.00210

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Conflict of Interest Statement: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

APPENDIX C: Manuscript Three

Title: Group versus individual differences in the neural representation of described and experienced risk

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Abstract

Risk taking is linked to important life outcomes, including health, wealth and criminality, hence individual differences therein have become attractive targets for developmental and longitudinal research. It is currently unclear, however, to what extent biomarkers such as brain function are informative in those contexts, mainly because there has been a lack of studies examining the role of measurement on the neural representation of risk and its explanatory power for behavior. This however is crucial: Contrary to standard economic theory, the same information encountered in different ways can lead to different choices. In this study we report results from the MRI sample (N=116 young adults) of the Basel-Berlin Risk Study, for which we collected functional neuroimaging data in response to the Balloon Analog Risk Task as an example of experienced risk, and monetary gambles as an example of described risk. In our analyses, we address (1) the overlap of the average neural representation of risk in BART and monetary gambles, (2) whether individual activation differences are preserved across these two measures, and (3) the explanatory power of neural indices from BART and monetary gambles for risky choice, within and across measures. Our results suggest joint activation increases in nucleus accumbens for BART and monetary gambles, but inconsistent individual differences in nucleus accumbens, insula and anterior cingulate cortex activation across the two measures. Within measure, we obtained brain-behavior associations only for monetary gambles, but not for BART. Across measures, we observed a link between anterior cingulate cortex activation in response to risk in the BART and risky choice in monetary gambles. Our findings further help to clarify the commonalities and differences between the neural correlates of experienced and described risk, suggesting that these two types of measures should not be used interchangeably to capture risk preference. As such, our results have strong implications for longitudinal and developmental designs targeting individual differences in risk taking.

Introduction

Risk preference—whether in the economic sense of preferring monetary high-variance options over more certain ones, or, more commonly, preferring options involving uncertain but potentially sizeable negative consequences[1]—pervades decisions across various life domains, including health, wealth and criminality[2,3]. Consequently, risk preference, its core components (e.g., valuation, risk sensitivity, loss aversion), related constructs (e.g., self-control, impulsivity), and the environmental influences upon them (e.g. peer environment, culture) have become promising targets for longitudinal, developmental and clinical research designs[4–7].

Description- and experience-based measures of risk taking

Unfortunately, the risk-preference literature offers numerous measures, but lacks a clear taxonomy of measures and the core capacities that they elicit[8,9]. Zooming in on behavioral measures of risk taking, recent studies observed divergent patterns of individual and age differences as a function of the measures used[10–12], as well as weak or no correlations between various measures, suggesting they cannot be used interchangeably[13–15]. One factor contributing to the divergence of behavioral measures resides in the way individuals come to know about risk-relevant information: Information about potential outcomes and their probabilities can either be fully described and thus known in advance, or over time have to be ascertained from experiencing choice outcomes[16]. Description- and experience-based measures (henceforth referred to as described risk and experienced risk, respectively) share central characteristics of decision-making under risk, including the processing of outcome magnitudes and probabilities, and their integration into a subjective value (i.e. utility) signal informing choice. They differ, however, with regard to the (coincidental or necessary) involvement of additional cognitive processes, including affect, memory, strategy usage, and learning[11,16–19]. As a likely consequence of these differences, described and experienced risk have been found to elicit different choices and thus lead to different average (e.g., younger

versus older) and individual risk profiles[11,14].

Given this *description-experience gap*[16], a prerequisite for understanding individual differences in risk taking and for finding suitable targets for intervention will be to address questions such as "Under what conditions does the description-experience gap arise?", "What are the underlying mechanisms?", "Are described and experienced risk equally predictive of risk-taking behavior and life outcomes?", and if not, "What drives the differential predictive validity of described and experienced risk?". Our aim in this study is to offer a neural perspective on these issues using brain activation differences for described and experienced risk.

Contribution of neural correlates of described and experienced risk to individual differences in risk taking

In a bid to understand the biological underpinnings of risk taking, neuroimaging methods have been used to understand the neural correlates and mechanisms of (individual differences in) risk taking. Both qualitative reviews[20,21] and quantitative meta-analyses of single neuroimaging studies[22–24] converge on several neural regions as key correlates of risk taking and its constituent processes, including striatum, insular cortex, anterior cingulate cortex, (ventro)medial prefrontal cortex, and dorsolateral prefrontal cortex. A subset of these regions has been advocated as being so crucial for risk taking that they form a neural "risk matrix", differentially promoting (nucleus accumbens in ventral striatum), inhibiting (insular cortex), and controlling (anterior cingulate cortex) risky choice[20].

Unfortunately, the role of the measurements adopted to study risk taking and its neural correlates has so far received very little attention. Both described and experienced risk have been used to understand the neural basis of risk preference and associated processes (e.g., valuation, risk sensitivity, loss aversion)[20,22–26], yet very few studies have directly compared the two. In a recent exception[27], the overlap of neural function during reflective and impulsive risk taking was examined using the Game of Dice Task and Balloon Analogue Risk Task,

respectively, revealing joint activation increases in two of the three "risk matrix" regions, namely bilateral caudate and insula. It is plausible that such neural functional commonalities are attributable to the central processes shared by described and experienced risk (e.g., valuation). In that case, these seemingly measure-invariant neural endophenotypes may present useful targets for prevention and intervention. Two fundamental issues need to be addressed before such a path should be taken.

First, the hitherto observed commonality of neural function in response to described and experienced risk does not necessarily indicate convergence (i.e. consistency) owing to the well-known but often neglected lack of a match between group-level (i.e., aggregate) and individual-level effects reported for instance in the developmental[28] and social preferences literature[29]. In the neuroimaging literature on decision making, the proposition that average activation differences do not necessarily reflect individual-level patterns has already found some support: In a study[30] which implemented three different reward paradigms and repeated MRI sessions, a reliable group-level reward-related BOLD signal was observed in striatum and orbitofrontal cortex, regardless of paradigm, session or contrast analysis (e.g., prediction or receipt of reward). However, at the level of the individual, intra-class correlation coefficients indicative of test-retest reliability for the different paradigms in ventral striatum and orbitofrontal cortex for most contrast analyses were low (0.1 – 0.2) and not significant[30].

In the current context of core substrates of decision making under risk, our current knowledge of common neural activation differences across measures is predominantly based on average activation patterns originating from different studies (i.e., different individuals). However, the same individual may respond very differently, neurally and behaviorally, to different measures of risk preference, for instance as a function of whether risk is described or experienced. It is therefore unclear whether repeated measures designs of neural activation for different measures would result in consistent neural activation, or if such designs would mirror the weak to no associations observed between behavioral indices of risk preference

obtained from repeated-measures designs (i.e., the same person completes multiple measures, and within-subject consistency is consequently evaluated)[13]. Thus, although the mechanisms underlying group-level convergence but individual-level divergence (e.g., low between-subjects variability[31]) can be debated, it is clear that it would be misguided to expect group-level results to reflect the individual level, and thus to be informative for individual differences analyses. Instead, studies are needed that investigate average (group-level) activation differences across different measures, but also target individual differences[6,32].

A second issue to address before using neural indices for intervention concerns the explanatory power of neural indices for risk-taking behavior and related life outcomes. At present, we do not know the extent to which neural indices of described and experienced risk are predictive of choice, both within and across measures. However, given that regional activation differences do not necessarily reflect useful, reliable predictors of observed behavior[33], this is an important prerequisite for understanding individual differences.

There is a regrettable absence of neuroimaging studies that have assessed described and experienced risk, addressed group- and individual-level effects, and investigated the explanatory power of neural indices for risky choice, both within and across measures. In the recent study which compared risk taking using the Game of Dice Task and the Balloon Analogue Risk Task[27], significant within-measure brain–behavior associations were observed for described but not experienced risk[27]. Whether individual differences in neural activity for one task were preserved in the other was unfortunately not reported, neither whether brain-behavior associations were observed across these two meaures[27]. This however is crucial for our understanding of individual differences in risk taking, especially where these inform studies investigating associated developmental trajectories[2,7] or clinical outcomes[4]: If joint neural activation differences were observed for experienced and described risk, which applied at group- and individual level, and which evidenced explanatory power for observed behavior, the case for measure-invariant neural indices of risk taking would be further supported.

The current study: Neuroimaging component of the Basel-Berlin Risk Study

To tackle the issues raised, the current study assesses the match between group- and individual-level effects for the neural representation of experienced and described risk, in two paradigms frequently adopted to examine individual differences in risk taking: the Balloon Analogue Risk Task (BART) and monetary gambles, respectively. Specifically, we report results from a neuroimaging component of the Basel-Berlin Risk Study (BBRS), a large-scale study assessing individual differences, psychometric structure and biological underpinnings of risk preference[13,15,34]. Participants in the BBRS completed a one-day laboratory session involving an extensive battery of measures assessing individual differences in risk taking (including self-report, frequency, and behavioral measures), cognitive capacity, personality, affect, and genetics (an overview of all subsamples, measures, and further details on the BBRS is reported on https://osf.io/rce7g). A subsample also completed an MRI session, which included structural and task-(in)dependent functional imaging sequences.

The current study is based on task-dependent functional imaging data from 116 individuals for experienced (BART) and described (monetary gambles) risk. These two measures were chosen because both are commonly used, relatively simple paradigms, for which average neural activation profiles[25,26,35,36] as well as individual differences have been extensively investigated[7,35,37–39]. Importantly, both measures feature similar concepts such as loss, reward, and risk. Yet, whereas these parameters are explicitly described for monetary gambles, some of them (in particular "risk") must be explored and learned from experience in the BART[19,40]. Based on these two measures, we examine (1) common and distinct neural correlates of experienced and described risk, (2) the consistency of individual differences in the neural representation of experienced and described risk, and (3) the explanatory power of neural indices of experienced and described risk for behavior. Thus, our unique design of a) investigating group- and individual-level neural representations of risk, and b) implementing two prototypical measures capturing both experienced and described risk allows us to

systematically understand which components of the neural response to risk are measure-(in)variant. This approach therefore promises to bring us one step closer to understanding the role of task characteristics to individual differences in risk taking[14,41]. Zooming in on experienced risk, performance-based indices of the BART have been shown to predict outcomes as critical as drug use, teenage pregnancy and criminal activity[42,43] and neural indicators thereof have been used to understand developmental aspects of risk taking[7,38]. By examining the explanatory power of activation differences in response to risk in the BART, we aim to learn more about the mechanisms underlying the predictive success of this experience-based measure for real life risk-taking behaviors.

In what follows, we first report results concerning group-level analyses of similarities between average neural activation patterns in response to experienced and described risk. Specifically, we were interested in whether any or potentially all joint activation differences would be located in "risk matrix" regions, that is, whether overlapping activation differences in response to experienced and described risk would be found in nucleus accumbens, insula and/or anterior cingulate cortex[20]. Secondly, we report results from individual-level analyses examining whether group-level activation patterns are representative of individual-level patterns. Thirdly, we report results pertaining to the explanatory power of neural indices common to experienced and described risk, both within and across tasks.

Given task-specific demands, it is possible that the "risk matrix" regions (nucleus accumbens, insula and anterior cingulate cortex) are recruited more strongly by one task than the other. Thus, for those "risk matrix" brain regions that were not conjunction regions, we additionally probed their individual-level consistency and explanatory power.

Materials and Method

Participants

For this neuroimaging study, we recruited an imaging subsample of 133 young adults from an existing pool of individuals who had participated in a large study on individual

differences in risk preference, the Basel-Berlin Risk Study (BBRS)[13]. The sample size is reflective of oversampling to achieve an effective sample size of N~100[44] in case of participant exclusions (e.g. due to excessive head motion in the scanner, image artefacts). The BBRS was run in Basel and in Berlin, but for the current study we recruited only individuals from the Berlin site due to the location of the neuroimaging facilities available. Exclusion criteria for participation in the MRI session were safety-limiting permanent implants, a history of neurological or psychiatric conditions, usage of psychoactive medication or substances, and receiving psychiatric treatment. Two participants aborted the session before any functional sequences were collected, thus were removed from all subsequent analyses. We excluded a further five participants due to excessive head motion inside the scanner (see image preprocessing section for movement parameter thresholds), one participant due to incidental anatomical findings, four participants due to incomplete data (e.g., only one paradigm was completed inside the scanner), and five participants due to non-compliance with the scanner protocol (e.g., falling asleep, reports of having mixed up button box responses). The final sample included in all analyses comprised 116 participants (62 females, mean age at scan = 25.4 years, SD = 2.6 years, range = 20.4–30.1 years).

All participants provided written informed consent. Ethical approval for this study was obtained from the German Society for Psychology (Deutsche Gesellschaft für Psychologie), and the ethics committee of the Center for Adaptive Rationality, Max Planck Institute for Human Development.

Experimental paradigms

Inside the scanner, participants completed the BART[26] and a monetary gambles paradigm[35]; we describe the two paradigms in more detail below. The MRI session further involved the collection of data outside the scanner, such as various self-reported demographic data, including date of birth, gender, marital status, educational attainment, native language and current occupation. Of note, only gender and age at the MRI session (calculated from date

of birth) were included as covariates in the current analyses; all other demographic measures were merely collected to describe the sample and ascertain the external validity of our findings with respect to sample characteristics. We further assessed individual's height and weight, and collected data from a verbal fluency task, various self-report measures of impulsivity[45], eating-related behaviors and attitudes[46–48]; these measures were not part of the current analyses and are therefore not reported further. All measures and instructions were presented in German.

Balloon Analogue Risk Task (BART). The BART is a commonly used measure of risk preference[49] that has also found wide application in neuroimaging research[25–27,37,50–52]. Individuals sequentially inflate a series of virtual balloons in the absence of a priori knowledge about the underlying contingencies (i.e., the maximum capacity of the balloons, which determines the distribution of trial-specific explosion points); these, however, can be learned from experience as individuals move from trial to trial and receive feedback (i.e., whether or not a balloon exploded on the previous trial). Given this structure, successful performance in the BART is predicated on decisions that are informed by the construction and updating of a mental representation of explosion distributions for a given balloon type over time.

The BART version implemented in the current study featured two risky balloon types and a control balloon (Figure 1A). The maximum capacity for the two risky balloons was set to be 12, and 20; that is, on average balloons with a capacity of 12 burst earlier than balloons with a capacity of 20. Risky balloons were represented in blue and red to discriminate between balloon types based on capacity, with capacity-color assignment being randomized between participants but stable across the two runs. The two different risky balloon types have been shown to systematically influence the number of decisions to inflate[14,26]. Hence, we were interested in examining whether this pattern extends to choice-specific neural activation. Control balloons were presented in gray, had a maximum capacity of 16, and were added to

control for neural processes of no interest (e.g., motor or visual processes) hence required no decision-making (participants merely inflated the balloon until it disappeared from the screen).

On any given trial, balloon capacity was determined via a random draw from a uniform distribution of values between one and the maximum capacity for the presented balloon type. Participants completed two runs of the BART, with a short break in-between. Each run was programmed to continue for 10 min, after which the final balloon was presented. Given that decisions are made sequentially and may become more difficult as the number of successful pumps in a trial increases, we did not impose a time limit on the decision phase of a given trial, resulting in the number of balloons played to vary between individuals (Table 1). Intervals between trials and between successive stimuli within trials were randomized (mean inter-trial interval = 4.39 s, range = 1–11 s; mean inter-stimulus interval = 1.5 s, range = 1–2 s).

The outcome variable typically used in the BART to reflect individuals' risk preference is the average number of pumps administered on cash out trials[40,49,52,53]. In line with previous research[13,14], in the current study the adjusted average number of pumps was highly correlated with the average number of pumps across all balloons ($r = 0.97$, $p < 0.001$). Given these results, we used the average number of pumps across all balloons as outcome variable in the BART because it allowed us to retain a maximum number of trials for analysis while working with congruent trial numbers in both neural and behavioral analyses. To understand if individuals' behavior in the BART is reflective of a differentiation between balloon types, and also to check if some commonly observed BART effects (e.g. effect of explosion on pumping/stopping behavior[14,40]) were present in the MRI sample, we applied a mixed-effects model to individuals' trial-by-trial behavioral data.

It has been suggested that computational models of the BART can help to disentangle different cognitive processes underlying the observed behavior in this task, including gain and

loss sensitivity, response consistency, risk preference, or learning[18,40]. However, attempts to model the BART have frequently resulted in highly correlated model parameters and failed parameter recovery[18], suggesting that the purported benefit of using parameters obtained from currently available models may be limited. We set out to model the behavior in the BART with two standard models: a target model that assumes a fixed strategy is being used[19,54], and a Bayesian sequential risk-taking model that allows for dynamic updating processes[19]. Yet, in line with past research the estimation of the model parameters turned out to be unreliable, and we thus do not report the modeling attempt here (a possible reason for the unreliable model parameters may consist of the lack of strong learning effects). Consequently, we relied on the average number of pumps as a simpler and generic index of risk preference in all subsequent analyses.

Gambles paradigm. We adopted a monetary gambles paradigm with mixed outcomes as an example of a description-based risk-taking measure (i.e., both gains and losses were possible; Figure 1B, left panel)[35,36,39,55]. In brief, individuals made repeated choices between two options: a gamble offering a 50% chance of a gain and a 50% chance of a loss, or a sure outcome of zero. In contrast to experienced risk, monetary gambles simulate a different decision context, namely one where decisions are informed by known, described outcomes and associated probabilities (*decisions from description)*[16]. Participants made a total of 144 decisions between a sure zero-outcome and a 50/50 gamble. Individual gambles were constructed to populate an asymmetric 12x12 payoff matrix (Figure 1B, right panel) with gains between 10 and 32 (increments of 2) and losses between 5 and 16 (increments of 1). Each gamble was presented once, with the order of gamble presentation randomized between participants. On a given trial, once the gamble was presented, participants had 3 s to accept or reject the gamble via respective button presses. Although in previous studies participants gave responses indicating the strength of their decision[35,39], we collected binary responses (accept/reject) only. The rationale for this decision was that responses under time pressure

may bias individuals towards using more extreme responses[56] and that previously reported analyses were commonly conducted for collapsed (binary) responses[35,39], thus we expected no substantial benefit from adopting more fine-grained response options. Participants completed two runs with a short pause in-between, each run featuring 72 gambles. Jitters were introduced between trials (mean inter-trial interval = 4.32 s, range = 1–11 s).

For every individual we computed the proportion of accepted gambles out of all gambles for which a response was provided as a risk-preference index. A simple model that captures the sensitivity to gains versus losses has been used to capture decision-making for monetary gambles[35,36,39]. However, the critical parameter of this model, loss aversion, was highly correlated with the proportion of accepted gambles (r = -0.9, p < 0.001). Consequently, we relied on the proportion of accepted gambles as a simpler and generic index of risk preference in all subsequent analyses.

Experimental procedure

Participants who had previously completed the laboratory session of the BBRS were contacted via phone and informed about the MRI follow-up study. Interested individuals were screened for any contraindications regarding MRI safety. For the current analyses, we did not link participants' data from the laboratory and MRI session, and only used data collected during the MRI session. At the time of the MRI session, individuals completed a 2-min training run for the BART and monetary gambles before entering the scanner. The scanner protocol took 75 minutes and included a high-resolution structural scan, two functional sequences for the BART, two functional sequences for monetary gambles, a resting state sequence and a diffusion-weighted imaging sequence. For the current study, only the high-resolution structural scan and the functional sequences were utilized, with the structural scan only serving normalization purposes during preprocessing of functional imaging data. The resting-state and diffusion-weighted sequences were not part of the current analysis and are therefore not discussed further. The order of scanner sequences was fixed, the BART

preceding the gambles task. The risk-taking paradigms were presented using E-Prime 2.0 software (Psychology Software Tools, Pittsburgh, PA), and responses inside the scanner were collected via a COVILEX response box system (series 1.X, Magdeburg, Germany) using the right-hand index and middle finger.

After the MRI session, individuals reported demographic data and completed additional measures reported above. Individuals received a fixed fee of 25 Euro for their participation. In addition, individuals could increase their earnings based on performance in the two scanner paradigms. For the BART, participants received 0.05 Euro for each successful pump on a balloon that was cashed out, i.e. did not explode. For monetary gambles, one trial was drawn at random and, if the participant had accepted the trial, was played out. The resulting loss or gain was combined with money made in the BART. Trials which were drawn but which the participant had rejected resulted in a 0 Euro outcome. Participants were told about the incentive structure at the start of the MRI session and received cash earnings at the end of the session (average actual payment = 41.50 Euro, SD = 14.50 Euro).

Statistical analysis of behavioral data

First, we aimed to assess whether the behavioral patterns obtained for the two measures matched those found in past work, and to identify whether specific aspects of the paradigm (e.g., balloon types in BART, explosions[14,40]) need to be considered in neuroimaging analyses. Individuals' trial-by-trial risk preference in the two fMRI paradigms was examined using mixed-effects regression analyses. For the BART, number of pumps in a given trial was regressed onto average effects of balloon capacity (12/20), whether the previous trial ended in an explosion, age and sex, allowing for random effects for balloon capacity and previous explosion (nested within individual). Control balloons were not included in the mixed-effects modeling, as these merely constitute baseline balloons for the neural analyses and do not offer any insight with regards to decision-making in the BART.

For monetary gambles a logistic mixed-effects model was specified, in which the binary choice outcome (reject/accept) of a given trial was regressed onto average effects of magnitude of the gain, magnitude of the loss, age and sex, as well as individual effects for gain and loss magnitude.

Before running the models, all continuous variables were normalized and categorical variables dummy-coded. In the BART, number of pumps was normalized separately for each of the two experimental balloon types.

All behavioral analyses were run in R (R Project for Statistical Computing; RRID:SCR_001905 http://r-project.org), using the packages lme4 (lme4: Linear mixed-effects models using Eigen and S4; R package v 1.1–8; http://CRAN.R-project.org/package=lme4) and lmerTest (lmerTest: Tests in linear mixed effects models; R package v 2.0–25; http://CRAN.R-project.org/package=lmerTest). We used the functions lmer and glmer for the mixed-effects models of continuous and binary outcome variables, respectively. To obtain p-values for the fixed-effects test statistics in lmerTest, the calculation of the denominator degrees of freedom adopts Satterthwaite's approximation (cf. SAS proc mixed theory).

Behavioral and survey data collected during the MRI session, as well as R analyses scripts are accessible via the Open Science Framework (LINK FOR REVIEWERS).

MRI data acquisition and image preprocessing

Neuroimaging data were collected at the Magnetic Resonance Imaging Laboratory at the Max Planck Institute for Human Development (Berlin, Germany) on a 3T Siemens MRI system with 12-channel head coil. Participants received a magnetization-prepared rapid gradient echo (MP-RAGE) sequence (repetition time = 2500 ms, echo time = 4.77 ms, inversion time = 1100 ms, flip angle = 7 degrees, field of view = 256 × 256 mm^2, 192 slices, voxel size = 1 × 1 × 1 mm^3). In each of the four functional runs, up to 320 functional T2*-weighted BOLD echo-planar images were acquired for every person (repetition time = 2010

ms, echo time = 30 ms, flip angle = 78 degrees, field of view = 192 × 192 mm^2, voxel size = 3 × 3 × 3 mm^3, 33 transversal slices/volume with 15% distance factor). Resting-state and diffusion-weighted imaging sequences were not part of the current analyses, hence are not specified here.

Image preprocessing and analyses were carried out using standard procedures implemented in SPM 8 (http://www.fil.ion.ucl.ac.uk/spm/software/spm8/; cf. Penny et al., 2011). Preprocessing involved realignment and co-registration of functional to structural volumes. Volumes were nonlinearly warped into standard stereotactic (MNI) space based on structural scans using the New Segment method (Ashburner, 2008). To control for spatial noise and average effects that may arise as a function of residual anatomical differences between subjects, images were spatially smoothed using an 8-mm full-width half-maximum Gaussian kernel.

fMRI model specification

At the level of the individual, we concatenated the two runs collected for each of the two risk-taking paradigms, and specified one general linear model (GLM) for BART and one for monetary gambles (see details below). Activation parameter estimates were obtained by convolving event onsets with a canonical hemodynamic response function, filtering out of low-frequency components of the time-series data above 128 s (considered to be noise), and correcting for further temporal error autocorrelation by pre-whitening the data using an AR(1) model (cf. Henson, 2003). Movement parameters were entered as covariates. Given the current focus on neural correlates of decision making under risk rather than correlates of anticipation or feedback-related processes, all analyses involved modeling the time from trial onset (i.e., display of stimulus) to choice.

BART. To model the neural activation in response to experienced risk in the BART, we specified a first level design matrix for each individual which included the following regressors per run (see Figure S1 for an exemplary design matrix): Onset vector of pumps for

control balloons, two onset vectors for pumps on reward balloons, onset vector for cash outs, onset vector for explosions, and six motion parameters estimated during the realignment process. The design matrix was set up to estimate activation differences across the two BART runs. The two onset vectors for pumps on reward balloons differentiated between pumps on balloons that matched the trial history of cash-out decisions, and the second vector captured all remaining pumps on reward balloons. Including an onset vector with only those pumps that matched the number of preceding pumps observed for cash out decisions was deemed necessary to account for the fact that cash out decisions may happen earlier on in the trial, thus a contrast should attempt to match the time point at which a pump/cash out happened. We did not differentiate between onsets for high- and low-capacity balloons because preliminary analyses in which we contrasted pumps on high-capacity with pumps on low-capacity balloons yielded no significantly different neural activations as a function of balloon type; consequently, we collapsed pumps across high- and low-capacity balloons for all analyses. It was not possible to incorporate onset vectors for the two balloon types in the main analyses because for some individuals this resulted in empty onset vectors for cash out or explosion events. The onset vector for explosions was included in order to account for additional variance, better isolate the main effects of interest, and also remove neural responses to explosions from baseline activity.

For our main contrast of interest—risky versus safe decisions—we contrasted cash-out decisions with matched pumps, using the contrast weights [0 1 0 -1 0 0 0 0 0 0 0] to assess Pumps (matched) > Cash out, and [0 -1 0 1 0 0 0 0 0 0 0] for Pumps (matched) < Cash out. Neuroimaging analyses of BART data usually involve contrasting activation differences in response to pumps on risky balloons with pumps on control balloons[25,26,52]. This procedure, however, does not address the question of risk preference directly because it merely contrasts activation for conditions with and without a decision component, and thus provides a general picture of the neural correlates of decision-making, but not risk preference. The ubiquity of

contrasting risky and control pumps in the BART in the literature, however, allows for a direct comparison of group-based results originating from different studies. Thus, we supplemented our focal analysis with a contrast of all pumps on risky versus control balloons, using the contrast weights [-2 1 1 0 0 0 0 0 0 0 0] to compute Control pumps < Reward pumps, and [2 -1 -1 0 0 0 0 0 0 0 0] to compute Control pumps > Reward pumps.

Monetary gambles. For the individual-level modeling of monetary gambles decisions, we specified one GLM, which targeted the neural representation of risky versus safe decisions[36] and included the following regressors (see Figure S2 for an exemplary design matrix): Onset vector for all accept decisions, onset vector for all reject decisions, six motion parameters estimated during the realignment procedure. The design matrix was set up to estimate activation differences across the two runs of monetary gambles. The simplicity of the paradigm allowed for this comparatively straightforward design matrix with only two regressors of interest, nevertheless yielding clean (event-unrelated) baseline activity. Emulating previous analyses[36] and striving for a contrast analysis that is comparable for risk in both BART and monetary gambles, individuals' *Accept* decisions were contrasted with *Reject* decisions, using the contrast weights [1 -1 0 0 0 0 0 0] for Accept > Reject, and [-1 1 0 0 0 0 0 0] for Accept < Reject.

At the level of the group, we specified a flexible factorial design with subject and paradigm as separate factors in order to obtain statistical parametric maps for mean activation patterns in the two measures and compute a conjunction (see Figure S3 for design matrix). Within-subject contrast images from risky versus safe decisions in monetary gambles and the BART were entered as two blocks, one block per measure. We assumed independence for the subject and paradigm factors, but assumed equal variance only for the subject factor. Gender and age were entered as covariates of no interest.

All initial contrast analyses were conducted at the level of the whole brain. Accounting for multiple comparisons, a cluster-forming threshold ($p<.001$, uncorrected) was

applied, followed by family-wise error correction at peak level ($p<.05$) to account for multiple testing. To avoid putting too much emphasis on potentially uninformative single activated voxels, we applied an extent threshold of a minimum of 100 contiguous voxels for all whole-brain group-level analyses. As we were agnostic regarding the potential overlap of voxels activated by both fMRI paradigms, we removed the extent threshold from our conjunction analysis. We report voxel coordinates in MNI space (mm) and also report the extent of the cluster within which the voxel is located. For sake of brevity, we report only peak voxel coordinates with the highest t-value in the associated cluster. Anatomical labels were obtained from the Neuromorphometrics Atlas in SPM8. Results are displayed on a customized study-specific group template, which we created by averaging all normalized structural volumes of all participants.

Statistical analyses of fMRI data

Three main goals drove the specification of our fMRI analyses. First, we wanted to test whether we could find group-level neural activation common to both paradigms as a function of risk (i.e., pumping relative to cashing out in the BART; accepting relative to rejecting an offer for monetary gambles), and to see if these map onto "risk matrix" regions. For this purpose, we conducted a conjunction analysis of risky versus safe decisions in the BART and monetary gambles following standard implementation routines in SPM. Specifically, we performed a conjunction analysis over 2 orthogonal contrasts which tested the conjunction null hypothesis rather than the global null hypothesis, allowing us to infer a conjunction of two effects (risky versus safe in experienced and described risk) at significant voxels[57]. We used visualizations of group maps for BART and monetary gambles to establish whether average brain activity for contrasts of interests were comparable to published functional brain maps and whether our paradigms could capture typical neural reactions to risk[25,26,35,39].

Second, we wanted to assess whether individual differences in risk processing were

consistent across measures. Recall that common activation in response to risk at group-level is not necessarily synonymous with consistent individual differences: even if the majority of individuals shows comparable patterns in each measure, this majority does not need to be made up of the same individuals. For this purpose, we extracted mean beta values from risky versus safe contrast images obtained for individual-level analyses of the BART (pumps versus cash out) and monetary gambles (accept versus reject) using regions of interest (ROI), and then conducted correlational analyses between the neural indices of the two measures (brain–brain associations). The ROIs were informed by brain regions previously implicated in risk processing[20], namely bilateral nucleus accumbens (NAcc), bilateral insular cortex, and anterior cingulate cortex (ACC), and potentially any other regions revealed by conjunction analyses to be implicated in risk taking in BART and monetary gambles. The ROIs were structurally defined based on the Hammersmith atlas nr30r83 (http://brain-development.org/brain-atlases/adult-brain-maximum-probability-map-hammers-mith-atlas-n30r83-in-mni-space/).

Third, we aimed to examine the explanatory power of experience- and description-based risk-related neural activation for risk-taking behavior, both within and across measures. To this end, we conducted two whole brain multiple regression analyses, modeling whether individual differences in the neural response to risky versus safe decision-making (1) in the BART was associated with mean number of pumps, and (2) in monetary gambles was associated with proportion of accepted gambles. To establish whether the explanatory power of neural activation differences is measure-invariant, we conducted a third whole-brain multiple regression analysis, examining whether neural signal in response to risk in the BART was predictive of proportion of accepted gambles. Given the temporal order of the two measures, we did not test whether neural signal in monetary gambles accounted for BART behavior.

All analyses controlled for age and gender. Following-on from whole brain analyses,

we conducted brain–behavior associations focusing the ROIs described above. These additional ROI analyses were conducted (1) to further probe the results obtained from whole brain analyses given the ROIs' a priori importance for risk-related processing[20], and (2) because the expected effect sizes are likely to be modest and may not survive stringent whole brain correction thresholds. For this purpose, we estimated brain–behavior associations by means of linear regression analyses with standardized variables, yielding partial correlation coefficients for links between paradigm-specific mean beta values extracted from the ROIs and behavioral indices of risk preference in the BART (mean number of pumps) and in monetary gambles (proportion of accepted gambles).

Initial plotting of mean beta values extracted from ROIs indicated relatively normally distributed mean signals for both measures, except for a small number of possible outliers for signals extracted from ACC (n = 2) and insula (n = 1) in the BART, and ACC (n = 1) in monetary gambles. To account for any biasing effects, we computed robust regression analyses ("rlm" function in R package MASS using method "MM"; Venables and Ripley, 2002) and obtained a correlation coefficient of $r = 0.97$ ($p < 0.001$) between the coefficients from standard and robust analyses. Consequently, we only report estimates obtained from standard regression analyses. Results from ROI analyses were not confounded by laterality because similar findings were obtained from analyses extracting mean beta values from the two hemispheres separately. Concatenating the two runs from each paradigm to compute one neural index did not bias the results; comparable findings were observed for supplemental ROI analyses based on two separate runs per measure.

To control for the number of analyses conducted, we report which of the associations reach significance thresholds after family-wise error correction. For this purpose, we define four families of tests: (1) brain–brain associations (three tests); (2) brain–behavior associations for BART (four tests; one whole brain multiple regression analysis and three regression analyses based on extracted mean beta values from ROIs); (3) brain–behavior

associations for monetary gambles (four tests; one whole brain multiple regression analysis and three regression analyses based on extracted mean beta values from ROIs); and (4) brain–behavior associations across the two measures (four tests; one whole brain multiple regression analysis plus three regression analyses based on extracted mean beta values from ROIs).

ROI data and corresponding R analyses scripts are available from the Open Science Framework (LINK FOR REVIEWERS). Uncorrected group-level maps are available via NeuroVault (LINK FOR REVIEWERS). We can provide access to individual-level neuroimaging data and SPM/Matlab scripts upon request.

Results

Behavioral results

Group-based descriptive statistics for behavior in the two fMRI paradigms are given in Table 1. Collapsed across both types of risky balloons, number of pumps in the BART was approximately normally distributed (mean = 4.98, SD = 1.05; Figure 1C, left panel). As reported previously, participants generally showed risk-averse behavior in the BART, indicated by the mean number of pumps for the low-capacity (mean = 4.45, SD = 1.06) and high-capacity (mean = 5.50, SD = 1.52) balloons falling below the optimal mean number of pumps (6 and 10, respectively). Results from the mixed-effects modeling of the BART (Table 2) suggested main effects of gender (b = -0.16, SE = 0.08, p = 0.04) and previous explosion (b = -0.14, SE = 0.03, $p < 0.001$). As expected, the mean number of pumps was lower for low-capacity (mean pumps = 4.45, SD = 1.06) than high-capacity (mean pumps = 5.50, SD = 1.52) balloons (cf. Schonberg et al., 2012), yet this difference did not translate into a significant main effect of balloon capacity (b = 0.03, SE = 0.06, p = 0.70). The main effect of previous explosion was not informative for the planned fMRI analyses, but falls in line with previous studies showing downward adjustment of pumping immediately following an explosion trial[14,58].

Risk preference in monetary gambles—indexed by the proportion of gambles accepted—was approximately normally distributed (Figure 1C, middle panel). The results from the mixed-effects logistic regression model for monetary gambles yielded a main effect of age (b = -0.60, SE = 0.09, $p < 0.001$), gender (b = -0.37, SE = 0.19, p = 0.04), magnitude of gain (b = 0.39, SE = 0.02, $p < 0.001$) and loss (b = -0.84, SE = 0.03, $p < 0.001$) on individuals' decisions to reject or accept a risky gamble (Table 3).

Examination of risk preference across the two measures revealed a lack of consistency at the level of the individual because proportion accepted in monetary gambles was not significantly associated with mean number of pumps in the BART (r = -0.11, p = 0.24; Figure 1C, right panel). The lack of behavioral consistency was not a result of combining the two runs to compute one behavioral index for each task, as risky choice was consistent over the two runs in monetary gambles (r = 0.86, $p < 0.001$) and the BART (r = 0.63, $p < 0.001$).

Neuroimaging results

Group-level analyses.

In the BART, taking a risk (decisions to pump) versus going safe (decisions to cash out) was associated with increased activity in striatum (specifically bilateral NAcc), left anterior insula, and right precentral gyrus, extending into supplementary motor cortex (Table 4, Figure 2A); results for this contrast are comparable with previous results[27]. Due to the various cognitive and visual aspects surrounding cash-out decisions, examination of the reverse main effect revealed widespread bilateral decreased activity, particularly in thalamus extending into hippocampal and parahippocampal regions and lateral occipital cortex. Because of the very short temporal delay between cash-out decisions and the subsequent visual feedback (~ 1 s), inclusion of the onset and duration of the visual feedback for cash-out decisions in the GLM did not achieve a more localized cash-out signal. Replication analyses of average activation differences for pumps on risky versus control balloons yielded results comparable with those of previous studies[25,26], including increased activation for peak

coordinates located in bilateral ventral and dorsal striatum, bilateral anterior insular cortex, inter-hemispheric anterior cingulate and prefrontal cortex, as well as decreased activation in inter-hemispheric ventromedial prefrontal cortex, posterior cingulate and posterior parietal cortex, and bilateral parahippocampal gyrus and posterior insula (Table 4).

For monetary gambles, decisions to accept a risky gamble, when compared with decisions to reject, were associated with increased activation in several neural regions, including peak coordinates located in bilateral caudate extending into NAcc, right ACC, left angular gyrus, left inferior temporal and frontal gyrus (Table 4, Figure 2B). Examination of the reverse main effect yielded no significant deactivation. The pattern of activations found is compatible with those found in similar measures involving decisions from description[23,36,59].

One of our main goals was to examine the overlap of neural activation differences in response to experienced and described risk. A conjunction analysis of activation differences in response to risky versus safe options in the BART and monetary gambles revealed a common risk signal in the ventral striatum (Table 4). As can be seen in Figure 2C, joint activation differences are locally restricted to a small portion of the ventral striatum, the nucleus accumbens. Thus, on average, taking a risk seems to elicit a localized, measure-invariant neural signal in nucleus accumbens.

To summarize, at the level of the whole brain, group-level differences obtained for experienced risk in the BART and described risk in monetary gambles were in line with previous studies. Crucially, consistency of group-level activation differences across the two paradigms was observed for jointly increased activation in NAcc in response to risky versus safe decisions. Next, we turn to individual-level analyses to investigate if individual differences in the neural response to risk are preserved across the two measures, and to examine their explanatory power for risky choice.

Individual-level analyses.

Consistency of neural activation across paradigms. In a first step, we examined

whether the NAcc being a conjunction region for experience- and description-based risk activation means this region is informative for individual differences by examining the consistency of neural signal across measures. Contrary to what might be expected, mean activation in NAcc in the BART was not significantly predictive of NAcc activation in monetary gambles (Table 5, Figure 3A). Thus, although at the level of the group the two measures converged on NAcc activity, individual differences were not preserved across measures. In other words, we found group-level but not individual-level consistency for experience- and description-based risk-taking[28].

In a second step, we also examined the consistency of the neural signal in the remaining "risk matrix" regions. Mean activation in both insula and ACC in the BART were significantly predictive of activation differences in ACC and insula in monetary gambles, respectively; these associations, however, were small and negative rather than the positive correlations required to suggest consistency (Table 5, Figure 3A). The ROI-based results remained significant after application of correction thresholds (FWE) for the number of tests. Thus, although we observed associations between individual differences in regional neural activations in the two paradigms, we did not find consistency.

Explanatory power of risk-related neural signal for risk preference within and across paradigms. To present a useful target for intervention, neural indices should hold some explanatory power for behavior or critical outcomes, at least for the measure from which they were derived and ideally even across measures. In a first step, we used whole-brain analyses supplemented by ROI analyses for "risk matrix" regions to examine whether activation differences in the BART were predictive of mean number of pumps, and whether activation differences in monetary gambles were predictive of proportion of accepted gambles. For the BART, whole brain as well as ROI analyses revealed no significant associations between risk-related activation differences and performance as measured by mean number of pumps (see Table 5, Figure 3C for results from ROI analyses). In contrast, for monetary gambles, whole-

brain analyses revealed a set of neural regions for which the risk-related signal was associated with the proportion of gambles an individual accepted, including positive associations in voxel clusters in bilateral occipital pole, central operculum, and superior temporal gyrus, as well as negative associations in bilateral anterior insula, supramarginal gyrus, middle cingulate gyrus and inferior temporal gyrus (Table S1). ROI-analyses further supported the involvement of "risk matrix" regions in predicting choice in monetary gambles. Specifically, mean activation in NAcc, insula and ACC extracted from *Accept* versus *Reject* decisions in monetary gambles was significantly negatively associated with the proportion of risky gambles accepted (all $p < 0.001$; Table 5, Figure 3B). The links between neural signal and behavior in monetary gambles remained significant after controlling (FWE) for the number of tests conducted.

In a second step, we were interested in brain-behavior associations across measures, that is, whether activation differences in BART were predictive of risky choice in monetary gambles. Whole-brain analyses did not reveal a significant brain-behavior association across measures. Only when we probed ROI-specific neural signal did we observe mean activation in the ACC to be significantly positively associated with the proportion of gambles accepted ($p = 0.01$; Table 5; Figure 3D), which is suggestive of control and monitoring processes in the BART to account for some variance in choice in monetary gambles.

Discussion

In this study, we investigated the neural basis of risk taking under experienced and described risk. Specifically, our aim was to examine (1) commonalities and distinctions between the neural correlates of experienced and described risk, (2) the consistency of individual differences in the neural response to experienced and described risk, and (3) the explanatory power of neural indices of experienced and described risk for behavior. We investigated these propositions by focusing our analyses on group as well as individual activation differences in response to two risk-taking measures, namely the BART and

monetary gambles, which, respectively, are prototypical measures of experienced and described risk.

Average activation differences for experienced and described risk

At the level of the group, we replicated previously published activation differences for risky versus safe choices under experienced and described risk[25–27,35,36,39,52]. One of our main aims was to address the functional overlap of decisions made under experienced and described risk. Comparative examination of average activation differences in the two paradigms revealed bilateral NAcc as a source of jointly increased activation for risky versus safe decisions. Our conjunction results in NAcc support previous work[27] which identified striatal and insula activation to jointly increase for reflective and impulsive risk taking. The striatum in general has been implicated in reward processing[60,61], and if we consult neurosynth (neurosynth.org) to establish a reverse inference of process given location using meta-analytical procedures, the highest posterior probability for a cognitive process given our peak conjunction coordinates was indeed observed to be for reward processing (posterior probability= 0.81). In principle, the observed joint NAcc activation differences for risky versus safe decisions in BART and monetary gambles are not surprising. The motivation for risk-taking behavior lies in the potential for reward, and in the two measures used in this study, risk and reward always coincided; we return to this issue in the study limitations. Thus, we cannot completely isolate risk signal from reward, especially since both risk and reward have been found to be encoded, albeit temporarily differentiated, in striatum[60].

An alternative explanation for a common NAcc signal for experienced and described risk is the role of the ventral striatum in the coding of prediction error. The brain computes several mutually-informative choice-relevant signals (including goal value, decision value, and prediction error), and dissociation of these signals by means of a bespoke MRI task suggested ventral striatal activation to code prediction error instead of goal or decision value[62]. Prediction error is of course based on the comparison of expected and achieved

outcomes, and the structural as well as functional interconnectedness of different brain regions implicated in the computation of different choice-relevant signals suggests that ventral striatal activation is not devoid of a value signal. However, if we want to understand commonalities between different risk-taking measures, especially in the search for endophenotypes suitable for intervention, it is important to distinguish between these different decision components. Our measures do not allow us to disentangle different choice-relevant signals, leaving open the possibility that, instead of signaling reward processing, the main commonality between experience- and description-based risk taking may be comparison of the current option with the status quo.

In contrast to previous results[27], we obtained no further conjunction regions for the two tasks, including insular cortex. The insula is heavily implicated in signaling subjective feelings, interoception, and explicit motivation[63], and as such is thought to inhibit risky choice[20]. In this study, group-level activation differences in insula were observed for BART but not monetary gambles, supporting the argument that experienced risk involves potentially more affective and motivational processes compared with described risk[1,16]. To note, in the study comparing activation differences in the BART and Game of Dice Task[27], described risk on average activated the insular cortex. This insula-based discrepancy between the current and previous work emphasizes that even two seemingly similar measures of described risk do not necessarily result in overlapping neural signals, given comparable contrast analyses.

Individual-level consistency of activation differences for experienced and described risk

Given that averages are not necessarily reflective of individual-level behavioral[28,29] or even neural[30] patterns, we examined whether individual differences in neural activation are preserved across our two measures. Interestingly, we observed a lack of consistency of individual differences in neural activation for risky versus safe decisions under experienced and described risk. On aggregate, joint activation increases were localized in NAcc, but individual differences in NAcc activation were not preserved from BART to monetary

gambles. Examination of further regions previously identified as core functional correlates of risk and risk preference[20], i.e. insula and ACC, also failed to yield consistent (that is, positively correlated) individual differences across measures. From these results we take home that it is important to recognize that individuals respond very differently to different risk-taking measures, both behaviorally[13] and neurally, hence it is unrealistic to expect individual differences in neural function observed in one measure or context to be informative for neural function in another measure or context.

Explanatory Power of Neural Indices for Behavior

Our third major aim was to examine the explanatory power of neural activation differences in response to experienced and described risk for risky choice. In particular, we were interested in the extent to which activation differences previously suggested as differentially promoting (NAcc), inhibiting (insula) and controlling (ACC) risky choice[20] might play a different role for brain-behavior associations using indices related to experienced or described risk.

In a first step, we examined the explanatory power of neural indices for behavior within measure, and our results first and foremost suggest explanatory power to vary as a function of risk measure. In the BART, whole-brain and ROI analyses converged on activation differences in response to risky versus safe decisions not being predictive of mean number of pumps. In contrast, for monetary gambles, whole-brain and ROI analyses indicated activation differences in bilateral nucleus accumbens, insula, and ACC in response to risk to be predictive of proportion of accepted gambles. Based on the strength of the associations obtained, the strongest predictor of description-based risky choice was NAcc activation, followed by ACC and insula activation. Considering the proposed roles of NAcc, insula and ACC in the promotion, inhibition, and control of risky choice[20], the observed associations for ACC and insula were in the expected negative direction; the more affect-based inhibition and control-related processes are experienced, the lower the number of risky gambles that are

accepted. The negative association between NAcc activation and risky choice in monetary gambles is informative, if we recall that for monetary gambles we used a quantitative index of risky choice (proportion accepted gambles), and that our neural index is an average signal over all *Accept* decisions, meaning individuals with an on average lower NAcc signal accepted more risky gambles. It is possible that this association is a corollary of our payoff matrix not being calibrated to individuals, which may result in choice being less discerning for those who place a similar subjective value on all gambles. Future research could easily remedy this issue by calibrating payoff matrices, for instance via an adaptive willingness-to-pay measure.

In a second step, we were interested in whether brain-behavior associations could be established across measures, that is, whether neural activation differences in the BART are predictive of choice in monetary gambles. Whole brain analyses revealed no brain-behavior association across experienced and described risk; our results are in line with previous findings[27] indicating no association between neural and behavioral indices originating from different risk-taking measures. Additional ROI analyses identified a link between ACC activation in the BART and proportion of accepted gambles. However, we refrain from placing too much emphasis on interpreting this link, firstly because whole-brain analyses did not support this association, and secondly because an association across measures involving BART neural indices seems surprising, given the lack of within-measure brain-behavior associations for BART. Instead, we err on the side of caution and treat this association between BART activation and monetary gambles choice as an informative starting point for further investigation, which, if replicated, could pave the way for neural endophenotypes serving as targets for intervention efforts.

Taken together, individual-level analyses of brain-behavior associations within and across measures first and foremost hint at the explanatory power of single neural indices for behavior being measure-dependent. We suggest that this measure-dependent explanatory

power arises as a potential consequence of the specific processes afforded by experienced and described risk[16,41]. In an experience-based, sequential decision-making measure such as the BART, which involves dynamic balloons and initially unknown risk distributions, activation differences in a single region are less likely to be highly correlated with choice, because choice depends on many interconnected processes[1,18,19]. In contrast, the simple, perhaps monotonous nature of description-based monetary gambles lends itself very well to the use of a choice rule, which, at brain level, is evident in choice-relevant neural signal (e.g., a reward signal coding for the attractiveness of a particular gamble). As a consequence, any explanatory links between neural function and risk preference have to be interpreted with caution, for they may be measurement-specific rather than capturing general associations between brain function and risk preference.

Limitations

In this study we adopted two paradigmatic risk-taking measures as examples of experienced and described risk inside the scanner, which limits generalization. However, with regards to the main findings, past research has shown that other tasks do not fare much better regarding behavioral consistency[13,15,27,64], hence it is questionable whether selecting different measures would have resulted in more extensive convergence at group- and individual level, or higher explanatory power for behavior. To allow for a more comprehensive assessment of measure-invariant neural activation at group- and individual level for these two types of tasks, one might be tempted to implement additional paradigms, including further risk-taking measures based on experienced (e.g., Iowa Gambling Task[65] or Columbia Card Task[66]) and described risk (e.g., multiple price lists[67]). Adoption of multiple measures of each type would also facilitate further interesting analyses, including whether the neural differences between described and experienced risk are greater than the differences among different measures of experienced and among different measures of described risk.

Despite our best efforts to create contrasts targeting the neural risk component in BART and monetary gambles, risk and reward may not be easily distinguishable as the two components coincided in both measures. This is a special limitation for contrast analyses which average activation differences over particular events (e.g. *Pumps* or *Accept* decisions). One way to disentangle risk from reward is to use parametric analyses that map activation to specific functional forms, such as increases in risk or reward. Parametric analyses were not applied here because we aimed to compare and contrast BART and monetary gambles, and parametric analyses were in principle only possible for the former but not the latter. Moreover, standard implementations of the BART, like the one used here and elsewhere[7,26,37,38,49], do not allow for the isolation of risk from reward signal even by using parametric analyses, because risk and reward increase linearly over a given trial. Thus, task manipulations are required which can disentangle risk from reward in the BART. One way to do this would be to include non-linear payoff functions but keep a linear risk function, and then (non)linear parametric analyses could be applied to examine (non)linear activation differences.

In designing the study, we faced decisions regarding task order, that is, whether to run a fixed task order or counterbalance task order between participants. We opted for a fixed task order because randomization for some analyses would have required splitting the sample into two groups based on order, thus reducing power. Based on our sample's behavior, it is unlikely that BART preceding gambles prompted strong order effects on risky choice. The overall level of observed risk-taking in BART and monetary gambles was comparable to previous independent investigations[26,35], and risky choice within a task was relatively consistent across the two runs. The correlations between risky choice in BART and monetary gambles also did not change substantially as a function of run number.

Implications

Risk preference, including its related constructs (e.g., self-control) and associated cognitive processes (e.g., reward sensitivity), has been found to be predictive of crucial life outcomes, including psychopathology, health, wealth, and criminality[2–4,7]. As such, individual differences in risk taking and related constructs are also the focus of many developmental and longitudinal research efforts[7,68,69]. Interestingly, much progress has been made identifying the biological basis of risk preference, including its genetic basis[70], hormonal[71,72] and neural pathways[20,22]. In contrast, the extent to which biomarkers such as neural function differ depending on the risk-taking measure and whether they are reliable predictors of behavior is much less understood. To make progress, we make the following three recommendations.

Firstly, our direct comparison of experienced and described risk clearly implies that different risk-taking measures should not be used interchangeably and without a clear rationale for why a particular measure was chosen. There exists a tempting richness of risk-taking measures, and whenever a particular measure is used we recommend that researchers make their selection criteria transparent. On the one hand, increased transparency will help the individual researcher to make more informed choices between different risk-taking measures for their studies. On the other hand, increased transparency should help the research community to establish a taxonomy of risk-taking measures, their core capacities, biological underpinnings, and usefulness for research designs targeting individual differences, including longitudinal and intervention studies.

Secondly, whenever it is feasible to include multiple measures in their design, we encourage researchers to do so. The adoption of multiple risk-taking measures in the same study has two benefits. One, it enables direct comparison of measures for a given sample, which makes prediction analyses across measures possible. Two, and perhaps more importantly, adoption of multiple measures means psychometric models can be applied[13,33,71] which provide insights for our understanding of risk taking as a phenotype, and its dimensionality.

Thirdly, if state-based, task-dependent neural activation indices do not present useful, reliable targets for intervention purposes, perhaps more trait-like endophenotypes such as brain morphometry, structural connectivity, or task-independent functional connectivity measures offer more promising targets[39,73–77]. Multi-modal, multi-measure projects such as BrainTime[7], Adolescent Brain Cognitive Development Study[68], and a research framework aimed at establishing an ontology of self-regulation[69] could provide suitable research designs to address these open questions in the future.

Conclusion

Many longitudinal, clinically relevant and developmental research designs focus on risk preference as a critical predictor or outcome, and often aim to establish links between individual differences in risk preference and neural structure or function[2,4,7,77]. Until recently, neuroimaging studies investigated primarily group-level neural representations of risk and paid less attention to individual differences or measurement convergence. To successfully target individual differences in risk taking and understand the biological underpinnings, a switch is required –especially within neuroscience– from group-level to individual-level research[6,32], and from single to multi-measure research[33]. If the ultimate aim is to help individuals navigate an uncertain, risk-laden world and make better choices, we first need to be prepared to navigate and map the mainly unchartered territory of our risk-taking measures.

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FIGURES

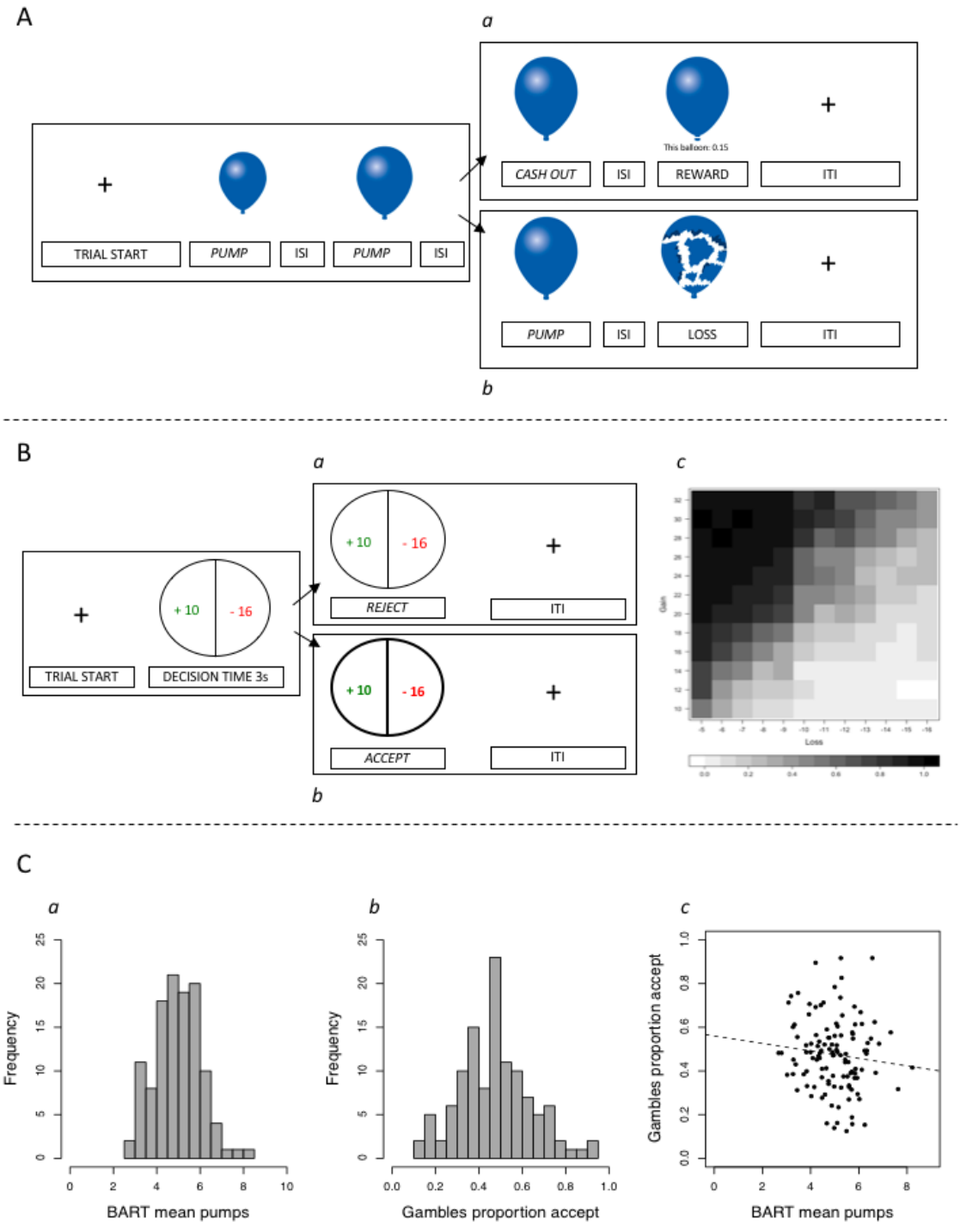

Figure 1. fMRI paradigms and performance. ***A,*** BART. ***a,*** Example cash-out trial. ***b,*** Example explosion trial. ***B,*** Monetary gambles. ***a,*** Example "Reject" trial. ***b,*** Example "Accept" trial. ***c,*** Payoff matrix overlaid with heatmap showing the observed probability of gamble acceptance. ***C,*** Risky choice in the two fMRI paradigms. ***a,*** Distribution of mean number of pumps in the BART, collapsed across

all risky balloons. ***b,*** Distribution of proportion accepted trials in monetary gambles. ***c,*** Association between risky choice in the BART and monetary gambles.

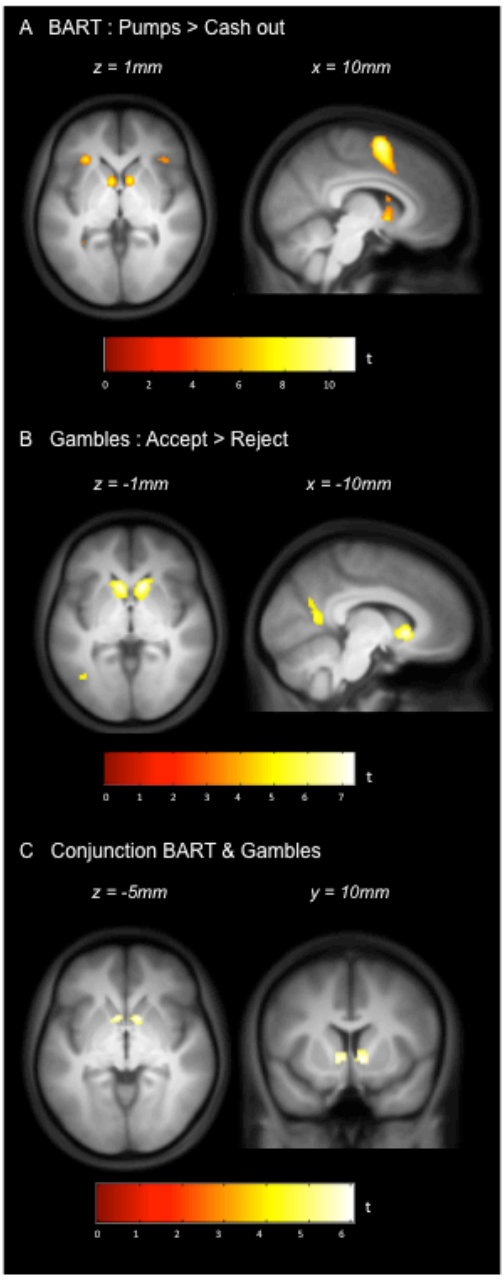

Figure 2. Statistical parametric maps of activation differences obtained for risky versus safe decisions under experienced and described risk. ***A,*** BART, Pumps > Cash out (FWE =.05, k>100). ***B,*** Monetary gambles, Accept > Reject (FWE =.05, k>100). ***C,*** Conjunction of joint increased activation differences in response to risky versus safe decisions in the BART (Pumps > Cash out) and monetary gambles (Accept > Reject) (FWE =.05). Activation differences are displayed on a customized study-group structural template. *Note:* The right (left) side of the image corresponds to the right (left) side of the brain.

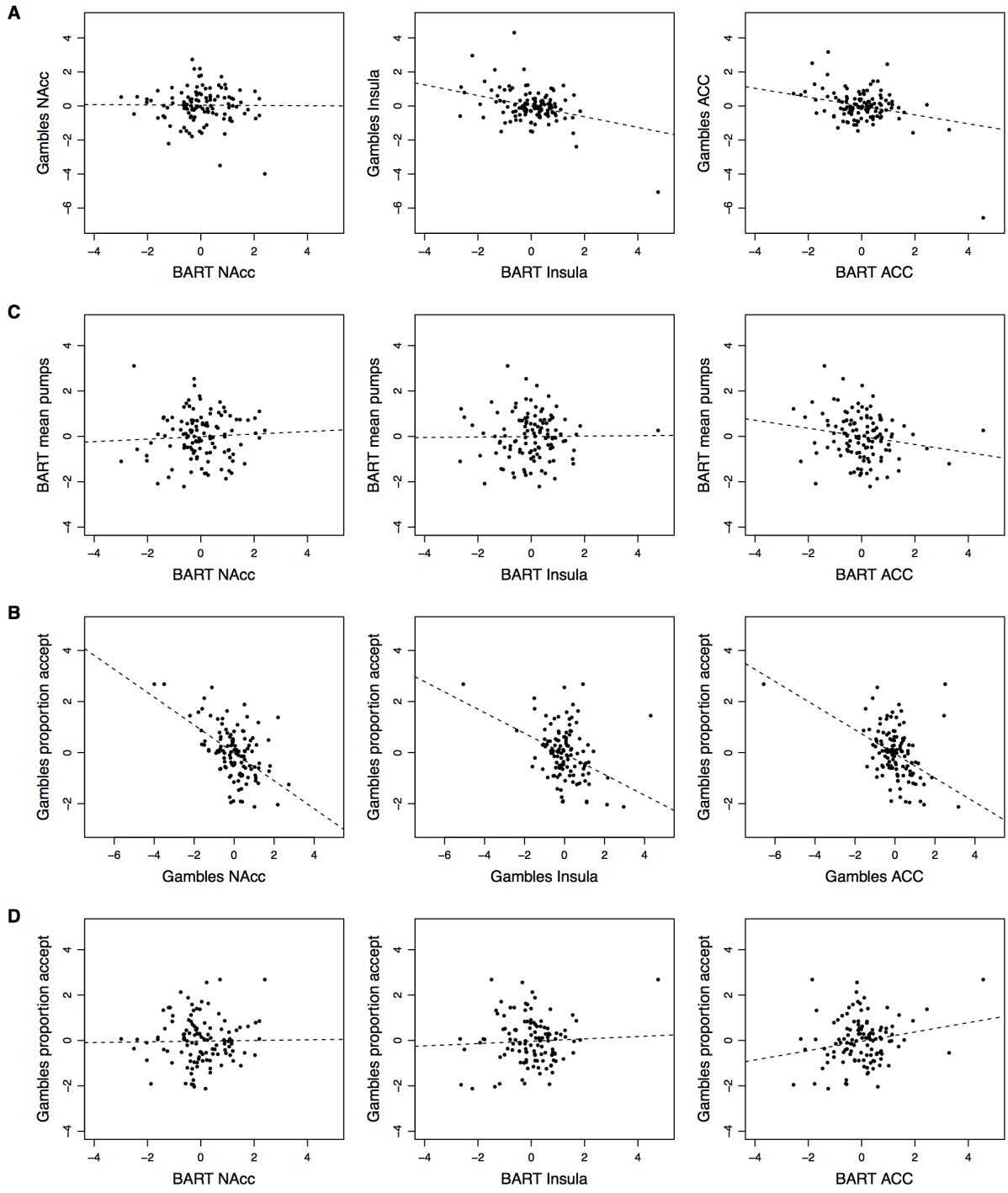

Figure 3. Partial correlations (controlling for age and gender) between mean neural signal extracted from ROIs for BART contrast (Pumps vs. Cash out), mean neural signal extracted from ROIs for monetary gambles contrast (Accept vs. Reject), mean number of pumps in BART, and proportion accepted trials in monetary gambles. ***A***, Association between regional neural signals across measures (brain–brain). ***B,*** Brain–behavior association BART. ***C,*** Brain–behavior association monetary gambles. ***D,*** Brain–behavior association across measures. *Note:* NAcc = nucleus accumbens;

ACC = anterior cingulate cortex. All variables were z-standardized prior to plotting and analysis. Intercepts and slopes were estimated using robust regression analyses.

TABLES

Table 1. Descriptive statistics for outcome measures for the BART and monetary gambles.

Outcome	Mean (SD)	Range
BART		
Number of completed trials (including controls)	60.72 (6.23)	37–73
Number of low-capacity balloons (max. 12)	20.12 (2.12)	12–25
Number of high-capacity balloons (max. 20)	20.25 (2.20)	12–25
Average pumps on low-capacity balloons (max. 12)	4.45 (1.06)	2.40–6.95
Average pumps on high-capacity balloons (max. 20)	5.50 (1.52)	2.25–9.93
Number of explosions experienced	15.81 (3.81)	6–24
Reaction time pumps control (seconds)	0.62 (0.47)	0.002–15.25
Reaction time pumps risky (seconds)	0.71 (0.53)	0.002–15.09
Reaction time cash out (seconds)	0.90 (0.70)	0.27–11.59
MONETARY GAMBLES		
Number of valid responses	142.67 (1.96)	133–144
Proportion accepted gambles	0.47 (0.16)	0.13–0.92
Reaction time accept decisions (seconds)	1.31 (0.47)	0.46–2.98
Reaction time reject decisions (seconds)	1.30 (0.44)	0.07–2.99

Table 2. Mixed effects linear regression model for trial-by-trial number of pumps in the BART.

	Estimate	SE	df	t	p
Intercept	0.16	0.07	136.63	2.44	0.02
Age	0.02	0.04	111.26	0.38	0.70
Sex	-0.16	0.08	111.22	-2.07	0.04
Capacity	0.03	0.06	113.21	0.49	0.63
Explosion on previous trial	-0.14	0.03	111.04	-4.19	< 0.001

Table 3. Mixed-effects logistic regression model for trial-by-trial decision making (0 = Reject, 1 = Accept) in monetary gambles.

	Estimate	SE	z	p
Intercept	0.52	0.15	3.55	< 0.001
Age	-0.59	0.09	-6.35	< 0.001
Sex	-0.37	0.19	-2.02	0.04
Gain	0.39	0.02	25.73	< 0.001
Loss (absolute)	-0.84	0.03	-25.47	< 0.001

Table 4. Significant peak coordinates obtained from group-level contrast analyses for main contrasts of interest in BART and monetary gambles.

Region	R/L	MNI (mm) x	y	z	T	k
BART: Pumps > Cash out						
Supplementary motor cortex	L	-6	-2	60	11.07	2534
Posterior cingulate gyrus	R	24	-42	14	8.26	206
Nucleus accumbens	L	-6	8	-4	7.84	106
Nucleus accumbens	R	8	8	-4	7.83	137
Anterior insula	L	-30	26	4	7.45	177
Anterior insula	R	40	22	6	7.10	170
Posterior cingulate gyrus	L	-14	-34	20	6.56	131
BART: Cash out > Pumps						
Inferior occipital gyrus	L	-38	-76	-12	20.00	100274
BART: $Pumps_{\_Risky} > Pumps_{\_Control}$						
Supplementary motor cortex	R	4	22	40	25.61	49140
Supramarginal gyrus	R	46	-42	44	17.26	5649
Occipital pole	L	-12	-102	-2	15.10	2845
BART: $Pumps_{\_Control} > Pumps_{\_Risky}$						
Angular gyrus	L	-48	-66	22	21.21	50828
Medial frontal cortex	L	-2	58	-12	18.65	5684
MONETARY GAMBLES: Accept > Reject						
Caudate / Nucleus accumbens	R	10	16	-2	7.31	278
Inferior frontal gyrus (triangular part)	L	-44	34	14	7.00	427
Caudate / Nucleus accumbens	L	-8	16	-2	6.94	209
Angular gyrus	L	-32	-72	36	6.89	1182
Inferior temporal gyrus	L	-50	-66	-12	6.21	449
Supramarginal gyrus	L	-46	-40	40	5.93	358
Precentral gyrus	L	-36	4	26	5.83	165
Middle frontal gyrus	L	-24	14	50	5.58	176
CONJUNCTION Pumps > Cash out & Accept > Reject						
Nucleus accumbens	R	8	12	0	6.03	49
Nucleus accumbens	L	-8	10	-4	5.72	36

All analyses whole-brain, with cluster-forming threshold ($p<.001$, uncorrected) and peak-level FWER-correction (extent threshold $k>100$; controlled for effects of age and gender); k = number of voxels in cluster within which peak coordinate is located. Extent threshold not applied to conjunction analysis.

Table 5. Partial correlations (controlling for age and gender) between regional (ROI) neural and behavioral indices of risk preference, computed within and across paradigms.

Analysis	Index	NAcc		Insula		ACC	
		b (SE)	***t (p)***	***b (SE)***	***t (p)***	***b (SE)***	***t (p)***
Brain–brain:	Monetary gambles activation ~ BART activation	-0.06	-0.67	**-0.46**	-5.37	**-0.47**	-5.54
		(0.09)	(.50)	(0.09)	**(<.001)**	(0.08)	**(<.001)**
Brain–behavior: Within paradigms	BART: Mean number of pumps	0.02	0.24	0.04	0.38	-0.17	-1.79
		(0.09)	(.81)	(0.09)	(.70)	(0.09)	(.08)
	Monetary gambles: Proportion Accept	**-0.50**	-5.97	**-0.31**	-3.56	**-0.39**	-4.63
		(0.08)	**(<.001)**	(0.09)	**(<.001)**	(0.08)	**(<.001)**
Brain–behavior: Across paradigms	Proportion Accept ~ BART activation	0.07	0.75	0.15	1.61	**0.24**	2.61
		(0.09)	(.46)	(0.09)	(.11)	(0.09)	**(.01)**

Note: Estimates obtained from linear regression analyses with standardized outcome and predictor variables. For models within paradigms, behavioral outcome paradigms and neural predictors originated from the same paradigm. For models across paradigms, the behavioral outcome originated from monetary gambles, and the neural predictors from BART. ACC = anterior cingulate cortex, NAcc = nucleus accumbens.

SUPPLEMENTARY MATERIALS

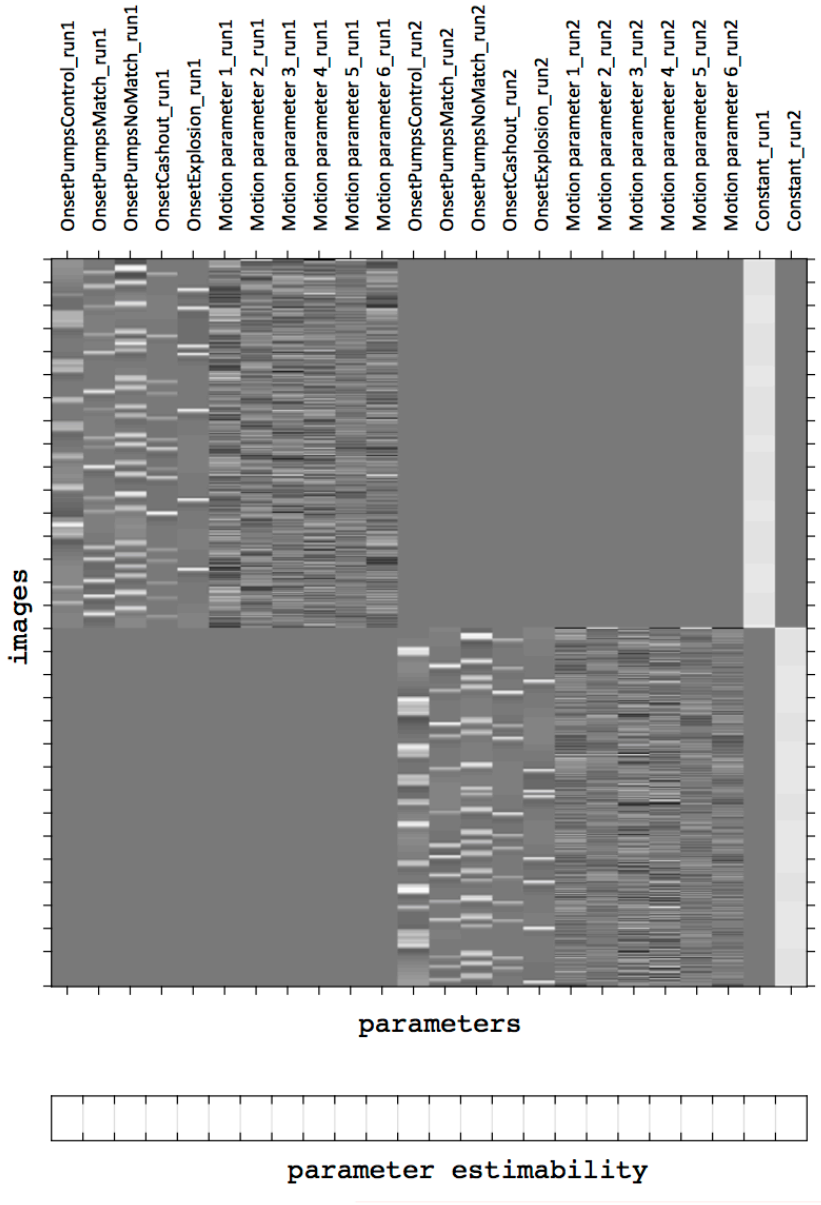

Figure S1. Exemplary SPM design matrix for first (i.e. individual) level modeling of neural activation in BART.

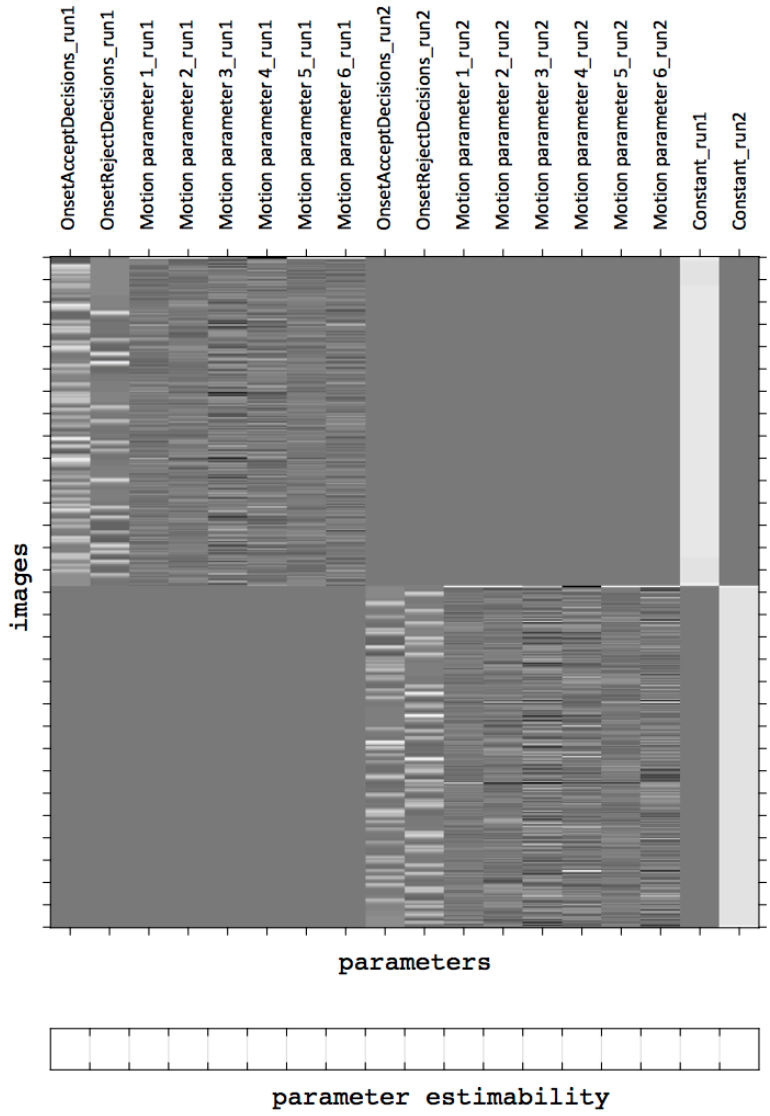

Figure S2. Exemplary SPM design matrix for first (i.e. individual) level modeling of neural activation in monetary gambles.

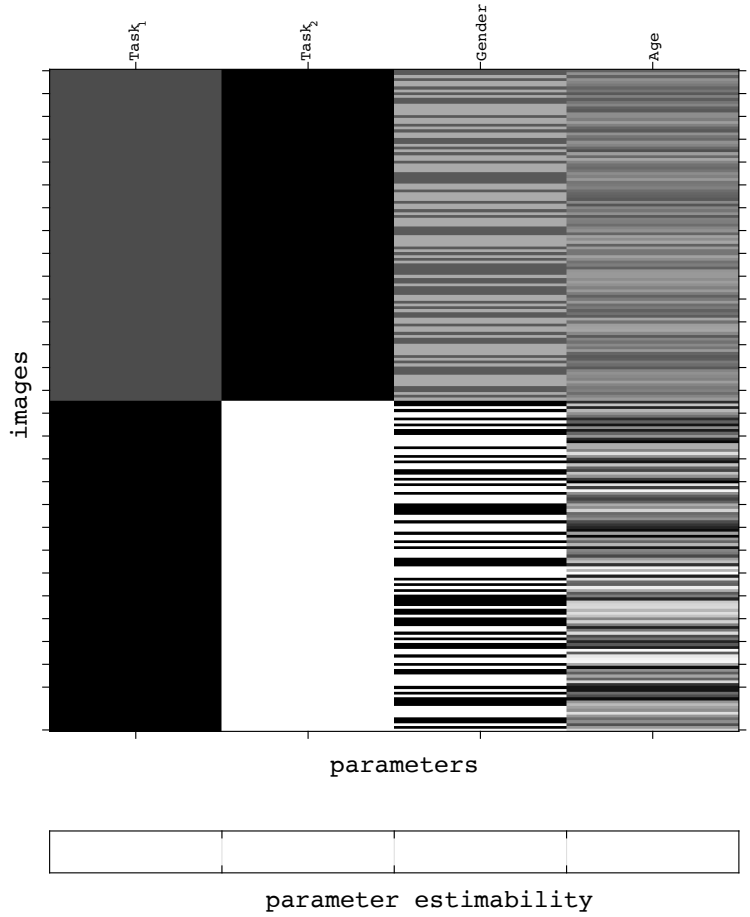

Figure S3. SPM design matrix for second (i.e. group) level modeling of main effects for BART, monetary gambles, and their conjunction.

Table S1. Significant peak coordinates obtained from multiple regression analysis to identify brain–behavior associations for monetary gambles.

Region	R/L	MNI (mm) x	y	z	T	Voxels
Accept>Reject ~ Proportion accepted gambles: Positive association						
Occipital pole	R	24	-96	16	8.39	376
Central operculum	R	40	-12	20	8.06	406
Precentral gyrus	R	26	-22	52	7.85	1017
Occipital pole	L	-26	-96	14	7.23	262
Medial frontal cortex	R	10	48	-14	7.12	358
Middle temporal gyrus	L	-56	-10	-20	6.75	437
Superior temporal gyrus	R	62	-30	12	6.57	205
Parietal operculum	L	-38	-40	18	6.14	101
Superior temporal gyrus	R	60	-8	-6	6.06	153
Accept>Reject ~ Proportion accepted gambles: Negative association						
Anterior insula	R	36	24	-4	12.89	10048
Anterior insula	L	-34	18	-6	11.82	1155
Supramarginal gyrus	R	42	-40	42	11.61	4519
Supramarginal gyrus	L	-50	-38	46	10.60	2888
Middle cingulate gyrus	L	-2	-26	30	8.22	313
Precentral gyrus	L	-52	8	28	7.76	700
Inferior temporal gyrus	R	56	-56	-14	7.36	240
Precentral gyrus	L	-28	-12	52	7.29	321

All analyses whole-brain, cluster-forming threshold ($p<.001$, uncorrected) with peak-level FWER-correction and extent threshold $k>100$, controlled for effects of age and gender; k = number of voxels in cluster within which peak coordinate is located.

APPENDIX D: Manuscript Four

Title: The risky brain: Local morphometry and degree centrality as neural markers of psychometrically derived risk preference factors.

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Author contributions: R.M., D.O., A.H. and R.H. designed the neuroimaging study; R.M., R.H., J.R., R.F., and A.P. designed the laboratory study; L.T., L.H., and R.M. acquired the neuroimaging data; L.T. and A.H. analyzed the neuroimaging data; R.F. and A.P. conducted the psychometric modelling of risk preference; L.T., R.M., A.H., and R.F. interpreted the neuroimaging results; L.T. drafted the first version of the manuscript; L.T., R.F., and R.M. wrote the final version of the manuscript. All authors edited the manuscript.

Conflict of Interest: none

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Abstract

The neural functional correlates of risk-related processes have been mapped extensively, but much less is known about the extent to which neural structure contributes to individual differences in risk preference. Given the life time impact of some decisions made under risk, gaining a better understanding of the biological underpinnings is a worthwhile endeavor which may hold some insights for prevention and intervention purposes. To overcome some of the shortcomings of previous studies, including the use of single indices for both risk preference and neural structure, we combine multi-modal imaging with psychometrically derived risk preference factors in the imaging sub-sample (N=131 young adults) of the Basel-Berlin Risk Study. We focus our analyses on volumetric and connectivity indices for a set of regions identified by Neurosynth-facilitated meta-analytical procedures as core correlates of the term 'risk'. To quantify the robustness of the contribution of individual neural indices, we compliment traditional multiple regression analyses with Bayesian model comparison and selection. Our results suggest that structural indices can account for variance in a general risk preference factor but are not predictive of domain-specific risk preferences. At the level of individual predictors, we observed grey matter volume in ventral striatum to be the most influential predictor of general risk preference, followed by grey matter volume in frontal regions and degree centrality of the amygdala. We did however not observe a role for previously identified indices, including insula and posterior parietal cortex. We discuss our findings in light of the suggestion of the general risk preference factor capturing the trait dimension of risk taking, and address reasons for the observed absence of previously established brain-behavior associations. We also provide a roadmap of methodological improvements for the study of risk taking in particular, and cognitive neuroscience in general.

Keywords: risk preference, brain structure, volume, degree centrality, psychometric factors

Introduction

Risk is ubiquitous, whether it is defined in the economic sense as outcome variance, or the more common notion of the prospect of a reward coupled with the chance of a sizeable loss (Nigg, 2017; Schonberg, Fox, & Poldrack, 2011). Which career should be pursued, stock invested in, or medical treatment selected? Who should one marry, lend money to, or vote for? The answer to any of these and related questions depends, in part, on the decision-maker's willingness to accept risk. Making decisions in a world of uncertain outcomes can be a highly challenging and consequential enterprise. The lifetime impact of individual differences in risk taking and related constructs such as self-control has been demonstrated for (mental) health, wealth, substance use, criminality and general well-being (Aklin, Lejuez, Zvolensky, Kahler, & Gwadz, 2005; Moffitt et al., 2011; Sharma, Markon, & Clark, 2014; Steinberg, 2013), and thus the following question arises: If variability in risk taking influences many critical aspects of a person's life, what influences variability in risk taking?

Individual Differences in Risk Taking

Many factors appear to contribute to intra- and inter-individual differences in risk taking, including gender (Byrnes, Miller, & Schafer, 1999; Wilson & Daly, 1985), age (Cavanagh et al., 2012; Defoe, Dubas, Figner, & van Aken, 2015; Josef et al., 2016; Mamerow, Frey, & Mata, 2016; Mata, Josef, Samanez-Larkin, & Hertwig, 2011), economic status (Payne, Brown-Iannuzzi, & Hannay, 2017), family background (Banducci, Felton, Dahne, Ninnemann, & Lejuez, 2015; Kennison, Wood, Byrd-Craven, & Downing, 2016), peer relationships (Telzer, Fuligni, Liebermann, Miernicki, & Galvan, 2014), reproductive cycle (Sylwester & Pawłowski, 2011), stress (Lighthall, Mather, & Gorlick, 2009), and affective state (Shao & Lee, 2014), to name but a few. More recently, the characteristics of risk-taking measures, specifically behavioral measures, have become a topic of interest. Studies have shown that differences between measures, regarding the format of information, the use of decision strategies, as well as the involvement of various cognitive and affective

processes, can lead to very different choices and consequently different risk profiles (Figner, Mackinlay, Wilkening, & Weber, 2008; Frey, Mata, & Hertwig, 2015; Hertwig & Erev, 2009; Mamerow et al., 2016; Mata et al., 2011; Schonberg et al., 2011; Shao & Lee, 2014).

For many of these factors, the distal mechanisms underlying their association with variability in risk taking are still unclear; nonetheless, biological pathways are frequently invoked as mediating said relationships. The final biological frontier may be to lay bare the genetic architecture of individual differences in risk taking and its mediating role for the association between various individual or measure-specific characteristics. Indeed, twin studies have estimated the genetic contribution to individual differences in risk taking to range between 25% and 50% (Benjamin et al., 2012; Wang, Zheng, Xuan, Chen, & Li, 2016), with as many (or as few) as 611 independent genetic loci associated with risk tolerance and risky behaviors (Karlsson Linnér et al., 2018). However, the complexity of this enterprise, at least for now, means higher-level pathways stand out as promising candidates for studying the biological underpinnings of risk preference and risk taking, including neural pathways.

Neural Structural Correlates of Risk Taking

Activation differences in the brain in response to risk have been extensively mapped, leading to various qualitative and quantitative reviews of the available literature implicating a core set of regions in the functional representation of risk and risk taking, including the striatum, insular cortex, anterior cingulate cortex, dorsolateral and medial prefrontal cortex (Bartra, McGuire, & Kable, 2013; Knutson & Huettel, 2015; Mohr, Biele, & Heekeren, 2010; Platt & Huettel, 2008; Wu, Sacchet, & Knutson, 2012). Much less is known about structural differences that may contribute to the biological basis of individual differences in risk taking. Why might structure play a role for risk taking? For one, because the identification of specific genetic loci that are common to different risk-taking domains (Karlsson Linnér et al., 2018) potentially hints at the existence of genetic predisposition for a general risk tolerance trait. In turn, this genetic predisposition may be expressed not only in how the brain responds and

processes risk ‘on the fly’, but is perhaps also expressed in more stable characteristics of the neural network, such as structural indices.

Grey matter volume. One variable which has received some attention in the past is grey matter volume (GMV). Studies have suggested a positive correlation between varying indices of risk taking and GMV in a range of brain structures, including right insula, amygdala, thalamus, orbitofrontal cortex for boys (Peper, Koolschijn, & Crone, 2013), and right posterior parietal cortex (Canessa et al., 2013; Gilaie-Dotan et al., 2014; Grubb, Tymula, Gilaie-Dotan, Glimcher, & Levy, 2016; Jung, Lee, Lerman, & Kable, 2018; Lin, Lin, & Wu, 2016; Nasiriavanaki et al., 2015). In contrast, there were no differences in local or global GMV between male adolescents classified as high- or low-risk takers, based on either their responses to a self-report inventory or their risky choices on a simulated driving test (Kwon, Vorobyev, Moe, Parkkola, & Ha, 2014).

Research on concrete risky behaviors or relevant psychopathologies has also yielded informative insights into the structural correlates of risk taking. For example, Cheetham and colleagues (2012) observed (lower) orbitofrontal cortex volume at age 12 to predict initiation of cannabis use at age 16. Moreover, in a sample of non-alcohol-dependent Japanese men, GMV in bilateral frontal gyri was negatively associated with lifetime intake of alcohol (Taki et al., 2006). In addition to substance use, research into the structural correlates of behavioral addictions has also provided enlightening findings. For example, GMV is higher in frontostriatal areas of pathological gamblers compared with controls (Koehler, Hasselmann, Wüstenberg, Heinz, & Romanczuk-Seiferth, 2013). For internet gaming disorder, symptom severity and deficits in cognitive control are also correlated with increased striatal GMV (Cai et al., 2015), but lower GMV in insula, anterior cingulate cortex, precuneus, superior parietal cortex, and dorsolateral prefrontal cortex (Lin, Dong, Wang, & Du, 2015).

In summary, GMV seems to be linked with different behavioral indices of risk taking. However, it is currently not known to what extent macro level GMV differences result from

micro level differences in tissue composition. Grey matter is composed of cell bodies, axon terminals, and dendrites, yet it is currently unclear how these neuronal components are linked with endophenotypes such as risk-taking behavior. Moreover, the influence of volumetric differences could also be limited by regional connectivity. Consider a very straightforward example: it has been observed that amygdala GMV is positively correlated with risk tolerance and negatively with anxiety (Jung et al., 2018; Milham et al., 2005). However, if an amygdala-based anxiety-signal cannot proliferate in the neural network due to compromised connections, this anxiety signal will likely be inconsequential. Thus, an understanding of structural connectivity differences may provide a fuller picture of the possible mechanisms underlying individual difference in risk taking.

Structural connectivity. In addition to a significant contribution of amygdala GMV, Jung and colleagues (2018) also found degree centrality (or node strength) of the bilateral amygdala to be predictive of risk tolerance. In the study by Kwon and colleagues (2014), which used a simulated driving task to classify adolescent males as high- or low risk-takers, high risk-takers were observed to have higher integrity of frontal subgyral white matter (WM) than low risk-takers, even though there were no volumetric differences between the two groups(Kwon et al., 2014). WM integrity in a prefrontal cortex-insula-midbrain-striatum network was also observed as being positively correlated with the number of risky choices in the Balloon Analogue Risk Task (Kohno, Morales, Guttman, & London, 2017), whereas lower fronto-limbic WM integrity at baseline was predictive of adolescent substance use and delinquent/aggressive risk-taking behaviors at 18-months follow up (Jacobus et al., 2013).

As with GMV, additional insights can be derived from studies involving specific (psychopathological) study populations. For example, teenagers with a family history of alcohol use disorders are more likely to initiate alcohol consumption during adolescence compared with teenagers without such a family history, and the former have been found to have higher WM integrity for connections between reward (nucleus accumbens) and salience

regions (orbitofrontal cortex) compared with the latter (Squeglia et al., 2015). Moreover, comparison of healthy controls with internet gaming addicts also suggest various differences in WM integrity that correlate with duration of pathology (Jeong, Han, Kim, Lee, & Renshaw, 2016).

In summary, research has started to emerge that studies the connection between the structural architecture of the brain and risk taking, yielding some insights into the involvement of different regions in individual differences in risk taking. Unfortunately, many study designs do not allow for conclusions about structural causes, effects or corollaries of pathology, and caution is warranted when interpreting observed links between neural structural indices and behavior. Furthermore, the aforementioned studies have applied a plethora of risk-taking measures. Recent research has argued that (especially behavioral) risk-taking measures do not correlate (Frey, Pedroni, Mata, Rieskamp, & Hertwig, 2017), which raises the question to what extent brain-behavior associations observed in the literature depend on the risk-taking measure itself. In the current study, we attempt to tackle some of the shortcomings of previous neuroimaging studies by combining psychometrically derived risk preference factors with multi-modal neuroimaging, in order to shed light on the association between indices of neural architecture and the psychometric structure of risk preference.

The Current Study

The current study is a follow-up to the Basel-Berlin Risk Study (BBRS), a large multi-site study of individual difference in risk taking that aims to address questions about the psychometric structure of risk taking and its biological underpinnings (an overview of all subsamples, measures, and further details on the BBRS is reported on https://osf.io/rce7g). One key contribution of the BBRS to date has been the extraction of psychometrically derived risk preference factors from a comprehensive battery of laboratory-based self-report, behavioral and frequency measures of risk taking completed by a large sample of young

adults (Frey et al., 2017). Here, we utilize these psychometric factors in order to circumvent issues of generalization stemming from the availability and (in some cases it could be argued arbitrary, criterion-free) usage of different risk-taking measures.

Risk preference factors versus single measures. Whilst previous analyses of structural brain-behavior associations have relied predominantly on single measures of risk preference (Canessa et al., 2013; Grubb et al., 2016; Jung et al., 2018; Kohno et al., 2017; Lin et al., 2016; Nasiriavanaki et al., 2015; Peper et al., 2013), several reasons stoked our interest in using psychometrically derived factors instead of specific measures.

One reason for using latent variables is that they present a more principled approach to understanding whether neuroanatomy is predictive of individual differences in risk preference, compared with brain-behavior associations based on single indices. A vast number of risk-taking measures exist (Appelt, Milch, Handgraaf, & Weber, 2011; Aven, 2012) and many different measures have found application in neuroimaging studies (Knutson & Huettel, 2015; Mohr et al., 2010; Wu et al., 2012). However, given the low convergence between different risk-taking measures, especially between behavioral measures (Frey et al., 2017; Pedroni et al., 2017) —implying that different measures cannot be used interchangeably and may even yield different life span trajectories (Mamerow et al., 2016; Mata et al., 2011; van den Bos & Hertwig, 2017)— previously established links between neural structure and risk taking or risk preference may be measure-specific. In other words, neural markers predictive of risk-related outcomes in one context may not be predictive of the same outcomes in another context. In the first instance, this would limit the utility of neural markers for understanding general mechanisms underlying individual differences in risk taking, and in a second instance limits their utility as targets for longitudinal (Braams, van Duijvenvoorde, Peper, & Crone, 2015; Casey et al., 2018) or pre-/intervention designs (Büchel et al., 2017; Cheetham et al., 2012; Conrod et al., 2013). Related to the issue of convergence, but zooming in on the measure itself, psychometric factors promise to reflect more error-free and thus

more reliable measures of risk preference. For instance, the general risk preference factor *R* extracted by Frey and colleagues (2017) was observed to have a 6-months retest reliability of .85, whereas many of the behavioral measures tested yielded retest reliabilities below .5. Much like convergence between measures, measurement reliability plays a critical role for brain-behavior associations: if the risk-taking measure used is unreliable, any associations between neural and behavioral indices are purely contextual and state-dependent, and would be unlikely to emerge at retest. Unreliable brain-behavior associations are not only uninformative for theoretical purposes but also of limited utility in practice.

A second reason stems from the finding that the general risk preference factor *R* accounts for 61% of the variance explained by all risk-taking measures (Frey et al., 2017). In conjunction with the high stability of *R* over time, this suggests that *R* potentially captures the trait-dimension of risk taking. If *R* can be thought of as a trait, it is reasonable to suggest that state-independent indices such as neuroanatomical variables fare well in accounting for individual differences therein. One of the goals of cognitive neuroscience has become to predict behavior, attitudes or outcomes from neuroimaging indices at ever greater remove (Braams et al., 2015; Büchel et al., 2017; Cheetham et al., 2012; Poldrack et al., 2018; Rosenberg, Casey, & Holmes, 2018), hence 'trait'-like neural variables such as grey matter volume or neural connectivity present attractive target variables to establish such brain-behavior associations. This is not to say that anatomy is fixed; clearly we expect changes over time in the structure of the brain (Koolschijn & Crone, 2013; Sowell, Thompson, & Toga, 2004). However, anatomical variables are not (as) susceptible to the influence of contextual factors which may play a role for brain-behavior associations based on neural functional indices, including on-task activation differences (Tisdall et al., submitted) and off-task resting state connectivity (Grigg & Grady, 2010).

A third reason for using factors concerns the domain-specificity of some of the factors. In addition to the general risk preference factor *R*, Frey and colleagues (2017) extracted seven

domain-specific risk preference factors which (due to being orthogonal) explained additional unique variance across the battery of risk-taking measures used. One of these domain-specific factors was composed of only behavioral measures of risk preference, including various types of risky gambles presented in the form of (monetary) lotteries. Importantly, many insights into brain-behavior associations using structural indices come not only from single risk-taking indices, but they also come predominantly from studies that have used variants of (monetary) lotteries or similar risk-laden decision scenarios (Canessa et al., 2013; Gilaie-Dotan et al., 2014; Jung et al., 2018; C. S. Lin et al., 2016). Using the BBRS risk preference factors, any associations between previously identified regions included in this study and the risk preference factor composed of lottery measures (F7) would provide a conceptual replication.

***Multi-modal imaging*.** It has been argued that function follows form, and that different functional networks in the brain map more or less directly onto the structural connectome (Horn, Ostwald, Reisert, & Blankenburg, 2014). The idea to combine imaging modalities to better understand individual differences in risk taking, especially the extent to which different functional and structural characteristics of the brain individually and in concert shape risky choice, has been appreciated for some time (Canessa et al., 2013; Jung et al., 2018; Kohno et al., 2017; Leong, Pestilli, Wu, Samanez-Larkin, & Knutson, 2016; C. S. Lin et al., 2016). For example, Kohno, Morales, Guttman and London (2017) studied risk taking in the widely-used Balloon Analogue Risk Task (Lejuez et al., 2002) using both functional and diffusion-weighted imaging. The results suggested that a core set of regions with established functional correlates in the task (including striatum, insula, prefrontal cortex and midbrain) are also structurally implicated: the higher the integrity of white-matter tracts in a risk network comprising striatum, insula, prefrontal cortex and midbrain, the more risky choices individuals made on the task. In another recent example, Jung, Lee, Lerman and Kable (2018) combined anatomical with resting state imaging to understand individual differences in risk tolerance on a monetary lottery task. A combination of amygdala grey matter volume and the

functional connectivity (at rest) between amygdala and the medial prefrontal cortex was observed to explain 27% of common variance in risk tolerance, whereas individually both indices explained only 10% and 19% of variance, respectively.

On the one hand, these findings highlight the need to expand the neural risk network to include the amygdala, a region which so far —perhaps due to signal drop out in subcortical regions as a function of scanning parameters selected to maximize signal associated with higher-order cognitive processes in cortical regions— has not received as much attention as for instance the striatum, frontal cortex and insula (Knutson & Huettel, 2015; Mohr et al., 2010; Platt & Huettel, 2008). On the other hand, these findings clearly demonstrate the benefit of multi-modal imaging for explaining additional variance and formulating predictions. Whether or not to include additional modalities in a neuroimaging study will largely depend on a cost-benefit analysis of additional scanning time against additional variance explained. Jung and colleagues' (2018) results suggest that adding some modalities to the model of risk tolerance can indeed lead to a better understanding not only of individual neural predictors, but ultimately also their respective roles, interaction and mutual influence within a neural network that contributes to individual differences in risk taking.

In the current study, we combine volumetry and structural connectomics to examine the extent to which different anatomical (i.e. off-task, context-independent) aspects of the neural risk network account for individual differences in risk preference factors. As discussed above, previous studies have observed both grey matter volume and structural connectivity indices to account for risky choice. Our unique contribution is threefold. (1) Using both volumetric and connectivity indices, we try to predict psychometrically derived risk preference factors given their discussed advantages over single measures. (2) We examine the contribution of individual regions within a comprehensive neural risk network which we built using meta-analytical procedures implemented via Neurosynth. (3) We supplement the traditional approach using multiple regression analyses with Bayesian model comparison to

quantify the evidence for a specific model (i.e. neural index) having generated the risk preference factor over a baseline model. Taken together, we view our contribution as a well-powered conceptual replication attempt of previous findings as well as the discovery of potentially new links between neural structure and risk preference.

Materials and Method

Participants

Participants of this study were members of the "imaging subsample" of the Basel-Berlin Risk Study (BBRS; https://osf.io/rce7g), a multi-site study investigating individual differences in risk preference in a large sample of young adults via collection of multiple measures of risk preference (N=1507; about 50% of the sample tested at each site). For the "imaging subsample", 133 healthy young adults were recruited at the Berlin site for participation in a neuroimaging study investigating the neural correlates of individual differences in risk preference (i.e., as a follow-up to the main lab session of the BBRS; see https://osf.io/rce7g for an overview of all subsamples). The final sample size reflects oversampling to achieve an effective sample size of N=100 (Yarkoni, 2009). To be eligible for participation, individuals were required to have completed the BBRS laboratory session, be right-handed, and be free of any contraindications concerning health and safety inside the MR scanner (e.g. permanent (electrical) implants, usage of psychoactive substances or medications, neurological or psychiatric conditions). Two individuals ended their participation before any neuroimaging data was acquired, hence these two individuals were removed from all analyses. A further seven participants aborted the MRI session early, which meant diffusion-weighted imaging sequences could not be acquired for these individuals. The final sample for all volumetric analyses thus comprises 131 individuals (69 females, mean age at scan = 25.3 years, SD = 2.6 years, range = 20.4 to 30.2 years), and for all structural connectivity analyses the final sample comprises 124 individuals (66 females, mean age at scan = 25.3 years, SD = 2.6 years, range = 20.4 to 30.1 years).

All participants provided written informed consent. Ethical approval for this study was granted by the German Society for Psychology (Deutsche Gesellschaft für Psychologie), and the ethics committee of the Max Planck Institute for Human Development.

Risk preference factors

As indices of risk preference in the current study, we utilized psychometric factors that were extracted across 39 widely used risk-taking measures collected from the full BBRS sample (for a comprehensive list of measures and details on latent variable modeling analyses, see (Frey et al., 2017). The implemented bifactor model gave rise to a general risk preference factor, *R* (akin to the general factor of intelligence) that captured 61% of the explained variance across risk-taking measures, and seven specific orthogonal factors that captured additional domain- or situation-specific variance. These seven factors were suggested to represent attitudes and behaviors associated with health risk taking (F1), financial risk taking (F2), recreational risk taking (F3), impulsivity (F4), traffic risk taking (F5), occupational risk taking (F6), and choices among (monetary) lotteries (F7).

Experimental procedure

Details concerning the laboratory component of the BBRS, including individual measures and study protocol, are reported by Frey and colleagues (2017). For the MRI study, individuals who had completed the BBRS (Berlin-site) were contacted via phone and informed about the follow-up MRI session. Individuals who expressed an interest in participating in the MRI session were screened for any conditions or circumstances preventing them from entering the scanner, and were preliminarily included in the MRI study. Due to a temporal overlap between the end of behavioral data collection in the laboratory and the start of the MRI component, individuals were contacted with varying delays after having completed the BBRS laboratory component. As a result, the MRI sample was heterogeneous with regards to the delay between the laboratory and MRI session (mean delay = 196 days, SD = 121 days, range = 1 to 453 days).

On the day of the MRI session, participants were once again checked for MRI contraindications, fully informed about the study protocol and prepared for the scanner. Participants also completed brief training runs for the tasks completed inside the scanner, as part of functional analyses not included in the current analyses (Tisdall et al., submitted). The full MRI protocol took around 75 minutes, and included a high-resolution structural scan, four functional runs, a resting state scan and a diffusion-weighted imaging sequence. Given our aim to investigate the structural correlates of the psychometric structure of risk preference in this study, we only used data coming from the high-resolution structural scan for volumetric analyses and diffusion-weighted imaging data for structural connectivity analyses. The functional and resting state sequences were not included in the current project, hence are not discussed further. The order of the MRI sequences was fixed for all participants. Following the scan, individuals responded to demographic questions (date of birth, gender, marital status, educational attainment, native language and current occupation). Following the MRI session, we collected demographic data from the participants, including their age, gender, marital status, and educational background. Furthermore, individuals completed several questionnaires and a verbal fluency task; these measures were part of a separate project (i.e. these additional measures were not used for the current analyses) and are thus not discussed further.

At the end of the MRI session, individuals received their participation fee of 25 Euro (1 Euro ~ 1.1 USD), and any additional earnings achieved based on performance in the two MRI paradigms used to address the functional neural correlates of risk preference (Tisdall et al., submitted). On average, participants in the MRI session were paid 41.50 Euro (SD=14.50 Euro) for their participation.

Regions of interest

We focused our investigation on neural regions of interest (ROIs) which we identified via Neurosynth meta-analysis (http://neurosynth.org/analyses/terms/risk/, accessed December

7, 2016) as core neural correlates of the search term 'risk'. At the time of the Neurosynth search, close to 500 individual studies contributed to the statistical parametric map of clusters associated with the search term 'risk'. We selected voxels identified via forward inference as *consistently* activated in studies given the term 'risk', rather than voxels which reverse inference indicates as *preferentially* associated with the term 'risk' (Yarkoni, Poldrack, Nichols, Van Essen, & Wager, 2011). Reverse inference maps are commonly preferred over forward inference maps, as the former are more diagnostic of the search term and thus more specific to a particular cognitive process, whereas the latter are usually more inclusive and thus may include incidental activations (Yarkoni et al., 2011). For this study however, we were interested in the forward inference maps, considering the plethora of definitions, measures and processes connected with risk preferences (Appelt et al., 2011; Dohmen et al., 2011; Schonberg et al., 2011). For example, depending on the adopted definition and measurement of risk, affective components might be incidental or central components of risk preference (Figner, Mackinlay, Wilkening, & Weber, 2009; Samanez-Larkin & Knutson, 2015). Thus, to capture the diversity of processes connected with the search term, we based all our analyses on activation in voxels yielded by forward inference, from 'risk' to brain regions.

The resulting z-score map (corrected for multiple comparisons with a false discovery rate of 0.01 and containing only positive activations) was smoothed (3x3x3 full-width at half maximum kernel) to increase the signal to noise ratio, followed by application of thresholding and clustering procedures to identify suitable clusters of activated voxels. The final binary 'risk' parcellation contained 18 regions (Figure 1), including eight bilateral (amygdala, ventral striatum, thalamus, anterior insular cortex, precentral gyrus, superior lateral occipital cortex, superior parietal lobule/angular gyrus, and middle frontal gyrus), and two medial (paracingulate gyrus, precuneus) regions. These 18 regions served both as ROIs for morphometry analyses and nodes for structural connectivity analyses. In line with the idea of

presenting a conceptual replication of past findings, many regions which previous studies have identified as being linked to risk-taking indices are included in the risk network derived from Neurosynth, including amygdala, thalamus, parietal regions and insula (Canessa et al., 2013; Gilaie-Dotan et al., 2014; Jung et al., 2018; C. S. Lin et al., 2016).

MRI data acquisition and image preprocessing

All neuroimaging data was collected on a 3T Siemens MRI system with 12-channel head coil at the Magnetic Resonance Imaging Laboratory at the Max Planck Institute for Human Development (Berlin, Germany). We acquired high-resolution T1-weighted images via a standard magnetization-prepared rapid gradient echo (MP-RAGE) sequence (repetition time = 2500ms; echo time = 4.77ms; inversion time = 1100ms; flip angle = 7º; FoV = 256 x 256 mm^2; 192 slices; voxel size = 1 x 1 x 1 mm^3). Structural connectivity data was acquired via one diffusion-weighted imaging (DWI) sequence (transverse orientation; 69 slices; voxel size = 2.0 x 2.0 x 2.0 mm^3; 61 diffusion directions; TR=10s; TE=94ms; TA=684s).

Preprocessing of MRI data for morphometry analyses. Preprocessing of the T1-weighted images prior to statistical analysis was performed using the Computational Anatomy Toolbox (CAT12; http://www.neuro.uni-jena.de/cat/) implemented in SPM12 (Functional Imaging Laboratory, Wellcome Department of Imaging Neuroscience, London; www.fil.ion.ucl.ac.uk/spm), and comprised the following six steps: (1) Segmentation of individuals' images into grey matter, white matter, and cerebrospinal fluid components, (2) spatial normalization to Montreal Neurological Institute space using diffeometric anatomical registration through exponentiated lie algebra (DARTEL) normalization, (3) removal of noise using the default procedures implemented in CAT12, (4) performance of quality checks including inspection of the segmentation and normalization results via display of the same slice for all images, and screening for outliers by visualizing the covariance between volumes, (5) smoothing of the grey matter images with a 8mm (full-width half-maximum) Gaussian kernel, and (6) computation of total intracranial volume for every individual.

The workflow created normalized modulated grey matter images, which allow for the comparison of absolute amount of grey matter tissue. For ROI analyses, mean grey matter volume (GMV) was extracted for the 18 ROIs included in the risk parcellation. All subsequent statistical analyses included total intracranial volume to control for individual differences in absolute brain size.

Preprocessing of MRI data for structural connectivity analyses. For analyses of diffusion-weighted imaging data, we used the structural connectivity analysis pipeline implemented in LEAD Connectome v2.1.0 (http://www.lead-connectome.org; (Horn & Blankenburg, 2016; Horn et al., 2014)). The Gibbs ringing removal tool was applied to dMRI data (Kellner, Dhital, & Reisert, 2016). To estimate a whole-brain fiber-set, a white-matter mask was estimated based on the T1-image using the Unified Segmentation approach (Ashburner & Friston, 2005) as implemented in SPM12, followed by the sampling of individual fibers within the white matter mask (co-registered to the b0-images). After initial co-registration and spatial normalization of T1 and b0 anatomical volumes into MNI space (using the MNI 152 NLIN Asym 2009b template), a whole-brain fiber set of 500'000 tracks was estimated by seeding randomly from each voxel in the white matter mask, using the Generalized *q*-Sampling Imaging approach (GQI; Yeh, Wedeen, & Tseng, 2010) as implemented in DSI-Studio (http://dsi-studio.labsolver.org). This model-free approach computes the orientational distribution of the density of diffusing water. Notably, the described procedure represents an established and automated default pathway implemented in Lead-Connectome (Darby, Laganiere, Pascual-Leone, Prasad, & Fox, 2017; Fox et al., 2014; Horn, Kühn, et al., 2017; Horn, Reich, et al., 2017; Horn, Neumann, Degen, Schneider, & Kühn, 2017).

Also implemented in LEAD Connectome, we computed degree centrality as our local connectivity measure of interest for the 18 nodes contained in the risk parcellation map. Nodal degree centrality is a graph-theoretic metric indicative of how central a particular node

is in a network (Rubinov & Sporns, 2010), the neurobiological interpretation being that nodes with a high degree of centrality are linked to (and thus are potentially interacting with) many other nodes in the network. Mathematically, degree centrality is calculated as the sum of all direct connections between a given node and the remaining nodes in the network: the more direct connections (i.e. the more first neighbors), the more central a node is in a network. As such, nodal degree centrality represents a simple yet highly informative local connectomic indicator, which we used in all subsequent analyses pertaining to structural connectivity.

Statistical analyses

All statistical analyses were performed in the software R (R Core Team, 2014) and aimed to identify the explanatory power of neuroanatomical substrates for individual differences in risk preference, measured as latent variables (i.e. general and specific factors of risk preference). To ensure that the values for the general and the seven specific factors observed for the MRI sample were representative of the relationships between factors observed in the full BBRS sample, we computed zero-order correlations and compared these with the correlations reported by Frey and colleagues (2017). Distributions of factor values and neural structural indices were plotted to determine the validity of parametric statistical approaches for factors as outcome measures, and to identify any potential outliers which might unduly influence the results. Given that we used two sets of neural predictors derived from the same unit (i.e., the brain), we computed zero-order correlation coefficients between all volumetric (GMV) and between all connectivity (degree centrality) indices to test for multicollinearity. For those individuals contained in both the volumetric and the connectivity analyses, we also computed correlation coefficients for the association between volume and connectivity of a given ROI (node) to test for multicollinearity between local GMV and degree centrality, respectively.

In a first step, we used a traditional approach to estimate the proportion of variance in inter-individual differences in risk preference that can be accounted for by individual

differences in neuroanatomy, assessed as local mean GMV and nodal degree centrality. To this end, for each of the eight psychometric factors we estimated a) a full model regressing the factor of risk preference factor on all indices of GMV and nodal degree centrality; and b) two separate models only including either the indices of GMV *or* the indices of nodal degree centrality, in order to investigate how much (more) variance in risk preference can be captured by information about volumetric and connectivity. For this first set of analyses, we report adjusted R-squared and alpha for every model. To account for the increase in the rate of false positives as a result of running a large number of regression models, we report which of the nominally significant results remain significant after adoption of a family-wise error rate. Specifically, we maintained an alpha level of .05 within each set of analyses involving combined, volumetric, and connectivity indices for a total of eight factors, yielding an alpha level for these first analyses of .05/8 = .00625. For significant associations between the risk preference factors and GMV, we employed multiple regression analyses (controlling for total intracranial volume, age and gender) to test whether ROI regions would also show significant effects at the level of the whole brain.

In a second step, we assessed the robustness of these linear regression results, and in particular focused on the contribution of each neural index to the overall amount of variance explained in risk preference. For this, we pursued a Bayesian approach to model comparison and model selection using the *BayesFactor* package in R (Morey & Rouder, 2015). Model comparisons based on Bayes factors provide a likelihood for a specific model over a baseline model given the data. In our context, we implemented all models consisting of only one neural index as predictor and tested these models' likelihoods over an intercept-only model. Thereby, the resulting Bayes factors are indicative of the importance of each single predictor.

Prior to analysis, all predictor (GMV, degree centrality) variables were z-standardized and regressed onto age and gender. Accounting for overall brain size in our volumetric analyses, we also regressed mean grey matter volume in the 18 risk network ROIs on total intracranial

volume. The risk preference factors were extracted from z-standardized measures, thus were already on a comparable scale, and were introduced into the current analyses as residuals after regressing out age, gender and test site of the BBRS laboratory session.

Results

Distribution of neural structural indices and BBRS risk preference factors

Initial inspection of the distribution of GMV and degree centrality revealed an approximately normal distribution for GMV but also identified one outlier in amygdala connectivity (6.5 SDs and 4.6 SDs above the mean for degree centrality in right and left amygdala, respectively). Transformation approaches were unsuccessful in removing the influence of this observation, hence we excluded this individual from connectivity analyses and from analyses of volume and connectivity combined. As shown in Figure 2, correlations between the two hemispheres of bilaterally represented structures were high for both GMV (mean correlation between hemispheres of 8 ROIs r =.74, SD=0.24) and for degree centrality (mean correlation between hemispheres of 8 nodes r=.72, SD=0.14). To account for the observed multicollinearity between predictors, we computed a mean GMV score and a mean degree centrality score across the two hemispheres for every individual and proceeded with analyses including 10 regions (Figure 3).

All risk preference factors were approximately normally distributed in both samples (Figure 4), justifying parametric statistical analyses. Pearson correlations between factors (Figure 5) were comparable to the correlation coefficients observed by Frey and colleagues (2017) and indicated orthogonality between the risk preference factors. Moreover, we obtained no significant associations between GMV and degree centrality in a given region (range of correlation coefficients r=-.08 to .17, all p>.05), suggesting these to be (neurobiologically) independent indices that warrant separate as well as combined examination.

Brain – behavior associations

We now turn to the results from analyses assessing the link between neural structure and psychometric (risk preference) factors. First, we report the results from multiple linear regression analyses aimed at estimating the amount of common variance in risk preference factors accounted for by volumetric indices, connectivity indices, and volumetric and connectivity indices combined. Regression coefficients are reported as standardized coefficients and the percentage of variance explained is based on adjusted R-squared (R^2). In a second step, we report the results from a model comparison approach to better understand the (robustness of the) contribution of individual structural markers for risk preference. For this second set of analyses, we report Bayes factors quantifying the likelihood of one model over an intercept model given the data.

Multiple regression analyses. Reported in Table 1 are the results from a first set of analyses aimed at estimating common variance in risk preference factors captured by individual differences in neural structure. Specifically, we found that volumetric indices alone accounted for almost 9 percent of variance in *R* (R^2=.087, *p*=.02), connectivity indices alone accounted for 5% of variance (R^2=.051, *p*=.10) and both volumetric and connectivity indices combined explained over 15 percent of variance in *R* (R^2=.152, *p*=.009). At the level of individual predictors, for the model containing only volumetric indices, only mean GMV in ventral striatum was significantly predictive of *R* (*b*=0.39, *SE*=0.13, *t*=2.93, *p*=.004). Despite the overall non-significance of the model containing only connectivity indices, at the level of individual predictors, degree centrality of amygdala emerged as significantly associated with *R* (*b*=0.32, *SE*=0.11, *t*=2.85, *p*=.005), as well as degree centrality of thalamus (*b*=-0.39, *SE*=0.16, *t*=-2.40, *p*=.018) and precuneus (*b*=0.37, *SE*=0.18, *t*=2.00, *p*=.049). For the model containing both volumetric and connectivity indices, GMV in ventral striatum (*b*=0.32, *SE*=0.15, *t*=2.21, *p*=.029), degree centrality of the amygdala (*b*=0.25, *SE*=0.11, *t*=2.19, *p*=.031), and degree centrality of the precuneus(*b*=-0.33, *SE*=0.16, *t*=-2.01, *p*=.047) remained

significant predictors of *R*. In addition, we observed GMV in the ROI covering lateral frontal cortex (*b*=-0.34, *SE*=0.17, *t*=-2.01, *p*=.047) to also be significantly predictive of *R*.

In contrast to the findings for the general risk preference factors, structural indices appear to bear no relation to differences in situation or domain-specific risk factors (F1 to F7). As a likely reflection of the small effect sizes revealed by ROI analyses, whole brain regression analyses did not reveal significant associations at voxel-level between grey matter volume and the general risk preference factor. Moreover, although the effects of neural volume as well as neural volume and connectivity combined on *R* survived the false positive threshold of p<.05, they did not survive family-wise error correction thresholds. However, by employing model comparisons based on Bayes factors in the next step, we achieved a test of the contribution of neural structural markers to general risk preference independently of an alpha increase due to multiple comparisons.

Model comparison with individual predictors. To assess the independent contribution of neural structural markers to individual differences in risk preference, we ran a model comparison of individual predictors using Bayes factor analysis. Specifically, a Bayes factor of 1 indicates that the tested model is as likely as the intercept model to have generated the data, whereas a value above 1 would indicate the tested model is more likely to have generated the data than the intercept model. Several models turned out to obtain positive (strong?) evidence that the respective neural markers are predictive for inter-individual differences in *R*, namely (Figures 6 and 7): ventral striatal GMV (BF=5.93, R^2=5.2%), frontal GMV (BF=2.70, R^2=3.9%), superior frontal gyrus GMV (BF=2.39, R^2=3.7%), amygdala degree centrality (BF=1.97 R^2=3.3%), and precuneus degree centrality (BF=1.06 R^2=2.2%). In other words, compared with an intercept model, a model containing ventral striatal GMV is close to 6 times as likely to have generated the observed general risk factor values, and a model containing amygdala degree centrality almost twice as likely. Interestingly, our analyses for *R* revealed Bayes factors below 1 –suggestive of the tested

model being less likely than the intercept model to have generated the observed outcome variable data– for regions which have previously been reported to be associated with a variety of risk-taking measures, including GMV in amygdala, insula and parietal cortex (Gilaie-Dotan et al., 2014; Jung et al., 2018; C. S. Lin et al., 2016; Nasiriavanaki et al., 2015).

Discussion

The aim of the current study was to estimate the contribution of neural structure to individual differences in risk preference. To overcome some of the limitations of previous studies, including the use of single measures of neural structure and risk taking, we used multi-modal imaging and psychometrically derived risk preference factors, focusing specifically on regions of interest which meta-analytical procedures suggested as core neural correlates of 'risk'. Our results suggest that neural structure can explain variance in risk preference, but also that this depends on the structural index used, as well as whether we try to account for general or domain-specific risk preference. Volumetric indices alone explained almost 9% of variance in the general risk preference factor *R*. Though degree centrality as an indicator of local connectivity alone only accounted for a small (5%) amount of variance, in combination with volumetric indices over 15% in common variance in general risk preference was explained.

Contribution of variability in neural structures to individual differences in risk preference

At the level of individual predictors, Bayesian model comparison yielded evidence in support of GMV in ventral striatum as an influential predictor of general risk preference, individually accounting for over 5% of common variance in *R*. In particular, we observed a positive association between GMV in ventral striatum and *R*, which falls in line with observations of increased frontostriatal volume in pathological gamblers and internet gaming addicts (Cai et al., 2015; Koehler et al., 2013). Functionally, the ventral striatum has a dominant role in reward-related processes (Hare, O'Doherty, Camerer, Schultz, & Rangel,

2008; Knutson & Huettel, 2015; Mohr et al., 2010; Wu et al., 2012), thus our findings of a positive association between ventral striatal GMV and general risk preference support and extend the proposition of reward-sensitivity as a key component of risk taking to the structural domain. Interestingly, our findings for ventral striatal GMV are mirrored by research on pathologies, but not by studies investigating the neural structural correlates of risk taking in healthy adults using behavioral risk-taking measures, such as monetary lotteries or the Balloon Analogue Risk Task (Canessa et al., 2017; Gilaie-Dotan et al., 2014; Jung et al., 2018; Nasiriavanaki et al., 2015). One explanation for this pattern of results resides in the interpretation of *R*. The general risk preference factor captures common variance across all risk-taking measures adopted in the Basel-Berlin Risk Study (BBRS), it accounts for over 60% of the explained variance, and has high retest reliability (Frey et al., 2017). Consequently, *R* may represent the trait dimension of risk preference. With this in mind, the trait dimension may predispose an individual to (psycho)pathology, and the domain-specific components may drive the concrete expression of aberrant behaviors (e.g. pathological gambling). Thus, *R* and pathology may share biological substrates. It is unfortunately not possible to test this hypothesis within the scope of the BBRS, because the sample comprises only healthy young adults who are free of neurological or psychiatric disorders. However, in principle, gaining an understanding of the extent to which *R* is implicated in and shares biological substrates with pathology would substantially boost our understanding of this risk taking.

In addition to ventral striatal GMV, we also obtained evidence for a contribution to general risk preference by GMV in lateral frontal cortices, and superior frontal gyrus, each explaining around 3% of common variance. The negative associations observed for lateral frontal cortices and superior frontal gyrus with risk preference support previous results implicating smaller frontal volume in the commencement of cannabis usage in adolescence (Cheetham et al., 2012), lifetime intake of alcohol (Taki et al., 2006) and internet gaming

disorder (X. Lin et al., 2015). As with ventral striatal GMV, our results fall in line with findings from studies of pathological samples. One possible route from smaller frontal volume to higher risk preference is to consider the predominant involvement of fronto-parietal networks in control processes, including the inhibition of (motor) responses (Hampshire & Sharp, 2015); we return to the role of the frontal cortex below.

For the connectivity indices, we obtained some evidence for degree centrality of amygdala and precuneus; the latter's contribution barely extended beyond the intercept model, thus we will not speculate on the role of precuneus for general risk preference and instead await studies which provide further evidence for the importance of this neural structure. In contrast, the observed positive association between amygdala degree centrality and general risk preference mirrors recent findings for risk tolerance reported by Jung and colleagues (2018). In their study, amygdala centrality in isolation was predictive of risk tolerance, but this effect did not remain significant in a model containing volumetric and functional connectivity indices (Jung et al., 2018). In our study, amygdala centrality was observed to account for less variance in general risk preference as reported for risk tolerance (3.3% versus 7%, respectively). However, the fact that we obtained evidence for amygdala centrality for a risk preference factor psychometrically derived from a large battery of risk-taking measures, could be taken to suggest that amygdala connectivity does indeed play a role for risk taking, regardless of the index used. As such, this strengthens our earlier recommendation to reserve a place for the amygdala on the neural correlates of risk's most wanted list. The amygdala has been implicated in fear processing as well as various anxiety-related pathologies (Davis, 1992; Ledoux, 2003; Milham et al., 2005), and as such may indeed play a central role for risk taking in the course of stimuli being evaluated as potentially threatening or inciting fear. A well-connected amygdala not only facilitates fear-related signals to travel through the neural risk network, but it also receives control signals from the (pre)frontal cortex (Aron, Robbins, & Poldrack, 2014; Hampshire, 2015; Hampshire & Sharp,

2015). This fronto-limbic feedback loop may ameliorate the initial evaluation of a threatening stimulus, for example by inhibiting / dampening somatic markers. Corroborating the role of a well-connected amygdala in risk preference, Jung and colleagues (2018) found off-task functional connectivity between amygdala and the medial prefrontal cortex to be positively associated with risk tolerance.

Failed conceptual replication: Implications for research on risk taking

Interestingly, and somewhat against our expectations, structural indices were not predictive of domain-specific factors, including F7. Considering that this factor comprises behavioral measures most often used in (neuroimaging) research on risk, such as monetary lotteries (Frey et al., 2017), we expected to find some overlap between our results and previously identified structural correlates of risk for F7, including volume in posterior parietal cortex, amygdala, thalamus, and insula (Canessa et al., 2013; Gilaie-Dotan et al., 2014; Grubb et al., 2016; Jung et al., 2018; Nasiriavanaki et al., 2015). What does our failed attempt at a conceptual replication imply for the robustness of previously identified neural structural correlates of risk taking?

We suggest that the lack of an overlap between the current study and previous work highlights the gap between behavioral measures of risk taking (Frey et al., 2017; Mamerow et al., 2016; Mata et al., 2011; Pedroni et al., 2017; Sharma et al., 2014) and the contextual specificity of established biomarkers for risk taking. For example, some (behavioral) risk-taking measures rely heavily on the representation and processing of numbers, especially outcome magnitudes and probabilities. One seemingly robust finding in the literature is the positive association between GMV in right posterior parietal cortex and risk tolerance as assessed using monetary lotteries (Gilaie-Dotan et al., 2014; Grubb et al., 2016). Feeding the reported peak coordinates for right posterior parietal cortex (MNI-space, x=27, y=-78, z=48) into meta-analytic procedures on Neurosynth (neurosynth.org), the term describing cognitive processes for these coordinates with the highest posterior probability was indeed ‘numerical’

(P(term|coordinates)=0.86, z=4.6), followed by 'navigation' (P(term|coordinates)=0.87, z=4.42). We acknowledge that these terms are associated with voxel activations rather than structural indices, but they provide a first principled approach to identifying associated candidate cognitive processes. Different measures place different demands on the individual, and whilst they may all have a 'risk-taking tag', some measures may differentiate individuals first and foremost based on cognitive capacity or affective components rather than risk preference (Figner et al., 2009; Frey et al., 2015; Hertwig & Erev, 2009; Mata et al., 2011). Thus, it is possible that the reported association between risk tolerance and the right posterior parietal cortex really is an association between numerical ability and right posterior parietal cortex (Gilaie-Dotan et al., 2014; Grubb et al., 2016).

Importantly, our results do not suggest that previously established brain-behavior associations are false positives. Instead, our results imply that trying to generalize findings obtained using one measure is, at best, difficult given the low convergence between measures (Frey et al., 2017; Mamerow et al., 2016; Pedroni et al., 2017). In the worst case, attempts at generalization are misleading and hindering progress because we may believe that an established link addresses a phenotype as a whole, when in actual fact the link is contextual and measure-specific. Akin to research on the role of hormones for sensation seeking and reward sensitivity in adolescence (Harden et al., 2017), our approach to risk preference as a latent construct ambitiously tries to break away from single indices, instead addressing the phenotype as a whole.

Limitations

Even by combining volumetric and connectivity indices we explained a maximum of 15% of variance in the general risk preference factor. Perhaps we are missing vital portions of variance by using additive linear models which do not include interaction terms. In a complex neural network, regional characteristics are likely to interact. While the current study is underpowered for an exhaustive search of the best possible model in the (theoretically)

complete model space, it is also questionable whether any such model, including interaction terms, is interpretable or informative. At present, it may be more critical to understand the building blocks of our models, which includes how best to measure the outcome variable (i.e. risk taking) and understand the extent to which different neural indices (e.g. functional, structural) contribute to our understanding of individual differences.

On a related note, we are seeking to explain variance in a risk preference factor that represents -to a large degree- variance across self-report inventories. If there was a mismatch between what individuals report (and thus makes up *R*) and what they actually do in real life, neural indices would be hard-pressed to account for variance in such a factor. However, self-report inventories and frequency items in the Basel-Berlin Risk Study were moderately correlated, suggesting that what people do and what they say does not substantially diverge.

Given that we were only able to look at GMV at the macro level, we refrain from speculating about the microstructural mechanisms driving the aforementioned relationships. Grey matter comprises cell bodies, axon terminals and dendrites, which in turn play different roles for neuronal signaling. How different neuronal components and their functions relate to risk-taking behavior and attitudes is however at present not understood. Thus, by looking at macro level characteristics such as GMV we only discover associations, but not the underlying mechanisms. This however is crucial for bridging the gap between different variables and risk taking using biological pathways and ultimately understanding the biological basis of risk preference.

Conclusion

By combining multi-modal imaging with psychometrically informed risk preference factors, we hope to contribute to the growing field of research on inter-and intra-individual differences in risk taking. For a phenotype with potentially grave consequences, we still know comparatively little about how best to measure it, which / how different factors influence risk taking, what its biological underpinnings are, and whether / how an understanding of

individual differences can help with prevention and intervention efforts. We present a first step in this direction by looking past the narrow scope of single measures, whilst encouraging others to follow suit. Beyond the current phenotype of interest and intended as a general recommendation for cognitive neuroscience, mapping the convergence of different (neural) indices will provide a basic scientific understanding of the organization of the human mind and brain that exceeds the sum of its parts.

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FIGURES

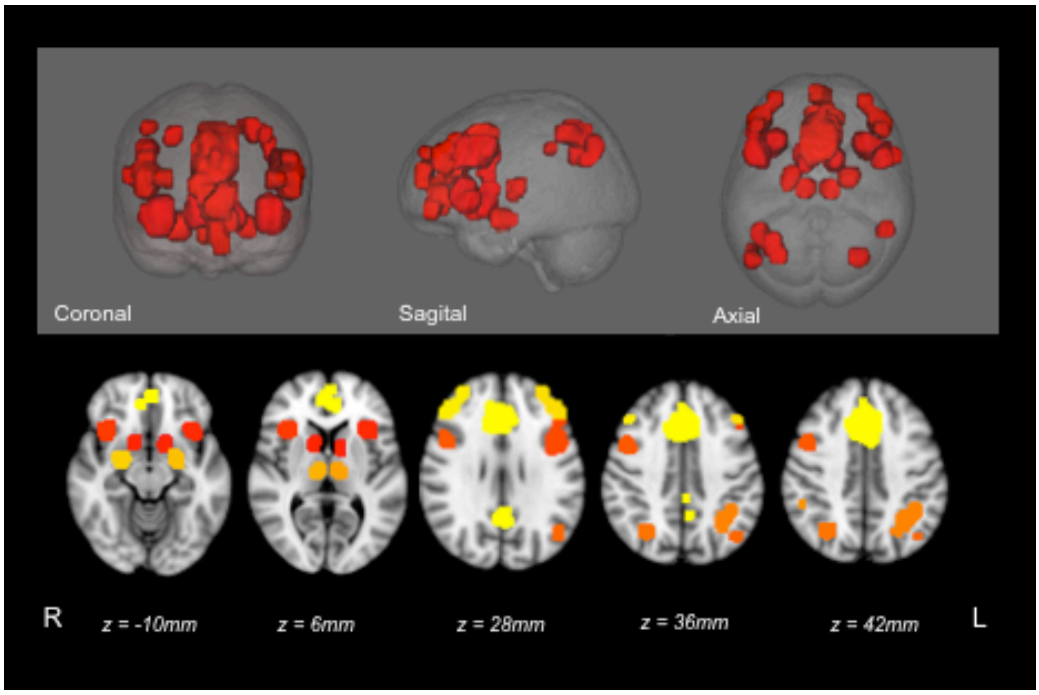

Figure 1. Risk mask used for extraction of mean grey matter volume for volumetric analyses and as nodes for connectivity analyses.

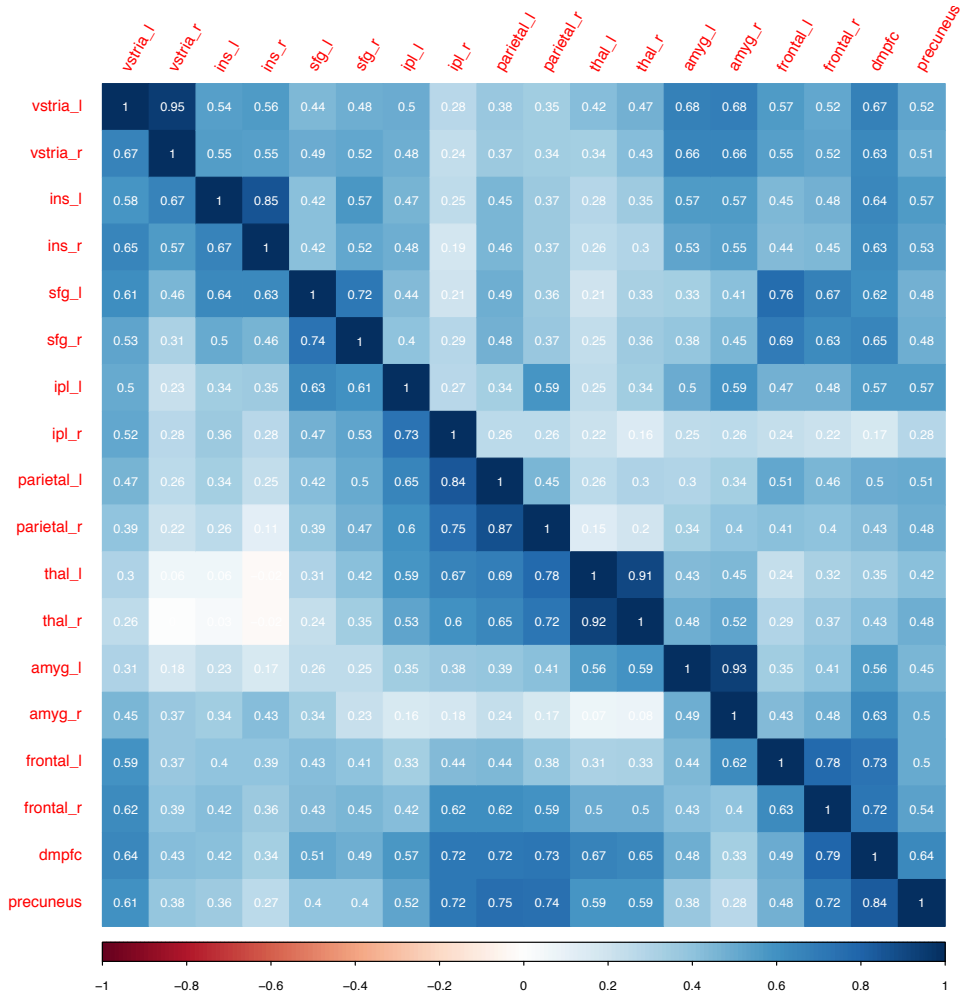

Figure 2. Correlation matrix of neuroanatomical indices in risk network. Upper triangle reflects Pearson correlation coefficients for associations between GMV in 18 risk network ROIs. Lower triangle reflects Pearson correlation coefficients for associations between degree centrality in 18 risk network nodes.

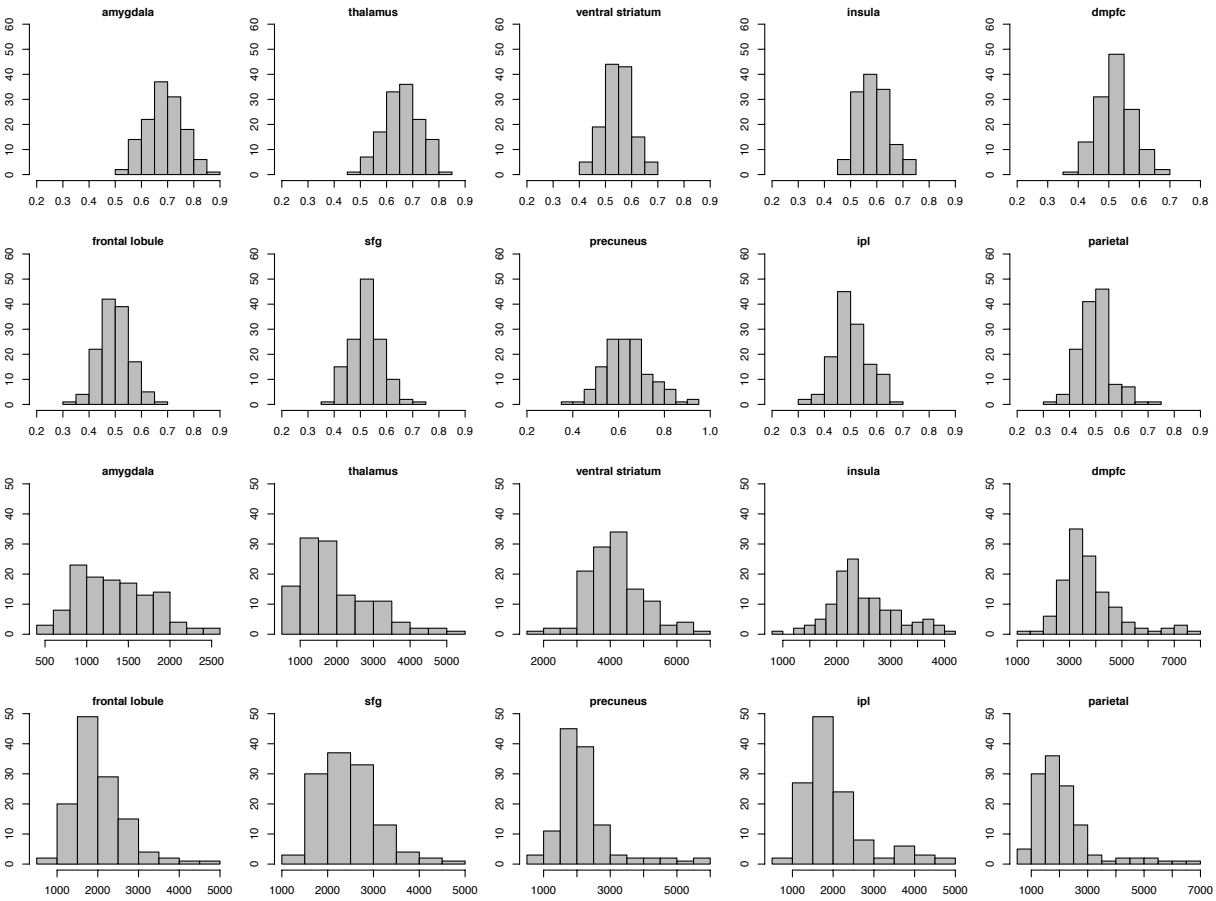

Figure 3. Frequency distribution of neuroanatomical indices in the risk network. Distribution of mean grey matter volume (N=131, upper two rows), and distribution of nodal degree centrality (N=123, lower two rows).

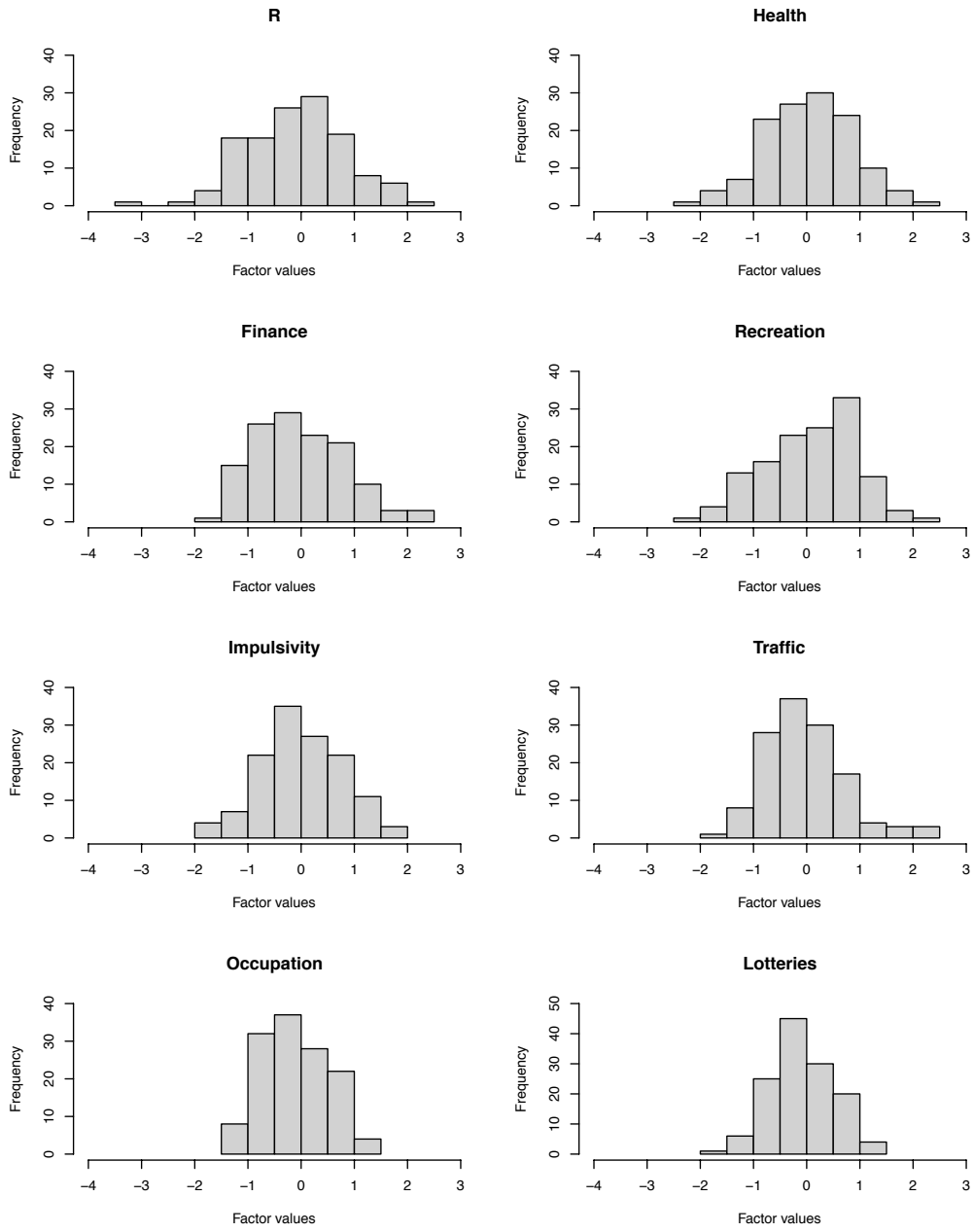

Figure 4. Frequency distribution of risk preference factors (N=131).

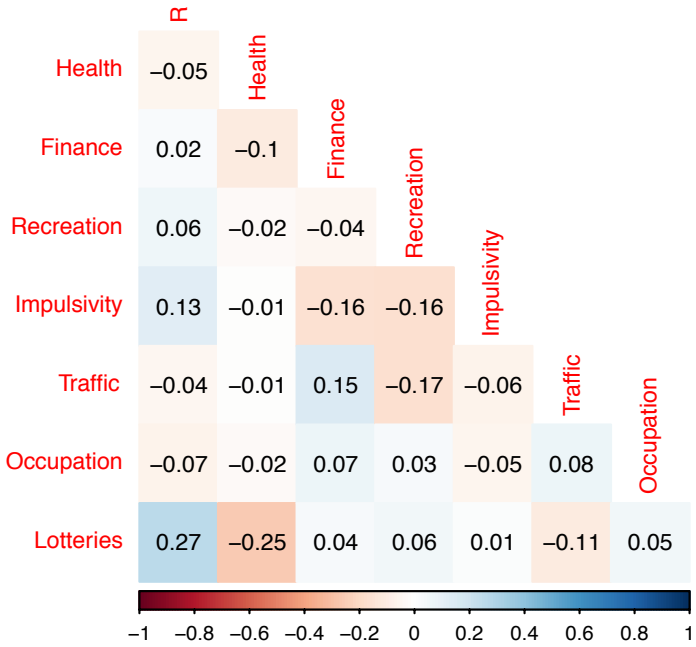

Figure 5. Pearson correlation coefficients for associations between risk preference factors (N=131). *Note:* As expected, correlations between risk preference factors for the connectivity subsample (N=123) were almost identical to those obtained from the full sample (mean correlation between factor correlation matrices obtained from volumetric sample and from connectivity sample r=.99, p<.001).

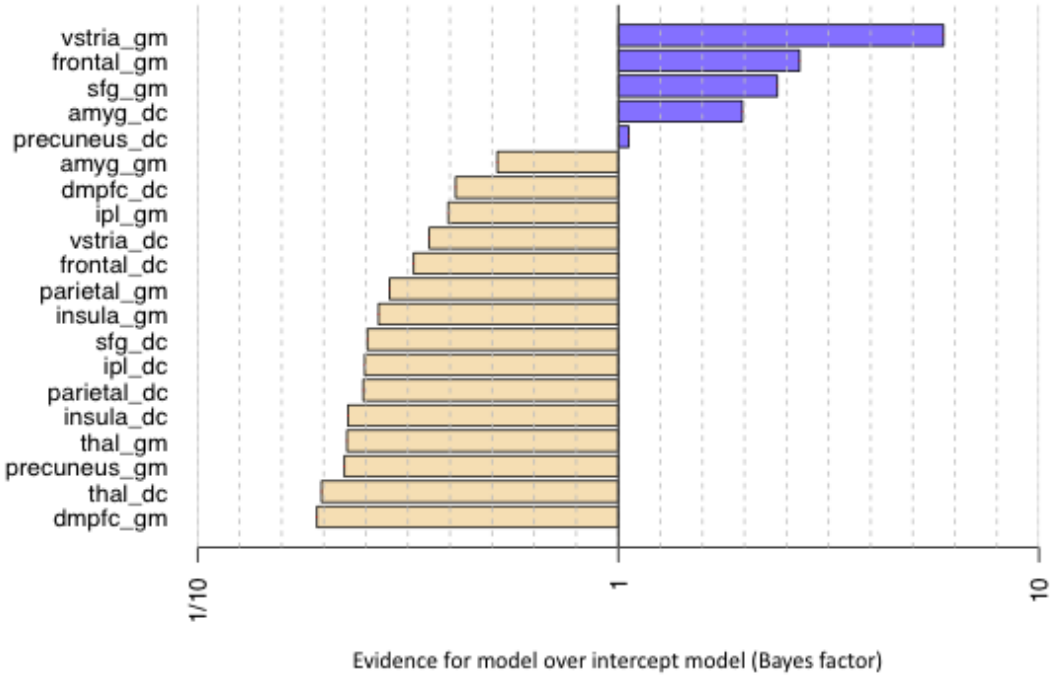

Figure 6. Bayes factor model comparison for all volumetric and connectivity indices as predictors of the general risk preference factor (N=123). All models were compared against an intercept model. A value of 1 indicates the tested model to be as likely as the intercept model to have generated the observed values of the outcome variable. *Note:* dc=degree centrality, gm=grey matter volume, vstria=ventral striatum, amyg=amygdala, ipl=inferior parietal lobule, dmpfc=dorsomedial prefrontal cortex, sfg=superior frontal gyrus, thal=thlalamus

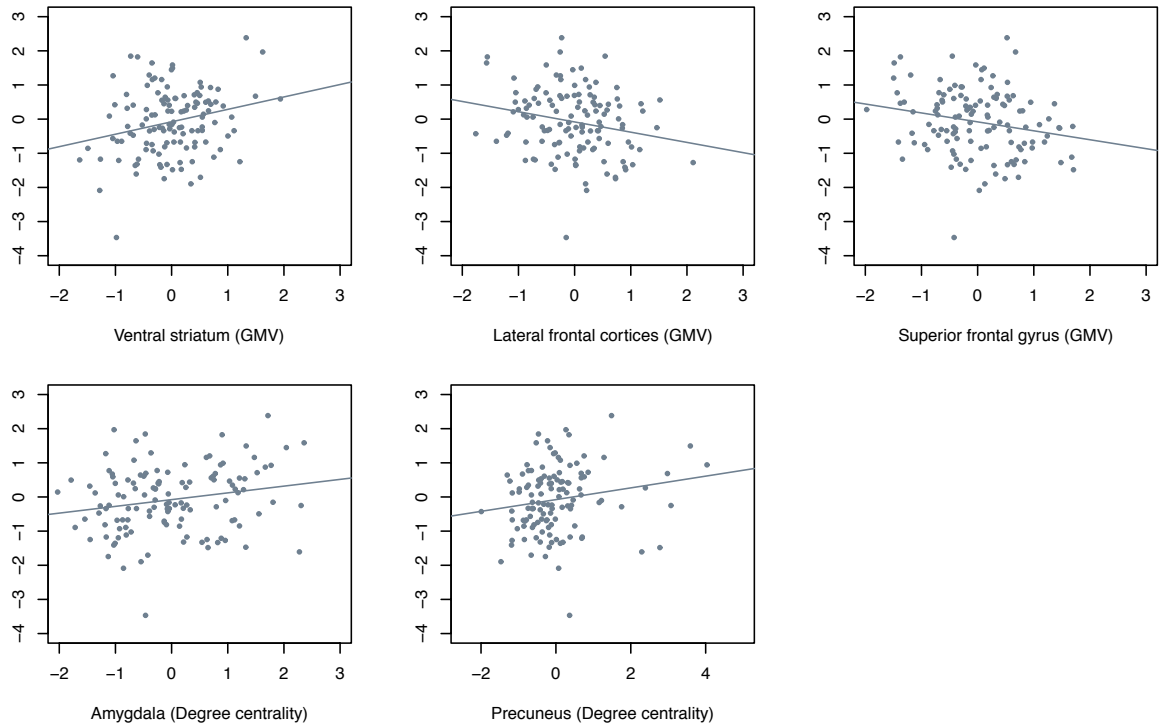

Figure 7. Scatterplots showing the association between general risk preference factor R and the five structural markers favored by model comparison as more likely generators of individual differences in risk preference than an intercept model. GMV = mean grey matter volume.

TABLES

Table 1. Results from multiple regression models assessing the link between neuroanatomy and risk preference factors. Reported are model-specific alpha and variance explained by (1) all volumetric indices (GMV, N=131), (2) all connectivity indices (DC, N=123), and (3) volumetric and connectivity indices together (GMV & DC, N=123).

BBRS Factor	**GMV** ***adjR^2***	**GMV** ***p***	**DC** ***adjR^2***	**DC** ***p***	**GMV & DC** ***adjR^2***	**GMV & DC** ***p***
R	0.087	0.02*	0.051	0.101	0.152	0.009*
Health	-0.037	0.864	0.03	0.197	0.002	0.456
Finance	-0.023	0.71	-0.035	0.82	-0.088	0.958
Recreation	-0.013	0.595	0	0.452	-0.065	0.884
Impulsivity	0.033	0.17	-0.005	0.498	0.034	0.261
Traffic	0.005	0.399	0.004	0.403	0.006	0.429
Occupation	-0.056	0.978	-0.066	0.991	-0.114	0.992
Lotteries	0.008	0.359	0.028	0.21	0.022	0.326

GMV = grey matter volume, DC=degree centrality, adjR^2 = adjusted R-squared, p = alpha level, * p<.05.