Improving Rationality by Increasing Attention

Authors

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Abstract

Models of limited attention have the potential to become a new unifying paradigm that could replace the rational choice approach. In this paper, we test the limited attention hypothesis by enhancing attention using pharmacological substances. A total of 160 subjects participated in our randomized, placebo-controlled, and double-blind experimental study. We find that enhancing attention through boosting the noradrenergic system with reboxetine improves the quality of choice as captured by multiple different measures of rationality. Eye-tracking suggests that boosting noradrenaline promotes more rational choice by efficiently directing attention to more valuable options. Other attention-enhancing drugs (methylphenidate, which boosts the dopaminergic system, and nicotine, which boosts the cholinergic system) improve rationality to a lesser extent. Aside from testing the limited attention hypothesis directly, our results have implications for welfare economics, policy-design, and public health.

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1. Introduction

Rational choice requires that our actions are consistent with our beliefs and goals. The assumption of rationality has been a defining feature of economics for most of its history (Samuelson, 1937; Arrow, 1959; Afriat, 1973). Rational choice is of interest also to political science (Simon, 1995; Austen-Smith and Banks, 2005), philosophy (Sugden, 1991), psychology (Tversky and Kahneman, 1986; Schwartz, 2002; Samuels and Stich, 2004; Regenwetter et al., 2011; Sturm, 2012; Nitsch et al., 2022), biology (Stevens, 2008; Lea and Ryan, 2015; Lazzaro et al., 2016; Cohen et al., 2019; Katzen et al., 2023), neuroscience (Camille et al., 2011; Chung et al., 2017; Kurtz-David et al., 2019), and medicine (McFadden, 2010).

The economics literature has developed consistency criteria that indicate rationalizability of choices by utility maximization (Houthakker, 1950; Afriat, 1967; Varian 1982). Based on these criteria, substantial evidence shows that decision-makers violate rationality in the lab and in the real world (Harbaugh et al., 2001; Choi et al., 2007; Choi et al., 2014; Stanovich et al., 2016; Kim et al., 2018; Natenzon, 2019). Choices often exhibit stochasticity and preference reversals or cycles.

While there is not yet agreement on the source of these rationality violations, theories of limited attention have gained prominence among numerous competing hypotheses during the last decades (Sims, 2006; Masatlioglu et al., 2012; Matějka and McKay, 2015; Woodford, 2020; Maćkowiak et al., 2023). The logic is that paying attention to the choice options and processing information is costly, so that rationality violations are (possibly constrained optimal) mistakes in the decision-maker's attempt to maximize their true deterministic preferences. To date, the hypothesis of limited attention is a leading contender for a new unifying paradigm that could replace the rational choice approach (Gabaix, 2019).

Here, we test the limited attention hypothesis by boosting different components of attention using pharmacological substances in a randomized, placebo-controlled, double-blind, and incentive-compatible experimental design. Current evidence suggests that the neurotransmitters noradrenaline, dopamine, and acetylcholine increase selective, motivational, and cognitive aspects of attention respectively (Thiele and Bellgrove, 2018; Ranjbar-Slamloo and Fazlali, 2019; Lockhofen and Mulert, 2021). Our intervention therefore involves the selective noradrenaline reuptake inhibitor *reboxetine*, the dopamine reuptake inhibitor *methylphenidate* (better known as Ritalin®), and the acetylcholine receptor agonist *nicotine*, to enhance these different aspects of attention underpinned by the different neurotransmitter systems.

Our approach directly tests, and confirms, the limited attention hypothesis. We find that reboxetine (and to a lesser extent the other substances) systematically improves the quality of choice as captured by multiple measures of rationality. Moreover, it does so by increasing selective attention to undominated options as measured by eye gaze.

The causal intervention of our study is motivated by the fact that multiple theories can explain a given behavior by invoking distinct underlying mechanisms. For example, in addition to limited attention, theories that can explain rationality violations include, among others, failure to maintain goals (Laibson, 1997), context-dependent preferences (Natenzon, 2019; Tversky and Simonson, 1993), salience (Bordalo et al., 2013; Kőszegi and Szeidl, 2013), or limited working memory (Nitsch and Kalenscher, 2021; Guan, 2023). Understanding the specific mechanism that gives rise to a rationality violation is important because different mechanisms can lead to drastically different interpretations of the same behavior, with far-reaching implications for out-of-sample prediction of behavior (e.g. reactions to a novel economic policy), welfare economics, and even the concept of freedom of choice more broadly (Thaler and Sunstein, 2003; Bernheim, 2009; Manzini and Mariotti, 2014). For example, when the true value of the choice options changes with the context, possibly because the context provides information, then apparent behavioral inconsistencies are not mistakes that require correction but simply reflect context-dependent preferences. Alternatively, the same observed choices may reflect mistakes when the decision-maker cannot hold all choice options in working memory. Helping the decision-maker overcome these mistakes will, in turn, require very different interventions than if the mistakes are due to limited attention (Benkert and Netzer, 2018). Finally, understanding the cognitive and neural mechanisms underlying non-rationality is of its own interest to neuroscience and cognitive psychology, which increasingly recognize that attention matters for choice (Orquin et al., 2021; Rosner et al., 2022) while the biological and psychological implementation of attention remains underspecified.

A total of 160 healthy subjects participated in our study (40 in each of the treatment groups: reboxetine, methylphenidate, nicotine, placebo). Following Kim et al. (2018), participants were presented with 20 decision problems, in each of which they chose one out of 11 binary lotteries. These problems were presented as choice from linear budget lines, like in conventional consumer choice, with each axis representing one of two equiprobable states. To measure the degree of rationality of choices, we use conventional approaches proposed in the literature: the number of stochastic dominance violations, the number of revealed preference cycles, and the rationality indices of Varian (1990) and Afriat (1972). To explicitly account for the stochastic nature of choice, we also apply the recently proposed swaps index by Apesteguia and Ballester (2015). To establish a connection to a specific model of inattention, we use structural estimates of the information cost parameter in the rational inattention model of Matějka and McKay (2015).

We find that reboxetine significantly improves many measures of rationality compared to placebo. This result holds controlling for cortisol level, as reboxetine is known to not only enhance attention through

the noradrenergic channel but to also increase the stress hormone cortisol (see Hennig et al., 2000). The effects of the other drugs go in the same direction but are less often significant. Methylphenidate does not systematically enhance economic rationality despite its reputation as a smart drug, which supports and expands recent research (Bowman et al., 2023; Sambeth et al., 2025).

For a confirmatory analysis, we collected eye-tracking data as a direct measure of attention. Compared to placebo, we find that the reboxetine group fixates undominated lotteries faster, longer, and with larger pupil dilations, indicating a more efficient direction of attention towards more valuable options. Taken together, our findings highlight the role of noradrenaline in improving economic rationality through enhanced attention.

A possible confound of the analysis is that reboxetine may also affect risk attitudes, which in turn may have direct effects on rationality of choice behavior, for example because risk-neutral optimal options are particularly easy to identify in our task. We find, however, no evidence for an effect of reboxetine on risk preferences measured in multiple ways, including a separate task where participants report their willingness to pay for different lotteries.

The literature has associated differences in rationality with differences in age (Harbaugh et al., 2001; Choi et al., 2014; Chung et al., 2017; Brocas et al., 2019), education (Choi et al., 2014; Kim et al., 2018; Brocas et al., 2019), cognitive resources (Burghart et al., 2013; Castillo et al., 2017; Drichoutis and Nayga, 2020), biological properties (Lazzaro et al., 2016), and emotion states (Lee et al., 2009; Cettolin et al., 2020; Nitsch et al., 2021). Rationality matters because it has been associated with higher household income (Choi et al., 2014, Banks et al., 2019) and it serves as the cornerstone of normative welfare economics (Bernheim, 2009; Manzini and Mariotti, 2014). Despite its importance, we are aware of only one study (Kim et al., 2018) that causally enhanced rationality. That study found an educational program to reduce stochastic dominance violations in younger (9th grade) adolescents by 1.8 percentage points but to have no effect in older (10th grade) adolescents. It is noteworthy that, in our study, the group of adults receiving reboxetine demonstrated a reduction of approximately 1.5 percentage points in stochastic dominance violations compared to the placebo group in the raw data, and the effect increases to more than 3 percentage points when adding controls. Thus, economic rationality can be improved substantially even in young, highly educated, and healthy adults.

The paper is organized as follows. Section 2 describes the experiment in greater detail. Section 3 presents the results. Section 4 concludes. Additional material can be found in the Appendix.

2. Experimental Design

2.1 Recruitment Procedures

Our study was conducted in the Laboratory for Social and Neural Systems Research at the University Hospital Zurich, Switzerland, between December 2020 and December 2021. The study was approved by the Ethics Committee of the Canton of Zurich (BASEC-NR. 2020-00044) and the project was registered at ClinicalTrials.gov (NCT04384562).[1]

Based on previous research using the same substances (Vossel et al., 2008; Harrison et al. 2010; Evers et al., 2017), a sample size of 40 per drug treatment is required to obtain a statistical power of 80% for detecting significant differences between drug conditions at a significance level of 5%, accounting for an average drop-out rate of 20%. Therefore, a total of 160 healthy volunteers (80 women and 80 men) participated in our study.

Participants were recruited from the participant pool maintained by the University Registration Center for Study Participants. They came to the laboratory twice, once on the screening day and once on the experimental day. On the screening day, after having given written informed consent for the screening session, participants were carefully assessed and selected. The inclusion and exclusion criteria are described in Appendix A.1. All participants had normal or corrected-to-normal vision. For each participant, an electrocardiogram (ECG) and cardiovascular measures (blood pressure and heart rate) were acquired, and their ECG was analyzed by medical doctors. A urine test was taken to screen out participants who were positive for psychoactive substances (benzodiazepines, cocaine, 3,4-methylenedioxymeth-amphetamine, methamphetamine, morphine, methadone, and delta9-tetrahydrocannabinol) and the nicotine metabolite cotinine. Female participants were screened for pregnancy. All participants completed the Symptom Checklist-90-Revised (Derogatis and Unger, 2010) to screen for psychiatric symptoms in general, the trait part of the State-Trait Anxiety Inventory (Spielberger, 1983), the Adult ADHD Self-Report (Kessler et al., 2005), and the Symbol Digit Modalities Test (Smith, 1973) to screen for cognitive impairment. Moreover, we measured handedness with the Edinburgh Handedness Inventory (Oldfield, 1971), reward and punishment sensitivity with the Behavioral Inhibition/Activation System (Carver and

[1] The registration describes the drug treatments, inclusion and exclusion criteria for the participants, the number of subjects, and four decision-making tasks. The prespecified outcome measures are choice data, response time data, pupil dilation, and computational parameters. Specific examples like measures of choice sub-optimality, randomness, or estimated preferences are mentioned, but the registration does not prespecify a definite and exhaustive list. The gaze data discussed in Section 3.3 below were not prespecified. The present paper reports results for the two tasks with stable environments. Results for the other two tasks with dynamic environments can be found in Doren et al. (2023) and Doren et al. (2025), and additional results concerning imprecision and randomness of choice can be found in Chung et al. (2025).

White, 1994), and impulsivity with the Barratt Impulsiveness Scale (Patton et al., 1995). Participants were compensated with a flat fee for the screening day of CHF 45 (about USD 50 in 2021).

On the experimental day, participants arrived at the lab at 9:30am and completed the entire experimental session in approximately 5h. They fasted for 6h before they commenced the session, to ensure comparable drug absorption. Moreover, participants were not allowed to consume any substances containing caffeine or alcohol 12h prior to the experiment. They were recommended to have 8h of sleep on the night before the experiment. All participants signed the consent form for the experimental session, answered the safety questions, and underwent the urine tests to ensure continued eligibility. Participants were compensated with an average of CHF 240 (about USD 264 in 2021), which included earnings from one randomly selected decision problem.

2.2 Pharmaceutical Treatments

Participants were randomly and double-blindly assigned to either the placebo group or a psychoactive drug group, with 40 participants per group. To achieve this, all pills and gums (see below for details) were sequentially and randomly numbered by the pharmacy partner before the experiment. Participants were sequentially and randomly assigned ID numbers and received the corresponding pills and gums. They were informed about the substances used in the study, but not about the group they belonged to. Demographic characteristics and general traits of the drug groups were statistically indistinguishable from those of the placebo group (see Table 1).

	Sex			Age			BMI			Education		
	Male	Female	χ2 test (df=1)	Mean	SD	t test (df=78)	Mean	SD	t test (df=78)	Mean	SD	t-test (df=78)
Reboxetine (N=40)	16	24	2.45, p=0.12	23.68	3.78	0.47, p=0.64	22.08	2.20	-0.70, p=0.49	15.90	2.26	-1.01, p=0.32
Methylphenidate (N=40)	20	20	0.45, p=0.50	23.75	3.88	0.55, p=0.59	22.92	2.86	0.53, p=0.60	16.70	2.45	0.48, p=0.63
Nicotine (N=40)	21	19	0.20, p=0.65	23.82	3.08	0.70, p=0.49	22.82	2.50	0.61, p=0.54	16.76	2.56	0.33, p=0.74
Placebo (N=40)	23	17	NA	23.27	3.90	NA	22.63	2.66	NA	16.35	3.39	NA

Table 1. Demographic characteristics of participants in the four treatment groups. Statistical tests assess differences between each drug group and the placebo group. BMI denotes body mass index. Age and education are in years.

Based on previous findings (Valentine and Sofuoglu, 2018; Lockhofen and Mulert, 2021; Webber et al., 2021), we used the selective noradrenaline reuptake inhibitor reboxetine, the dopamine reuptake inhibitor methylphenidate, and the acetylcholine receptor agonist nicotine to enhance the different aspects of attention underpinned by the different neurotransmitter systems (see Appendix A.2 for a more detailed discussion of the neurophysiological foundations). Low doses of each drug were chosen to achieve

enhancements rather than decrements in functioning (Vossel et al., 2008; Gelbard-Sagiv et al., 2018; Roberts et al., 2020) and to minimize adverse side effects. A controlled breakfast was served before drug administration. Participants were then administered a tasteless, identical-looking pill containing either 20mg of methylphenidate, 4mg of reboxetine, or a placebo 90 minutes before the start of the decision-making tasks. One hour later, participants received a gum containing 2mg of nicotine or a placebo gum. One drop of hot sauce was placed on the gum to mask its taste. Participants were asked to chew the gum once every 3 seconds for 30 minutes, following recorded instructions (based on Vossel et al., 2008, and Meyhöfer et al., 2019). They were advised to place the gum in the cheek when the taste became too strong and to try not to swallow excessively, thus avoiding the rapid release of nicotine. Therefore, each participant received both a pill and a gum, with only one or neither (placebo group) being active.

Cardiovascular measures were assessed three times, once before drug administration and twice after. Saliva samples were collected for the analysis of cortisol and testosterone levels before drug administration, before the decision-making tasks, and after the decision-making tasks. This allows us to control for the cortisol-enhancing effects of reboxetine (Hennig et al., 2000) and the testosterone-enhancing effects of chewing gum itself (van Anders, 2010). Two blood samples were taken, one before and one after the decision-making tasks, to assess metabolization of the drugs. Subjective drug effects were assessed with Visual Analog Scales at five time points following drug administration, where participants rated general drug effects (“I feel the drug’s effect”), agitation (“I feel agitated”), fatigue (“I feel tired”), negative mood (“I feel bad”), and positive mood (“I feel good”). Additionally, working memory (Wechsler, 1997; Maier et al., 2020), perceived positive and negative affect (Watson et al., 1988) and numeracy score (Langa et al., 2005) were assessed before and after drug administration. Participants completed the Pittsburgh Sleep Quality Index (PSQI) for quantifying general sleep quality and the Sleep Questionnaire (SQ) for an additional check of data quality at the end of the experiment. A complete list of all used questionnaires can be found in Appendix A.3.

To ascertain that the drugs were metabolized, we measured plasma levels of the drugs and/or their metabolites (reboxetine in the reboxetine group, methylphenidate and ritalinic acid in the methyl-phenidate group, cotinine in the nicotine group) twice after drug administration. The first measurement was before participants performed the decision-making tasks and the second one after they completed them. As illustrated in Figure 1, drug-related plasma concentrations were elevated in all drug groups at both timepoints after drug administration. We also conducted Bayesian regression models (as throughout the paper) with a constant to compare the average concentrations to zero. Plasma levels were considered

significantly elevated if the 5% lower credible interval (CI) of the coefficient distribution was higher than zero. The concentrations of all substances were significantly elevated from zero at both timepoints.

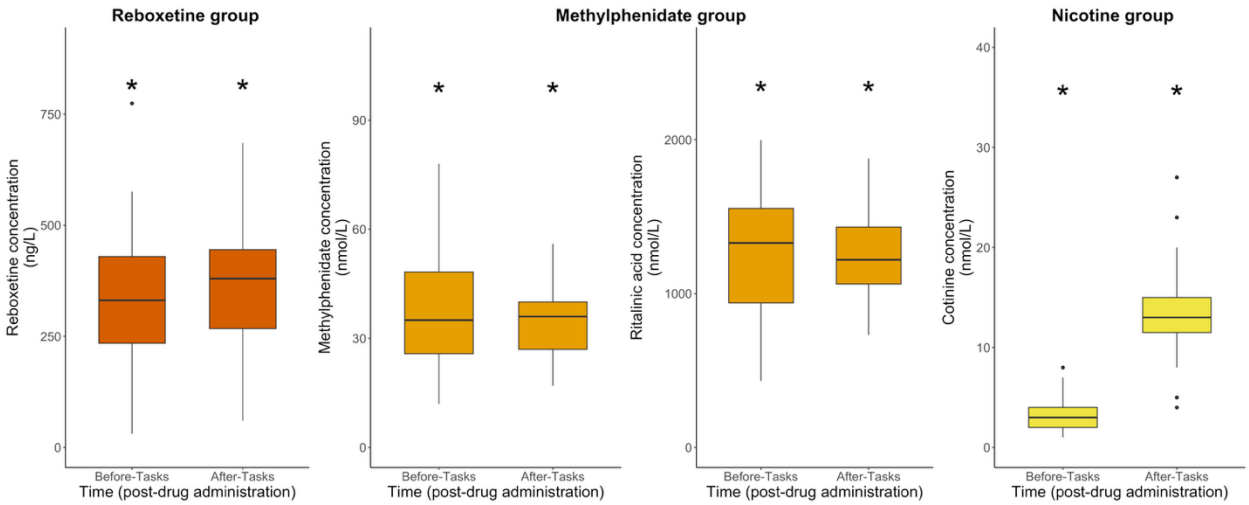

Figure 1. Drug and metabolite concentration in blood plasma. The center line inside the box corresponds to the median of the data, while the box limits denote the 25th and 75th percentiles. The upper (lower) whisker represents the maximum (minimum) value of the data falling within 1.5 times the interquartile range above the 75th (below the 25th) percentile. Any data points beyond the whiskers are considered outliers and represented as individual points. Asterisks indicate significant differences to zero. Note that ritalinic acid is the main metabolite of methylphenidate.

2.3 Decision-Making Tasks

Our main decision-making task was adopted from Kim et al. (2018). This task allows us to compute measures of rationality of choice. Participants were given 20 different decision problems in randomized order, in each of which they had to choose one out of 11 binary lotteries. These lotteries were presented on a budget line like in conventional consumer choice situations. They corresponded to different distributions of tokens between two accounts, referred to as the X account and the Y account (see Figure 2a). Both accounts had an equal chance of being realized. For instance, the lottery (27, 108) in Figure 2c indicates that the participant would receive 27 tokens if the X account was realized and 108 tokens if the Y account was realized, each of which would happen with a probability of 50%. Tokens were converted into CHF at a rate of 1:1. Participants had up to 15 seconds to decide, and responses were followed by a 1-second confirmation display. Decision problems were separated by a variable inter-trial interval, which had a mean duration of 3.5 seconds plus any remaining time from the preceding 15 seconds decision period (see Figure 2b). This variable interval allowed for pupil dilation to return to baseline between trials. The entire task lasted approximately 6.5 minutes.

We implemented an additional task that allows us to measure the participants' risk preferences more specifically. In this task, participants received an initial endowment of 150 experimental units and evaluated the lotteries by reporting minimum and maximum willingness to pay. Their response can be considered as implementing many binary choices between a given lottery and the safe option of keeping the endowment. For prices below the minimum willingness to pay, they chose the lottery, for prices above the maximum willingness to pay, they chose the safe option, and for prices between the minimum and the maximum, a probabilistic choice with linearly interpolated probabilities was implemented. In this task, one experimental unit was converted into 0.55 CHF to ensure comparable earnings across tasks.

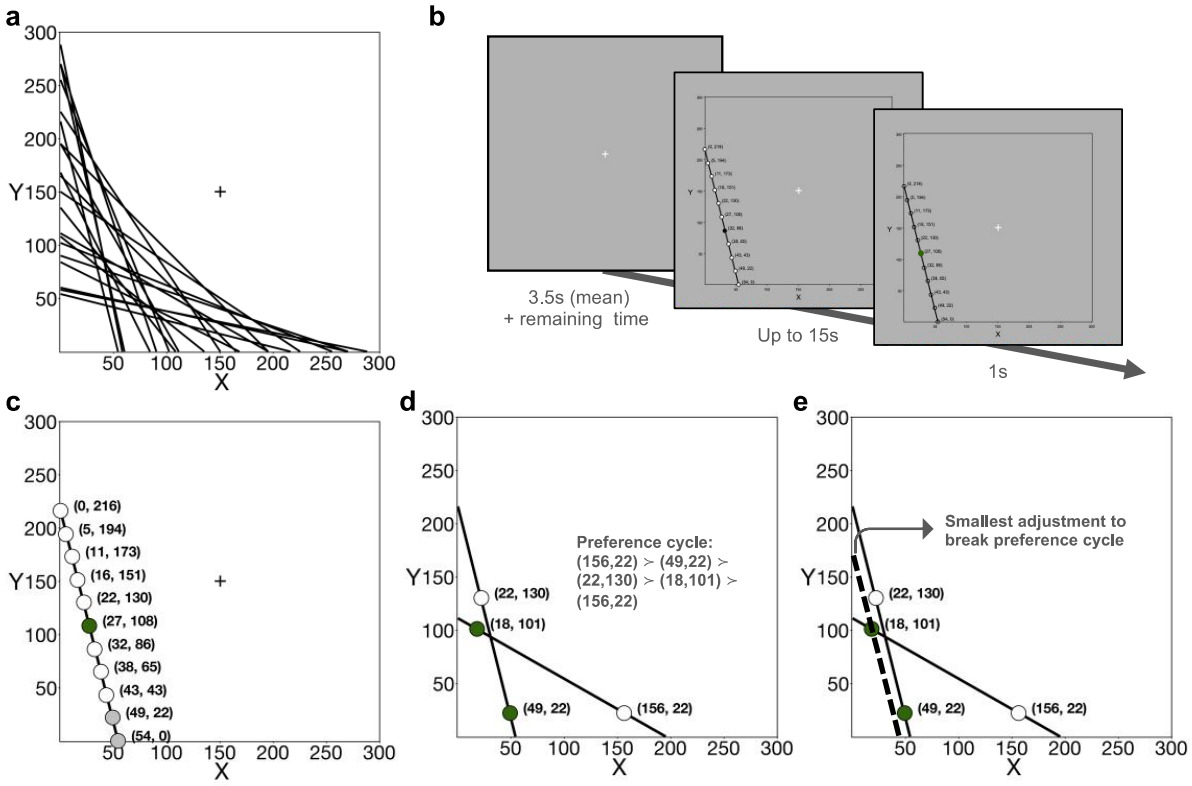

Figure 2. Main decision task used in the experiment. In each of the 20 different decision problems (a), participants had up to 15s (b) to choose between 11 binary lotteries presented on a budget line (c). The task allows to compute different measures of rationality, including preference cycles (d) and the Varian Index (e).

2.4 Measures of Rationality

We measure the degree of rationality of each participant in our main decision-making task. There are different ways in which behavior can be irrational, including the choice of dominated options, random choice, or choice that displays systematic cyclical patterns. We therefore use different measures of

rationality that have been suggested in the literature. For all these measures, a lower value will indicate greater rationality.

A first and straightforward measure is the *number of first-order stochastic dominance violations*. A lottery is first-order stochastically dominated (FOSD) if there exists another lottery that guarantees winning any given amount or more with a weakly higher probability. The two grey lotteries in Figure 2c, which are located on the shorter side of the 45-degree line, are examples of dominated lotteries. To see why, observe that a lottery like (49, 22) is dominated by a lottery like (27, 108), which pays 27 instead of 22 and 108 instead of 49, each with probability 50%. The stochastic dominance principle requires that a decision-maker should not choose dominated lotteries, regardless of risk preferences (see Choi et al., 2014; Kim et al., 2018). Our measure is therefore defined as the number of choices of a dominated lottery, divided by the number of decision problems in which dominated lotteries exist (no dominated lotteries exist if the budget line has a slope of -1).

Another measure of rationality relies on *revealed preference cycles*, a choice pattern which suggests that a decision-maker prefers option A over option B, option B over option C, and so on, and the last option in the chain over option A (Houthakker, 1950). Formally, in each decision problem $t = 1, \dots, T$, participants selected an option c^t (a bundle of two quantities) at prices p^t (one for each quantity, thus defining the slope of the budget line), forming a collection of choice data $D = ((p^1, c^1), \dots, (p^T, c^T))$. Then, for any $1 \leq i, k \leq T$, option c^i is revealed preferred to option c^k, written $c^i \; R \; c^k$, if $p^i \cdot c^i \geq \; p^i \cdot c^k$, because in that case c^i was chosen over other options on the budget line that weakly dominated c^k.[2] Analogously, c^i is strictly revealed preferred to c^k, written $c^i \; P \; c^k$, if $p^i \cdot c^i > \; p^i \cdot c^k$. A revealed preference cycle is a sequence of options $c^i, c^j, \dots, c^k$ such that $c^i \; R \; c^j \; R \dots R \; c^k$ and $c^k \; P \; c^i$. Figure 2d illustrates an example of a binary revealed preference cycle, where lottery (49, 22) was selected on the steeper budget line and lottery (18, 101) was selected on the flatter budget line, leading to a cyclical pattern where each of the two lotteries is revealed preferred over the other. The Generalized Axiom of Revealed Preference (GARP) requires the absence of revealed preference cycles and is known to be a necessary and sufficient condition for the rationalizability of choices by maximizing a utility function (Afriat, 1967). One measure of rationality is therefore the number of ordered pairs of chosen options (c^i, c^k) that are part of a revealed preference cycle. We implement the procedure proposed by Burghart at al. (2013, Online Appendix B) to calculate this number. We compute the measure on the mirrored data (i.e., the original data extended by adding all choice problems and choices mirrored along the 45-degree line) as described previously (Choi et al., 2014;

[2] Following the literature, we use this procedure to derive revealed preferences even though our setting is discrete and not all bundles on the budget line are feasible options.

Cettolin et al., 2020). Mirroring implies that violations of the stochastic dominance principle automatically turn into a revealed preference cycle, and GARP of the mirrored data is a necessary and sufficient condition for the rationalizability of choices by maximizing a symmetric utility function, which in our context with equiprobable states respects the stochastic dominance principle (Polisson et al., 2020).[3]

Simply counting the number of times preference cycles are present constitutes a relatively coarse measure of rationality that does not consider the severity of a rationality violation. To quantify the severity of irrationality in a rigorous yet non-parametric fashion, we calculate the *Varian index* (Varian, 1990). The index can be interpreted as the average fraction of income that the decision-maker could have saved without reducing their welfare. Formally, in each decision problem t, a budget line-specific adjustment factor $e^t \in [0,1]$ is used to shift the budget line towards the origin, and a revealed preference $c^i \; R \; c^k$ is removed when $(1 - e^t)p^i \cdot c^i < \; p^i \cdot c^k$. This is illustrated in Figure 2e. The Varian index is the smallest average (across problems) adjustment factor that is necessary to remove all revealed preference cycles and thus make the data rationalizable. A higher value indicates a greater severity of irrationality. We again calculate the index on the mirrored data and constrain the budget line-specific adjustment factors to be identical in a budget set and its mirror, to obtain a measure of distance between the data and rationalizability by a symmetric utility function (Halevy et al., 2018; Polisson et al., 2020). Calculating the Varian index is known to be computationally complex. We use a recently developed computational toolbox that makes the computation feasible (Mononen, 2023).

Another measure commonly used in the literature is the *Afriat index* (Afriat, 1972). The index is similar to the Varian index but restricts the adjustment factors e^t to be the same in all problems t, capturing the maximum (rather than average) fraction of income that the decision-maker could have saved without experiencing a welfare loss. We again calculate this measure on the mirrored data, using the toolbox of Mononen (2023).

We finally consider two less established parametric measures of rationality. First, the *swaps index* is a novel measure that was proposed by Apesteguia and Ballester (2015). Suppose a participant is confronted with a set $S = \{x, y, z\}$ of alternatives and chooses alternative $x \in S$. If this choice is to be explained with a preference that ranks the alternatives in the order $y \succ x \succ z$, then the choice would involve one mistake, in the sense that one needs to argue that the participant overlooked the one better

[3] Counting revealed preference cycles is complicated by the fact that not all options are exactly on the same budget line in the task of Kim et al. (2018) but are rounded to integers. We account for this issue by adding a weak revealed preference but removing a strict revealed preference whenever two chosen bundles are available in the same decision problem. We ignore the issue in the calculation of the other measures (Varian, Afriat, Swaps) as these are continuous in the data.

alternative y in the set. The swaps approach counts these mistakes, weighted by their empirical frequency of occurring, to calculate a "swaps distance" between observed choices and any candidate preference. The swaps index is the minimal swaps distance between the choices and any preference. The approach can be extended to cover choice under uncertainty from budget sets, where the number of better alternatives is replaced by their Lebesgue measure (Lu and Netzer, 2023). We derive this swaps index assuming a parametric expected utility function with constant relative risk aversion (Arrow, 1965). We use the resulting coefficient of risk aversion for additional analyses later (see Subsection 2.5).

Second, we structurally estimate a parametric *rational inattention model* (Matějka and McKay, 2015). In this model, a cost parameter scales the marginal cost of information acquisition. As the parameter rises, choices become less sensitive to the actual payoffs. We apply the model by equating the states about which participants learn with the budget sets from the 20 different decision problems (assuming a uniform prior) and the actions with the 11 options from left to right based on the reading direction. We employ a maximum likelihood approach to estimate the cost parameter (as in Dean and Neligh, 2023), again assuming expected utility with constant relative risk aversion. Due to the structure of the model and our data, fine distinctions between parameters are difficult to identify. We therefore use a binary classification for risk aversion (high/low) and information cost (high/low). We specify CRRA coefficients of 0 and 2, representing risk neutrality and an empirically reasonable level of risk aversion, respectively. Information costs can be 0 (fully rational agents) and 1 (equal weighting of information cost and payoff). The estimated coefficient of risk aversion will again be used later.

2.5 Measures of Risk Preferences

We measure the participants' risk preferences to assess drug effects, primarily because potential effects of the drugs on risk attitudes may constitute a confound for our analysis of rationality. For example, the risk-neutral optimal lottery (the one with highest expected value) in our main decision-making task is always the larger of the two corner options and hence is particularly easy to identify. If the drugs made a participant risk-neutral, then this participant may find it easy to select the optimal lottery and may almost mechanically behave in a more rational way. An analogous argument applies if the drugs increased risk aversion and made it optimal for a participant to always choose the safe option on the 45-degree line.

As a first measure of individual risk aversion, which is particularly simple and non-parametric, we calculate the *proportion of safe options* chosen across the 20 decision problems.

The parametric models described in the previous subsection, which we use for quantification of rationality, also generate estimates of risk attitudes. These models assume expected utility maximization

with constant relative risk aversion. We thus obtain one coefficient of relative risk aversion of the *swaps preference* and one of the *rational inattention preference*.

We additionally measure risk preferences in our dedicated willingness-to-pay task, where participants report minimal and maximal willingness to pay for different lotteries. We explain the mean of the minimal and the maximal value in a regression model, using several explanatory variables including the expected value and the variance of the respective lottery. The estimated *coefficient on the variance* for each treatment group serves as a coarse yet robust index of risk attitude in the respective group (Tobler and Weber, 2013).

Finally, in the willingness-to-pay task we apply a maximum likelihood approach to estimate a parametric model of expected utility maximization with constant relative risk aversion. As outlined earlier, reporting minimal and maximal willingness to pay can be interpreted as implementing many stochastic choices between the given lottery at different prices and the safe endowment. Our likelihood function calculates the likelihood of these many binary choices, integrating over the possible lottery prices and summing across the different lotteries. The estimated *coefficient of relative risk aversion* in the willingness-to-pay task is our final measure of risk preferences.

3. Results

3.1 Drug Effects on Rationality

Our main question of interest is whether improving attention by the pharmaceutical intervention affects the participants' degree of rationality. Table 2 summarizes the average values of our measures of rationality for all participants and separately by treatment group.

The average proportion of choices that violate FOSD is 0.025 in the full sample, and 118 out of the 160 participants never violate FOSD. In the reboxetine group, the average proportion is only 0.019 and thereby the smallest value among all groups, followed by 0.021 in the methylphenidate group and 0.028 in the nicotine group. The largest average value of 0.034 is observed in the placebo group. The number of participants who never violate FOSD is largest in the reboxetine group (34), followed by the methylphenidate group (30) and the nicotine group (29), and smallest in the placebo group (25). Hence, rationality as measured by obedience to the stochastic dominance principle is increased compared to placebo for all drugs, but most strongly so for reboxetine.

	All (N=160)		Reboxetine (N=40)		Methylphenidate (N=40)		Nicotine (N=40)		Placebo (N=40)	
Measures	Mean Scores	Rational N	Mean Scores	Rational N	Mean Scores	Rational N	Mean Scores	Rational N	Mean Scores	Rational N
FOSD Violations	0.025	118	0.019	34	0.021	30	0.028	29	0.034	25
GARP Violations	49.825	54	38.350	15	57.700	12	43.500	14	59.750	13
Varian Index	0.007	64	0.006	17	0.006	15	0.007	16	0.010	16
Afriat Index	0.075	64	0.053	17	0.074	15	0.062	16	0.110	16
SWAPS Index	317.897	6	419.473	1	283.846	3	343.483	2	224.784	0
Rational Inattention Cost	0.406	95	0.450	22	0.225	31	0.450	22	0.500	20

Table 2. Rationality of all participants and by treatment group. Mean Scores are the average group values of the measure of rationality, and Rational N is the number of participants without violations (for FOSD and GARP), with an index value of zero (for Varian, Afriat and Swaps), and zero attention cost (for rational inattention).

Similar patterns hold for the number of GARP violations and the indices of Varian and Afriat. In all these cases, the average value of the measure is smallest in the reboxetine group (indicating greatest rationality) and largest in the placebo group (indicating greatest irrationality), and the number of participants who achieve the best possible rationality score is maximal in the reboxetine group.[4] For the rational inattention model, the results are only slightly different. The placebo group is still the least rational among all groups (i.e., smallest number of participants with an estimated information cost of zero), but rationality is higher in the methylphenidate group than in the other two drug groups for this measure. The only measure for which we see a very different pattern is the swaps index, where the results are almost reversed.[5] These findings are again illustrated in Figure 3, which shows the cumulative distribution functions of the measures of rationality by treatment.

[4] For FOSD and GARP, these are participants who have no violations. For the three indices (Varian, Afriat and Swaps), these are participants who achieve a value of zero. For the rational inattention model, these are subjects with zero attention cost. Note that the existence of a GARP violation does not imply a strictly positive value of the Varian or Afriat index, whenever the violation disappears for infinitesimal adjustments of the budget lines. It is therefore not a contradiction to observe more participants with zero index value than without GARP cycles. Note further that a swaps index of zero is achieved only when the choices are rationalizable by an expected utility function with constant relative risk aversion, which reduces the number of participants with zero score. By contrast, since our rational inattention model generates only a binary classification, the number of participants classified as rational is increased.

[5] While the reason for this opposite effect remains unknown, we speculate that it arises because the swaps index is particularly well-suited for data generated by conventional random utility models but not by models of rational inattention (Lu and Netzer, 2023). We also remark that the effect is not significant, as shown below.

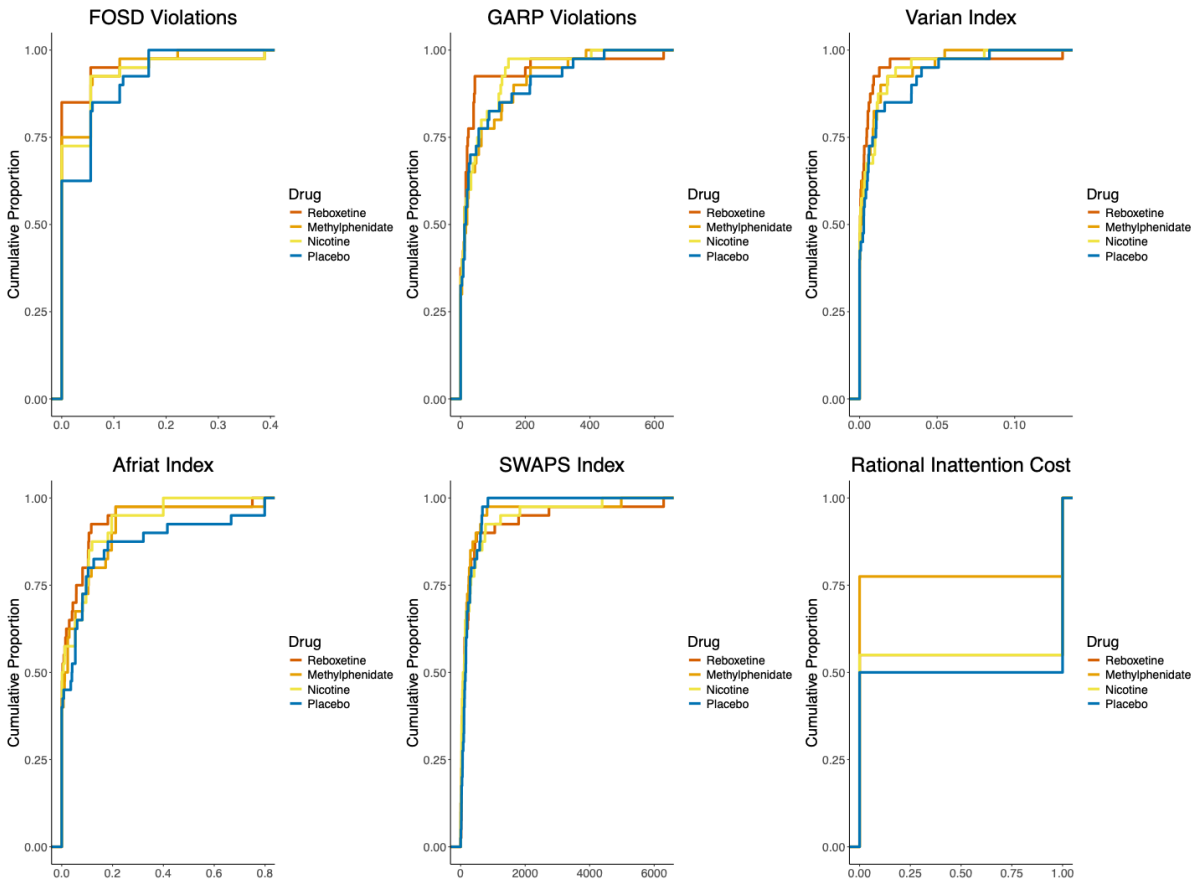

Figure 3. Distribution of rationality by treatment group. The subfigures refer to the different measures of rationality. The lines are cumulative distribution functions of the respective measure in the respective treatment group.

For a more rigorous statistical analysis of the effect of the drugs, we run Bayesian regressions.[6] These regressions explain the respective measure of rationality by drug dummy variables, age, sex, and cortisol level (averaged across the two time points after drug administration). We control for cortisol because reboxetine is known to not only enhance attention through the noradrenergic channel but to also increase the stress hormone cortisol (Hennig et al., 2000; also confirmed in our data), which can have opposing effects on rationality (see Appendix A.4 for a more detailed discussion; note that we did not find group differences in testosterone). Cortisol would be a bad control if our interest was in the overall causal effect

[6] The Bayesian regression models were implemented in R (version 4.5.1; R Core Team, 2025) through the brms package (version 2.22.0; Bürkner, 2017). We used non-informative uniform priors for regression coefficients and a half Student-t prior with 3 degrees of freedom for the Gaussian parameter sigma. Each model was fitted with 4 chains of 3000 iterations each (1500 burn-in iterations), and we assessed convergence using R-hat and effective sample size (ESS). We followed the recommendation (Vehtari et al., 2021) that ESS values should be greater than 400 and R-hat should be not larger than 1.01 for reliable estimation. For the binary rational inattention cost parameter, we used Bayesian logistic (Bernoulli) regression.

of the drugs on rationality. Our interest, however, is in the effect through the attention-related neurotransmitter channels. We thus deliberately control for cortisol to shut off the stress hormone-related channel.

The results are given in Table 3, which reports the difference β_{drug} - $\beta_{placebo}$ between the estimated coefficient of each drug dummy and the placebo dummy. The credible intervals (CIs) aid interpretation. Given our directional hypothesis that the drugs reduce rationality violations, the mitigation effect (β_{drug} - $\beta_{placebo} < 0$) is considered statistically significant if the 95% upper CI is smaller than zero.

	FOSD Violations			GARP Violations			Varian Index			Afriat Index			SWAPS Index			Rational Inattention Cost		
	Estimates	5% CI	95% CI	Estimates	5% CI	95% CI	Estimates	5% CI	95% CI	Estimates	5% CI	95% CI	Estimates	5% CI	95% CI	Estimates	5% CI	95% CI
RBX-PBO	-0.033	-0.057	-0.009	-58.077	-95.388	-21.067	-0.009	-0.016	-0.002	-0.095	-0.155	-0.032	73.729	-254.468	398.232	-0.825	-1.816	0.126
MPH-PBO	-0.020	-0.041	0.002	-15.287	-49.080	18.655	-0.005	-0.011	0.001	-0.051	-0.105	0.003	26.197	-261.226	316.213	-1.548	-2.476	-0.682
NIC-PBO	-0.008	-0.029	0.013	-20.190	-53.743	13.590	-0.003	-0.009	0.003	-0.055	-0.109	-0.002	124.105	-160.594	406.787	-0.278	-1.041	0.488
Age	0.002	0.000	0.004	2.814	-0.457	6.161	0.000	0.000	0.001	0.005	-0.001	0.010	2.981	-25.112	31.436	0.015	-0.059	0.089
Sex	-0.020	-0.035	-0.006	-36.781	-60.571	-12.929	-0.005	-0.010	-0.001	-0.057	-0.095	-0.019	86.616	-105.053	283.435	-0.428	-0.996	0.133
Cortisol Level	0.004	0.001	0.006	7.226	2.849	11.696	0.001	0.000	0.002	0.006	-0.001	0.013	33.493	-4.353	70.714	0.122	0.004	0.249

Table 3. Coefficients in Bayesian regression models. Estimated coefficients and credible intervals (CIs) from regressions of the respective rationality measure on drug dummy, age, sex, and cortisol level.

As can be seen in the table, the reboxetine group shows significantly fewer dominated choices than the placebo group ($\beta_{reboxetine}$ - $\beta_{placebo}$ = -0.033, 95% CI = -0.009), whereas no significant differences with the placebo group are observed for both the methylphenidate group ($\beta_{methylphenidate}$ - $\beta_{placebo}$ = -0.020, 95% CI = 0.002) and the nicotine group ($\beta_{nicotine}$ - $\beta_{placebo}$ = -0.008, 95% CI = 0.013). The results for the number of GARP violations are analogous, with a significant effect of reboxetine compared to placebo on revealed preference cycles ($\beta_{reboxetine}$ - $\beta_{placebo}$ = -58.077, 95% CI = -21.067) and no significant effect for the methylphenidate and nicotine groups. Furthermore, reboxetine significantly decreases the value of the Varian index ($\beta_{reboxetine}$ - $\beta_{placebo}$ = -0.009, 95% CI = -0.002) and the Afriat index ($\beta_{reboxetine}$ - $\beta_{placebo}$ = -0.095, 95% CI = -0.032). The other drugs do not have a significant effect on these indices, apart from nicotine for the Afriat index ($\beta_{nicotine}$ - $\beta_{placebo}$ = -0.055, 95% CI = -0.002). When measured by the rational inattention cost parameter, all drugs increase rationality relative to placebo, but only the effect of methylphenidate is significant ($\beta_{methylphenidate}$ - $\beta_{placebo}$ = -1.548, 95% CI = -0.682). The seemingly contradictory effects for the swaps index are never statistically significant, for any of the drugs.

We can also compute Bayes Factors (BFs) for each directional drug effect (β_{drug} - $\beta_{placebo} < 0$ against the alternative hypothesis). Using the interpretative framework proposed by Lee and Wagenmakers (2013), a BF between 1 and 3 indicates anecdotal evidence, between 3 and 10 indicates moderate evidence, between 10 and 30 indicates strong evidence, between 30 and 100 indicates very strong evidence, and greater than 100 indicates extreme evidence. According to this approach, the evidence that reboxetine

reduces dominance violations is very strong (BF = 89.91), reduces GARP violations is extreme (BF = 239), decreases the Varian index is very strong (BF = 72.17), reduces the Afriat index is extreme (BF = 165.67), and reduces the rational inattention cost is strong (BF = 12.33).

As we show in Appendix A.5, our results are robust to including additional explanatory variables for working memory, numeracy skill, education, body mass index, fatigue, sleep quality, and various measures of psychological traits and symptoms elicited in our questionnaires.

In summary, we find that reboxetine has systematic and significant effects on rationality, highlighting the role of noradrenaline for rational choice, in line with an attention-based account of rationality. The other drugs have typically smaller and less significant effects, providing no strong evidence for dopamine and acetylcholine to play a relevant role for rationality.

3.2 Drug Effects on Risk Preferences

We next examine whether the pharmacological intervention affected participants' risk preferences. Our main motivation for asking this question is that effects on risk preferences may constitute a confound for our analysis of rationality, as some preferences may be easier to implement than others in our decision-making tasks.

Table 4 summarizes the main findings. It again reports the difference β_{drug} - $\beta_{placebo}$ between the estimated coefficient of each drug dummy and the placebo dummy. Due to the mixed nature of drug effects on risk taking (Yang et al., 2016; Petzold et al., 2019; Waheed, 2023), we have no strong ex-ante hypothesis about the direction of potential effects. We therefore report 2.5% lower and 97.5% upper CIs and consider an effect significant if these intervals do not contain zero. As before, the regressions control for age, sex, and cortisol level.

	Safe option chosen			SWAPS CRRA risk			RI CRRA risk			ML CRRA risk (WTP)			Variance risk (WTP)		
	Estimates	2.5% CI	97.5% CI	Estimates	2.5% CI	97.5% CI	Estimates	2.5% CI	97.5% CI	Estimates	2.5% CI	97.5% CI	Estimates	2.5% CI	97.5% CI
RBX-PBO	0.04779	-0.04733	0.14658	0.16565	-0.42220	0.76314	-0.54211	-1.81369	0.68596	-0.00443	-0.16508	0.16132	-0.00030	-0.00079	0.00018
MPH-PBO	0.01919	-0.07198	0.10691	0.28188	-0.23849	0.80493	-0.63581	-1.70618	0.38333	-0.12979	-0.27097	0.01664	-0.00051	-0.00099	-0.00003
NIC-PBO	0.05532	-0.02870	0.14135	0.24871	-0.25340	0.75959	-0.01066	-0.99734	0.96094	-0.11047	-0.25407	0.03378	-0.00033	-0.00080	0.00015
Age	0.00357	-0.00489	0.01238	0.01308	-0.03525	0.06340	0.06659	-0.03089	0.17237	-0.00664	-0.02103	0.00737	0.24567	0.05183	0.44764
Sex	-0.01497	-0.07897	0.04724	-0.48055	-0.83509	-0.12175	-1.17196	-1.89142	-0.48729	-0.19590	-0.29643	-0.09323	-2.78597	-7.16225	1.43493
Cortisol Level	0.00340	-0.00813	0.01467	-0.01607	-0.08434	0.05290	0.21394	0.03990	0.41022	-0.01622	-0.03516	0.00283	-0.24651	-0.98170	0.45575

Table 4. Coefficients in Bayesian regression models. Estimated coefficients and credible intervals (CIs) from regressions of the respective preference measure on drug dummy, age, sex, and cortisol level.

There are no systematic effects of the drugs on risk aversion. The first column of Table 4 reveals that all drug groups chose safe options more often than the placebo group, indicating greater risk aversion, but the effect is never significant (e.g. $\beta_{reboxetine}$ - $\beta_{placebo}$ = 0.048, 95% CI interval = [-0.047, 0.147]). The second

column shows that swaps preferences in the drug groups have a higher coefficient of relative risk aversion than in the placebo group, again indicating greater risk aversion, but the effects are also not significant (e.g. $\beta_{reboxetine} - \beta_{placebo} = 0.166$, 95% CI interval = [-0.422, 0.763]).[7] If we measure risk preferences within the rational inattention framework, the sign of the effects is reversed, with the drug groups showing a greater tendency to be classified as risk neutral than the placebo group, but none of the effects is significant (e.g. $\beta_{reboxetine} - \beta_{placebo} = -0.542$, 95% CI interval = [-1.814, 0.686]).

The remaining columns in Table 4 report results when we use individual measures of risk preferences obtained from the separate willingness-to-pay task (see Appendix A.6 for a comparison of the risk attitudes estimated in the two tasks). As can be seen in column four, the maximum likelihood coefficients of relative risk aversion are smaller in all drug groups compared to the placebo group, but not significantly so. According to column five, an increased variance of the lottery (holding the expected value fixed) reduces the willingness to pay for the lottery more strongly in the placebo group than in any of the drug groups.[8] The effect is significant only in the methylphenidate group, indicating, if anything, a tendency for reduced risk aversion under methylphenidate (compatible with the findings of Mandali et al., 2021).

In summary, we find little to no evidence that the drugs systematically altered risk preferences. In particular, the increased rationality of the reboxetine group described earlier is not a simple consequence of increased or reduced risk aversion.

3.3 Eye-Tracking and Response Times

In addition to choice data, we collected oculomotor data using an eye-tracker. These data allow us to examine how participants look at the options and how this is affected by the drug treatments, which makes it possible to directly study the allocation of limited attention during the decision-making process.

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[7] In this regression, we exclude 4 participants for whom the swaps coefficient of risk aversion is infinite, which leaves us with 156 participants.

[8] The regression equation underlying these results is

$$\begin{aligned} WTP = {} & +\beta_1 * EV * PBO + \beta_2 * EV * RBX + \beta_3 * EV * MPH + \beta_4 * EV * NIC \\ & - \beta_5 * VA * PBO - \beta_6 * VA * RBX - \beta_7 * VA * MPH - \beta_8 * VA * NIC \\ & + \beta_9 * Age + \beta_{10} * Sex + \beta_{11} * Cortisol \\ & + u_{(participant)} + u_{(participantEV)} * EV + u_{(participantVA)} * VA + \epsilon, \end{aligned}$$

where WTP is the mean of the lowest and highest reported willingness to pay, EV and VA denote expected value and variance of the lottery, and PBO, RBX, MPH, and NIC are dummies for the drug treatments. Random effects were modelled as random intercepts for each participant and random slopes for EV and VA. We pooled all choices from all participants (21867 observations in total). The coefficient referred to as RBX-PBO in Table 4 corresponds to $\beta_6 - \beta_5$, and analogously for the other drugs. Note that the reported coefficients for Age, Sex, and Cortisol Level are not interacted with variance.

During the experiment, participants were seated in front of a computer screen with a resolution of 1920x1080 pixels. They rested their chin on a support with a viewing distance of approximately 60 cm. We recorded oculomotor data in our main decision-making task using an EyeLink 1000 Plus eye-tracker (SR Research, Ottawa, Canada).[9] We measure visual fixations as an indicator of overt attention (Russo and Rosen, 1975; Mahanama et al., 2022; Wedel et al., 2023). To detect fixations, we use a velocity-based algorithm based on an adaptive method (Nyström and Holmqvist, 2010). Furthermore, we measure pupil dilation as another indicator of overt attention (Hoeks and Levelt, 1993; Joshi and Gold, 2020; Mahanama et al., 2022). Pupil data were preprocessed with a custom in-house script (Maier and Grueschow, 2021). Blinks were defined as periods of signal loss lasting longer than 80ms and shorter than 2000ms. The identified blinks vector was convolved with a Gaussian window of 40, transformed back into a box-car function, and linearly interpolated. A low-pass filter with a cutoff frequency of 4Hz, implemented using the EEGLAB toolbox (Delorme and Makeig, 2004), was applied to the pupil data. Finally, the pupil data was z-scored within each participant, and pupil dilation was calculated as the deviation from the 1000ms baseline before the onset of the decision problem.

Figure 4 shows the difference between the reboxetine group and the placebo group in terms of where the participants look at. In the figure, red indicates that the reboxetine group spends more time on average fixating the respective region, and blue indicates the reverse. It is immediately visible that the reboxetine group looks systematically longer at undominated options relative to the placebo group, indicating a more efficient allocation of attention to valuable options.

[9] The quality of the eye-tracking data was ensured with a spatial 9-point calibration and validation process. Four participants (one from the methylphenidate group and three from the nicotine group) were excluded from the oculomotor data analysis due to loss of eye-tracking data in more than half of the trials. Due to technical issues, the sampling rates during data collection varied between 500, 1000, and 2000 Hz. However, in each group, the majority of participants were recorded at 1000Hz. We therefore downsample or upsample all data to 1000Hz. When additionally controlling for eye-tracker frequency as a random effect in our regression models, our main results concerning the drug effects remain qualitatively unchanged.

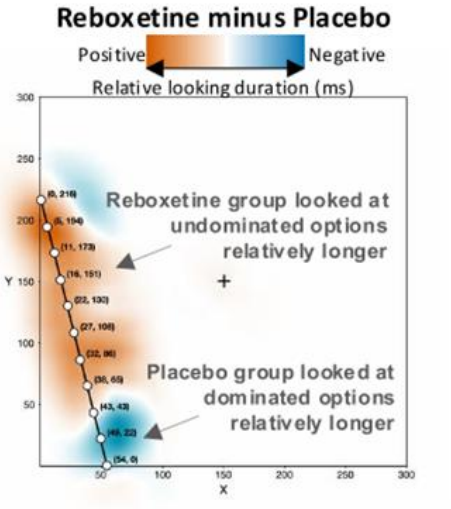

Figure 4. Difference in cumulative fixation duration between the reboxetine group and the placebo group. Group-specific heatmaps were produced first and the difference heatmap then calculated for each pixel. The heatmap was smoothed using a Gaussian kernel with a sigma value of 20 and a size of 80 for the purpose of data visualization.

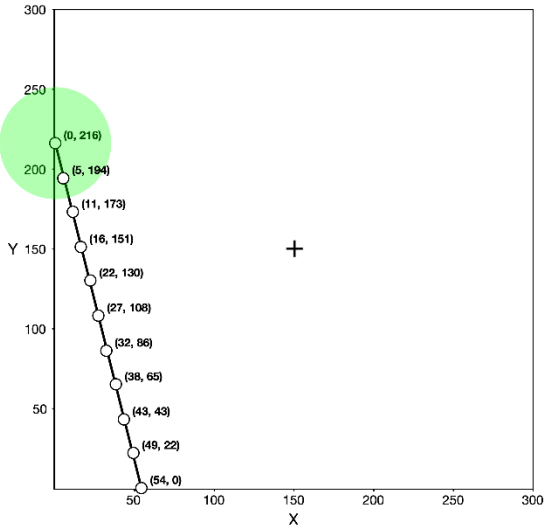

Figure 5. Area of interest (AOI) for fixation analysis. For each option, an area of interest is defined as a circle with a radius five times larger than the option dot, covering both option dot and number display.

For our statistical analysis, we determine fixation of an option by defining a circular area of interest (AOI) for each option, covering both the option dot and the number display (see Figure 5). The AOI radius is five times larger than the option dot. While the AOI partially covers other options, each fixation is

assigned solely to one option. To determine the option to which a fixation belongs, we follow the conventional reading direction, moving from left up to right down.[10]

Our first measure is the proportion of time participants spend looking at the undominated options during each trial. As shown in Table 5, the reboxetine group fixates the undominated options longer compared to the placebo group and the difference is statistically significant ($\beta_{reboxetine} - \beta_{placebo} = 0.048$, 5% CI = 0.012). No significant differences with the placebo group are observed for both the methylphenidate and the nicotine group.

	Proportion of fixation time on undominated options			**Fixation delay to undominated options**			**Pupil dilation of undominated optioins**		
	Estimates	5%	95%	Estimates	5%	95%	Estimates	5%	95%
RBX-PBO	0.048	0.012	0.085	-107.562	-174.498	-40.680	0.217	0.014	0.420
MPH-PBO	0.010	-0.022	0.043	-38.718	-100.810	23.678	0.254	0.067	0.445
NIC-PBO	-0.006	-0.039	0.028	-18.337	-80.382	44.251	0.139	-0.041	0.321
Age	0.002	-0.001	0.005	-0.518	-6.296	5.213	-0.014	-0.031	0.003
Sex	-0.039	-0.062	-0.016	38.800	-3.587	81.065	-0.138	-0.265	-0.010
Cortisol Level	-0.003	-0.007	0.002	10.382	2.402	18.441	-0.005	-0.029	0.020

Table 5. Coefficients in Bayesian regression models. Estimated coefficients and credible intervals (CIs) from regressions of the respective eye-tracking measure on drug dummy, age, sex, and cortisol level.

As an alternative measure, we calculate the delay until participants first fixate an undominated option, with enhanced attention being reflected in shorter delays. We exclude any delays shorter than 70ms from our analysis, because attentional effects are not detectable for such short delays (Nakayama and Mackeben, 1989; Li et al., 2017). As Table 5 shows, participants in the reboxetine group fixate undominated options more quickly than the placebo group ($\beta_{reboxetine} - \beta_{placebo} = -107.562$, 95% CI = -40.680). Again, no significant differences are observed for the methylphenidate and the nicotine groups.

Next, we compute the average pupil dilation while participants look at undominated options, with enhanced attention being reflected in larger pupil size. Compared to placebo, undominated options elicited stronger pupil dilations in the reboxetine group ($\beta_{reboxetine} - \beta_{placebo} = 0.217$, 5% CI = 0.014). A similar and also significant effect is observed for the methylphenidate group ($\beta_{methylphenidate} - \beta_{placebo} = 0.254$, 5% CI = 0.067) but not for the nicotine group.

These results indicate that reboxetine improved multiple measures of selective attention to undominated options. From an alternative perspective, one may argue that distributing attention across all available options is more rational in a general sense, because it ensures that nothing is overlooked. To

[10]Our main results concerning the reboxetine effects are robust to using an AOI radius that is four or six times larger than the option dot and to determining fixations in the reverse reading direction from right down to left up.

examine this hypothesis, we performed several control analyses (Table 6). In comparison to the placebo group, none of the drug groups exhibits a significant change in the overall number of options they examine or in the total time they spend looking at options. We also find no significant drug effects on the delay to fixate the eventually chosen option or on the total time fixating the eventually chosen option.

	Number of options viewed			Fixation duration on options			Fixation delay to chosen option			Fixation duration to chosen options		
	Estimates	5% CI	95% CI	Estimates	5% CI	95% CI	Estimates	5% CI	95% CI	Estimates	5% CI	95% CI
RBX-PBO	0.177	-0.377	0.731	490.151	-151.270	1128.554	-83.041	-350.669	177.004	144.844	-25.966	315.222
MPH-PBO	-0.008	-0.512	0.500	173.871	-392.537	743.712	64.977	-172.522	300.999	26.160	-129.675	188.432
NIC-PBO	-0.228	-0.727	0.278	-86.663	-660.298	496.957	-24.594	-299.898	250.157	-46.723	-199.086	105.724
Age	-0.049	-0.099	-0.001	-16.037	-70.590	37.656	-17.121	-40.043	5.927	-0.840	-14.902	13.487
Sex	-0.487	-0.852	-0.130	-459.612	-860.662	-53.684	-197.203	-367.817	-31.863	57.781	-49.785	165.401
Cortisol level	-0.007	-0.072	0.056	-42.152	-115.960	33.898	9.736	-21.981	40.824	-15.780	-35.665	4.366

Table 6. Coefficients in Bayesian regression models. Estimated coefficients and credible intervals (CIs) from regressions of the respective eye-tracking measure on drug dummy, age, sex, and cortisol level.

Finally, one may conjecture that reboxetine improved deliberation in general. More careful decisions may take longer (Ratcliff and Rouder, 1998). Additional time spent examining the options often provides more accurate estimates of their value and allows for better decisions, typically referred to as the "speed-accuracy trade-off" (Hick, 1952; Bogacz et al., 201). Under this assumption, one could expect the reboxetine group to show longer response times (participants had up to 15s to decide but under placebo used only 5.6s on average). Contrary to this prediction, neither reboxetine nor any of the other drugs did significantly affect response times (Table 7).

	Response time		
	Estimates	5% CI	95% CI
RBX-PBO	0.13	-0.67	0.93
MPH-PBO	-0.05	-0.77	0.65
NIC-PBO	-0.35	-1.05	0.38
Age	-0.03	-0.10	0.04
Sex	-0.25	-0.77	0.26
Cortisol Level	-0.04	-0.14	0.06

Table 7. Coefficients in Bayesian regression models. Estimated coefficients and credible intervals (CIs) from regressions of response time on drug dummy, age, sex, and cortisol level.

Taken together, the information processing in the reboxetine group is more rational in that individuals process the undominated options more quickly, for a longer duration, and more attentively. Enhancing noradrenaline appears to improve economic rationality by effectively directing selective attention towards

more valuable options and away from less valuable ones, rather than by increasing attention to all available options non-discriminately or improving deliberation in general.

4. Conclusion

Rational choice requires that we consistently choose the option with the highest subjective value. However, choice behavior is often random and displays revealed preference cycles or systematic reversals. Dominant theoretical accounts try to explain such rationality violations with limits in attention. We test the theory of limited attention by enhancing attention with pharmacological interventions. We find that the noradrenergic intervention (using reboxetine) increases rationality. Our eye-tracking analysis suggests that the effect arises through a more efficient allocation of selective attention to better options. The dopaminergic and cholinergic interventions (using methylphenidate and nicotine) had qualitatively similar but smaller and often not significant effects on rationality. Taken together, our findings not only test and support the limited attention theory of irrationality but also establish for the first time a pharmacologically specific and causal neurobiological foundation of human choice rationality.

In addition to testing the general idea of limited attention, our study has important implications for normative welfare economics. First, the fact that rationality is far from perfect and can be improved even in young and healthy adults demonstrates the limits of conventional revealed preference analysis. Preferences elicited from choices can be severely misguided when incomplete attention is not taken into account. Second, our results underscore the importance of developing novel tools for improving the quality of choices. It is important to consider what makes it easy for decision-makers and consumers to allocate their attention efficiently, and how one can help them direct attention to where it matters most. Such tools have the potential to improve welfare substantially, demonstrated by the reduction of the Afriat and Varian indices in our study, which have interpretations as income wasted and welfare lost due to suboptimal choices.

The impact of psychoactive drugs on rationality is potentially also important for medicine and public health because psychiatric disorders like depression and attention disorders manifest themselves in reduced decision quality, a symptom which should be considered in treatment. Improving optimal allocation of limited attention aligns with behavior therapy's goal to teach cognitive strategies, advocating for resource rationality as a guiding principle in the development of more effective therapies.

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A. Appendix

A.1 Inclusion and Exclusion Criteria

To be eligible, participants had to fulfil the following criteria:

1. Physically and psychiatrically healthy
2. Aged 18-35 years
3. Able and willing to participate in the study
4. Willing not to eat or drink any food/beverage containing caffeine or alcohol 12 hours prior to the administration of study medication
5. Willing not to eat or drink grapefruit or grapefruit related citrus fruits (e.g., Seville oranges, pomelos) from 7 days prior to the administration of study medication
6. Have good command of English language (be able to understand the task instructions and in the unlikely case of adverse effects inform the examiner)
7. Provide signed informed consent

Individuals who met one of the following criteria were ineligible:

1. Serious past brain disease or injury
2. Frequent headaches (of any sort, > 1/week) or migraine (irrespective of frequency)
3. History of epileptic seizures
4. Any neurological disorder
5. Known cardiac or cardiovascular disease or anomaly
6. Family history of sudden death due to cardiac arrhythmia
7. High or low blood pressure, history of heart attack, infrequent heartbeat
8. Respiratory problems (including difficulty with breathing through the nose)
9. Glaucoma (present or past)
10. Insufficiency of kidney or liver, acute liver disease
11. Any psychiatric disorder (especially depression, mania, schizophrenia, addiction panic and suicidality)
12. Severe vocal or motor tics (methylphenidate, data quality)
13. Severe psychosomatic disorder (somatic complaints without clear medical cause, has a mental component)
14. Pregnancy, nursing, or currently planned pregnancy
15. Allergy to drugs, particularly methylphenidate, reboxetine or nicotine

16. Severe intolerance to lactose including strong diarrhoea after only a few mg (weak lactose intolerance was no exclusion criterion as medication only contained a very small dose (around 4 mg) of lactose)
17. Oversensitivity to hot pepper sauce (e.g., tabasco)
18. Currently taking any medication or recently participated in other clinical trials that might interfere with Methylphenidate and Reboxetine, especially MAO-Inhibitors (e.g., Aurorix (Moclobemid) and Azilect (Rasagilin), antipsychotics, antibiotics, and medication for heart diseases
19. Currently taking any further medication (besides birth control) or natural products (infrequent intake of natural products and/or food supplements had to be mentioned to the examiner)
20. Drug abuse (exclude people with a positive test)
21. Serious acute or chronic disease that could interfere with participation in the experiments
22. Inability to understand the instructions
23. Participants with BMI < 18
24. Clinically relevant score in STAI T (anxiety), measured in screening session
25. ECG demonstrating QTcF >450 msec or a QRS interval >120 msec at screening. If QTcF exceeded 450 msec, or QRS exceeded 120 msec, the ECG was repeated two more times and the average of the three QTcF/QRS values used to determine participant eligibility, measured in screening session
26. Participants who ate or drank grapefruit or grapefruit related citrus fruits (e.g., Seville oranges, pomelos) or drinks from 7 days prior to the administration of study medication
27. Participants who ate or drank any food/beverage containing caffeine or alcohol 12 hours before the study
28. Had little sleep last night
29. Current smokers/tobacco consumers (i.e., people whose cotinine level is higher than 50ng/ml in urine test)
30. Phenylketonuria
31. Dental or jaw condition prohibiting gum chewing
32. Pheochromocytoma
33. Thyroid disorders
34. Diabetes
35. Type of angina where chest pain occurs at rest
36. Unpredictable severe constricting chest pain
37. Prickling or tingling of fingers and toes

38. Buerger's Disease
39. Throat irritation
40. Peptic ulcers
41. Esophagitis

A.2 Neurophysiological Foundations

The representation of value in the brain relies on the activity of populations of neurons processing the choice options and their values in a noisy fashion (Glimcher and Tymula, 2023). Higher signal-to-noise ratios lead to more stable outcomes. In contrast, low signal-to-noise ratios result in unstable and random outcomes, providing a possible explanation for irrational choices. Given that all three substances in our study are thought to enhance attention (Thiele and Bellgrove, 2018; Ranjbar-Slamloo and Fazlali, 2019; Lockhofen and Mulert, 2021) and, at a basic level, to facilitate the distinction between neural signal and noise, each of them could potentially increase rationality of choices.

Reboxetine. Considering our low dose of reboxetine (4 mg), it is plausible to assume a moderate elevation of noradrenaline activity in our study (note that inverted U-shaped effects have been reported for many neurotransmitters, including noradrenaline). The administration of acute reboxetine has been demonstrated to elevate noradrenaline levels in the frontal cortex and hippocampus by inhibiting noradrenaline reuptake (Sacchetti et al., 1999), whereas in the locus coeruleus it reduces firing activity by activating inhibitory α2-autoreceptors (Szabo and Blier, 2001). Attention and the noradrenergic system have been widely studied (Thiele and Bellgrove, 2018; Ranjbar-Slamloo and Fazlali, 2019; Lockhofen and Mulert, 2021). For example, studies have shown that the noradrenergic drug reboxetine improves accuracy of human visual perception and enhances BOLD fMRI responses in high-order visual cortex (Gelbard-Sagiv et al., 2018). Reboxetine has little affinity for the dopamine transporter (Millan et al., 2001) but binds weakly to the serotonin transporter (SERT), although its affinity for the norepinephrine transporter (NET) is much higher (NET/SERT affinity ratio 130; see Hajós et al., 2004). Therefore, our results for reboxetine primarily reflect an effect of increased noradrenaline rather than dopamine or serotonin.

Methylphenidate. Traditional motivational effects are underpinned by neuromodulators such as dopamine (Westbrook et al., 2020; Bowman et al., 2023), which also plays a key role in attention (Thiele and Bellgrove, 2018; Ranjbar-Slamloo and Fazlali, 2019; Lockhofen and Mulert, 2021). Although

methylphenidate can also impact noradrenaline reuptake, it exerts a stronger effect on the dopamine than the noradrenaline transporter (Heal and Pierce, 2006; Jaeschke et al., 2021). Methylphenidate also inhibits norepinephrine transporters, and, in rodents, the norepinephrine transporter clears dopamine in prefrontal cortex (Morón et al., 2002; Williams and Steketee, 2004). In human prefrontal cortex, this double role is less-well established. In any case, dopamine is cleared also by the monoamine oxidases (Wayment et al., 2001), and clearance of prefrontal monoamines is relatively slow, enabling them to travel further than in regions with higher transporter density (Mundorf et al., 2001). Our observed effects of methylphenidate clearly differ from those of reboxetine, which speaks against a common prefrontal dopamine mechanism explaining our findings.

Nicotine. Like for dopamine, boosting acetylcholine using nicotine improved economic rationality moderately. Acetylcholine has long been associated with attention and nicotinic acetylcholine receptors are widely distributed in the brain (Thiele and Bellgrove, 2018; Lockhofen and Mulert, 2021). Notably nicotinic receptors primarily up-regulate dopamine release in the ventral striatum (Grenhoff et al., 1986; Mereu et al., 1987), while methylphenidate influences prefrontal functions (Fallon et al., 2017) and dopamine release particularly in the dorsal striatum (Birn et al., 2019).

It is generally worth noting that the distribution of noradrenaline, dopamine and acetylcholine receptors varies across brain regions and cortical layers. For example, the visual cortex contains many noradrenaline receptors but relatively few dopamine receptors while the opposite holds in the striatum (Jacob et al., 2018). Moreover, within cortical regions with both receptors, noradrenergic axons innervate layers I and II/III more strongly than dopaminergic axons, and vice versa for layers V and VI (Vander Weele, 2018). We speculate that the selective attention-enhancing effect of noradrenaline on rational choices may be attributed to increases in signal-to-noise ratio particularly in regions and layers with a relatively high density of noradrenaline receptors, and that these are involved in the suppression of the (noise) signal associated with dominated options. This effectively ensures that the most preferred options are more consistently selected. Our finding of reboxetine enhancing selective attention aligns with theories which suggest that noradrenaline plays a crucial role in facilitating the ability to direct attention towards relevant information (Servan-Schreiber et al., 1990; Berridge and Waterhouse, 2004; Aston-Jones and Cohen, 2005). Of course, there is need for future electrophysiological and neuroimaging studies to characterize the neural correlates of the present study.

A.3 List of Questionnaires

Screening session:

1. Adult ADHD Self-Report (ASRS-v1.1; Kessler et al., 2005)
2. Barratt Impulsiveness Scale (BIS-11; Patton et al., 1995)
3. Behavioral Inhibition/Activation System (BIS/BAS; Carver and White, 1994)
4. Edinburgh Handedness Inventory (Oldfield, 1971)
5. Trait part of State-Trait Anxiety Inventory (STAI-Y2; Spielberger, 1983)
6. Symbol Digit Modalities Test (SDMT; Smith, 1973)
7. Symptom Checklist-90-Revised (SCL-90-R; Derogatis and Unger, 2010)

Experiment session:

1. Numeracy test (Ofstedal, 2005)
2. Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989)
3. Positive and Negative Affect Schedule (PANAS; Watson et al., 1988)
4. Sleep Questionnaire (SQ; Görtelmeyer, 2011)
5. Visual Analog Scales (VAS) about drug and mood effects
6. Wechsler Digit Span task (Wechsler, 1997)

A.4 Cortisol and Testosterone

We measured the participants' salivary cortisol levels (Gao et al., 2015) three times: before drug administration, after drug administration but before the decision-making tasks, and after the decision-making tasks. As shown in Table A.1., group differences before drug administration are small. After drug administration, salivary cortisol appears to be elevated in the reboxetine group relative to the other groups, in line with the well-known cortisol-enhancing effects of reboxetine (Hennig et al., 2000).

	Pre-drug administration		**Post-drug administration**			
	Baseline		Before decision-making tasks		After decision-making tasks	
	Mean	SD	Mean	SD	Mean	SD
Reboxetine group	8.75	9.36	8.12	4.71	6.83	2.95
Methylphenidate group	7.80	5.26	4.96	3.57	4.49	2.55
Nicotine group	7.68	4.66	3.25	1.93	3.80	2.55
Placebo group	7.96	5.07	3.28	3.13	3.61	5.23

Table A.1. Descriptive statistics of salivary cortisol levels at three timepoints: pre-drug administration, post-drug administration & before decision-making tasks, post-drug administration & after decision-making tasks.

We conduct Bayesian regression models with drug dummy variables to test for differences between the groups. Compared to the placebo group, a drug effect ($\beta_{drug} \neq \beta_{placebo}$) is considered statistically significant if the 95% CI range of the difference distribution does not include zero. Based on this approach, we find no significant group differences in cortisol levels before drug administration, but higher levels in the reboxetine group compared to the placebo group after drug administration, both before and after the decision-making tasks. The methylphenidate group exhibits higher cortisol levels compared to the placebo group before the decision-making tasks, but this difference does not last until after completion of the tasks. Together, these findings highlight the need to control for salivary cortisol levels in the main analyses. We remark here that our main analyses indicate that higher cortisol may decrease rationality, in contrast to the findings of Cettolin et al. (2020).

We also determined salivary testosterone levels from the same saliva samples we used to determine cortisol. It has been suggested that chewing gum increases salivary testosterone (van Anders, 2010). While in our experiment all participants received a gum and followed the same chewing instructions, the chewing patterns could in principle have differed between placebo and nicotine gum. However, we observe no significant differences in testosterone levels across the four groups and across the three timepoints. We therefore do not control for testosterone in our regressions.

A.5 Robustness of Regression Results

This appendix contains regression results analogous to those reported in Table 3 but with additional controls. Our results are largely robust to these additional controls.

	FOSD Violations			GARP Violations			Varian Index			Afriat Index			SWAPS Index			Rational Inattention Cost		
	Estimates	5% CI	95% CI	Estimates	5% CI	95% CI	Estimates	5% CI	95% CI	Estimates	5% CI	95% CI	Estimates	5% CI	95% CI	Estimates	5% CI	95% CI
RBX-PBO	-0.033	-0.059	-0.007	-60.785	-100.802	-20.875	-0.009	-0.016	-0.002	-0.096	-0.162	-0.030	107.377	-218.356	431.031	-1.009	-2.243	0.134
MPH-PBO	-0.018	-0.041	0.004	-10.443	-45.062	24.648	-0.005	-0.011	0.002	-0.049	-0.107	0.009	65.016	-218.395	353.090	-1.999	-3.096	-0.967
NIC-PBO	-0.013	-0.035	0.010	-30.423	-64.788	3.982	-0.005	-0.011	0.002	-0.064	-0.121	-0.007	2.121	-282.792	283.345	-0.796	-1.758	0.160
Age	0.002	-0.001	0.005	4.982	-0.255	10.185	0.001	0.000	0.002	0.005	-0.004	0.013	-4.263	-46.179	37.606	-0.102	-0.263	0.050
Sex	-0.025	-0.045	-0.006	-47.966	-78.237	-17.860	-0.009	-0.014	-0.003	-0.071	-0.121	-0.022	26.443	-209.380	267.386	-0.427	-1.253	0.382
Cortisol	0.003	0.000	0.006	6.845	2.102	11.550	0.001	0.000	0.002	0.006	-0.002	0.013	37.268	-0.351	75.404	0.098	-0.046	0.256
Baseline Working Memory	-0.003	-0.005	0.000	-1.130	-5.303	3.085	0.000	-0.001	0.000	-0.002	-0.008	0.005	-5.556	-39.033	28.172	-0.053	-0.167	0.059
Baseline Numeracy Skill	-0.009	-0.026	0.007	-23.051	-47.744	2.052	-0.004	-0.008	0.001	-0.024	-0.064	0.018	-98.639	-299.925	99.679	-0.715	-1.438	-0.006
Baseline Positive Affect	0.001	-0.001	0.003	3.254	0.465	6.037	0.000	0.000	0.001	0.002	-0.003	0.006	23.771	1.631	45.763	-0.164	-0.249	-0.083
Baseline Negative Affect	-0.001	-0.003	0.001	-2.586	-5.751	0.575	0.000	-0.001	0.000	-0.004	-0.009	0.001	-21.212	-46.976	4.057	0.084	-0.004	0.172
Session Last	0.000	-0.002	0.001	1.028	-1.304	3.373	0.000	0.000	0.001	-0.001	-0.005	0.003	-8.238	-27.068	10.593	-0.057	-0.124	0.009
Education	0.000	-0.005	0.004	-2.818	-9.652	4.089	-0.001	-0.002	0.001	-0.001	-0.013	0.010	31.064	-24.287	85.492	0.114	-0.081	0.311
BMI	-0.002	-0.005	0.001	-3.976	-8.930	1.006	0.000	-0.001	0.000	-0.004	-0.012	0.004	-10.816	-50.864	28.909	0.018	-0.124	0.159
BIS11 Score	0.000	-0.002	0.001	-0.083	-2.100	1.943	0.000	0.000	0.000	0.000	-0.004	0.003	-6.312	-22.017	9.595	0.005	-0.048	0.059
ADHD Score	-0.001	-0.002	0.001	-1.697	-4.091	0.680	0.000	-0.001	0.000	-0.002	-0.005	0.002	-0.433	-19.598	18.764	-0.084	-0.152	-0.017
STAI Score	0.001	-0.001	0.002	1.722	-0.887	4.323	0.000	0.000	0.001	0.001	-0.003	0.005	-3.980	-24.777	16.597	0.016	-0.058	0.090
BAS Score	0.001	-0.001	0.002	0.055	-2.681	2.762	0.000	-0.001	0.000	-0.001	-0.006	0.003	-3.145	-24.795	18.529	0.128	0.043	0.218
BIS Score	-0.003	-0.006	-0.001	-2.986	-7.116	1.158	-0.001	-0.002	0.000	-0.006	-0.013	0.001	21.878	-11.079	55.279	0.056	-0.059	0.172
SCL Score	0.001	-0.001	0.002	1.573	-0.925	4.092	0.000	0.000	0.001	0.000	-0.004	0.004	4.490	-15.231	24.502	0.029	-0.041	0.098
PSQI Score	0.004	-0.001	0.009	11.209	3.732	18.781	0.002	0.000	0.003	0.013	0.000	0.025	64.473	4.542	124.237	-0.035	-0.245	0.176

Table A.2. Coefficients in Bayesian regression models. Estimated coefficients and credible intervals (CIs) from regressions of the respective rationality measure on drug dummy, age, sex, cortisol level, and additional controls measured pre-drug treatment. These controls are working memory from Wechsler digit span task (Baseline Working Memory), numeracy skill (Baseline Numeracy Skill), positive affect from PANAS (Baseline Positive Affect), negative affect from PANAS (Baseline Negative Affect), duration of experiment (Session Last), education (Education), body mass index (BMI), trait impulsivity (BIS-11 Score), ADHD symptoms from ASRS-v1.1 (ADHD Score), trait anxiety (STAI Score), behavioral activation (BAS Score), behavioral inhibition (BIS Score), general psychiatric symptoms (SCL score), and sleep quality (PSQI Score).

A.6 Comparison of Risk Aversion Across Tasks

This appendix compares the estimated coefficients of relative risk aversion between the willingness-to-pay task and our main decision task.

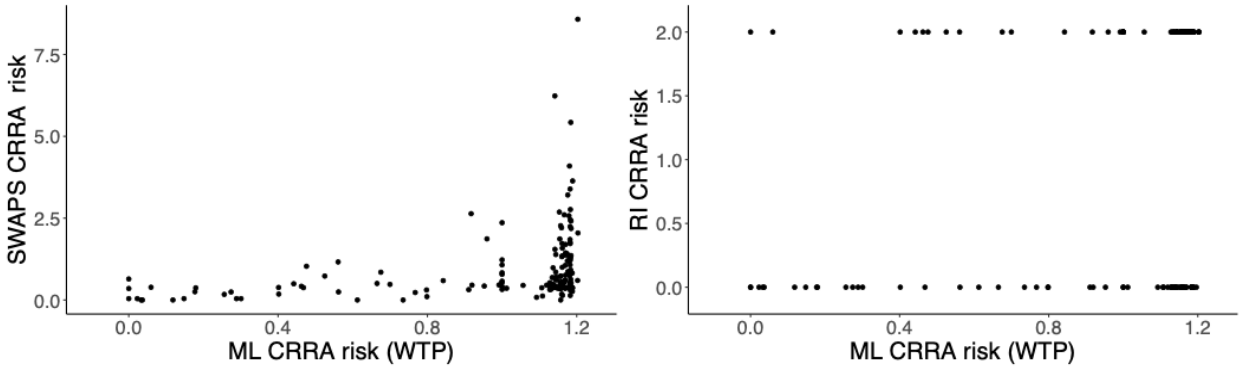

Figure A.1. Scatterplot of estimated CRRA coefficients. The left panel plots each participant's CRRA coefficient estimated in the willingness-to-pay task against the CRRA coefficient obtained with the swaps approach in the main decision-making task (omitting the participants with an estimated value of infinity). The right panel plots each participant's CRRA coefficient estimated in the willingness-to-pay task against the binary CRRA coefficient obtained with the rational inattention approach in the main decision-making task.