When Helping is Risky:

Behavioral and Neurobiological Mechanisms of Prosocial Decisions Entailing Risk

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Abstract

Helping others can entail risks. Doctors that treat infectious patients may risk their own health, intervening in a fight can lead to injury, and organ donations can lead to medical complications. When helping others comes with a risk to oneself, decisions depend on the individual's valuation of others' well-being (social preferences) and the degree of personal risk the individual finds acceptable (risk preferences). Here we identify how these distinct preferences are behaviorally (Study 1, N=292) and neurobiologically (Study 2, N=154) integrated when helping is risky. We independently assessed social and risk preferences using incentivized behavioral tasks, and manipulated dopamine and norepinephrine levels in the brain by providing methylphenidate, atomoxetine, or placebo. Results reveal that social and risk preferences are independent driving forces of risky helping, and that methylphenidate-altered dopamine concentrations lead to more helping under risk because of increases in risk-tolerance rather than increased social preferences. Implications for decision-theory and drug use are discussed.

Statement of Relevance

People help others at sometimes substantial costs to themselves. What has been largely overlooked is that helping can also be risky. When treating patients with infectious diseases doctors may become infected themselves, and volunteers trying to rescue ship-wrecked refugees risk injury and drowning. Here we identify individual differences in social preferences–predicting willingness to help–and risk preferences–predicting willingness to take risks–and examine how these distinct preferences alone and in combination predict decision-making when helping comes at a risk. Findings advance theory by showing that popular off-the-shelf drugs like methylphenidate increase risky helping because they alter risk rather than social preferences.

Findings have practical relevance because they reveal that refusals to help others often are rooted in risk-aversion rather than selfishness. Interventions geared at reducing risk-aversion may be as effective, if not more, than interventions geared at increasing people's moral obligation to care for the faith of others.

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Individuals differ in the extent to which they help others (Engel, 2011). Such behavioral variation in helping is often considered a reflection of differences in social preferences – the value individuals assign to the welfare of another person (Fehr & Schmidt, 1999; Van Lange, 1999). However, helping others often comes with risks – the possibility to incur additional personal costs (Do, Moreira & Telzer, 2017; Müller & Rau, 2016; Vives & FeldmanHall, 2018). Helping a victim of an assault comes with the risk of injury, organ donations can lead to medical complications, and helping a colleague to rescue a poor investment may result in own financial loss. As with social preferences, people also differ in the risks they take (Halek & Eisenhauer, 2001; Holt & Laury, 2002), reflecting differences in risk preferences – the degree to which a decision maker tolerates uncertain outcomes. Indeed, public debate about helping often revolve around the involved risks. Allowing entry to war refugees at Europe's borders, for example, has been met with concerns over risk for national security, attempts to bring Ebola patients to hospitals in the United States was met with strong public protest due to concerns over infection risks (Yang, 2015), and amidst the on-going Corona pandemic, the entanglement and dilemma of helping those in need and the risk it poses to the own welfare is all too evident.

Whereas both risk preferences and social preferences may be involved in risky helping (Brock, Van Lange, & Ozbay, 2013; Saito, 2013), the precise form and function of these two preferences in producing risky helping remains poorly understood. Although risk and social preferences have been mostly studied in isolation, neuroimaging studies suggests that the brain represents decision options on a common subjective value scale by integrating and trading off

different option features or conflicting internal motives, like risk aversion, value maximization, and social preferences (e.g., Gross et al., 2014; Hunt, Dolan, & Behrens, 2014; Levy & Glimcher, 2012). For example, Kameda et al. (2016) showed that risky decision-making and fairness judgments share common neural processes, and studies in developmental neuroscience have shown that the emergence of pro-social attitudes during adolescence coincides with increased risk-taking (e.g., van den Bos, van Dijk, Westenberg, Rombouts, & Crone, 2011). Specifically, prefrontal areas like the DLPFC associate with both risk-taking (e.g., Fecteau et al., 2007; Mohr, Biele, & Heekeren, 2010), and with social preferences (e.g., Gross, Emmerling, Vostroknutov, & Sack, 2018; Ruff, Ugazio, & Fehr, 2013). Likewise, dopamine signals in the midbrain and striatum have been shown to modulate both risk-taking (Fiorillo, Tobler, & Schultz, 2003) and pro-social behavior (e.g., Harbaugh, Mayr, & Burghart, 2007).

Combined, these earlier studies point to the possibility that risk and social preferences may be integrated in risky helping decisions, and that such integration involves (dopaminergic modulation of) prefrontal and striatal structures in the human brain. We performed two studies to examine these possibilities. Across both studies, we used incentivized behavioral tasks that confront the individual with a decision to help (or not) another person, when helping is not only costly but also risky. By manipulating risk and social consequences independently within the same task, we measured social and risk preferences in isolation, allowing us to see whether and how these preferences are behaviorally integrated when helping is risky.

In Study 2 we complemented the behavioral approach with a double-blind neuropharmacological intervention to manipulate dopaminergic and noradrenergic neurotransmission and test if risky helping can be causally manipulated. Specifically, we compared placebo treatment against treatment with methylphenidate, and with atomoxetine. Both

are used as off-label drugs for cognitive enhancement, yet with distinctly different effects on brain activation. Methylphenidate functions as a reuptake inhibitor of both dopamine and norepinephrine by blocking the dopamine (DAT) and norepinephrine transporter (NET; Kuczenski & Segal, 1997). Methylphenidate is believed to increase dopamine concentration in the striatum (e.g., Kim et al., 2009), and dopamine and norepinephrine concentration in the vMPFC and DLPFC (Arnsten & Li, 2005), neurotransmitters that have been associated with both risk taking and social preferences. Atomoxetine, in contrast, is a selective NET reuptake inhibitor. In the prefrontal cortex it increases synaptic levels of both norepinephrine and dopamine; however, it does not influence striatal dopamine levels (e.g., Smith, Beveridge, & Porrino, 2006). The divergent effects of these two drugs, therefore, allow us to examine how the manipulation of subcortical and prefrontal dopamine and noradrenaline impacts the trade-off between social and risk preference when decisions to help entail risks.

Methods

The Risky Helping Task. To investigate risky helping as a function of social and risk preferences, we designed a two-player incentivized "risky helping task" (Figure 1a). It involves a decider who repeatedly decides whether or not to help another participant, the receiver. Deciding not to help leads to a sure outcome of 15 monetary units (MU) for the decider and zero MU for the receiver, which are exchanged into cash at the end of the experiment. Deciding to help is risky: With a probability p helping is unsuccessful, in which case both decider and receiver earn zero MU. In case helping is successful (with probability 1-p), both earn 13 MU. Probability p, i.e. risk, is systematically varied between 0 and 1 across trials. Note that helping always reduces inequality between decider and receiver to zero. Further, helping is costly to the decider: even

when successful, the decider incurs a cost of 2 MU. Hence, the risky helping task confronts the decision maker with a dilemma between avoiding risk to oneself and helping another person.

To measure the relative influence of the decider's social and risk preferences, we developed two variants of the risky helping task. In the "risk task", we removed the social component of the task to measure risk preferences in isolation – the receiver is not affected by the decider's decisions. The decider chooses – for themselves – between a certain outcome and a lottery with outcomes linearly related to the risky helping outcome for each possible *p*-value (Figure 1b and Table 1). Hence, the risk task measures risk preferences by reducing the risky helping task to a set of paired lottery choices with no social consequences. In the "helping task" we removed the risk component of the task to measure social preferences in isolation (Figure 1c) – the outcome of each choice in the risky helping task is replaced with the expected value for each *p*-value (i.e. risk level). Thus, the decider chooses between two sure outcomes – one that maximizes their own payoff but also leads to inequality, or one that requires to sacrifice own MUs to benefit the receiver and eliminates inequality (similar to a dictator game with different efficiency gains). Hence, the helping task measures social preferences unaffected by risk. In the experiments, neutral labels for helping or risk were used to avoid demand or framing effects and decisions were completely anonymous to avoid reputation effects.

Participants, Sample Size and Research Ethics. In Study 1, a sample of 292 participants (mean age = 22.3±3.7, 142 female) were recruited and split into 146 deciders and 146 receivers. Sample size for this fully within-subjects design was based on earlier work assessing individual differences in social value orientation (Van Lange, 1999), typically finding roughly 45% (40%) of the sample holding pro-social (pro-self) preferences. Participants were free to withdraw from participation at any time. Only individuals who voluntarily entered the

experiment recruiting database were invited and informed consent was obtained from all participants by electronic acceptance of an invitation to attend an experimental session. The experiment was conducted following the peer-approved procedures established by the Center for Research in Experimental Economics and Political Decision Making (CREED) at the University of Amsterdam, and was fully incentivized and void of deception.

In Study 2, a sample of 154 participants (mean age = 23.7±3.9, 77 female) were recruited in the role of deciders, with only one participant taking part per experimental session. Participants were free to withdraw from participation at any time. Participants completed a pre-screening questionnaire to select only those who had no history of drug consumption, a limited alcohol and caffeine uptake, no clinically relevant depression scores, and who were generally in a healthy condition. Participants underwent another screening via telephone the day before the study, to make sure they had, for example, not consumed any alcohol or other medication. The experiment received ethical approval from the University of Oxford's Medical Sciences Interdivisional Research Ethics Committee. Because dopamine function can vary throughout the day (e.g. López, Howell, Canúl, Leyton, & Gobbi, 2014), each experimental session took place in the same place at roughly the same time of the day (starting around 9 AM). Before substance administration and after pre-administration checks, female participants took a test to confirm they were not pregnant and we measured height, weight, and blood pressure of all participants. Supervised by a medical doctor throughout the study, participants either received 30mg of methylphenidate, 60mg of atomoxetine, or placebo, contained in an identical blue capsule. The active substance was unknown to both the experimenters and participants. To make sure that testing coinciding with peak absorption rate, the experiment started 90 minutes after drug administration (Sauer, Ring, & Witcher, 2005). Participants then took part in the risk task, the

helping task, and the risky helping task as part of a larger test battery. Before drug administration and after the experiment, participants completed a 15-item visual analogue mood scale. For each item participants had to place a mark along a straight line with two opposing adjectives on either side (e.g. 'muzzy vs. clear-header', 'happy vs. sad', 'tense vs. relaxed'), resulting in a continuous measure between 0 and 1 for each item. Hence, we measured subjective mood states before and after the experiment (also allowing to compute mood changes).

Experimental Procedure. In both studies, each participant performed the tasks in front of a personal computer terminal. Comprehension checks were used to make sure that participants understood the instructions. To measure risk- and social preferences and the extent of helping in a risky environment on the individual level, each decider engaged in the three separate tasks in random order. The decisions of the decider had real financial consequences for decider and receiver that were paid out in cash at the end of the experimental session (one round of each task was selected at random for payment; 1 MU was worth 20 Euro cents in Study 1 and 36 British pennies in Study 2). In Study 1, receivers were also confronted with every decision that the decider had to face for risk, helping, and risky helping across all risk levels, but instead had to guess how the decider would decide in each situation. Guessing correctly was financially incentivized (see Supplementary Information).

Behavioral Task Implementation. In the "risky helping task", the decider has to repeatedly decide whether or not to help the receiver. Helping is risky. With probability p, helping fails and both decider and receiver earn zero MU. When helping is successful (with probability 1-p), both, decider and receiver receive a payoff of 13 MU. Across trials, probability p varies between 0 and 1 across 21 randomly presented trials. In the "risk task", the decider faces a set of lottery choices that do not affect the payoff of the receiver. Hence, the risk task confronts

the decision maker with a dilemma between choosing a sure outcome or a gamble that can lead to a higher payoff (with probability 1-*p*) but also entails the risk to earn nothing for that trial (with probability *p*). In the "helping task", the decider again chooses between the option to help or not help. Compared to the risky helping task, helping is not risky and the decider knows the outcome of both options in advance. Not helping leads to 15 MU for the decider and 0 MU for the receiver, as in the risky helping task. The outcome of helping varied across trials. Specifically, the outcome of helping was equal to the expected value of the risky helping decisions in the risky helping task. To avoid reputation and demand effects, neutral labels for helping or risk were used in the instructions and tasks and decider and receiver were unaware with whom they were matched.

Responses. In each task, deciders made the binary decision to help or not (helping task and risky helping task), or to take the risky or the safe choice (risk task) across multiple trials that differed in risk (risk task / risky helping task) or helping consequences (helping task). Trials were presented sequentially and in random order. Thus, in the risk task, each participant indicated whether to take the sure outcome or the lottery for each risk-level. In the risky helping task, each participant indicated whether to take the sure but unequal outcome (option A, 15 MUs for the decider, 0 MUs for the receiver), or the risky but equal outcome (option B) for each risk-level. Lastly, in the helping task, each participant indicated whether to take the sure and unequal outcome (15 MUs for the decider, 0 MUs for the receiver), or the sure and equal outcome, where the outcome was equal to the expected value of the risky helping lottery for each risk level. Table 1 shows the expected outcomes for decider and receiver across risk-levels for risk task and helping / risky helping task.

Analyses. In each trial we observed a binary decision. For each task and each decider, we observed a decision pattern that revealed when a participant would take the risky/social option and when they would switch to the certain/selfish option. We used these switching points as a measure for risky helping, social preferences and risk preferences, respectively. For example, if a participant decided to choose the lottery for a risk p = [0, 0.05, 0,10, 0.15, 0.20, 0.25] and switched to the sure outcome for p = [0.30, …, 1], the participant was assigned the value 0.30 as their measure of risk-preference. Note that this implies that a switching point of 0.45 or lower indicates risk aversion (or risk neutrality) in the risk task, since the decision maker switched to the safe option before the expected value of the lottery decreased below the fixed payoff of the safe option. A switching point of 0.5 and higher indicates risk seeking, since the decision maker accepted lotteries for which the expected value of the lottery was lower than the fixed payoff of the safe option. Between 14% and 18% of our participants had multiple switching-points, thereby violating monotonicity. This is not unexpected, because trials were randomly and sequentially presented within each task. Accordingly, we interpret violations of monotonicity as noise and averaged the switching points in these cases. Thus, a decision pattern like [**B**, **B**, **B**, A, **B**, A, A, …, A] for risk-levels of [0, 0.05, 0,10, 0.15, 0.20, 0.25, 0.30, … 1] led to a switching point of 0.20. Excluding participants who violated monotonicity from the analysis did not alter the reported conclusions below.

To analyze switching points, we used Tobit regressions. Tobit regressions deviate from a linear regression model in that they account for the (left) censoring in the data by assuming instead of y, a latent variable y* that linearly depends on the predictors (y is equal to y* if y > 0, but deviates in the lower bound). For y = y*, we used the likelihood function of a simple t-distribution $t(y|bX, \sigma^2, df)$. For $y \neq y^*$, the likelihood function is based on the normal cumulative

distribution and hence models the probability that y* will take a value less than or equal to y, given the observed predictors. This approach allows us to derive unbiased estimates for predictors and error variance in the presence of heteroscedasticity.

Results

The Supplementing Information gives full reports of the regression models and results, alongside additional models that include control variables and robustness checks. Here we summarize the main findings from our two experiments.

Study 1 Results. In the risk task, in line with previous literature (e.g., Holt & Laury, 2002), participants were predominantly risk-averse, meaning that they switched to the risky option only for gambles for which the expected value of the risky option exceeded the value of the safe option (Figure 2a). In the helping task, a majority of participants revealed other-regarding preferences, willing to give up own resources to help the other person at least once (Figure 2b). In the risky helping task, participants who engaged in risky helping did so up to a risk of $p = 0.15$ on average, and then switched to not helping (Figure 2c). When participants engaged in risky helping, the gain to social welfare (i.e. the combined expected earnings of decider and receiver) significantly exceeded their own monetary sacrifice, showing that risky helping was not unconditional (reduction in earnings for deciders vs. expected value of decider and receiver; Wilcoxon Signed Rank test, $p < 0.001$). On average, deciders were willing to sacrifice 1 MU to achieve an expected welfare increase of 1.8 MU.

To test if risky helping decisions can be modelled as a function of risk and social preferences, we analyzed if and how switching points in the risk and helping task predicted risky helping decisions. We found, first of all, that risk preferences derived from the risk task and social preferences derived from the helping task, were linearly independent of each other

(Spearman $r = -0.02$, $p = 0.787$). Thus, an individual's social preference could not be inferred from knowing their risk preference and vice versa, pointing to a behavioral dissociation of risk and social preferences. Yet, already indicating that risk preferences are integrated in risky helping decisions, the change in switching points between helping and risky helping was significantly correlated with the switching point in the risk task (Spearman $r = 0.25$, $p = 0.002$). Conversely, a switching point change between the risk task and the risky helping task was significantly correlated with the extent of helping in the helping task (Spearman $r = 0.57$, $p < 0.001$). In other words, the change in helping under risk (compared to helping under no risk) was associated with risk preferences, as measured by the risk task. Likewise, the change in risk-taking when taking risks also had social consequences vs. not was associated with social preferences.

Another approach to test the extent to which risky helping can be predicted by risk preference, social preference, or their combination, is to use regression models. Since switching points were censored at zero, we fitted Tobit regressions for this purpose. As shown in Figure 3a, risky helping decisions were a function of both social preferences and risk preferences (Tobit regression: risk preference estimate = 0.26, $p = 0.005$, social preference estimate = 0.83, $p < 0.001$). Comparing the relative weight of social and risk preferences also revealed that, while both variables were independently associated with risky helping, social preferences were a stronger predictor of risky helping than risk preferences. In other words, helping under risk emerged only with moderate to high social preference combined with low to moderate risk aversion. Taken together, Study 1 provided first evidence that risk and social preferences are behaviorally independent of each other, yet systematically integrated in risky helping decisions.

Study 2 Results. As in Study 1, individuals' risk and social preferences were uncorrelated (Spearman $r = 0.03$, $p = 0.800$). Also replicating the results of Study 1, within-individual changes in helping across helping and risky helping task were predicted by risk preferences (Spearman $r = 0.24$, $p = 0.003$) and, vice versa, within-individual changes in risk-taking across risk and risky helping task were predicted by social preferences (Spearman $r = 0.61$, $p < 0.001$). As shown in Figure 3b, risky helping decisions could be meaningfully modelled as linear combination of social preferences and risk preferences (Tobit regression: risk preference estimate = 0.31, $p = 0.001$, social preference estimate = 0.73, $p < 0.001$), meaning that knowing a person's social preferences does not allow accurate predictions of risky helping without also knowing the person's risk preferences and vice versa.

Methylphenidate causally affected risk preferences (Figure 4a). Participants under methylphenidate accepted significantly more risk than participants under placebo (Tobit regression, methylphenidate vs. placebo estimate = 0.05, $p = 0.018$). While 90% of the participants under placebo were classified as risk-averse (similar to Study 1), this dropped to 78% under methylphenidate. Risk-taking was not significantly different between placebo and atomoxetine (Tobit regression, atomoxetine vs. placebo estimate = 0.02, $p = 0.295$). We also did not find a statistically significant difference in risk-taking between atomoxetine and methylphenidate (Tobit regression, atomoxetine vs. methylphenidate estimate = 0.03, $p = 0.185$).

Importantly, we found no evidence that methylphenidate or atomoxetine affected social preferences (Figure 4b, Tobit regression: methylphenidate vs. placebo estimate = 0.02, $p = 0.728$; atomoxetine vs. placebo estimate = 0.00, $p = 0.947$; methylphenidate vs. atomoxetine estimate = 0.02, $p = 0.664$). However, due to their higher willingness to take risks, participants under methylphenidate helped more often in the risky helping task compared to placebo (Figure

4c, Tobit regression: methylphenidate vs. placebo estimate = 0.07, p = 0.047) and atomoxetine (Tobit regression: atomoxetine vs. methylphenidate effect estimate = 0.08, p = 0.030). In contrast, atomoxetine did not significantly increase risky helping compared to placebo (Tobit regression: atomoxetine vs. placebo estimate = -0.01, p = 0.787). Consequently, participants under methylphenidate were willing to sacrifice more resources in the risky helping task, resulting in higher overall welfare compared to placebo and atomoxetine. Under methylphenidate, 22.4% of participants were even willing to help beyond the social efficiency point under risk, compared to 11.5% and 13.2% under placebo and atomoxetine, respectively.

The effects of methylphenidate on risk attitudes and risky helping may be driven by interindividual differences in drug-absorption rates, drug interactions with age or gender, or effects of drugs on mood. To help rule out these alternative explanations, we controlled for age and gender in the reported regressions and computed additional regressions controlling for body-mass index and regular medication (e.g. contraceptive pill) that both may influence absorption rates of the drug agent. We also controlled for mood and mood changes that have been shown to influence risk-taking and social decision making. Drug treatment remained a robust predictor of risk and risky helping decisions (see also Table S3-S5).

Furthermore, 38% of the participants correctly guessed the treatment they received (which is consistent with random guessing, i.e. chance level of 33%, chi-square test, $\chi^2(1) = 1.44$, p = 0.231). Repeating the reported analyses only with participants that incorrectly guessed the treatment did not change the above conclusions (see also Table S3-S5). Lastly, drug treatment may render decision makers more erratic, increasing choice inconsistency that could spuriously increase or decrease switching points. In our tasks, choice consistency can be measured by looking at the number of switching points. A perfectly consistent decision maker should have

one unique switching point in each task, while multiple switching points indicate intransitive choice. In the risk task, 76% of the participants had a unique switching point (compared to 86% in Study 1). In the helping task, 73% of the participants had a unique switching point (compared to 82% in Study 1). Lastly, in the risky helping task, 77% of the participants had a unique switching point (compared to 83% in Study 1). While participants were slightly less consistent than in Study 1, we found no statistical evidence that the drug treatments made decisions noisier and more inconsistent compared to placebo in the risk task (Tobit regression, methylphenidate vs. placebo estimate = 0.50, $p = 0.609$, atomoxetine vs. placebo estimate = 0.89, $p = 0.337$), helping task (Tobit regression, methylphenidate vs. placebo estimate = -1.55, $p = 0.141$, atomoxetine vs. placebo estimate = 0.74, $p = 0.306$), or risky helping task (Tobit regression, methylphenidate vs. placebo estimate = -0.24, $p = 0.862$, atomoxetine vs. placebo estimate = 1.64, $p = 0.201$).

Discussion

Helping can often have unforeseen consequences, rendering pro-social behavior not only costly but also risky. Here, we show that people react to both the risk- and social consequences of their decisions and systematically integrate their risk and social preferences when helping is not only costly, but also risky. Because risk and social preferences were uncorrelated in our sample, it is not possibly to simply substitute or estimate social preferences from risk preferences and vice versa. One cannot reliably predict decision-making in risky helping situations based on risk- or social preferences alone. Instead, observing someone refusing to help under risk could be equally driven by a lack of social preferences or by risk aversion (or a combination of both).

Our results suggest that risk and social preferences are, to some degree, independently processed and then systematically integrated when social concerns need to be traded off against

risk concerns. In line with this behavioral separation, methylphenidate selectively altered risk preferences, while social preferences were unaffected. In comparison, atomoxetine changed neither risk nor social preferences. One possible explanation is that the specific effects of methylphenidate on subcortical dopamine transmission selectively influence risk tolerance without affecting social concerns. This resonates with earlier work linking subcortical dopamine levels to the propensity to gamble (e.g., Christopoulos, Tobler, Bossaerts, Dolan, & Schultz, 2009; Clark, Lawrence, Astley-Jones, & Gray, 2009; Fiorillo et al., 2003; Onge et al., 2010). Our results reveal that these risk-related changes can indirectly affect social decision-making when helping is risky and open the possibility that previously observed effects of dopaminergic manipulations on social preferences might be due to an influence on (unmeasured) risk preferences.

Our findings also have implications for policy and ethics concerned with the use of psychostimulants in nonclinical populations . Psychostimulants are regularly prescribed for treatment of attention deficit hyperactivity disorder, but they are also widely taken off-label by a large, uncontrolled number of users as "smart drugs", as they can improve cognitive functioning also in healthy individuals. In the Global Drug Survey, 3.2% and 6.6% of responders reported to use psychostimulants like methylphenidate for cognitive enhancement in 2015 and 2017, respectively (Maier, Ferris, & Winstock, 2018). The finding that methylphenidate alters helping behavior through increased risk-seeking (see also Campbell-Meiklejohn et al., 2012; Schlösser et al., 2009) demonstrates that substances aimed at changing cognitive functioning can also influence social behavior. Such “social” side-effects are currently unknown to both users and administrators and are thus far not considered in the societal debate about psychostimulant use, whether for treatment or enhancement purposes (Faulmüller, Maslen, & Santoni de Sio, 2013).

Decision makers are rarely sure about how a decision will be perceived, affect other parties, or influence their own welfare. Accordingly, social decisions often require integration of social preferences and risk concerns that, theoretically, can have divergent effects on decisions and create a dilemma between helping and avoiding risk to oneself. Our results highlight that risk and social concerns are behaviorally dissociable and orthogonal preferences, making it difficult to infer preferences from decisions when they entail both social consequences and risk. A failure to help can reflect a lack of concerns for others and/or a personal aversion to risk. Our data on receivers reveal that individuals often misattribute failures to help to a lack of social concern rather than a lack of risk tolerance (see *Supplementary Information* for these results).

While previous research has shown that risk and social consequences are processed in overlapping neural circuitries, our drug manipulation suggests largely independent mechanisms. We showed that methylphenidate selectively alters risk tolerance but keeps social preferences unchanged. Accordingly, methylphenidate increased helping under risk but not helping without risk. Atomoxetine, on the other hand, had no effect on either risk or social preferences. More generally, the drug induced changes in risk-taking and risky helping demonstrate a neurobiological dissociation of the processing of risk and social consequences in humans.

References

Arnsten, A. F. T., & Li, B.-M. (2005). Neurobiology of executive functions: catecholamine influences on prefrontal cortical functions. *Biological Psychiatry*, *57*(11), 1377–1384. http://doi.org/10.1016/j.biopsych.2004.08.019

Brock, J. M., Lange, A., & Ozbay, E. Y. (2013). Dictating the risk: experimental evidence on giving in risky environments. *American Economic Review*, *103*(1), 415–437. http://doi.org/10.1257/aer.103.1.415

Campbell-Meiklejohn, D., Simonsen, A., Scheel-Krüger, J., Wohlert, V., Gjerløff, T., Frith, C. D., et al. (2012). In for a penny, in for a pound: methylphenidate reduces the inhibitory effect of high stakes on persistent risky choice. *The Journal of Neuroscience*, *32*(38), 13032–13038. http://doi.org/10.1523/JNEUROSCI.0151-12.2012

Christopoulos, G. I., Tobler, P. N., Bossaerts, P., Dolan, R. J., & Schultz, W. (2009). Neural correlates of value, risk, and risk aversion contributing to decision making under risk. *The Journal of Neuroscience*, *29*(40), 12574–12583. http://doi.org/10.1523/JNEUROSCI.2614-09.2009

Clark, L., Lawrence, A. J., Astley-Jones, F., & Gray, N. (2009). Gambling near-misses enhance motivation to gamble and recruit win-related brain circuitry. *Neuron*, *61*(3), 481–490. http://doi.org/10.1016/j.neuron.2008.12.031

Do, K. T., Moreira, J. F. G., & Telzer, E. H. (2017). But is helping you worth the risk? Defining prosocial risk taking in adolescence. *Developmental Cognitive Neuroscience*, *25*, 260–271. http://doi.org/10.1016/j.dcn.2016.11.008

Engel, C. (2011). Dictator games: a meta study. *Experimental Economics*, *14*(4), 583–610. http://doi.org/10.1007/s10683-011-9283-7

Faulmüller, N., Maslen, H., & Santoni de Sio, F. (2013). The indirect psychological costs of cognitive enhancement. *The American Journal of Bioethics*, *13*(7), 45–47. http://doi.org/10.1080/15265161.2013.794880

Fecteau, S., Pascual-Leone, A., Zald, D. H., Liguori, P., Théoret, H., Boggio, P. S., & Fregni, F. (2007). Activation of prefrontal cortex by transcranial direct current stimulation reduces appetite for risk during ambiguous decision making. *The Journal of Neuroscience*, *27*(23), 6212–6218. http://doi.org/10.1523/JNEUROSCI.0314-07.2007

Fehr, E., & Schmidt, K. M. (1999). A theory of fairness, competition, and cooperation. *Quarterly Journal of Economics*, *114*(3), 817–868. http://doi.org/10.2307/2586885

Fiorillo, C. D., Tobler, P. N., & Schultz, W. (2003). Discrete coding of reward probability and uncertainty by dopamine neurons. *Science*, *299*(5614), 1898–1902. http://doi.org/10.1126/science.1077349

Gross, J., Emmerling, F., Vostroknutov, A., & Sack, A. T. (2018). Manipulation of pro-sociality and rule-following with non-invasive brain stimulation. *Scientific Reports*, *8*(1), 1–10. http://doi.org/10.1038/s41598-018-19997-5

Gross, J., Woelbert, E., Zimmermann, J., Okamoto-Barth, S., Riedl, A., & Goebel, R. (2014). Value signals in the prefrontal cortex predict individual preferences across reward categories. *The Journal of Neuroscience*, *34*(22), 7580–7586.

Halek, M., & Eisenhauer, J. G. (2001). Demography of risk aversion. *Journal of Risk and Insurance*, *68*(1), 1–24.

Harbaugh, W. T., Mayr, U., & Burghart, D. (2007). Neural responses to taxation and voluntary giving reveal motives for charitable donations. *Science*, *316*, 1622–1625.

Holt, C. A., & Laury, S. K. (2002). Risk aversion and incentive effects. *American Economic Review*, *92*(5), 1644–1655.

Hunt, L. T., Dolan, R. J., & Behrens, T. E. J. (2014). Hierarchical competitions subserving multi-attribute choice. *Nature Neuroscience*, *17*(11), 1613–1622. http://doi.org/10.1038/nn.3836

Kameda, T., Inukai, K., Higuchi, S., Ogawa, A., Kim, H., Matsuda, T., & Sakagami, M. (2016). Rawlsian maximin rule operates as a common cognitive anchor in distributive justice and risky decisions. *Proceedings of the National Academy of Sciences*, *113*(42), 11817–11822. http://doi.org/10.1073/pnas.1602641113

Kim, Y., Teylan, M. A., Baron, M., Sands, A., Nairn, A. C., & Greengard, P. (2009). Methylphenidate-induced dendritic spine formation and ΔFosB expression in nucleus accumbens. *Proceedings of the National Academy of Sciences*, *106*(8), 2915–2920. http://doi.org/10.1073/pnas.0813179106

Kuczenski, R., & Segal, D. S. (1997). Effects of methylphenidate on extracellular dopamine, serotonin, and norepinephrine: comparison with amphetamine. *Journal of Neurochemistry*, *68*(5), 2032–2037. http://doi.org/10.1046/j.1471-4159.1997.68052032.x

Levy, D. J., & Glimcher, P. W. (2011). Comparing apples and oranges: using reward-specific and reward-general subjective value representation in the brain. *The Journal of Neuroscience*, *31*(41), 14693–14707. http://doi.org/10.1523/JNEUROSCI.2218-11.2011

López, S. D., Howell, R. D., Canúl, M. G. L., Leyton, M., & Gobbi, G. (2014). Electrophysiological characterization of dopamine neuronal activity in the ventral tegmental area across the light–dark cycle. *Synapse*, *68*(10), 454–467. http://doi.org/10.1002/syn.21757

Maier, L. J., Ferris, J. A., & Winstock, A. R. (2018). Pharmacological cognitive enhancement among non-ADHD individuals—A cross-sectional study in 15 countries. *International Journal of Drug Policy*, *58*, 104–112. http://doi.org/10.1016/j.drugpo.2018.05.009

Mohr, P. N. C., Biele, G., & Heekeren, H. R. (2010). Neural processing of risk. *The Journal of Neuroscience*, *30*(19), 6613–6619. http://doi.org/10.1523/JNEUROSCI.0003-10.2010

Müller, S., & Rau, H. A. (2016). The relation of risk attitudes and other-regarding preferences. A within-subjects analysis. *European Economic Review*, *85*, 1–7. http://doi.org/10.1016/j.euroecorev.2016.02.004

Onge, J. R. S., Chiu, Y. C., & Floresco, S. B. (2010). Differential effects of dopaminergic manipulations on risky choice. *Psychopharmacology*, *211*(2), 209–221. http://doi.org/10.1007/s00213-010-1883-y

Ruff, C. C., Ugazio, G., & Fehr, E. (2013). Changing social norm compliance with noninvasive brain stimulation. *Science*, *342*, 482–484. http://doi.org/10.1126/science.1241399

Saito, K. (2013). Social preferences under risk: equality of opportunity versus equality of outcome. *American Economic Review*, *103*(7), 3084–3101. http://doi.org/10.1257/aer.103.7.3084

Sauer, J.-M., Ring, B. J., & Witcher, J. W. (2005). Clinical pharmacokinetics of atomoxetine. *Clinical Pharmacokinetics*, *44*(6), 571–590. http://doi.org/10.2165/00003088-200544060-00002

Schlösser, R. G. M., Nenadic, I., Wagner, G., Zysset, S., Koch, K., & Sauer, H. (2009). Dopaminergic modulation of brain systems subserving decision making under uncertainty: A study with fMRI and methylphenidate challenge. *Synapse*, *63*(5), 429–442. http://doi.org/10.1002/syn.20621

Smith, H. R., Beveridge, T. J. R., & Porrino, L. J. (2006). Distribution of norepinephrine transporters in the non-human primate brain. *Neuroscience*, *138*(2), 703–714. http://doi.org/10.1016/j.neuroscience.2005.11.033

Strobel, A., Zimmermann, J., Schmitz, A., Reuter, M., Lis, S., Windmann, S., & Kirsch, P. (2011). Beyond revenge: Neural and genetic bases of altruistic punishment. *NeuroImage*, *54*(1), 671–680. http://doi.org/10.1016/j.neuroimage.2010.07.051

van den Bos, W., van Dijk, E., Westenberg, M., Rombouts, S. A. R. B., & Crone, E. A. (2011). Changing brains, changing perspectives: the neurocognitive development of reciprocity. *Psychological Science*, *22*(1), 60–70. http://doi.org/10.1177/0956797610391102

Vives, M.-L., & FeldmanHall, O. (2018). Tolerance to ambiguous uncertainty predicts prosocial behavior. *Nature Communications*, *9*(1), 1–9. http://doi.org/10.1038/s41467-018-04631-9

Yang, Z. J. (2015). Altruism during Ebola: risk perception, issue salience, cultural cognition, and information processing. *Risk Analysis*, *36*(6), 1079–1089. http://doi.org/10.1111/risa.12526

Table 1

Expected value (EV), social welfare and earnings change (when choosing to help, option B) across risk levels (p) in the risk and helping/risky helping task.

		Option A		Option B			
	p	EV decider	EV receiver	EV decider	EV receiver	(expected) welfare change of option B	decider's (expected) earnings change
risk task	**1**	15	–	0	–	–	-15
	0.9	15	–	2.6	–	–	-12.4
	0.8	15	–	5.2	–	–	-9.8
	0.7	15	–	7.8	–	–	-7.2
	0.6	15	–	10.4	–	–	-4.6
	0.5	15	–	13	–	–	-2
	0.4	15	–	15.6	–	–	0.6
	0.3	15	–	18.2	–	–	3.2
	0.2	15	–	20.8	–	–	5.8
	0.1	15	–	23.4	–	–	8.4
	0	15	–	26	–	–	11
helping / risky helping task	**1**	15	0	0	0	-15	-15
	0.9	15	0	1.3	1.3	-12.4	-13.7
	0.8	15	0	2.6	2.6	-9.8	-12.4
	0.7	15	0	3.9	3.9	-7.2	-11.1
	0.6	15	0	5.2	5.2	-4.6	-9.8
	0.5	15	0	6.5	6.5	-2	-8.5
	0.4	15	0	7.8	7.8	0.6	-7.2
	0.3	15	0	9.1	9.1	3.2	-5.9
	0.2	15	0	10.4	10.4	5.8	-4.6
	0.1	15	0	11.7	11.7	8.4	-3.3
	0	15	0	13	13	11	-2

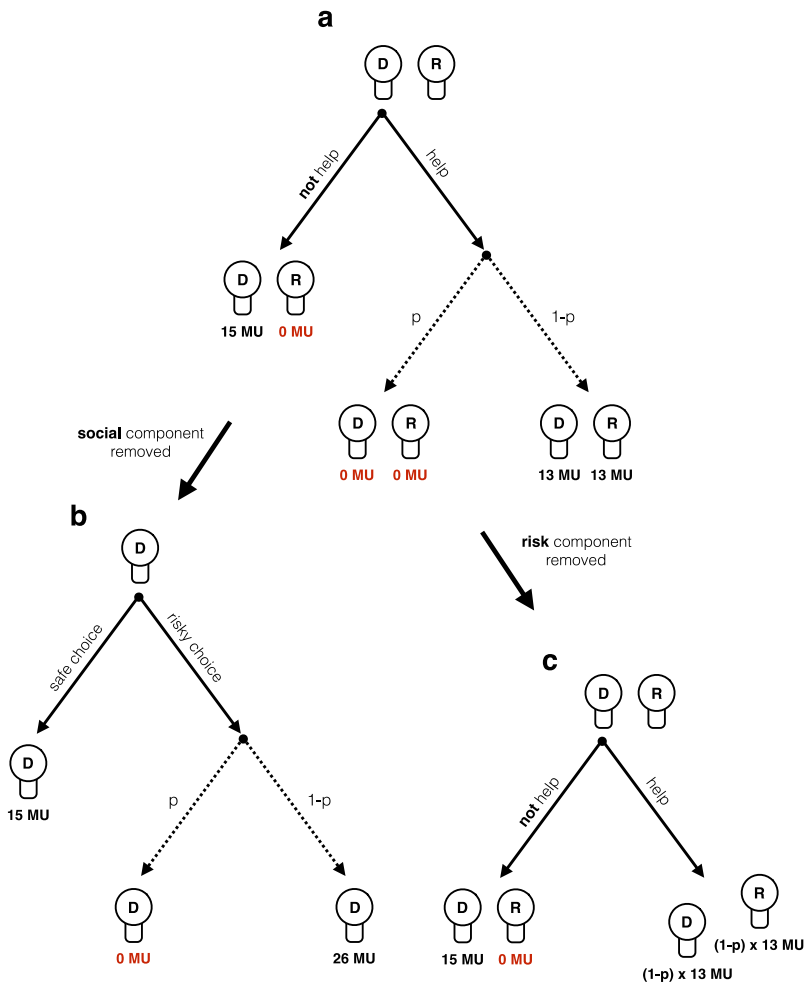

Figure 1. risky helping, risk, and helping task. In the risky helping task (**a**) the decider (D) decides whether to help or not to help the receiver (R). In case the decider decides to help, helping is unsuccessful with risk *p* and both decider and receiver earn 0 monetary units (MU). With risk 1-*p* helping is successful, leading to an equal outcome of 13 MU for both. In the risk task (**b**), the decider decides between a safe option leading to a payoff of 15 MU and a risky option, leading to either a payoff of 0 MU with risk *p* or a payoff of 26 MU with risk 1-*p*. In the risk task, the receiver is not affected by the decider's decision, thus removing the social component of the risky helping task. In the helping task (**c**), the decider decides whether to help or not to help the receiver. Across trials, helping leads to a sure outcome equal to the expected value of possible outcomes of the risky helping task, thus removing the risk component of the risky helping task. Note: in the actual task, the labels “help”, “not help”, “risky choice”, “safe choice” were replaced with neutral labels.

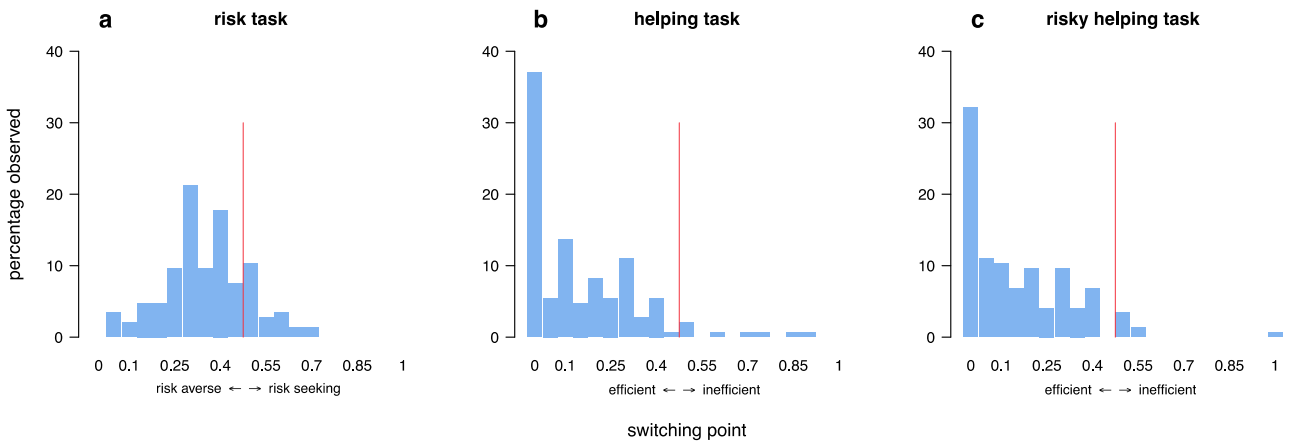

Figure 2. Switching points. Distribution of switching points across the risk (**a**), helping (**b**), and risk helping task (**c**). Red line indicates the border of risk aversion to risk seeking (risk task) and social efficiency to inefficiency (helping and risky helping task).

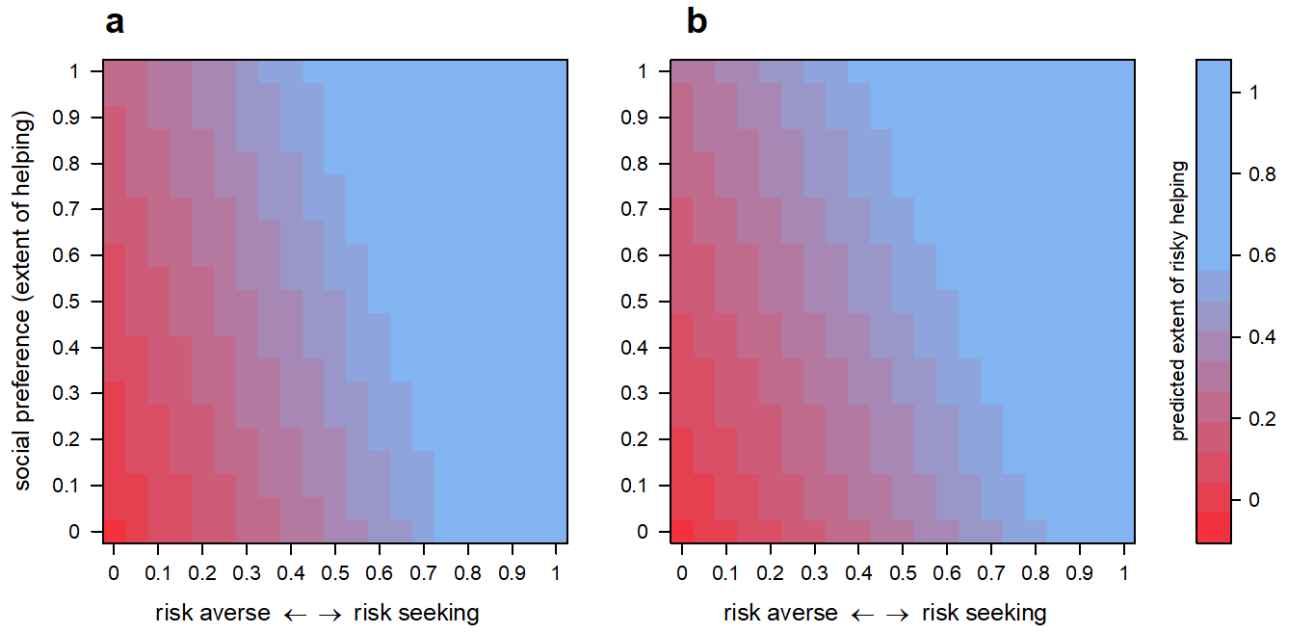

Figure 3. Predicting risky helping. Contour plot showing the predicted extent of risky helping (color surface) based on a linear combination of risk preference and social preference in Study 1 (**a**) and Study 2 (**b**). Dots show the observed risk- and social preference of deciders.

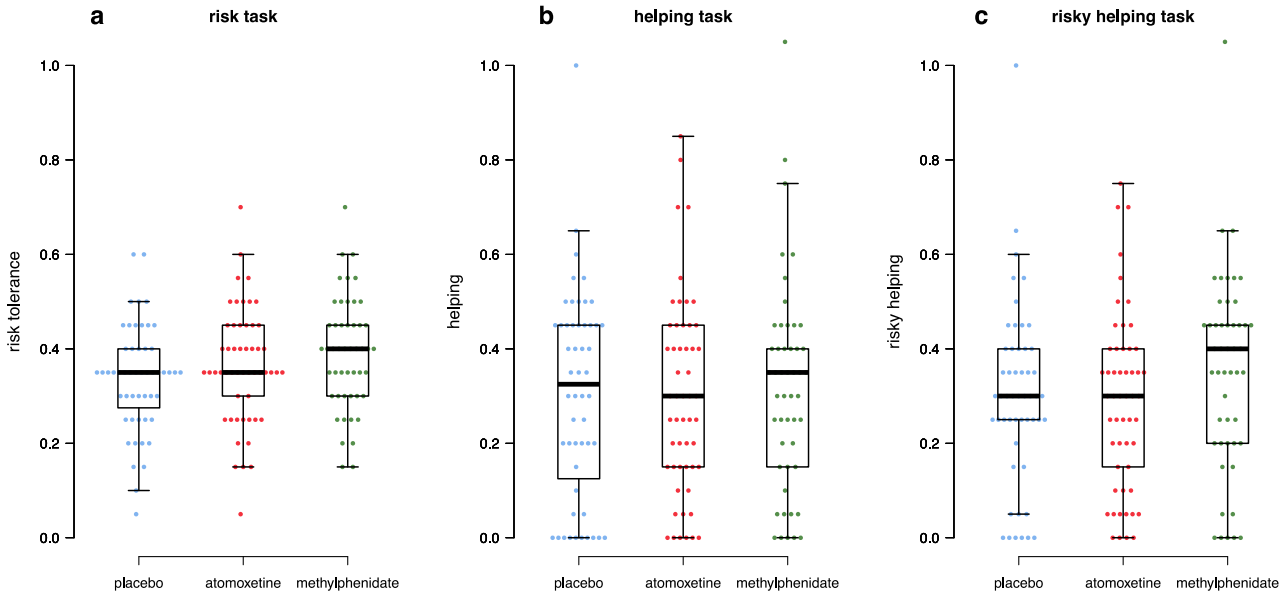

Figure 4. Preference changes across drug conditions. Boxplots showing the distribution of switching points in the risk (**a**), helping (**b**), and risky helping task (**c**). Points show individual data points (blue = placebo, red = atomoxetine, green = methylphenidate).

Supplementary Information

Regression models. As shown in Table S1 and S2, we fitted separate nested regression models to the risky helping choice data. In both data-sets a model that predicted risky helping based on a combination of risk- and social preferences fitted the data better according to Likelihood Ratio tests compared to a model that tried to predict risky helping based on risk preferences or social preferences alone (Study 1: Model 1 vs. Model 3: Likelihood Ratio test, $p < 0.007$, Model 2 vs. Model 3: Likelihood Ratio test, $p < 0.001$; Study 2: Model 1 vs. Model 3: Likelihood Ratio test, $p < 0.001$, Model 2 vs. Model 3: Likelihood Ratio test, $p < 0.001$). In Study 1, an interaction model that assumes that the slope (in other words: the relationship) between risky helping and social (risk) preferences changes as a function of risk (social) preferences even outperformed the linear combination model (Study 1: Model 3 vs. Model 4: Likelihood Ratio test, $p < 0.020$). This, however, was not the case in Study 2 (Study 2: Model 3 vs. Model 4: Likelihood Ratio test, $p = 0.123$).

Control regressions. Table S3-S5 shows the influence of the drug treatment on risk-taking, helping, risky helping behavior based on a stepwise inclusion of control variables and either fitting the model to the whole sample or a subsample of participants who guessed their drug-treatment incorrectly. To control for mood and mood changes we reduced the dimensions of the mood questionnaire using factor analysis to avoid overspecification of the model (and resulting convergence problems due to multicollinearity of mood-items). Specifically, we performed exploratory factor analyses on the pre-treatment responses, the post-treatment responses, and the difference between pre- and post-treatment responses (i.e. mood change) of the 15-item mood questionnaire. In all three cases, a three-factor structure (determined by Horn's parallel analysis

using Oblimin rotation) could parsimoniously capture the covariance-matrix of the 15-item mood items (accounting for 59%, 64%, and 59% of the variance of the pre-, post-, and mood-change responses, respectively). We labelled these three factors alertness (e.g. 'muzzy vs. clear-header', 'lethargic vs. energetic', attentive vs. dreamy'), affective valence (e.g. 'happy vs. sad', 'content vs. discontent', 'friendly vs. antagonistic'), and arousal (e.g. 'excited vs. calm', 'tense vs. relaxed', 'troubled vs. tranquil'). We either controlled for differences in mood before the experiment, after the experiment, or changes in mood (i.e. difference between pre- and post-measure; see Table S3).

Additional results. While not the main aim of this investigation, Study 1 also allowed us to test how receivers attributed helping decisions under risk based on their estimated social and risk preferences of their deciders, since they had to guess each decision of the decider. Receivers did not expect more helping with (Figure S1c, Mann Whitney U-test, $p = 0.18$) or without risk (Figure S1b, Mann Whitney U-test, $p = 0.35$, see also Figure S1). They did however, estimate the deciders to be more risk-seeking than they actually were (Figure S1a, Mann Whitney U-test, $p < 0.001$). Importantly, receivers' risky helping expectations were mainly driven by their estimated social preferences of their decider (Tobit regression, social preference estimate = 0.96, $p = 0.01$), but not by their estimated risk preferences of their decider (Tobit regression, risk preference estimate = -0.21, $p = 0.18$), or the interaction of expected risk and social preferences (Tobit regression, risk preferences × social preferences estimate = -0.19, $p = 0.80$). Put differently, receivers did not consider that both social and risk preferences condition the extent of helping under risk and instead misattributed risky helping (or the lack thereof) to social preferences alone.

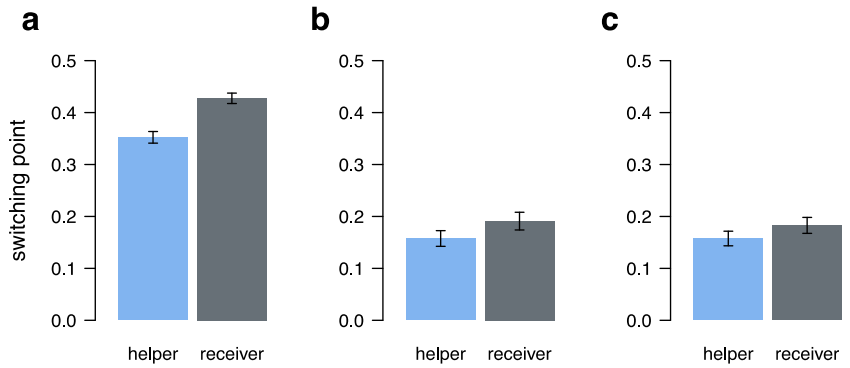

Figure S1. Receiver's expectation vs. decider's actual helping behavior. Average expected switching points of receivers (grey) versus actual switching points of deciders (blue) across (**a**) risk, (**b**) helping, and (**c**) risky helping task.

Table S1

Tobit regression models predicting risky helping based on risk and social preferences in Study 1.

	(1)	**(2)**	**(3)**	**(4)**
	B (p)	B (p)	B (p)	B (p)
Intercept	0.03 (0.550)	-0.01 (0.470)	-0.10 (0.007)	-0.04 (0.411)
risk preference	0.23 (0.110)		0.26 (0.006)	0.09 (0.448)
social preference		0.84 (< 0.001)	0.83 (< 0.001)	0.37 (0.061)
risk x social				1.28 (0.012)
AIC	77.19	-16.48	-21.81	-25.25
Log-Likelihood	-34.60	12.24	15.90	18.62

Table S2

Tobit regression models predicting risky helping based on risk and social preferences in Study 2.

	(1)	**(2)**	**(3)**	**(4)**
	B (p)	B (p)	B (p)	B (p)
Intercept	0.10 (0.003)	0.08 (< 0.001)	-0.03 (0.42)	-0.09 (0.101)
risk preference	0.66 (< 0.001)		0.31 (0.001)	0.48 (< 0.001)
social preference		0.73 (< 0.001)	0.73 (< 0.001)	0.92 (< 0.001)
risk x social				-0.55 (0.121)
AIC	-27.44	-113.19	-121.45	-121.83
Log-Likelihood	17.72	60.59	65.72	66.91

Table S3

Tobit regression models predicting risk-taking based on drug treatment, control variables, and excluding participants that guessed their drug treatment correctly.

	(1)	**(2)**	**(3)**	**(4)**	**(5)**	**(6)**	**(7)**	**(8)**	**(9)**	**(10)**
	B (p)	B (p)	B (p)	B (p)	B (p)	B (p)	B (p)	B (p)	B (p)	B (p)
Intercept (placebo)	0.36 (0.000)	0.42 (0.000)	0.36 (0.004)	0.56 (0.000)	0.35 (0.003)	0.63 (0.000)	0.28 (0.023)	0.42 (0.002)	0.33 (0.003)	0.57 (0.000)
atomoxetine	0.02 (0.295)	0.02 (0.255)	0.03 (0.173)	0.04 (0.171)	0.01 (0.542)	0.01 (0.701)	0.03 (0.199)	0.05 (0.088)	0.02 (0.277)	0.02 (0.380)
methylphenidate	**0.05 (0.018)**	**0.05 (0.015)**	**0.06 (0.006)**	**0.14 (0.000)**	**0.06 (0.012)**	**0.13 (0.000)**	**0.06 (0.009)**	**0.14 (0.000)**	**0.05 (0.025)**	**0.12 (0.000)**
age	0.00 (0.511)	0.00 (0.454)	0.00 (0.329)	**-0.01 (0.017)**	0.00 (0.163)	-0.01 (0.000)	0.00 (0.653)	0.00 (0.128)	0.00 (0.227)	**-0.01 (0.001)**
sex (1 = male)	**0.04 (0.027)**	**0.05 (0.011)**	**0.05 (0.015)**	**0.07 (0.002)**	**0.05 (0.013)**	**0.08 (0.000)**	**0.05 (0.018)**	**0.07 (0.002)**	**0.05 (0.013)**	**0.08 (0.000)**
BMI		0.00 (0.438)	0.00 (0.344)	**-0.01 (0.010)**	0.00 (0.694)	**-0.01 (0.011)**	0.00 (0.627)	-0.01 (0.051)	0.00 (0.490)	**-0.01 (0.009)**
alertness (pre)					-0.01 (0.707)	-0.02 (0.259)				
affect (pre)					0.00 (0.982)	0.03 (0.166)				
arousal (pre)					-0.01 (0.663)	-0.01 (0.348)				
alertness (post)							**0.04 (0.007)**	**0.04 (0.017)**		
affect (post)							**0.04 (0.006)**	**0.04 (0.037)**		
arousal (post)							**0.04 (0.000)**	**0.04 (0.003)**		
alertness (change)									-0.02 (0.137)	-0.02 (0.303)
affect (change)									**-0.04 (0.003)**	-0.03 (0.168)
arousal (change)									**0.03 (0.006)**	0.02 (0.145)
medication dummies incl.	no	no	yes	yes	yes	yes	yes	yes	yes	yes
correct guessers excl.	no	no	no	yes	no	yes	no	yes	no	yes

Note: BMI = Body Mass Index; medication dummies incl. = inclusion of dummy controls for contraceptive implant, contraceptive pill, ibuprofen (3/day), mefenamic acid, salbutamol; $p < 0.05$ highlighted in bold.

Table S4

Tobit regression models predicting helping based on drug treatment, control variables, and excluding participants that guessed their drug treatment correctly.

	(1)	**(2)**	**(3)**	**(4)**	**(5)**	**(6)**	**(7)**	**(8)**	**(9)**	**(10)**
	B (p)	B (p)	B (p)	B (p)	B (p)	B (p)	B (p)	B (p)	B (p)	B (p)
Intercept (placebo)	0.15 (0.227)	0.46 (0.016)	0.21 (0.415)	0.31 (0.351)	0.20 (0.382)	0.29 (0.232)	0.14 (0.598)	0.12 (0.718)	0.21 (0.456)	0.23 (0.489)
atomoxetine	0.00 (0.947)	-0.01 (0.891)	-0.02 (0.771)	0.00 (0.985)	-0.02 (0.684)	-0.02 (0.753)	0.02 (0.647)	0.03 (0.659)	0.02 (0.843)	0.03 (0.686)
methylphenidate	0.02 (0.728)	0.03 (0.484)	0.04 (0.415)	0.10 (0.141)	0.03 (0.520)	**0.15 (0.018)**	0.04 (0.371)	0.11 (0.103)	0.04 (0.449)	0.13 (0.060)
age	0.01 (0.103)	0.01 (0.061)	0.01 (0.076)	0.00 (0.575)	**0.01 (0.030)**	0.00 (0.778)	**0.01 (0.039)**	0.01 (0.221)	0.01 (0.057)	0.01 (0.254)
sex (1 = male)	**-0.10 (0.012)**	-0.08 (0.060)	-0.07 (0.137)	-0.06 (0.278)	-0.05 (0.331)	-0.02 (0.731)	-0.07 (0.074)	-0.06 (0.258)	-0.07 (0.142)	-0.07 (0.212)
BMI		**-0.02 (0.024)**	**-0.02 (0.007)**	**-0.02 (0.039)**	**-0.02 (0.006)**	-0.02 (0.076)	**-0.02 (0.007)**	-0.02 (0.071)	**-0.02 (0.009)**	-0.02 (0.116)
alertness (pre)					-0.02 (0.504)	-0.01 (0.634)				
affect (pre)					**0.06 (0.062)**	**0.10 (0.012)**				
arousal (pre)					-0.03 (0.178)	**-0.07 (0.007)**				
alertness (post)							0.05 (0.071)	**0.09 (0.021)**		
affect (post)							**0.08 (0.014)**	**0.09 (0.021)**		
arousal (post)							0.02 (0.432)	0.01 (0.789)		
alertness (change)									-0.03 (0.565)	**-0.11 (0.010)**
affect (change)									-0.03 (0.586)	-0.05 (0.253)
arousal (change)									0.01 (0.571)	0.00 (0.976)
medication dummies incl.	no	no	yes	yes	yes	yes	yes	yes	yes	yes
correct guessers excl.	no	no	no	yes	no	yes	no	yes	no	yes

Note: BMI = Body Mass Index; medication dummies incl. = inclusion of dummy controls for contraceptive implant, contraceptive pill, ibuprofen (3/day), mefenamic acid, salbutamol; $p < 0.05$ highlighted in bold.

Table S5

Tobit regression models predicting risky helping based on drug treatment, control variables, and excluding participants that guessed their drug treatment correctly.

	(1)	**(2)**	**(3)**	**(4)**	**(5)**	**(6)**	**(7)**	**(8)**	**(9)**	**(10)**
	B (p)	B (p)	B (p)	B (p)	B (p)	B (p)	B (p)	B (p)	B (p)	B (p)
Intercept (placebo)	0.19 (0.059)	0.43 (0.004)	0.26 (0.103)	0.69 (0.000)	0.27 (0.103)	1.10 (0.000)	0.19 (0.167)	0.32 (0.305)	0.20 (0.157)	0.35 (0.248)
atomoxetine	-0.01 (0.787)	-0.01 (0.819)	-0.01 (0.657)	0.00 (0.944)	-0.01 (0.684)	0.01 (0.838)	-0.02 (0.518)	0.02 (0.725)	0.00 (0.967)	0.03 (0.576)
methylphenidate	**0.07 (0.047)**	**0.08 (0.026)**	**0.10 (0.001)**	**0.15 (0.001)**	**0.10 (0.004)**	**0.15 (0.001)**	**0.08 (0.025)**	0.11 (0.075)	**0.10 (0.003)**	**0.13 (0.032)**
age	0.00 (0.260)	0.01 (0.233)	0.00 (0.466)	**-0.01 (0.048)**	0.00 (0.452)	**-0.03 (0.000)**	0.00 (0.318)	0.00 (0.612)	0.00 (0.469)	0.00 (0.441)
sex (1 = male)	0.00 (0.901)	0.02 (0.521)	0.06 (0.075)	**0.14 (0.000)**	0.05 (0.091)	**0.14 (0.002)**	0.05 (0.091)	0.05 (0.300)	0.05 (0.131)	0.04 (0.411)
BMI		**-0.01 (0.030)**	**-0.01 (0.022)**	**-0.02 (0.002)**	**-0.01 (0.026)**	-0.01 (0.070)	-0.01 (0.059)	-0.02 (0.080)	-0.01 (0.053)	-0.01 (0.167)
alertness (pre)					0.00 (0.991)	0.05 (0.126)				
affect (pre)					0.03 (0.291)	0.04 (0.242)				
arousal (pre)					-0.02 (0.210)	**-0.05 (0.000)**				
alertness (post)							0.02 (0.416)	0.06 (0.097)		
affect (post)							**0.05 (0.008)**	0.06 (0.126)		
arousal (post)							0.02 (0.154)	-0.01 (0.842)		
alertness (change)									-0.01 (0.694)	**-0.10 (0.012)**
affect (change)									-0.03 (0.157)	-0.03 (0.459)
arousal (change)									0.01 (0.594)	-0.02 (0.510)
medication dummies incl.	no	no	yes	yes	yes	yes	yes	yes	yes	yes
correct guessers excl.	no	no	no	yes	no	yes	no	yes	no	yes

Note: BMI = Body Mass Index; medication dummies incl. = inclusion of dummy controls for contraceptive implant, contraceptive pill, ibuprofen (3/day), mefenamic acid, salbutamol; $p < 0.05$ highlighted in bold.