Body appearance values modulate risk aversion in eating restriction

P. M. Jenkinson[1,2,3]*, A. Koukoutsakis[2], E. Panagiotopoulou[2,4], E. Vagnoni[5], B, Demartini[6,7], V, Nistico[6,7], O. Gambini[6,7], A. Christakou[8] & A. Fotopoulou[2]

[1]School of Life and Medical Sciences, University of Hertfordshire, UK.
[2]Research Department of Clinical, Educational and Health Psychology, University College London, UK.
[3]Institute for Social Neuroscience, ISN Innovations, Melbourne, Australia.
[4]Postgraduate Studies, Anna Freud National Centre for Children and Families, London, UK.
[5]Department of Psychology, Bournemouth University, UK.
[6]Department of Health Sciences, University of Milan, Italy.
[7]"Aldo Ravelli" Centre for neurotechnology and experimental brain therapeutics, University of Milan, Italy.
[8]School of Psychology and Clinical Language Sciences, University of Reading, UK.
*pjenkinson@isn.edu.au

ABSTRACT

Eating decisions depend on both appetitive and social motivation processes but the interactions between these motivations are poorly understood. We examine how women from nonclinical and clinical samples with variable levels of eating restriction make value-based decisions during an experimental task where monetary values are coupled with values related to body appearance, such as the social value of thinness. We show that eating restriction is associated with less risk taking and greater hesitancy when making decisions, but this general tendency is modulated by subjective body-related valuations in both subclinical and clinical samples; risk taking is decreased and hesitancy is increased when monetary reward is coupled with larger body stimuli, particularly in 'at risk' samples, and the reverse when reward is coupled with thinner body stimuli, particularly in clinical samples. Computational modelling further indicated that these behaviours are driven by an aversion to risk rather than loss, with desirable body outcomes being associated with less risk aversion, and undesirable body outcomes linked to greater risk aversion. These findings have important implications for current explanations of eating, indicating that cognitive and social factors may influence eating decisions by distinct mechanisms.

Keywords: risk-taking; decision-making; uncertainty; value; reward; punishment; restrictive eating; anorexia nervosa; computational modelling,

Introduction

The neurobiological regulation of eating, and of metabolism more generally, is of fundamental homeostatic importance. In contemporary societies where food availability and variety is high,

eating regulation may involve eating restrictions for health purposes[1]. However, eating restriction is also increasingly pursued to achieve certain societal body ideals[2] despite documented risks for adverse physical and psychological effects[3–5]. These health risks are even greater in psychopathologies like Anorexia Nervosa (AN), an eating disturbance characterised by an intense fear of gaining weight despite being underweight, a disturbed body image, and a relentless pursuit of thinness[6]. Unfortunately, the multifactorial aetiology of AN remains poorly understood despite the fact that the disorder has the highest mortality rate of any psychiatric illness[7]. Importantly, disordered eating at subclinical levels is among the most common indicators of the development of a clinical eating disorder[2,8]. It is, therefore, important to understand the biopsychosocial factors that drive restrictive eating across the healthy population and psychiatric diseases.

Eating restriction motivations have been hitherto addressed mainly within two parallel, research lines, namely psychosocial and neurobiological approaches. Firstly, psychosocial accounts stress that eating is motivated by social reasons, such as impression management, with hunger and satiety circuits playing relatively minor roles[9,10]. A major sociocultural influence particularly affecting women in Western cultures is the dominant "thin ideal", which places a positive value on a slim body and a negative value on larger body appearances. Thin ideal internalisation (i.e. the extent to which an individual ascribes to this social value[11]) has been proposed as a key explanation of eating restriction, with supporting evidence from various correlational, cross-sectional, and experimental studies[12,13].

By contrast, in neurobiology, eating is understood as a product of bodily systems that mediate energy homeostasis (metabolism) and the complex appetitive motivation systems regulating hunger and satiation. For instance, neuroimaging studies suggest that restrictive eating is the behavioural result of decisions based on skewed interactions between dopamine-based reward-learning systems and serotonin-based control or inhibitory systems (see [14,15] for reviews). Moreover, while altered reward and punishment processing in response to food and taste stimuli has been noted in AN[16–19], it has become clear that a generalized blunting of reward responsivity to food is insufficient to explain eating restriction. Instead, studies on eating should take into account more general and commonly entangled components of value-based decisions, such as valuation, risk preference and aversion, loss aversion, and the handling of uncertainty.

Crucially, in order to experimentally manipulate and computationally model the complex interrelations between such parameters, and particularly as the valuation of food is complex in individuals with disordered eating[16,17], most behavioural and neuroimaging studies have used monetary decision making paradigms[20–25]. Such tasks are able to quantify not only how much people normally prefer larger over smaller rewards, but how this situation changes when rewards are associated with costs, such as delays, or uncertainties. People integrate such costs into a value function according to their preferences, make choices accordingly and learn from the experienced outcomes. Delay discounting, for instance, is defined as the degree to which a reward is devalued as a function of the delay to its receipt[26]. Recent studies found that patients with restrictive Anorexia Nervosa (AN) discount rewards as a function of delay at a rate that is significantly lower than healthy peers, consistent with their everyday behaviour of increased 'self-control' for immediate rewards (e.g. food) in pursuit of long-term outcomes (e.g. a 'thin' body)[20,24]. AN patients were found to make less risky choices than healthy controls in another, widely-used decision-making paradigm, the Balloon Analogue Risk Task[27], that assesses how people balance potential reward against the possibility of loss under uncertain conditions (i.e. when the particular probability of loss is unknown during the task). Such findings are consistent with self-reported intolerance of uncertainty[28] and hypersensitivity to punishment[29] in AN. Yet there have been contrary findings in such *monetary* tasks in AN[22,30,31] and in community samples with eating restriction[32,33], with some studies, for example, finding

no differences in loss aversion between restricting and non-restricting groups[34], while others finding that eating restriction is associated either with *hyper*sensitivity[23] or *hypo*sensitivity to the possibility of loss or punishment[35]. There can be at least three reasons for such discrepancies that the present study aims to address, as described in turn below.

First, existing paradigms examine decision-making as driven by the evaluation of abstract monetary rewards, but fail to manipulate or model parameters that most relate to eating restriction (see [23,36] for discussion). For example, it is unclear whether there are additional variables that can explain the seemingly paradoxical finding that AN patients, who are risk-averse and hypersensitive to the possibility of loss[23,27], nevertheless tolerate the severe health risks of malnutrition. One possibility is that AN individuals' risk-aversion is modulated by independent motivations, such as the aforementioned social value of being thin, and the corresponding negative value of larger body appearances. Indeed, a systematic review of the value of cues used in AN research revealed that acute and weight-restored individuals with AN show aversive reactions to non-thin, or overweight body appearances in both explicit and implicit tasks[19]. Moreover, the pursuit of thinness, as well as the fear of gaining weight, are central to the symptomatology of disordered eating and have been implicit in previous interpretations of decision-making in restrictive AN (increased 'self-control' over immediate rewards like food in pursuit of later 'thin' body outcomes), yet to our knowledge no study on clinical or subclinical eating restriction has explicitly examined the value ascribed to body outcomes in value-based, decision-making. Here, we aimed to investigate the role of subjective values regarding body thinness in how individuals with subclinical and clinical eating restriction make reward-based decisions under risk.

To this end, we developed a new risk analogue task (based on one of the most widely used and well-validated, risk-taking paradigms[37,38]), which assesses body-related risk taking. In our Body and Balloon Analogue Risk Task (B-BART; see Figure 1) participants made consecutive decisions to 'click' a button in order to accumulate money, or stop clicking and 'collect' the money already accumulated in the trial. In separate conditions, each click causes a virtual body or balloon to increase (get bigger/fatter) or decrease (get smaller/thinner) in size, but carries the risk of reaching a limit ('loss limit'), which ends the trial, at which point the accumulated money not 'collected' is lost. The loss limit point is unknown, so each decision to click or collect involves risk (i.e. the potential to lose) and uncertainty (i.e. although the probability of loss increases with every click in each trial, the actual probability of losing on the subsequent click is unknown to the subject). The *number of clicks* in trials in which the limit was not reached was used as a primary index of *explicit risk-taking*, as in most other studies with this paradigm[37,39]. We also calculated a recommended alternative reaction time measure[39] as an *implicit index* of *behavioural uncertainty (i.e. hesitancy)* in the decision-making process, i.e. a measure indicative of reflective decision processes based on the amount of time (in ms) it takes between the last decision to click and the decision to collect the accumulated gain rather than to continue clicking. We hypothesise that desired and/or undesired body options may be overvalued in relation to more neutral stimuli in individuals with subclinical or clinical eating restriction and hence influence explicit reward-based decisions and hesitancy, over and above any more general tendency to avoid risk[27].

Second, existing discrepancies in the literature may relate to the separation of studies on clinical and community samples. To our knowledge, no single study has assessed the relationship between eating restriction and value-based decision making across community and psychiatric samples with subclinical and clinical eating restriction behaviours, respectively. This was the second aim of the present study; we tested the above risk-taking paradigm (B-BART) on several independent samples covering both the 'at risk' and clinical ends of the spectrum, the latter targeted at different time points in the course of the disease, and controlled for by a well-matched subset of our larger population. Specifically, we first examined the above

behavioural measures of risk-taking in a large (n = 485) sample of women without any psychiatric history, and with a wide range of restrictive eating tendencies as measured by the Eating Disorder Examination Questionnaire (EDE-Q[40]). We examined how risk-taking is affected by individual differences in self-reported level of restrictive eating (indexed by EDE-Q restraint score), stimulus Type (body or balloon) and stimulus Direction (increasing or decreasing size). Based on previous studies[23,27], we predicted that risk-taking would be lower overall in people with higher levels of restrictive eating, and particularly when reward was coupled with an 'undesirable' body outcome, (i.e. a female body getting gradually larger in size) rather than a neutral stimulus. By contrast, we expected that risk-taking would increase in conditions where the outcome was a 'desirable' body (i.e. a gradually thinning body) rather than a neutral stimulus. To test these hypotheses, we examined how risk taking is modulated by restrictive eating in general, as well as in two key comparisons: i.e. comparing each body condition with its respective balloon control condition (i.e. body increase vs. balloon increase; body decrease vs. balloon decrease), while controlling for age and BMI.

We complemented these studies with a clinical study in which we compared the risk-taking behaviour of acute, restrictive subtype Anorexia Nervosa patients (AN; N = 31), and weight-restored AN patients (AN-WR; N = 23), with two non-clinical control groups: a low restrictive eating group (HC-L, N = 38), and a high restrictive eating group (HC-H, N = 35), created from our larger non-clinical sample, following a targeted recruitment strategy (see [41] and Methods) and controlling for key clinical and psychometric variables such as mood and eating disorder severity (see Methods and Supplementary Materials). In line with the predictions made above and our conceptualisation of an eating restriction spectrum, we predicted that both acute AN and AN-WR groups would behave like healthy individuals with higher levels of restrictive eating, i.e. showing lower risk taking in comparison to healthy individuals with lower eating restriction, which would nevertheless be modulated by desirable and/or undesirable body outcomes. These multiple samples allowed us to not only examine multiple levels of eating restriction ranging from subclinical to clinical populations, but also to disentangle state (i.e. present only during the acute AN phase) from trait disease mechanisms (i.e. deficits that endure beyond the acute phase and are present during remission, or in at risk populations). Disentangling these mechanisms is important for identifying state-independent aetiological traits and vulnerability indicators of AN (or 'endophenotypes'[42]), versus secondary effects that are a consequence of the acute malnourished state, comorbidities, or medication. Therefore, confirmation of the above hypotheses across our samples would suggest that appearance-based, risk-aversion is a marker of an eating restriction spectrum rather than the expression of a categorical disease state like AN.

Finally, existing studies have failed to differentiate between different components of decision-making which produce different responses, such as risk (when the outcome is *unknown* but the outcome probabilities are *known*) and uncertainty (when both the outcome and the probability distribution are *unknown*). Indeed, it has been long noted that there is ambiguity regarding these dimensions in the original Balloon Analogue Risk Task (BART[37,38]), which seems to involve a transition from initial uncertainty to later risk[43,44]. Unfortunately, the point when this transition occurs is typically unknown, meaning that without computational modelling, the processes governing behaviour are underspecified. Similarly, without computational modelling, it is not possible to know whether behaviour on the task is driven by hypersensitivity to loss (the potential to lose increasing rewards as the task progresses) or to risk (taking an increasing risk as the task progresses), both of which have been previously linked to disordered eating. Leveraging computational modelling to disentangle these latent explanatory levels was the third aim of the present study. Specifically, we applied, and compared between, existing computational models of the BART task[45,46] to examine which parameters best described the risk taking behaviours associated with our critical conditions and

samples, and particularly risk-aversion and loss-aversion. Before proceeding to this main aim, however, we also computationally addressed the difficulty to disambiguate between BART trials when the subject is making decisions under risk versus under uncertainty (see above;[43,44]). We thus present a novel model of the potential transition from uncertainty to risk in our control sample, tested it against a previously-used baseline model[45] (see Supplementary Materials) and then assessed whether these transitions had an effect on our critical conditions and groups.

In summary, our study combined insights from existing psychosocial and neurobiological perspectives on eating, as well as methods from dimensional[47,48] and computational[49,50] approaches to mental health to develop, and apply to multiple samples across the eating restriction spectrum, a novel, decision-making paradigm. This paradigm allowed us to examine the degree to which eating restriction in both subclinical and clinical samples is associated with an increased value ascribed to certain socially-valued, body appearances (the thin ideal) in reward decisions under uncertainty and risk, over and above the more general, potential tendencies for risk and loss aversion.

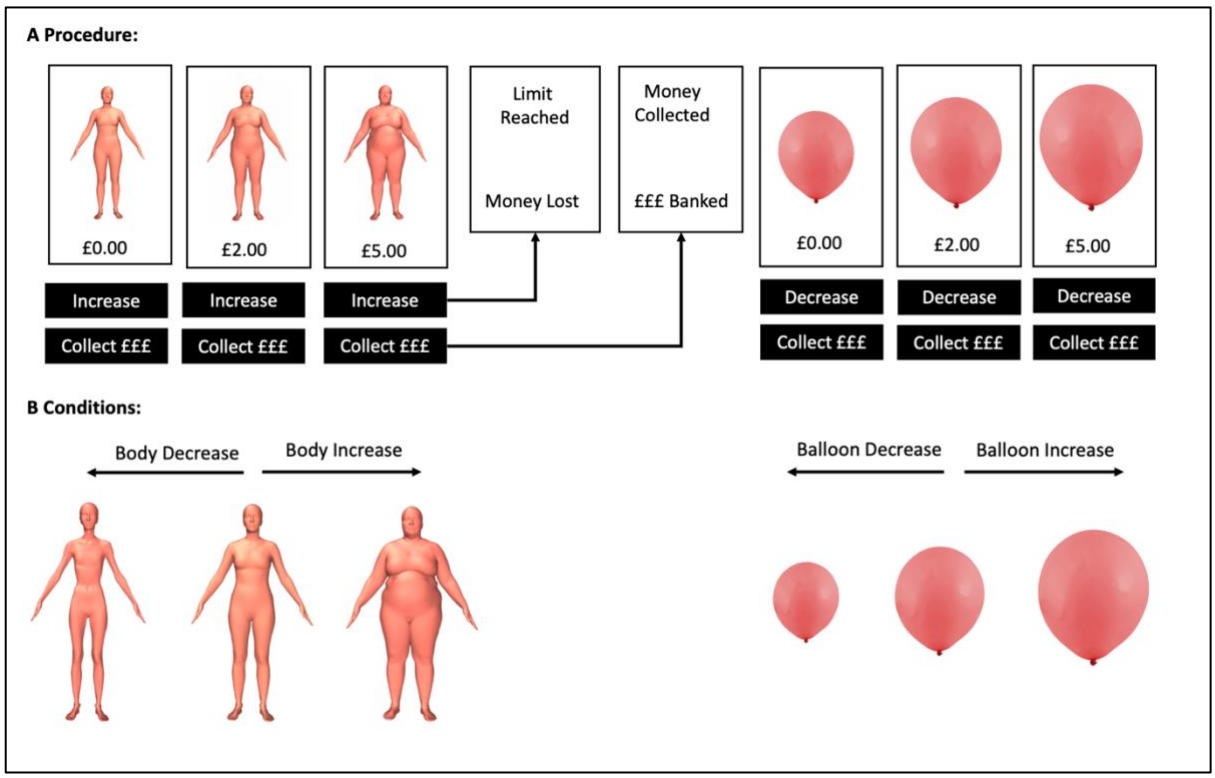

Figure 1. The B-BART.

Results

Risk Taking in Subclinical Eating Restriction

Our first set of analyses was in a sample of women without any reported history of eating disorder. Following initial pilot work in which we developed the B-BART and established the presence of expected effects (see Methods and Supplementary Materials), we conducted a fully-powered study in a large sample of women (N = 485). We used multilevel models to examine how individual differences in restrictive eating (EDE-Q restraint score) influence overall risk taking (i.e. irrespective of different conditions), and body-related risk taking (using

the key comparisons and measures outlined above), while controlling for experimental order effects, age and BMI variability. The results of these analyses are reported in Table 2. In complementary control analyses, in a subset of participants, we explored whether key psychometric variables in eating restriction research, such as body image disturbances and concerns (BIDQ[51]), the related psychological dimensions of impulsiveness (Barratt Impulsiveness Scale; BIS-11[52]) and obsessive-compulsive behaviours (Obsessive-Compulsive Inventory Short Version; OCI-R[53]), and affective traits such as depression, anxiety and stress (DASS-21[54]), influenced the effects found in our main analyses. We first examined whether they correlated with each other as well as the outcome variables (to check for multicollinearity), and then after removing highly correlated variables, included each relevant variable in our MLM as a fixed covariate to identify any substantive change in significance or variance explained.

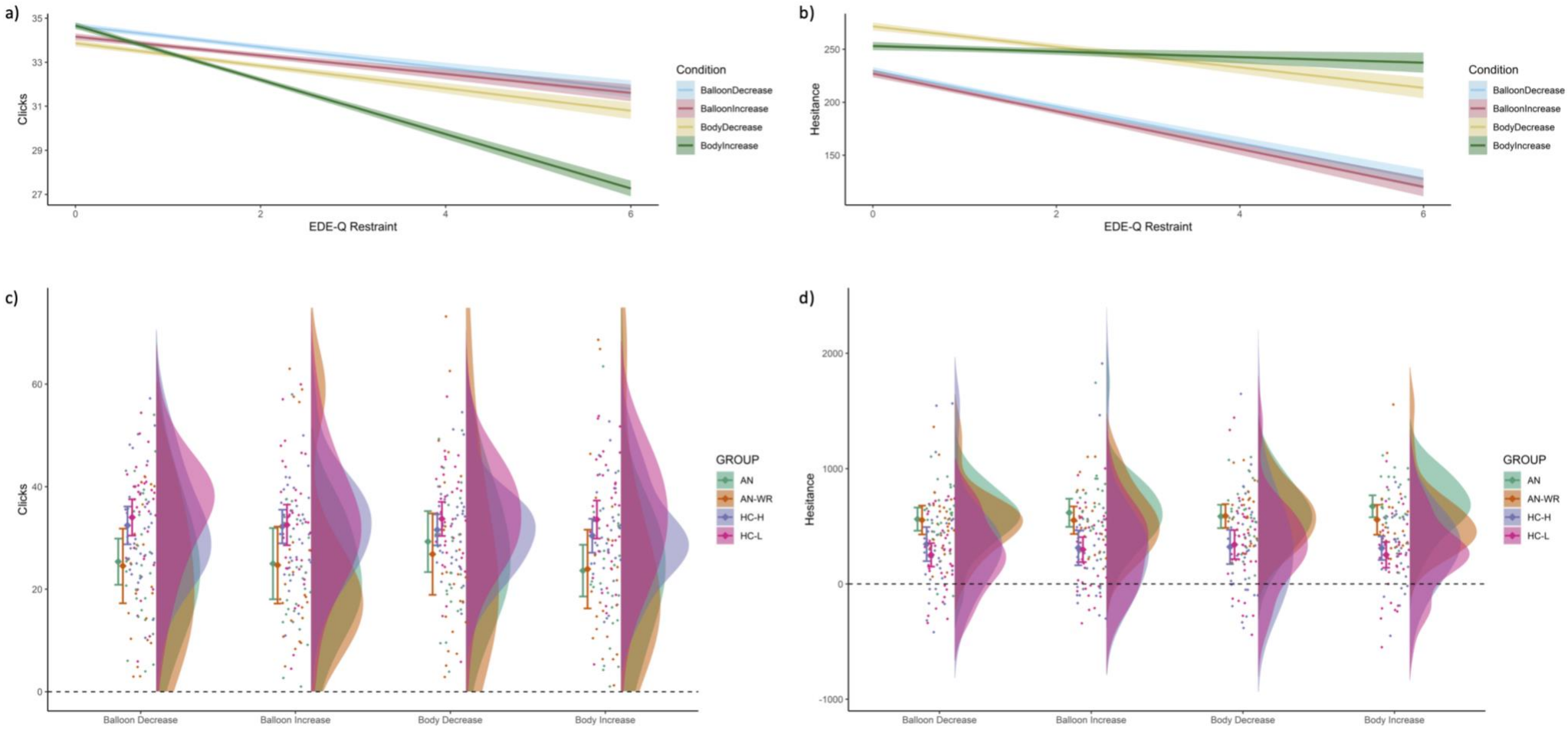

Figure 2. Overall clicks per condition (panel a) and hesitance (panel b) in the subclinical sample study. Overall clicks per condition (panel c) and hesitance (panel d) in the clinical sample study. NB: illustrating slopes from multilevel models takes only fixed effects into account by necessity, so the exact direction of the slopes should be interpreted with caution.

Women with subclinical eating restrictions take less risk when monetary reward is coupled with an undesired body outcome.

We found that higher levels of self-reported eating restriction were predictive of significantly less risk taking (fewer clicks) overall (Table 2). A significant three-way interaction indicated that this effect varied depending on Stimulus Type and Direction (Table 2 & Figure 2a). Specifically, women with greater self-reported eating restriction clicked significantly fewer times when monetary reward was coupled with an increasing body compared to an increasing balloon. There was no significant difference between the decreasing body and decreasing balloon conditions (Table 2). This interaction remained significant when accounting for general body image disturbances, impulsiveness, compulsiveness, and affective traits (see Supplementary Materials).

Women with subclinical eating restrictions show greater hesitancy when monetary reward is coupled with an undesired body outcome.

Performing these same analyses with hesitancy (i.e. time taken to collect earnings after the final click; see above and Methods) as the dependent variable revealed similar results. The overall effect of restrictive eating on hesitancy was not significant (Table 2), however, there was a significant three-way interaction between Stimulus Type, Direction and EDE-Q restraint (Table 2; Figure 2b), and this remained significant when controlling for secondary body image, cognitive and affective factors (see Supplementary Materials). In the same way as for explicit risk-taking, women with higher levels of restrictive eating showed significantly greater hesitancy when an increasing body was compared with an increasing balloon, but no significant difference when a decreasing body was compared with a decreasing balloon (Table 2).

Risk Taking Across Subclinical and Clinical Eating Restriction

We conducted the above analyses comparing acute AN patients, AN-WR patients, and two BMI-and age-matched non-clinical samples characterised by either low or high restrictive eating (i.e. HC-L and HC-H groups, respectively), following a targeted recruitment strategy[41] based on the aforementioned dimensional approach to eating restriction. Specifically, we first selected individuals from our larger non-clinical sample based on matching their BMI to that of the AN-WR patients (thereby controlling for BMI differences that could inflate any eating restriction effects as well as the possible extraneous effect of BMI on cognition[55]; noting that matching BMI to the acute AN group would not be appropriate, or possible since low BMI is a diagnostic feature of AN) and then we divided them into two groups based on their level of restrictive eating (EDE-Q restraint scores; see Methods and Supplementary Materials). This approach ensured that any differences between non-anorexic individuals with low versus high eating restriction were not driven by differences in BMI or sample size. We also controlled for condition order and age in all analyses, and performed additional analyses to rule out affective factors such as depression, anxiety and stress and overall severity of eating disorder symptomatology as primary explanations for our results (reported in Supplementary Materials). We examined the effect of Group (AN, AN-WR, HC-H, HC-L) on overall risk taking, and then the three-way interaction between Group, Stimulus Type and Direction. When performing the two critical comparisons to determine whether risk taking is moderated by body outcome desirability, we compared each clinical group to the sub-group with no behavioural evidence of eating restriction (HC-L), with 'trait deficits' identified when both clinical groups differed from controls, while 'state deficits' (including the effects of low BMI) identified when only the acute group differed from controls. Given the unclear EDE-Q cut-offs for at risk populations[56,57], clear state-trait distinctions were not possible based on comparisons with the HC-H and these were not performed. Results of these analyses are reported in Table 2.

Acute AN and AN-WR patients' risk taking is modulated by body outcome.

Our MLM including all four groups (AN, AN-WR, HC-H, HC-L) showed that overall risk taking differed significantly between groups, with AN and AN-WR patients taking significantly less risk (i.e. making fewer clicks) compared to the HC-L group (see Table 2 & Figure 2c). The expected three-way interaction between stimulus Type, Direction and Group was significant (Table 2), and this interaction was unaffected when affective variables (DASS scores) were included in the analyses (see Supplementary Materials). Examining this interaction using our key comparisons, we found that AN patients showed a (non-significant) tendency to take *less* risk than HC-L when examining the difference between the *increasing* body condition and the *increasing* balloon condition. By contrast, acute AN patients took significantly *more* risk than HC-L when examining the difference between the *decreasing* body condition and the *decreasing* balloon condition.

Performing these same analyses comparing AN-WR patients and HC-L revealed the same results (Table 2). AN-WR patients took significantly *less* risk than HC-L when looking at the difference between the *increasing* body and *increasing* balloon conditions, and significantly *more* risk when looking at the difference between the *decreasing* body and *decreasing* balloon conditions.

Acute AN and AN-WR patients' hesitancy is modulated by body outcome.

Our MLM including all four groups (AN, AN-WR, HC-H, HC-L) confirmed the results from our analysis of explicit risk-taking, with overall hesitancy being significantly different between groups, and both AN and AN-WR patients showing significantly greater hesitancy compared to the HC-L group (see Figure 2d & Table 2). Hesitancy also showed the expected 3-way interaction between Stimulus Type, Direction and Group, and this interaction was unaffected when affective variables (DASS scores) were included in the analyses (see Supplementary Materials). Examining this interaction with our standard pairwise comparisons, we confirmed that patients with acute AN exhibited significantly *more* hesitancy than the HC-L group when considering the difference between the *increasing* body and *increasing* balloon conditions. However, there was no significant difference between AN patients and the HC-L group when considering the difference between the *decreasing* body and *decreasing* balloon conditions.

These same analyses performed to compare the AN-WR patients with the HC-L group showed no significant differences (Table 2).

Computational Modelling

In addition to our behavioural analyses, we used computational modelling to understand the latent (hidden) processes driving risk-taking behaviour, and to disentangle the contribution of general cognitive versus social-motivational (thin-ideal) factors in the risk-taking of restrictive eaters. We conducted two sets of modelling as detailed below; first, examining uncertainty versus risk, and then risk- versus loss-aversion, in the risk-taking behaviour of our non-clinical and clinical samples. For both sets of modelling we created a set of cognitive models based on previous studies of the BART[45,58], and compared the fit of these models with baseline models that assume no change in uncertainty, nor any influence of loss avoidance, in the behaviour of the non-clinical sample. We tested the fit of the data in all cases using Maximum Likelihood Estimation (MLE), the Akaike Information Criterion (AIC) and the Bayesian Information Criterion (BIC). For each of the two sets of modelling we selected a winning model based on best fit as detailed in the Supplementary Materials. Subsequent analysis was performed using the parameters of the winning model from each set of model comparison.

Our modelling of uncertainty vs. risk involved a cognitive model (i.e. herein called the *Exploration-Exploitation* model; described fully in Supplementary Materials and summarised

in the Methods section) which assumes that participants go through two phases: *Exploration* (during which there is greater uncertainty) and *Exploitation* (i.e. during which uncertainty is reduced). In both phases, the participant is assumed to have a fixed belief about the probability of reaching the maximum (loss) limit each time they click (hereafter referred to as 'loss belief'), providing two parameters: *Prior Probability of Loss Belief* (loss belief during Exploration) and the *Posterior Probability of Loss Belief* (loss belief during Exploitation). The transition from Exploration to Exploitation takes place at a trial that gives the final parameter: *Threshold*. After validating the two phases of the Exploration-Exploitation model, these three parameters were used in further analyses to examine if loss beliefs and hesitancy during the exploration and exploitation phases, and the threshold click, varied based on restrictive eating levels (i.e. EDE-Q Restraint) in our subclinical sample, and when comparing across the groups of our clinical study. In so doing, we aimed to ascertain if our samples tended to shift between uncertainty and risk at different rates depending on their level of restrictive eating (i.e. EDE-Q restraint score or clinical diagnosis) or experimental condition (body vs. balloon; increasing v. decreasing), and whether this could explain the observed differences in risk-taking behaviour. Results of these analysis are reported in Table 3.

Our main modelling of risk-parameters (*risk aversion vs. loss aversion*) used the Exponential-Weight Model (EW model)[46]. The EW model was developed to overcome the limitations of the earlier 4-parameter model[45], which has been criticised for failing to reproduce accurate parameters in parameter recovery, and being difficult to interpret within a general reinforcement learning (RL) framework (see [59,60] for details). The EW model (described fully in [46] and summarised in Supplementary Materials) contains five parameters, two of which are of direct relevance to the current aims (for brevity the remaining three parameters and results relating to these parameters are described in Supplementary Materials only). *Risk aversion* (ρ) indicates an individual's sensitivity to the value of reward change, such that individuals with higher risk avoidance take less risk to get the same amount of reward. *Loss-aversion* (λ) indicates an individual's sensitivity to negative outcomes, such that potential loss is perceived as more severe at higher λ. After assessing the fit of these models and performing model comparison as detailed above, we used the parameters of the winning model in subsequent analyses to identify whether overall risk-taking, and body-related risk taking (i.e. differences in behaviour observed when reward is coupled with an increasing/decreasing body vs. balloon) in individuals with different levels of restrictive eating (i.e. in relation to EDE-Q restraint score in our non-clinical sample, and clinical diagnosis in our clinical samples) is best accounted for by risk aversion or loss aversion. Results of these analysis are reported in Table 4.

Hesitancy and click variability is greater during Exploration compared to Exploitation

Our first set of modelling supported the existence of two phases in the B-BART, characterised by initially high and subsequently lower levels of uncertainty (see Supplementary Materials for full model fit results). To validate this two-phase model, we examined if two behavioural measures that were not used to create the model (i.e. hesitancy and behavioural (click) variability [i.e. SD/Average]) were different between the two phases in the subclinical sample (see Supplementary Materials for results). Based on existing theory[61,62] we expected behavioural hesitancy and variability to be greater during exploration compared to exploitation. Using the data from our subclinical sample, we found that the degree of hesitancy and click variability was significantly greater during exploration compared to exploitation (see Supplementary Materials).

Women with greater subclinical eating restrictions believe that loss is more probable

Women with higher levels of self-reported eating restriction believe that loss is more probable overall (i.e. irrespective of condition), with this effect being significant during exploitation but not exploration (see Table 3). The 3-way interaction between Stimulus, Direction and EDE-Q restraint was not significant for exploration or exploitation and so we did not conduct further planned comparisons. Running these analyses same with hesitancy as the dependent variable, there was no main effect of EDE-Q restraint or significant 3-way interaction (Table 3). The relationship between EDE-Q and threshold was small and non-significant (see Supplementary Materials).

Acute AN and AN-WR patients believe that loss is more probable and show more hesitancy.

There was an overall effect of group on the probability of loss belief, both for exploration and exploitation (Table 3). Specifically, when compared to HC-L individuals both acute AN and AN-WR patients behaved as if the probability of reaching the loss limit was significantly greater overall (i.e. irrespective of condition' see Table 3). This effect was present for both Exploration (at trend level in acute AN) and Exploitation. However, the 3-way interaction between Group, Direction and Stimulus was not significant for either exploration or exploitation (Table 3). Running these same analyses with hesitancy as the dependent variable replicated and confirmed the same pattern of findings (Table 3). There was no significant difference in threshold between groups (see Supplementary Materials).

In sum, our first set of computational modelling supports the idea that the B-BART involves two phases, which are characterised by initially high and subsequently lower degrees of uncertainty, as suggested by recent methodological discussions of the BART[43,44]. Importantly, although loss beliefs were generally higher in our subclinical sample with high levels of eating restriction, and in our two clinical samples (AN and AN-WR) relative to the HC-L group, these beliefs did not explain the behavioural differences in body-related risk-taking. Similarly, the transition point from one phase to the other did not depend on level of eating restriction (EDE-Q restriction) or clinical grouping (AN vs. AN-WR vs. HC-L) and does not relate to the behavioural differences observed between our samples.

Women with greater subclinical eating restrictions are less risk averse overall

Higher levels of self-reported eating restriction were predictive of small but significantly lower risk aversion (ρ) overall (i.e. irrespective of condition). The 3-way interaction between Stimulus, Direction and EDE-Q restraint was not significant for risk aversion. There were no significant effects for loss aversion (λ).

Acute AN and AN-WR patients' risk aversion is modulated by body outcome

Risk aversion was significantly greater overall (i.e. irrespective of condition) in AN-WR women compared to the HC-L group, and a similar, non-significant tendency in the same direction was found in acute AN women compared to the HC-L group (Table 4). In addition, the 3-way interaction between Stimulus, Direction and Group was significant for risk aversion. Examining this interaction with our standard contrasts, we found no significant difference in risk aversion between acute AN patients and the HC-L group when considering the difference between the *increasing* body and *increasing* balloon conditions. However, acute AN patients showed a non-significant tendency for *less* risk aversion than the HC-L group when considering the difference between the *decreasing* body and *decreasing* balloon conditions.

AN-WR patients showed significantly *more* risk aversion than the HC-L group when considering the difference between the *increasing* body and *increasing* balloon conditions, and also significantly *less* risk aversion than the HC-L group when considering the difference

between the *decreasing* body and *decreasing* balloon conditions (Table 4). Similar patterns of results were observed for the selected HC-H vs. HC-L groups, as reported in Supplementary Materials.

Acute AN and AN-WR patients' loss aversion is unaffected by body outcome

There was no significant effect of group (AN, AN-WR, HC-L, HC-H) on loss aversion overall, nor a significant 3-way interaction between Stimulus, Direction and Group.

Discussion

We used a new, body and balloon analogue risk-taking task (B-BART) and computational modelling to disentangle the social and cognitive mechanisms underlying restrictive eating behaviours. We examined risk taking and eating restriction across clinical and nonclinical samples, taking into consideration the influence of the social value of thin body ideals. We confirmed existing behavioural findings of heightened risk aversion in patients with AN[23,27,29], and established the presence of this trait in weight-restored AN patients and in a non-clinical 'at risk' sample of women with higher levels of restrictive eating. Moreover, using our new task, which involves body-related stimuli, we found that both clinical and subclinical eating restriction is associated with greater risk aversion and greater behavioural uncertainty (hesitancy) when the decision is coupled with an undesirable (bigger) body outcome than merely a neutral balloon. Moreover, in both our clinical samples we also discovered that, in spite of the above general and larger body-related risk aversion tendencies, patients take *more* risk when the same monetary reward is coupled with a desired, thinner body compared to a neutral (balloon) stimulus. These findings indicate that body appearance values influence reward-based decisions under uncertainty in women with subclinical and clinical levels of eating restriction.

Our findings cast new light on recent research which suggests that restrictive eating is the result of aberrant decision and learning processes, caused by dysfunctional punishment and reward brain circuitry[14,17]. In particular, we aimed to address three major limitations of this existing research. First, by regarding eating restriction as a dimension varying along a continuum of severity from non-clinical to clinical samples and by applying our task to a large, general population sample, as well as patients at different periods of a diagnosed eating disorder, we were able to examine risk-taking over and above the confounding effects of medication, malnutrition and multiple comorbidities. We were thus able to show that low risk-taking and high behavioural uncertainty were characteristic not only of the acute AN state, but were also an enduring trait that is present in 'at risk' healthy individuals with subclinical levels of eating restriction and weight-restored AN patients.

Secondly, by extending an existing, generic risk-taking paradigm to couple monetary reward with both neutral and body-related stimuli, we were able to provide behavioural evidence that body appearance may have a role in the value-based decisions individuals take under uncertainty. Several decision-making studies in AN suggest that these patients are risk-averse[29], or loss averse[23,27], but fail to consider why AN patients, as well as healthy individuals restricting their diets, are paradoxically willing to take significant health risks in their pursuit of thinness. Based on our findings, one likely explanation is that eating restriction decisions are influenced by social, motivational values regarding body appearance, such as the thin ideal[2,11], or the reverse, the aversive value non-thin bodies may have for some individuals. Indeed, we found that both clinical and subclinical eating restriction is associated with greater risk aversion and greater behavioural uncertainty when the decision is coupled with an undesirable (bigger) body outcome. Individuals may restrict their eating to conform with such

body-related, social or internalised valuations, so that options related to different body outcomes may influence value-based decisions under uncertainty, over and above any more general tendency to avoid loss, or uncertainty. These findings may explain why individuals who have been found to be risk-averse[29] and intolerant of uncertainty[28] in self-report measures, nevertheless engage in behaviours such as extreme eating restriction or excessive exercise, that are known to have severe health risks[3–5]. Indeed, previous studies have shown that individuals make daily decisions about what, when and how much to eat by taking into account not only bodily signals (e.g. hunger, stomach fullness) and food parameters (availability, desirability[1]), but also the potential effects of eating on their body weight and size and more generally their body appearance[63,64]. For example, numerous questionnaire studies have found that exposure to the 'thin ideal' plays a determining role in how women perceive their own body and feel pressured to lose weight and be thin (reviewed by [65]). However, to our knowledge no experimental, decision-making study has provided mechanistic insight regarding such motivations by examining how women value different body appearances when making value-based decisions under uncertainty. The present findings suggest that values relating to increasing and decreasing body size may influence decision-making, over and above a general risk-aversion that is associated with individuals prone to eating restrictions.

Alternatively, our findings could be explained by low-level perceptual or attentional biases in our population, i.e. certain individuals may have changed their behaviour because they processes body stimuli with less attention, or accuracy than balloon stimuli. Although there are conflicting results regarding the role of perceptual, as opposed to attitudinal and emotional, abnormalities in body image research[66,67], at least some studies claim that subclinical and clinical populations with disordered eating have perceptual deficits, and not just different attitudes and emotional responses to body stimuli[68]. We do however think this interpretation is unlikely in the present study, as our results were not only stimulus-specific but also direction specific. It is not clear how low-level perceptual deficits could explain our directional, risk-taking findings.

Thirdly, existing studies using the BART in patients with AN have failed to disentangle different components of the task, such as the role of risk versus uncertainty, and avoidance of risk versus loss (e.g. [27]; see also [43,44] for discussion). To address these limitations we used computational modelling to first establish whether our non-clinical and clinical samples were making decisions under risk or uncertainty, and whether this would provide an explanation for observed differences in behaviour. Our first set of modelling supports the idea that the BART involves two phases, characterised by initially high levels of uncertainty and later risk. However, neither of these two phases, nor the point when people transition from uncertainty to risk, were able to explain the behavioural differences between our samples in body-related risk-taking. That is, non-clinical individuals with increased eating restriction showed an increased belief in the probability of experiencing a loss during the lower uncertainty, higher risk (exploitation) phase. Acute AN, as well as AN-WR patients show a similar increase in loss beliefs and greater hesitancy during both uncertainty and risk phases. These findings suggests that the risk taking observed generally in women with higher levels of restrictive eating is underscored by a belief that loss or punishment is more likely to occur, and this increased loss belief is a trait that spans acute AN and recovering AN-WR patients, as well as subclinical eating restriction tendencies. However, there were no statistically significant differences in the threshold between the two phases, nor any interactions with body-specific variables in either phase of the exploration-exploitation model. Thus, there was no evidence suggesting that eating restriction, or associated values ascribed to body ideals, were associated with a faster or slower transition between uncertainty and risk during the B-BART. Our modelling of risk-parameters, however, indicated that decision-making in individuals with greater levels of restrictive eating is linked to differences

in risk aversion but not loss aversion. In particular, despite both acute AN and AN-WR patients showing a general increase in risk aversion (when not specifically considering the body), they are less risk averse (i.e. willing to take more risk) when monetary rewards are coupled with a desired, decreasing body size. Additionally, AN-WR patients showed greater risk aversion when monetary rewards are coupled with an undesirable, increasing body size. Overall, this pattern of results indicates that risk aversion rather than loss aversion plays a key role in the decisions of individuals with increased levels of restrictive eating. Our findings are also consistent with the paradoxical behaviour of individuals with AN, whereby patients take considerable risk to obtain their desired body size, despite a more general aversion to risk observed in their everyday behaviour and recent experimental work (Adoue at al., 2015). Further research is needed to determine whether this behavioural risk-aversion is related to the self-reported avoidance of risk[29] and intolerant of uncertainty[28] that has been found in individuals who engage in extreme eating restriction or excessive exercise.

A limitation of our study was that we tested only women. Eating restrictions, body image concerns and anorexia nervosa are more common in women than in men, and body appearance values in men involve more than just weight variables[69,70], and hence they are more complex to experimentally manipulate and directly compare to those of women. We also noted that our AN group had a longer illness duration than the AN-WR patients, although we examined and found not relationship between severity of eating disorder symptoms and degree of risk-taking. Moreover, although we tested many samples across the spectrum of eating restriction, our study remains cross-sectional and hence with limited explanatory potential regarding developmental variables. To further specify the interpretation of our findings, future studies could also use explicit measures of the degree to which each participant values body appearance ideals, instead of only measuring body image concerns and preoccupations with one's own body image, as we did in this study. Additionally, while we did not find an effect of body image concerns or preoccupations on our findings, future studies could use more detailed measures for this multifaceted dimension. Also, it went beyond the scope of the current study to combine the two modelling approaches that we applied separately to our B-BART results, and we did not examine whether the observed level of risk taking is 'rational' or 'optimal'[71,72], nor whether using food stimuli, or manipulating hunger level might influence decision making. However, our study paves the way for future research to consider these issues in unison rather than in isolation. Finally, although we tested multiple samples and we controlled for a number of confounding variables such as mood and compulsivity symptoms, it remains possible that risk-aversion as observed in the present study relates to some other pathogenic dimension in eating restriction.

In conclusion, our study combined neurobiological and psychosocial perspectives on eating restriction into a common decision-making and computational framework that allowed us to test the interrelations between key determinants of eating restriction across subclinical and clinical samples. We found that values related to body appearance influence how individuals with eating restriction take value-based decisions, over and above their general risk-aversion and their tendency to anticipate greater loss under uncertainty. These findings cast new light on current debates concerning the psychosocial and neurobiological factors that motivate eating behaviour.

Methods

The study was conducted following the Declaration of Helsinki[73]. Institutional ethics approval was obtained from all organisations involved in the research and all participants give written, informed consent.

Participants

Non-clinical samples. In an initial, online pilot study (Study 0) we developed a body-only version of the BART (including only increasing body and decreasing body conditions, without the traditional balloon conditions) and tested the newly-developed (B-BART) task in 35 non-clinical females. Participants were aged 18 or over, had 18.5<=BMI<=30, with no reported history of eating disorder, neurological disease, or brain damage. Exclusion criteria were any history of psychiatric illness; substance abuse or dependency; or first degree relative with an eating disorder. Pilot participants were randomly assigned to take part in a between-subject design, comprising either the increasing body (N = 23) or decreasing body condition (N = 12). We used this version of the task to explore feasibility and design parameters of the newly-developed task, and to establish the existence of the effects of interest.

A subsequent non-clinical sample of 206 women, fitting the same inclusion/exclusion criteria, were recruited to an experiment conducted at a public science UK event (Study 1). We excluded any participant who did not complete the B-BART task according to instructions, or did not complete the EDE-Q Restraint questionnaire (our primary independent variable), yielding a final sample size of N=135. Study 1 used a between-subject design in which participants completed either the increasing body (N = 67) or decreasing body (N = 68) conditions, due to practical time restrictions. To ensure motivation, participants tested at the public event were told they would receive a gift for passing a pre-defined "winning" threshold (the threshold and the science-themed gifts, e.g. brain erasers, were not disclosed to the participant in advance).

A final, non-clinical sample of 318 women were recruited to an experiment conducted in a laboratory at University College London (Study 2). The same inclusion/exclusion criteria as used in Study 0 and 1 were applied (resulting in 3 exclusions; final sample 315), with the exception that a lower BMI limit of >16.5 was used to account for an observed lower average BMI in the student population obtained. Full details of the non-clinical sample demographics are provided in Table 1. For this experiment participants received a fixed amount of money for taking part (£8), plus a performance-based bonus (£2) if a pre-defined threshold (unknown to the participant) was reached.

AN Patients. Thirty-one females with acute Anorexia Nervosa (AN) were recruited from an eating disorder clinic at the San Paolo University Hospital in Milan, Italy. AN patients were aged 18 years or over, with a BMI < 18.5 and met DSM-5[6] criteria for restrictive subtype anorexia nervosa, as diagnosed as by an experienced psychiatrist, using standard clinical interview procedures. Exclusion criteria for the AN group were any documented history of brain injury, substance abuse or dependency, or concurrent psychotic disorder. Weight-restored AN patients (AN-WR; N = 23) were diagnosed by a clinician as no longer meeting DSM-5 criteria for AN, with no binge eating, purging, or restrictive eating patterns for at least 1 year before the study[20]. Because strict weight-based criteria fail to capture the clinical complexity of AN, we did not used BMI as a primary criteria for recovery. AN-WR patients had a BMI >16.5 and showed substantial improvement for at least 6 months. Characteristics of the AN and AN-WR groups, including data on current psychiatric comorbidities and medication, are summarised in Table 1.

Design

Non-clinical Studies. In the above samples, we measured the extent of restrictive eating (using the EDE-Q; see Measures) and analysed how risk taking varies when reward is coupled with desired or undesired body outcomes (see Experimental Task). Our pilot and public event studies (study 0 & 1) employed a between-subject design, with body-related stimuli (see Experimental Task, below) only due to practical and time-limitations, whereas our laboratory study used a fully-factorial, within-subject design to examine the effect of Stimulus Type (balloon vs. body) and direction (increasing vs. decreasing) on risk taking. We also explored in certain sub-samples whether key psychometric variables in eating restriction research, such as body image disturbances and concerns (BIDQ[51]), as well as related psychological dimensions of impulsiveness (Barratt Impulsiveness Scale; BIS-11[52]), obsessions and compulsions (Obsessive-Compulsive Inventory Short Version; OCI-R[53]), and affective factors such as depression, anxiety and stress (DASS-21[54]) influenced the effects found in our main analyses.

Clinical Study in AN and AN-WR patients. Our clinical study compared risk taking (B-BART task performance) in individuals with acute AN, AN-WR, and non-clinical controls (as detailed above). We manipulated the stimulus Type (balloon vs. body) and stimulus Direction (increasing vs. decreasing) in a repeated-measure design, with condition order counterbalanced across participants. Similar to the non-clinical sample, we examined in control analyses the influence of affective traits (depression, anxiety and stress) on risk-taking. Participants received vouchers of 10EUR per hour for their participation.

Balloon and Body Risk Task (B-BART)

We developed the Balloon and Body Analogue Risk Task (B-BART) by adapting a well-established behavioural measure of risk-taking, i.e. the Balloon Analogue Risk Task (BART[38]; see also [74,75] for a similar adaptation of the BART). In the B-BART, monetary reward is coupled with desirable or undesirable (body) outcomes (i.e. changes to either a female body avatar or a balloon). During the task participants click a button to win money. Each click increases the amount of money won by £0.05, and simultaneously causes the avatar body (or balloon) to increase in size (or in separate conditions, decrease in size). Importantly, each body/balloon has a random maximum limit (herein referred to as the 'loss limit') that is unknown to the participant (see below for task parameters), and when the loss limit is reached any money not collected into a permanent bank is lost. Thus, on each trial participants must choose between collecting the money they have won so far, or risk losing their winnings and increasing/decreasing the body/balloon size further in order to earn more money.

Stimuli. Stimulus bodies (avatars) were created using a Body Shape Vizualizer (© 2011, Copyright Max Planck Gesellschaf), which generates a 3D rendered model of a female body using specified body measurements (e.g. height and weight). In order to generate bodies within a visually realistic range, we fixed the height of the model (164cm) and generated three bodies corresponding to World Health Organisation Body Mass Index (BMI) values in the normal weight (64kg, BMI = 23.7), underweight (34kg, BMI = 12.6), and obese (125kg, BMI = 46.4) categories. Using these three 3D models, a computerised morphing procedure implementing a mesh warping algorithm (ABROSOFT FANTAMORPH™) was then used to morph the average body model into the maximally decreased (underweight) and maximally increased (obese) body size (see Figure 1; Panel B), generating 116 morphed frames for each of the two directions (decreasing and increasing). We followed a similar method to generate balloon stimuli: beginning with threes image of a (red) balloon of approximately the same overall image size as the average, underweight, and obese bodies, we morphed the average balloon into the maximally inflated (increased) and deflated (decreased) balloon, generating 116

images in each direction. This resulted in each pump of the balloon increased or decreased the image by approximately one pixel in all directions.

Probability of reaching the maximum limit. The probability that a body or balloon would reach its 'loss limit' was determined following the method described by Lejuez et al[38], using an array of *N* numbers. The number 1 was designated as the limit being reached. On each click, a number was randomly selected without replacement from the array. The body/balloon reached its limit if the number 1 was selected. The array contained the integers 1-116 (reflecting the 116 image frames in each condition). Thus, the probability that the limit would be reached on the first pump is 1/116. If the limit was not reached after the first click, the probability that it would be reached was 1/115 on the second click, 1/114 on the third click, and so on until the 116th click, at which point the probability of reaching the limit was 1/1 (i.e. 100%). According to this algorithm, the optimal number of clicks is 58, after which point the possible increase in earnings is reduced relative to the increased likelihood of reaching the maximum limit and losing any money accrued in the temporary bank.

Task procedure. The B-BART comprised four possible conditions: increasing body, decreasing body, increasing balloon, and decreasing balloon. In all studies, participants completed 20 trials of the relevant body/balloon conditions. Participants were given standardised, written instructions as part of the computerised task before the first trial commenced. At no point was the maximum number of pumps possible or probability of reaching the maximum limit mentioned to participants. Throughout the task, the number of trials (balloons or bodies) remaining and the total amount of money in the temporary and permanent bank were displayed on-screen. This information was provided to participants following careful piloting. The decision to display the temporary bank balance allowed us to examine learning rates based on feedback in our statistical modelling.

Measures. A number of measures can be derived directly from the BART/B-BART task, based on the pumping behaviour of the participant. In the present study we took the following measures: 1) as a measure of *explicit risk taking* we recorded the number of clicks ('pumps') on 'winning' trials (i.e. trials in which the participant collected the accumulated money before the maximum limit was reached), and 2) as an *implicit* measure o*f risk taking* behaviour we recorded behavioural uncertainty (i.e. the amount of time (ms) between the last pump and collecting the accumulated money from the trial; also referred to as *hesitancy* in collecting earnings). Further parameters derived from our computational modelling were analysed as described in the Computational Modelling section below.

Questionnaires and Scales

Eating Disorders Examination Questionnaire (EDE-Q[40]). The EDE-Q is a widely-used self-report measure of eating disorder symptoms, used in research with both clinical and non-clinical samples. The EDE-Q provides measures of dietary Restraint, Eating Concern, Shape Concern, and Weight Concern – plus a Global scale score computed from the average of the four subscales. Higher scores reflect greater eating-related pathology. Cronbach's alpha for the present study samples ranged from 0.85 to 0.95 (see Supplementary Materials).

Body Image Disturbance Questionnaire (BIDQ[51]). The BIDQ comprises quantifies body image impairment in terms of body dissatisfaction, distress and dysfunction. Low scores indicate a low level of concern, no distress, or functional limitation, whereas high scores indicating a high level of concern, emotional distress, or impairment of function. Previous research[51,76] has established the BIDQ as a valid assessment in non-clinical samples with good internal consistency and test-retest reliability.

Barratt Impulsiveness Scale Version 11 (BIS-11[52]). The BIS-11 is a widely used, self-reported assessment of impulsiveness, which has also been linked to other personality traits including risk-taking propensity and compulsiveness[77]. The BIS-11 assesses three aspects of

impulsiveness: (1) Motor Impulsiveness – i.e. acting without thinking, (2) Non-Planning Impulsiveness – i.e. a lack of forethought, and (3) Attentional Impulsiveness – i.e. an inability to focus attention or concentrate. Higher scores indicate greater levels of impulsiveness. Psychometric data for the BIS-11 indicates good convergent validity, test-retest reliability and internal consistency[77].

Depression Anxiety Stress Scales – 21-Item Version (DASS-21[54]). The DASS-21 is a set of three self-report scales designed to measure depression, anxiety and stress. Scores for each scale are calculated by summing the relevant subscale items, with higher scores indicating a greater occurrence of the specific dimension. The DASS-21 has demonstrated good psychometric properties, with good concurrent validity and internal consistency[78].

Obsessive-Compulsive Inventory (short version, OCI-R[53]). The OCI-R is a self-report questionnaire designed to assess a variety of obsessions and compulsions, including obsessing, washing, hoarding, ordering, checking and neutralizing behaviours. The outcome measure is a sum of all items, with a possible range from 0-72, and higher scores indicating greater obsessive-compulsive behaviours. The OCI-R has good to excellent internal consistency, test-retest reliability, and convergent validity[53].

General Procedures

Non-clinical studies. Study 0 (pilot) was conducted online using Inquisit (Millisecond software; https://www.millisecond.com) to administer the B-BART and Online Surveys (https://www.onlinesurveys.ac.uk) for completion of questionnaires. Study 1 took place at a public event, with the B-BART (programmed in C++) and completion of questionnaires taking approximately 20 minutes to complete. Study 2 was conducted at an experimental psychology laboratory located at University College London. Participants completed the B-BART and all questionnaires in a single session lasting approximately 1.5 hours.

Clinical study. Participants were tested in a single session at an eating disorder research clinic in Milan, Italy, by an experienced psychology research assistant. The session included a clinical assessment (including measurement of height and weight), B-BART, and questionnaires. Note that 4 out of the 31 AN patients completed only two out of the four B-BART conditions due to an administrative error.

Statistical Analyses

All statistical analyses were performed with R[79] with figures generated using ggplot 2[80].

Non-clinical studies. We were interested in testing how risk-taking in a non-clinical sample is affected by the level of restrictive eating (indexed by EDE-Q restraint score), stimulus Type (body or balloon) and stimulus Direction (increasing or decreasing). Our pilot study (Study 0) and public event (Study 1) utilised a between-subject design, with participants randomly allocated to either increasing body or decreasing body conditions (see Design). We performed separate simple linear regression on each condition to determine how risk taking changes as EDE-Q Restraint increases. We used as dependent variable the average number of clicks per participant for trials where the participant collected winnings prior to the body reaching the loss limit. Independent variables were EDE-Q Restraint score, Age and BMI. This analysis did not allow us to directly assess the interaction between Direction and EDE-Q Restraint; however, inspection of the regression slopes of each condition provided a guide to the effect direction and magnitude of the relative change in risk-taking dependent on EDE-Q Restraint for each stimulus Direction. We analysed the data from these two studies both separately and together (reporting the results in Supplementary Materials for brevity).

Subsequently, we collected the data for Study 2, in which participants undertook all four conditions (*Increasing Body*, *Decreasing Body*, *Increasing Balloon*, *Decreasing Balloon*)

in a within-subject design. To evaluate the relative difference in risk taking between conditions as EDE-Q Restraint increases, we performed step-wise Multilevel modelling analysis on the combined data from Studies 0, 1 and 2, culminating in the effect of the interaction between stimulus Type (*Body/Balloon*), Direction (*Increasing/Decreasing*), and EDE-Q Restraint score. As Random Effects we used the intercepts (not slopes) of the Subject (Participant ID), Condition Order and Experimenter. As Fixed Effects, participant Age and BMI were used as covariates, and Stimulus, Direction and EDE-Q Restraint were used as independent variables of interest. The same analysis was run twice; firstly, to assess explicit *risk-taking*, we used as dependent variable the number of pumps made by the participant on "winning" trials, i.e. where earnings were collected prior to the limit being reached. Secondly, as a measure of decision-making uncertainty, we used as dependent variable the Logarithm of the time taken between the last pump and the collection of temporary earnings (multiplied by 1000 to make the results more readable). We labelled this variable *hesitancy (in collecting earnings*, which we collected in Study 1 and 2 but not the pilot (Study 0) due to limitations of the software used. Within these step-wise analyses, from each model to the next, we added one independent variable and measured with R's "anova" function (of the "stats" package) if this addition increased in a statistically significant way the explanatory power of our model. The "anova" function of R takes as input the step-wise sequentially constructed nested Multilevel models, and compares them sequentially, two at a time, using a Chi Square test of the deviances of the two sequential models, to test whether the more general model fits significantly better than the simpler model.

The following sequential steps took place in this process: a) we used the Random Effects as the baseline model, b) we evaluated the effect of Age and BMI with respect to the baseline, c) we evaluated the effects of Stimulus and Direction independently as well as their interaction with respect to the previous steps, d) we evaluated the effect of EDE-Q Restraint and its interaction with Stimulus and Direction with respect to the previous steps (i.e. the three-way interaction between EDE-Q x Stimulus x Direction). Where this three-way interaction was the winning model (i.e. the model providing a significantly better fit for the data than any of the simpler models, as determined by our hierarchical MLM procedure) we subsequently broke down this interaction by performed a similar step-wise Multilevel analysis to evaluate the significance and effect of two planned comparisons/interactions of interest. We were specifically interested in establishing if individuals with higher levels of eating restraint took less risk when reward was coupled with an 'undesirable' body outcome (i.e. increasing size), while taking more risk when the outcome was a 'desirable' body (i.e. decreasing size). We therefore compared the regression slopes of each body condition with its respective balloon control condition (i.e. body increase vs. balloon increase; and body decrease vs. balloon decrease).

In results tables we present summary information comprising a) the slope *b*, and its *SD* taken from the most complex model in the step-wise MLM analysis, b) Chi squared and *p*-values are the results of the 'anova' comparison between the model of the current step (most complex model) and the model of the previous step, c) Cohen's f squared is computed comparing the Fixed and Random Effects variance between the current model and the "empty" model (the model with no Fixed Effects). The full statistical models and results from these analyses are presented in the Supplementary Materials.

Clinical study. To examine risk-taking in acute AN and AN-WR patients compared to HCs we first performed a series of preliminary analyses (reported in Supplementary Materials) to create and validate a healthy control group from our large, non-clinical, healthy control sample. Briefly, this involved creating two sub-groups of healthy controls characterised by high or low disordered eating (HC-L and HC-H respectively). We created these two groups by selecting from our Study 2 healthy control sample individuals with (1) a BMI matching that of the AN-WR patients (in order to remove, as much as possible, the effect of BMI in Risk-taking;

in the clinical study BMI was used as a covariate, but cannot be used as such here since BMI is one of the dimensions used to define the clinical groups), and (2) an EDE-Q Restraint score in the lowest quartile of the sample (i.e. 25th percentile; HC-L; N = 38) and highest quartile (i.e. 75th percentile; HC-H; N = 35), in order to see if the HCs with high levels of restrictive eating (similar to the same subscale scores of the AN group) and the HCs with low levels of restrictive eating performed differently in risk-taking compared to the AN group in the four experimental conditions.

Our main analysis followed the same strategy to that used to analyse the non-clinical data. We examined the relative difference in risk-taking across the four within-subject conditions (increasing body, decreasing body, increasing balloon, decreasing balloon) and between groups (AN, AN-WR, HC-L, HC-H) using step-wise Multilevel modelling that culminated with the three-way interaction between the Stimulus Type (*Body/Balloon*), Direction (*Increasing/Decreasing*) and Group. As Random Effects we used the intercepts (not slopes) of the Subject (Participant ID), and Condition Order. As Fixed Effects, Age was used as a covariate, and Stimulus, Direction and Group were used as the independent variables of interest. The dependent variables were 1) our explicit measure of *risk taking*, i.e. the number of clicks for the collected trials, and 2) *hesitancy* (as above). Within these step-wise analysis, from each model to the next, we added one independent variable and measured with R's 'anova' package if this addition increased in a statistically significant way the explanatory power of our model.

Following the same procedure used to analyse the non-clinical data: a) we used the Random Effects as the baseline model, b) we evaluated the effect of Age with respect to the baseline, c) we evaluated the effects of Stimulus and Direction independently as well as their interaction with respect to the previous steps, d) we evaluated the effect of Group and its interaction with Stimulus and Direction with respect to the previous steps. Subsequently, we performed a step-wise Multilevel analysis to evaluate the significance and effect of the three-way interaction, using the same planned comparisons described above for the non-clinical data analysis. However, instead of looking at the differences between conditions dependent on EDE-Q restraint scores, we compared in each analysis the relative difference in the slopes of the clinical groups to that of the HC-L group.

Computational Modelling

We implemented two sets of cognitive models to examine 1) uncertainty vs. risk, and 2) aversion to risk vs. loss, as drivers of observed risk-taking behaviour. Based on previous modelling studies on the BART[45,58], we created two sets of models to assess these two objectives (see Introduction for an overview and Supplementary Materials for full modelling details). In each case we tested the fit and compared the models in the non-clinical data using Maximum Likelihood Estimation (MLE), BIC and AIC. We selected the winning model based on best fit as detailed in the Supplementary Materials. Further analysis, as detailed below, was conducted on the parameters of the winning models. The Exploration-Exploitation model has three parameters: (a) *Prior Probability of Loss Belief* (loss belief during Exploration), (b) *Posterior Probability of Loss Belief* (loss belief during Exploitation), and (c) *Threshold* (the trial at which the transition from Exploration to Exploitation takes place. The EW model (described fully in [46] and summarised in Supplementary Materials) contains five parameters, two of which are of direct relevance to the current study aims and are summarised here (see Supplementary Materials for full details). *Risk aversion* (ρ) indicates an individual's sensitivity to the value of reward change, such that individuals with higher risk avoidance take less risk to get the same amount of reward. *Loss-aversion* (λ) indicates an individual's sensitivity to negative outcomes, such that potential loss is perceived as more severe for higher λ.

Model Validation. To validate the exploration-exploitation model of uncertainty and risk, we analysed in our non-clinical samples whether two independent behavioural measures not used to construct the model (i.e. hesitancy calculated as described above, and behavioural variability calculated as SD/Average clicks), showed an expected difference between exploration and exploitation phases, with greater hesitancy and behavioural variability expected during exploration compared to exploitation. We compared between Phases (exploration vs. exploitation) the hesitancy and behavioural variability (see Supplementary Materials). Secondary validation was performed via supervised learning clustering analysis as specified in Supplementary Materials.

Analysis. We conducted two sets of analyses on the modelling data, with the aim of first examining if decisions are made under uncertainty versus risk, and whether this might provide an explanation for general and body-related risk taking across different levels of non-clinical and clinical eating restriction. We then looked at whether risk- or loss-aversion might provide a better explanation of risky decision-making behaviours across the different levels of non-clinical and clinical restrictive eating. The analysis was the same for both sets of modelling. To analyse data from the non-clinical sample, we used separate MLMs with the winning models' parameters as dependent variables, and EDE-Q restraint, age and BMI as independent variables. For the modelling of uncertainty versus risk we also conducted this analysis with hesitancy as the dependent variable (separately for exploration and exploitation). For our clinical data we examined how these same dependent variables (model parameters) were affected in AN and AN-WR patients, following the same plan of analysis used for our behavioural data, i.e. by examining for each dependent variable the overall effect of group (HC-L, HC-H, AN, AN-WR) and the 3-way interaction between Group (HC-L, AN, AN-WR), Stimulus Type (Body, Balloon) and Direction (Increase, Decrease), with planned comparisons carried out if the main effect or three-way interaction was significant.

References

1. Lowe, M. R. & Levine, A. S. Eating motives and the controversy over dieting: eating less than needed versus less than wanted. *Obes. Res.* **13**, 797–806 (2005).
2. Thompson, J. K. & Stice, E. Thin-Ideal Internalization: Mounting Evidence for a New Risk Factor for Body-Image Disturbance and Eating Pathology. *Curr. Dir. Psychol. Sci.* **10**, 181–183 (2001).
3. Hawks, S. R., Madanat, H. N. & Christley, H. S. Behavioral and Biological Associations of Dietary Restraint: A Review of the Literature. *Ecol. Food Nutr.* **47**, 415–449 (2008).
4. French, S. A. & Jeffery, R. W. Consequences of dieting to lose weight: Effects on physical and mental health. *Health Psychol.* **13**, 195–212 (1994).
5. Polivy, J. Psychological Consequences of Food Restriction. *J. Am. Diet. Assoc.* **96**, 589–592 (1996).
6. American Psychiatric Association. *American Psychiatric Association, 2013. Diagnostic and statistical manual of mental disorders (5th ed.)*. (2013). doi:10.1176/appi.books.9780890425596.744053.
7. Arcelus, J., Mitchell, A. J., Wales, J. & Nielsen, S. Mortality Rates in Patients With Anorexia Nervosa and Other Eating Disorders: A Meta-analysis of 36 Studies. *Arch. Gen. Psychiatry* **68**, 724–731 (2011).
8. Heatherton, T. F. & Polivy, J. Chronic dieting and eating disorders: A Spiral Model. in *The etiology of bulimia nervosa: The individual and familial context* (eds. Crowther, J. H., Tennenbaum, D. L., Hobfoll, S. E. & Stephens, M. A. P.) 113–155 (Hemisphere Publishing Corp, 1992). doi:10.4324/9780203782286-15.
9. Roth, D. A., Herman, C. P., Polivy, J. & Pliner, P. Self-presentational conflict in social eating situations: a normative perspective. *Appetite* **36**, 165–171 (2001).
10. Herman, C. P. & Polivy, J. Normative influences on food intake. *Physiol. Behav.* **86**, 762–772 (2005).
11. Diedrichs, P. C. Sociocultural Environment and Internalization of the Thin Ideal as Eating Disorder Risk Factors. in *Encyclopedia of Feeding and Eating Disorders* (ed. Wade, T.) 1–5 (Springer, 2015). doi:10.1007/978-981-287-087-2_89-1.
12. Grabe, S., Ward, L. M. & Hyde, J. S. The role of the media in body image concerns among women: a meta-analysis of experimental and correlational studies. *Psychol. Bull.* **134**, 460–476 (2008).
13. Becker, A. E., Burwell, R. A., Gilman, S. E., Herzog, D. B. & Hamburg, P. Eating behaviours and attitudes following prolonged exposure to television among ethnic Fijian adolescent girls. *Br. J. Psychiatry J. Ment. Sci.* **180**, 509–514 (2002).
14. Kaye, W. H., Wierenga, C. E., Bailer, U. F., Simmons, A. N. & Bischoff-Grethe, A. Nothing Tastes as Good as Skinny Feels: The Neurobiology of Anorexia Nervosa. *Trends Neurosci.* **36**, (2013).
15. Treasure, J., Cardi, V., Leppanen, J. & Turton, R. New treatment approaches for severe and enduring eating disorders. *Physiol. Behav.* **152**, 456–465 (2015).
16. Lloyd, E. C. & Steinglass, J. E. What can food-image tasks teach us about anorexia nervosa? A systematic review. *J. Eat. Disord.* **6**, (2018).
17. Park, R. J., Godier, L. R. & Cowdrey, F. A. Hungry for reward: How can neuroscience inform the development of treatment for Anorexia Nervosa? *Behav. Res. Ther.* **62**, 47–59 (2014).
18. Keating, C., Tilbrook, A. J., Rossell, S. L., Enticott, P. G. & Fitzgerald, P. B. Reward processing in anorexia nervosa. *Neuropsychologia* **50**, 567–575 (2012).
19. Haynos, A. F., Lavender, J. M., Nelson, J., Crow, S. J. & Peterson, C. B. Moving towards specificity: A systematic review of cue features associated with reward and punishment in anorexia nervosa. *Clin. Psychol. Rev.* **79**, 101872 (2020).

20. Wierenga, C. E. *et al.* Hunger does not motivate reward in women remitted from anorexia nervosa. *Biol. Psychiatry* **77**, 642–652 (2015).
21. Bischoff-Grethe, A. *et al.* Altered brain response to reward and punishment in adolescents with Anorexia nervosa. *Psychiatry Res. Neuroimaging* **214**, 331–340 (2013).
22. King, J. A. *et al.* Altered Neural Efficiency of Decision Making During Temporal Reward Discounting in Anorexia Nervosa. *J. Am. Acad. Child Adolesc. Psychiatry* **55**, 972–979 (2016).
23. Bernardoni, F. *et al.* Altered Medial Frontal Feedback Learning Signals in Anorexia Nervosa. *Biol. Psychiatry* **83**, 235–243 (2018).
24. Decker, J. H., Figner, B. & Steinglass, J. E. On weight and waiting: delay discounting in anorexia nervosa pre- and post-treatment. *Biol. Psychiatry* **78**, 606–614 (2015).
25. Guillaume, S. *et al.* Impaired decision-making in symptomatic anorexia and bulimia nervosa patients: a meta-analysis. *Psychol. Med.* **45**, 3377–3391 (2015).
26. Bickel, W. K. & Marsch, L. A. Toward a behavioral economic understanding of drug dependence: delay discounting processes. *Addict. Abingdon Engl.* **96**, 73–86 (2001).
27. Adoue, C. *et al.* A further assessment of decision-making in anorexia nervosa. *Eur. Psychiatry* **30**, 121–127 (2015).
28. Frank, G. K. W. *et al.* Heightened Fear of Uncertainty in Anorexia and Bulimia Nervosa. *Int. J. Eat. Disord.* **45**, 227 (2012).
29. Jappe, L. M. *et al.* Heightened Sensitivity to Reward and Punishment in Anorexia Nervosa. *Int. J. Eat. Disord.* **44**, 317 (2011).
30. McClelland, J. *et al.* A Randomised Controlled Trial of Neuronavigated Repetitive Transcranial Magnetic Stimulation (rTMS) in Anorexia Nervosa. *PLoS ONE* **11**, (2016).
31. Wu, M. *et al.* Reward-related decision making in eating and weight disorders: A systematic review and meta-analysis of the evidence from neuropsychological studies. *Neurosci. Biobehav. Rev.* **61**, 177–196 (2016).
32. Leitch, M. A., Morgan, M. J. & Yeomans, M. R. Different subtypes of impulsivity differentiate uncontrolled eating and dietary restraint. *Appetite* **69**, 54–63 (2013).
33. Yeomans, M. R. & Brace, A. Cued to Act on Impulse: More Impulsive Choice and Risky Decision Making by Women Susceptible to Overeating after Exposure to Food Stimuli. *PLOS ONE* **10**, e0137626 (2015).
34. Chan, T. W. S. *et al.* Differential impairments underlying decision making in anorexia nervosa and bulimia nervosa: a cognitive modeling analysis. *Int. J. Eat. Disord.* **47**, 157–167 (2014).
35. Verharen, J. P. H. *et al.* Insensitivity to Losses: A Core Feature in Patients With Anorexia Nervosa? *Biol. Psychiatry Cogn. Neurosci. Neuroimaging* **4**, 995–1003 (2019).
36. Kroemer, N. B. Exaggerated Control Demands Over Reward-Related Behavior in Anorexia Nervosa. *Biol. Psychiatry* **83**, 194–196 (2018).
37. Lauriola, M., Panno, A., Levin, I. P. & Lejuez, C. W. Individual Differences in Risky Decision Making: A Meta-analysis of Sensation Seeking and Impulsivity with the Balloon Analogue Risk Task. *J. Behav. Decis. Mak.* **27**, 20–36 (2014).
38. Lejuez, C. W. *et al.* Evaluation of a behavioral measure of risk taking: The Balloon Analogue Risk Task (BART). *J. Exp. Psychol. Appl.* **8**, 75–84 (2002).
39. Schmitz, F., Manske, K., Preckel, F. & Wilhelm, O. The Multiple Faces of Risk-Taking. *Eur. J. Psychol. Assess.* **32**, 17–38 (2016).
40. Fairburn, C. G. & Beglin, S. J. Assessment of eating disorders: interview or self-report questionnaire? *Int. J. Eat. Disord.* **16**, 363–370 (1994).
41. Hauser, T. U., Moutoussis, M., Dayan, P. & Dolan, R. J. Increased decision thresholds trigger extended information gathering across the compulsivity spectrum. *Transl. Psychiatry* **7**, (2017).

42. Bulik, C. M. *et al.* Genetic epidemiology, endophenotypes, and eating disorder classification. *Int. J. Eat. Disord.* **40**, S52–S60 (2007).
43. De Groot, K. & Thurik, R. Disentangling Risk and Uncertainty: When Risk-Taking Measures Are Not About Risk. *Front. Psychol.* **0**, (2018).
44. Groot, K. D. Burst Beliefs – Methodological Problems in the Balloon Analogue Risk Task and Implications for Its Use. *J. Trial Error* **1**, (2020).
45. Wallsten, T. S., Pleskac, T. J. & Lejuez, C. W. Modeling behavior in a clinically diagnostic sequential risk-taking task. *Psychol. Rev.* **112**, 862–880 (2005).
46. Park, H., Yang, J., Vassileva, J. & Ahn, W.-Y. Development of a novel computational model for the Balloon Analogue Risk Task: The Exponential-Weight Mean-Variance Model. (2019) doi:10.31234/osf.io/sdzj4.
47. Heinz, A., Schlagenhauf, F., Beck, A. & Wackerhagen, C. Dimensional psychiatry: mental disorders as dysfunctions of basic learning mechanisms. *J. Neural Transm.* **123**, 809–821 (2016).
48. Hengartner, M. P. & Lehmann, S. N. Why Psychiatric Research Must Abandon Traditional Diagnostic Classification and Adopt a Fully Dimensional Scope: Two Solutions to a Persistent Problem. *Front. Psychiatry* **8**, 101 (2017).
49. Adams, R. A., Huys, Q. J. M. & Roiser, J. P. Computational Psychiatry: towards a mathematically informed understanding of mental illness. *J. Neurol. Neurosurg. Psychiatry* jnnp-2015-310737 (2015) doi:10.1136/jnnp-2015-310737.
50. Huys, Q. J. M., Maia, T. V. & Frank, M. J. Computational psychiatry as a bridge from neuroscience to clinical applications. *Nat. Neurosci.* **19**, 404–413 (2016).
51. Cash, T. F., Phillips, K. A., Santos, M. T. & Hrabosky, J. I. Measuring "negative body image": validation of the Body Image Disturbance Questionnaire in a nonclinical population. *Body Image* **1**, 363–372 (2004).
52. Patton, J. H., Stanford, M. S. & Barratt, E. S. Factor structure of the Barratt impulsiveness scale. *J. Clin. Psychol.* **51**, 768–774 (1995).
53. Foa, E. B. *et al.* The Obsessive-Compulsive Inventory: Development and validation of a short version. *Psychol. Assess.* **14**, 485–496 (2002).
54. Lovibond, S. H. & Lovibond, P. F. *Manual for the depression anxiety stress scales*. (Psychology Foundation of Australia, 1995).
55. Zakzanis, K. K., Campbell, Z. & Polsinelli, A. Quantitative evidence for distinct cognitive impairment in anorexia nervosa and bulimia nervosa. *J. Neuropsychol.* **4**, 89–106 (2010).
56. Brewin, N., Baggott, J., Dugard, P. & Arcelus, J. Clinical normative data for eating disorder examination questionnaire and eating disorder inventory for DSM-5 feeding and eating disorder classifications: a retrospective study of patients formerly diagnosed via DSM-IV. *Eur. Eat. Disord. Rev. J. Eat. Disord. Assoc.* **22**, 299–305 (2014).
57. Berg, K. C., Peterson, C. B., Frazier, P. & Crow, S. J. Psychometric evaluation of the eating disorder examination and eating disorder examination-questionnaire: a systematic review of the literature. *Int. J. Eat. Disord.* **45**, 428–438 (2012).
58. van Ravenzwaaij, D., Dutilh, G. & Wagenmakers, E.-J. Cognitive model decomposition of the BART: Assessment and application. *J. Math. Psychol.* **55**, 94–105 (2011).
59. van Ravenzwaaij, D., Dutilh, G. & Wagenmakers, E.-J. Cognitive model decomposition of the BART: Assessment and application. *J. Math. Psychol.* **55**, 94–105 (2011).
60. Wagenmakers, E.-J., Van Der Maas, H. L. J. & Grasman, R. P. P. P. An EZ-diffusion model for response time and accuracy. *Psychon. Bull. Rev.* **14**, 3–22 (2007).
61. Hills, T. T., Todd, P. M., Lazer, D., Redish, A. D. & Couzin, I. D. Exploration versus exploitation in space, mind, and society. *Trends Cogn. Sci.* **19**, 46–54 (2015).

62. Gopnik, A. *et al.* Changes in cognitive flexibility and hypothesis search across human life history from childhood to adolescence to adulthood. *Proc. Natl. Acad. Sci.* **114**, 7892–7899 (2017).
63. Keery, H., van den Berg, P. & Thompson, J. K. An evaluation of the Tripartite Influence Model of body dissatisfaction and eating disturbance with adolescent girls. *Body Image* **1**, 237–251 (2004).
64. Mills, J. S., Polivy, J., Herman, C. P. & Tiggemann, M. Effects of Exposure to Thin Media Images: Evidence of Self-Enhancement among Restrained Eaters: *Pers. Soc. Psychol. Bull.* (2016) doi:10.1177/014616702237650.
65. Mask, L. & Blanchard, C. M. The effects of 'thin ideal' media on women's body image concerns and eating-related intentions: the beneficial role of an autonomous regulation of eating behaviors. *Body Image* **8**, 357–365 (2011).
66. Cash, T. F. & Deagle, E. A. The nature and extent of body-image disturbances in anorexia nervosa and bulimia nervosa: a meta-analysis. *Int. J. Eat. Disord.* **22**, 107–125 (1997).
67. Hagman, J. *et al.* Body size overestimation and its association with body mass index, body dissatisfaction, and drive for thinness in anorexia nervosa. *Eat. Weight Disord. EWD* **20**, 449–455 (2015).
68. Esposito, R., Cieri, F., di Giannantonio, M. & Tartaro, A. The role of body image and self-perception in anorexia nervosa: the neuroimaging perspective. *J. Neuropsychol.* **12**, 41–52 (2018).
69. Smith, A. R., Hawkeswood, S. E., Bodell, L. P. & Joiner, T. E. Muscularity versus leanness: An examination of body ideals and predictors of disordered eating in heterosexual and gay college students. *Body Image* **8**, 232–236 (2011).
70. Talbot, D., Smith, E., Cass, J. & Griffiths, S. Development and validation of the New Somatomorphic Matrix–Male: A figural rating scale for measuring male actual–ideal body discrepancy. *Psychol. Men Masculinities* **20**, 356–367 (2019).
71. Keller, G., Novák, V. & Willems, T. A note on optimal experimentation under risk aversion. *J. Econ. Theory* **179**, 476–487 (2019).
72. Benjamin, A. M. & Robbins, S. J. The role of framing effects in performance on the Balloon Analogue Risk Task (BART). *Personal. Individ. Differ.* **43**, 221–230 (2007).
73. World Medical Association. World Medical Association Declaration of Helsinki: ethical principles for medical research involving human subjects. *JAMA* **310**, 2191–2194 (2013).
74. Salvato, G., De Maio, G. & Bottini, G. Interoceptive sensibility tunes risk-taking behaviour when body-related stimuli come into play. *Sci. Rep.* **9**, 2396 (2019).
75. Salvato, G. *et al.* Pathological risk-propensity typifies Mafia members' cognitive profile. *Sci. Rep.* **10**, 8559 (2020).
76. Cash, T. F. & Grasso, K. The norms and stability of new measures of the multidimensional body image construct. *Body Image* **2**, 199–203 (2005).
77. Stanford, M. S. *et al.* Fifty years of the Barratt Impulsiveness Scale: An update and review. *Personal. Individ. Differ.* **47**, 385–395 (2009).
78. Antony, M. M., Bieling, P. J., Cox, B. J., Enns, M. W. & Swinson, R. P. Psychometric properties of the 42-item and 21-item versions of the Depression Anxiety Stress Scales in clinical groups and a community sample. *Psychol. Assess.* **10**, 176–181 (1998).
79. R Core Team. R: A Language and Environment for Statistical Computing. (2013).
80. Wickham, H. ggplot2: Elegant Graphics for Data Analysis. (2016).

Acknowledgements
The study was supported by a European Research Council Consolidator Award [ERC- 2018-COG-818070] to A.K. We thank the following individuals for their assistance with data collection: Rita El Hachem, Sing Yee Wong, Benjamin Ang, Raluca-Alexandra Petre-Melinte, Yu Chow, Faith Shu Ting, Yvonne Van Hoecke and Alkistis Saramandi.

Author contributions statement
A.F. and E.P. designed the research with input from E.V. A.T. analysed the data and produced all tables and figures with input from A.F. and P.M.J. B.D. and V.N. contributed to the design planning of the clinical aspects of the research and collected the clinical data. O.G. provided clinical input and supervision. E.P. and E.V. collected the non-clinical data and supervised students that helped with data collection. P.M.J wrote the paper with input from A.F. and A.T. All authors provided feedback on the paper.

Data and code availability
All data and code used in the manuscript are available via GitHub: https://github.com/katlaboratory/RiskTaking

Table 1. Demographic and clinical characteristics.

	Clinical Study				Non-Clinical Study	
	AN	AN-WR	HC-L	HC-H	S0+S1	S2
	Mean (sd)	Mean (sd)	Mean (sd)	Mean (sd)	Mean (sd)	Mean (sd)
N	31	23	38	35	170	315
Age	24.9 (8.72)	26.13 (7.5)	22.89 (3.29)	22.46 (4.67)	32.28 (10.58)	23.92 (6.63)
BMI	15.88 (1.48)	19.7 (1.72)	19.51 (0.91)	19.73 (0.83)	22.23 (2.61)	22.21 (4.33)
Illness duration (years)	7.55(8.17)	4.66(4.53)	-	-	-	-
Avg Pumps	25.73 (12.62)	24.66 (15.35)	33.41 (9.47)	31.58 (7.67)	34.35 (14.56)	33.15 (10.99)
Hesitance	589.09 (246.08)	580.36 (220.66)	300.7 (301.69)	357.28 (422.54)	-	303.26 (322.48)
EDE-Q Total	2.98 (1.67)	2.19 (1.75)	0.44 (0.51)	2.53 (1.07)	1.53 (1.12)	1.61 (1.26)
EDE-Q Restraint	2.44 (1.89)	1.91 (1.8)	0 (0)	2.83 (1.15)	1.46 (1.47)	1.31 (1.42)
DASS-21 Anxiety	7.5 (4.52)	6.27 (4.98)	5.56 (5.61)	5.5 (4.83)	-	5.77 (4.46)
DASS-21 Depression	12.65 (5.94)	7.91 (6.02)	4.56 (4.19)	6.5 (4.65)	-	4.81 (4.09)
DASS-21 Stress	13.04 (4.89)	8.82 (5.32)	6.22 (5.02)	8.21 (4.28)	-	7.74 (4.53)
OCD-10 Total	-	-	12.67 (8.47)	23.86 (14.35)	-	21.09 (13.25)
BIS-11 Total	-	-	64.92 (11.99)	69.45 (11.32)	-	68.13 (11.42)
Psychiatric Comorbidities	14 Mood disorder	9 Mood disorder				
	5 GAD	6 GAD				
	9 OCD	4 OCD				
	3 Panic disorder	6 Panic disorder				
	2 Personality disorder	0 Personality disorder				
Current Medication	4 Antidepressants	1 Antidepressants				
	2 Sedatives	0 Sedatives				
	1 Anxiolytics	1 Anxiolytics				
	3 Antipsychotics	1 Antipsychotics				

Note: one AN participant was removed from the Hesitance overall mean because she was an outlier in overall slowness. Anxiety disorders includes panic disorder; mood disorders include MDD. Dashes indicate measure not taken.

Table 2. Results of main behavioural analyses in non-clinical (upper half) and clinical (lower half) samples

non-Clinical Explicit Risk-Taking				
Effect	**β(sd)**	**p**	**X^2(df)**	**f^2**
EDE-Q Restraint	-0.8(0.38)	**0.037**	4.35(1)	0.008
Stimulus x Direction x EDE-Q Restraint	-0.76(0.26)	**<0.001**	21.22(3)	0.008

non-Clinical Hesitance			
β(sd)	**p**	**X^2(df)**	**f^2**
-2.95(7.55)	0.696	0.15(1)	0.026
8.67(6.49)	**0.014**	10.57(3)	0.035

non-Clinical Risk-Taking Increase Body vs Balloon				
Effect	**β(sd)**	**p**	**X^2(df)**	**f^2**
Stimulus x EDE-Q Restraint	-0.64(0.18)	**<0.001**	12.14(1)	0.014

non-Clinical Hesitance Increase Body vs Balloon			
β(sd)	**p**	**X^2(df)**	**f^2**
13.45(4.56)	**0.003**	8.67(1)	0.023

non-Clinical Risk-Taking Decrease Body vs Balloon				
Effect	**β(sd)**	**p**	**X^2(df)**	**f^2**
Stimulus x EDE-Q Restraint	0.01(0.18)	0.957	0(1)	-0.008

non-Clinical Hesitance Decrease Body vs Balloon			
β(sd)	**p**	**X^2(df)**	**f^2**
4.66(4.6)	0.311	1.03(1)	0.037

Clinical Explicit Risk-Taking				
Effect	**β(sd)**	**p**	**X^2(df)**	**f^2**
ALL GROUPS		**0.005**	12.7(3)	0.055
AN vs HC-L	-7.44(2.6)	**0.005**	7.72(1)	0.062
AN-WR vs HC-L	-8.63(3.25)	**0.01**	6.66(1)	0.067
HC-H vs HC-L	-2.01(1.97)	0.308	1.04(1)	0.02
Stimulus x Direction x GROUP		**0.001**	27.36(9)	0.059

Clinical Hesitance			
β(sd)	**p**	**X^2(df)**	**f^2**
	<0.001	21.25(3)	0.103
316.6(68.51)	**<0.001**	18.65(1)	0.150
271.77(71.3)	**<0.001**	13.02(1)	0.111
48.51(78.34)	0.536	0.38(1)	0.014
	<0.001	41.84(9)	0.106

Clinical Risk-Taking Increase Body vs Balloon (AN vs HC-L)				
Effect	**β(sd)**	**p**	**X^2(df)**	**f^2**
Stimulus x GROUP	-1.93(1.01)	0.058	3.61(1)	0.085

Clinical Hesitance Increase Body vs Balloon (AN vs HC-L)			
β(sd)	**p**	**X^2(df)**	**f^2**
124.95(27.66)	**<0.001**	20.27(1)	0.202

Clinical Risk-Taking Decrease Body vs Balloon (AN vs HC-L)				
Effect	**β(sd)**	**p**	**X^2(df)**	**f^2**
Stimulus x GROUP	4.11(1.03)	**<0.001**	15.49(1)	0.049

Clinical Hesitance Decrease Body vs Balloon (AN vs HC-L)			
β(sd)	**p**	**X^2(df)**	**f^2**
12.87(26.64)	0.63	0.23(1)	0.142

Clinical Risk-Taking Increase Body vs Balloon (AN-WR vs HC-L)				

Clinical Hesitance Increase Body vs Balloon (AN-WR vs HC-L)			

Effect	β(sd)	p	X^2(df)	f^2	β(sd)	p	X^2(df)	f^2
Stimulus x GROUP	-2.37(1.1)	**0.031**	4.66(1)	0.069	35.73(27.59)	0.195	1.68(1)	0.121

	Clinical Risk-Taking Decrease Body vs Balloon (AN-WR vs HC-L)				Clinical Hesitance Decrease Body vs Balloon (AN-WR vs HC-L)			
Effect	**β(sd)**	**p**	**X^2(df)**	**f^2**	**β(sd)**	**p**	**X^2(df)**	**f^2**
Stimulus x GROUP	2.65(1.11)	**0.017**	5.68(1)	0.060	19.66(28.01)	0.483	0.49(1)	0.121

Table 3. Results of uncertainty vs. risk computational modelling analyses in non-clinical (upper half) and clinical (lower half) samples

	non-Clinical Prior Probability of Loss Belief (exploration)			
Effect	**β(sd)**	**p**	**X^2(df)**	**f^2**
EDE-Q Restraint	0.02(0.02)	0.325	0.97(1)	0.044
Stimulus x Direction x EDE-Q Restraint	0.01(0.04)	0.442	2.69(3)	0.044

	non-Clinical Prior Hesitance (exploration)			
β(sd)	**p**	**X^2(df)**	**f^2**
1.24(9.14)	0.892	0.02(1)	0.044
9.41(17.27)	0.825	0.9(3)	0.055

	non-Clinical Posterior Probability of Loss Belief (exploitation)			
Effect	**β(sd)**	**p**	**X^2(df)**	**f^2**
EDE-Q Restraint	0.04(0.02)	**0.018**	5.57(1)	0.025
Stimulus x Direction x EDE-Q Restraint	-0.01(0.03)	0.379	3.08(3)	0.025

	non-Clinical Posterior Hesitance (exploitation)			
β(sd)	**p**	**X^2(df)**	**f^2**
-1.99(8.48)	0.814	0.06(1)	0.050
11.75(13.82)	0.773	1.12(3)	0.068

	Clinical Prior Probability of Loss Belief (exploration)			
Effect	**β(sd)**	**p**	**X^2(df)**	**f^2**
GROUP		**0.043**	8.15(3)	0.048
AN vs HC_L	0.29(0.15)	0.065	3.4(1)	0.038
AN-WR vs HC_L	0.34(0.15)	**0.029**	4.79(1)	0.076
HC_H vs HC_L	0.05(0.09)	0.598	0.28(1)	0.026
Stimulus x Direction x GROUP		0.284	10.88(9)	0.075

	Clinical Prior Hesitance (exploration)			
β(sd)	**p**	**X^2(df)**	**f^2**
	<0.001	19.63(3)	0.121
307.32(65.85)	**<0.001**	18.72(1)	0.216
313.04(71.74)	**<0.001**	16.63(1)	0.172
68.93(81.48)	0.399	0.71(1)	0.147
	0.162	13(9)	0.135

	Clinical Posterior Probability of Loss Belief (exploitation)			
Effect	**β(sd)**	**p**	**X^2(df)**	**f^2**
GROUP		**0.002**	14.96(3)	0.105
AN vs HC_L	0.39(0.16)	**0.019**	5.46(1)	0.071
AN-WR vs HC_L	0.53(0.18)	**0.004**	8.07(1)	0.127
HC_H vs HC_L	0.01(0.1)	0.958	0(1)	0.052
Stimulus x Direction x GROUP		0.527	8.07(9)	0.116

	Clinical Posterior Hesitance (exploitation)			
β(sd)	**p**	**X^2(df)**	**f^2**
	<0.001	18.98(3)	0.145
310.53(67.94)	**<0.001**	18.15(1)	0.242
254.11(75.08)	**0.001**	10.49(1)	0.023
49.37(81.65)	0.546	0.36(1)	0.017
	0.297	10.69(9)	0.149

Table 4. Results of risk vs loss aversion computational modelling analyses in non-clinical (upper half) and clinical (lower half) samples

	Exponential Weighting Model (Risk-Aversion & Loss-Aversion parameters ρ & λ)					
	Risk-Aversion (ρ)			Loss-Aversion (λ)		
Effect	**β(sd)**	**p**	**f^2**	**β(sd)**	**p**	**f^2**
EDE-Q Restraint	-0.02(0.01)	**<0.001**	0.067	0.01(0.03)	0.675	0.001
Stimulus x Direction x EDE-Q Restraint	0(0)	0.25	0.068	0.01(0.01)	0.689	0.001

	Risk-Aversion Increase Body vs Balloon			Loss-Aversion Increase Body vs Balloon		
Effect	**β(sd)**	**p**	**f^2**	**β(sd)**	**p**	**f^2**
Stimulus x EDE-Q Restraint	0(0)	0.545	0.069	0(0.01)	0.503	0.001

	Risk-Aversion Decrease Body vs Balloon			Loss-Aversion Decrease Body vs Balloon		
Effect	**β(sd)**	**p**	**f^2**	**β(sd)**	**p**	**f^2**
Stimulus x EDE-Q Restraint	0(0)	0.152	0.065	0 (0.01)	0.562	0.001

	Exponential Weighting Model (Risk-Aversion & Loss-Aversion parameters ρ & λ)					
	Risk-Aversion (ρ)			Loss-Aversion (λ)		
Effect	**β(sd)**	**p**	**f^2**	**β(sd)**	**p**	**f^2**
ALL GROUPS	-0.01(0.05)	0.16	0.067	0(0.17)	0.42	0.023
AN vs HC-L	-0.09(0.05)	0.073	0.125	0(0.17)	0.994	0.000
AN-WR vs HC-L	-0.11(0.05)	**0.041**	0.072	0.31(0.18)	0.09	0.048
HC-H vs HC-L	-0.05(0.03)	0.089	0.120	0.01(0.13)	0.919	0.000
Stimulus x Direction x GROUP	0.03(0.02)	**0.006**	0.068	0.04(0.06)	0.123	0.024

	Risk-Aversion Increase Body vs Balloon (AN vs HC-L)			Loss-Aversion Increase Body vs Balloon (AN vs HC-L)		
Effect	**β(sd)**	**p**	**f^2**	**β(sd)**	**p**	**f^2**
Stimulus x GROUP	0.01(0.01)	0.417	0.108	-0.01(0.04)	0.751	0.004

	Risk-Aversion Decrease Body vs Balloon (AN vs HC-L)			Loss-Aversion Decrease Body vs Balloon (AN vs HC-L)		
Effect	**β(sd)**	**p**	f^2	**β(sd)**	**p**	f^2
Stimulus x GROUP	-0.02(0.01)	0.064	0.128	-0.05(0.04)	0.208	0.000

	Risk-Aversion Increase Body vs Balloon (AN-WR vs HC-L)			Loss-Aversion Increase Body vs Balloon (AN-WR vs HC-L)		
Effect	**β(sd)**	**p**	f^2	**β(sd)**	**p**	f^2
Stimulus x GROUP	0.03(0.01)	**0.015**	0.065	0.07(0.04)	0.141	0.048

	Risk-Aversion Decrease Body vs Balloon (AN-WR vs HC-L)			Loss-Aversion Decrease Body vs Balloon (AN-WR vs HC-L)		
Effect	**β(sd)**	**p**	f^2	**β(sd)**	**p**	f^2
Stimulus x GROUP	-0.02(0.01)	**0.044**	0.085	-0.08(0.04)	0.043	0.051

	Risk-Aversion Increase Body vs Balloon (HC-H vs HC-L)			Loss-Aversion Increase Body vs Balloon (HC-H vs HC-L)		
Effect	**β(sd)**	**p**	f^2	**β(sd)**	**p**	f^2
Stimulus x GROUP	0.02(0.01)	**0.032**	0.122	0.04(0.04)	0.268	0.000

	Risk-Aversion Decrease Body vs Balloon (HC-H vs HC-L)			Loss-Aversion Decrease Body vs Balloon (HC-H vs HC-L)		
Effect	**β(sd)**	**p**	f^2	**β(sd)**	**p**	f^2
Stimulus x GROUP	-0.02(0.01)	**0.022**	0.126	-0.03(0.03)	0.373	0.002