Variants at serotonin transporter and 2A receptor genes predict cooperative behavior differentially according to presence of punishment

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Punishment of free-riding has been implicated in the evolution of cooperation in humans, and yet mechanisms for punishment avoidance remain largely uninvestigated. Individual variation in these mechanisms may stem from variation in the serotonergic system, which modulates processing of aversive stimuli. Functional serotonin gene variants have been associated with variation in the processing of aversive stimuli and widely studied as risk factors for psychiatric disorders. We show that variants at the serotonin transporter gene (*SLC6A4*) and serotonin 2A receptor gene (*HTR2A*) predict contributions to the public good in economic games, dependent upon whether contribution behavior can be punished. Participants with a variant at the serotonin transporter gene contribute more, leading to group-level differences in cooperation, but this effect dissipates in the presence of punishment. When contribution behavior can be punished, those with a variant at the serotonin 2A receptor gene contribute more than those without it. This variant also predicts a more stressful experience of the games. The diversity of institutions (including norms) that govern cooperation and punishment may create selective pressures for punishment avoidance that change rapidly across time and space. Variant-specific epigenetic regulation of these genes, as well as population-level variation in the frequencies of these variants, may facilitate adaptation to local norms of cooperation and punishment.

public goods game | collective action | behavioral plasticity | 5-HTTLPR

Punishment has likely been a strong selective force in human evolutionary history. The punishment of free-riders enables cooperation (1), which is a hallmark of human evolution. Across diverse cultures, social norms, both within and outside of the domain of cooperation, are enforced with punishment ranging from gossip to exile and even death (2). Thus, natural selection should shape cognitive and affective mechanisms that enable the internalization of norms (3), sensitivity to the probability of punishment by others for norm violation, and aversion to imagined or experienced punishment (4).

However, mechanisms for punishment avoidance remain largely uninvestigated. Variation in the serotonergic system could underlie individual variation in psychological mechanisms for avoiding punishment. Prediction of (5) and response to (6) negative outcomes and social decision-making behavior (7) can be modified via manipulation of serotonin levels. A bias toward negative stimuli (8) characterizes mood and anxiety disorders; altered regulation of the serotonergic system has long been implicated in these disorders. Processing of aversive stimuli and sensitivity to the social environment are also linked to functional serotonin gene variants. A length polymorphism (5-HTTLPR) in the promoter region of the serotonin transporter gene, *SLC6A4* [*solute carrier family 6 (neurotransmitter transporter, serotonin)*, *member 4*, also referred to as 5-HTT], predicts increased observational fear conditioning (9) and amygdala activation in the presence of threatening social cues (10). This polymorphism, as well as a polymorphism in the promoter region of the serotonin 2A receptor gene, *HTR2A* [*5-hydroxytryptamine (serotonin) receptor 2A*, *G protein-coupled*, also referred to as 5-HT_{2A}], is also associated with the personality dimension of neuroticism (11, 12), increased risk for depression and other psychiatric disorders (13, 14), and increased cortisol response to a psychosocial stressor (12, 15). Intriguingly, a recent assessment of global variation at *SLC6A4* and *HTR2A* suggests unusual evolutionary histories at these loci in humans (16). Haplotypes (i.e., the combination of linked alleles that are inherited as a unit) at these loci have a striking geographic distribution, may have been under directional selection, and are estimated to have originated or spread relatively recently (16).

We hypothesized that individuals with particular variants at *SLC6A4* and *HTR2A* are more sensitive to punishment and will thus be more cooperative than individuals without these variants when noncooperative behavior can be punished. We used a standard of experimental economics, the Public Goods Game (PGG), to investigate the effect of *SLC6A4* and *HTR2A* haplotypes on cooperative behavior in the presence and absence of punishment and assessed sensitivity to punishment via changes in affect and cortisol secretion during the PGG. In the PGG, each player privately decides how much of her money to contribute to the public good, the total of which is multiplied by a number greater than one and divided equally among players. Although everyone in the group benefits equally from contributions, an individual maximizes her payoff by keeping her money. The PGG has been used to study how contributions change when players are given the opportunity to punish each other (via fines). In the absence of punishment opportunities, contributions decline over rounds. Punishment targeted at low contributors attenuates that decline (17, 18).

One hundred eighty-four students participated in the study at Newcastle University. Participants remained in groups of four for the duration of the experiment and played 10 rounds each of two versions of the PGG. In the No Punishment game, each player received 20 tokens per round and privately decided how many tokens (integer from 0 to 20) to contribute to the group fund. The Punishment game always followed the No Punishment game. It differed in that after players' contributions and incomes for a given round were revealed, players assigned 0 to 10 negative tokens to each other player. Each negative token cost the giver one token and the recipient three tokens. Participants were paid at the end of the experiment (one token: £0.015).

Before the games, self-reported assessments of personality and depression were collected. Self-reported positive and negative affect was also assessed at five times during the experiment. DNA was extracted from buccal swabs collected at the start of

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Table 1. Fixed-effects coefficients and variance components for the best models of the number of tokens contributed to the group fund

	Model 1			Model 2			Model 3		
Parameter	Estimate	2.5%	97.5%	Estimate	2.5%	97.5%	Estimate	2.5%	97.5%
Fixed effects									
Intercept	−0.02 (0.36)	−0.7	0.674	1.01 (0.45)	0.12	1.900	0.01 (0.54)	−1.046	1.075
SH1	1.33 (0.37)	0.6	2.058				1.38 (0.39)	0.619	2.132
SH1 × Lag MCO	−0.08 (0.03)	−0.1	−0.030				−0.09 (0.03)	−0.143	−0.034
SH1 × Lag punished	−0.22 (0.08)	−0.4	−0.073				−0.23 (0.08)	−0.382	−0.081
HH1				−0.17 (0.44)	−1.03	0.700	0.02 (0.45)	−0.855	0.897
HH1 × P game				1.11 (0.36)	0.40	1.818	1.01 (0.36)	0.298	1.719
P game	1.44 (0.17)	1.1	1.769	0.58 (0.34)	−0.08	1.241	0.66 (0.34)	−0.004	1.316
Round	−0.05 (0.03)	−0.1	0.004	−0.06 (0.03)	−0.11	−0.001	−0.06 (0.03)	−0.113	−0.003
First round	7.71 (0.31)	7.1	8.325	7.69 (0.32)	7.07	8.306	7.61 (0.32)	6.995	8.232
Lag contribution	0.33 (0.02)	0.3	0.359	0.32 (0.02)	0.29	0.355	0.32 (0.02)	0.286	0.355
Lag MCO	0.56 (0.03)	0.5	0.612	0.50 (0.02)	0.46	0.543	0.56 (0.03)	0.502	0.613
Lag punished	0.24 (0.06)	0.1	0.366	0.10 (0.04)	0.02	0.177	0.25 (0.07)	0.122	0.379
Variance components									
Participant	3.14 (1.77)			3.23 (1.80)			3.27 (1.81)		
Residual	16.21 (4.03)			16.10 (4.01)			16.04 (4.01)		

Predictions from models 1–3 are plotted in Figs. 1 and 2 and Fig. S1, respectively. Parentheses contain SEs or, for the variance components, SDs of the estimates. Lag refers to the previous round. *First round*, the initial round of either game (i.e., round 1 or round 11); *Lag contribution*, the lagged contribution of ego; *Lag MCO*, the lagged mean contribution of the group, excluding ego; *Lag punished*, the lagged number of negative tokens ego received; *P game*, the punishment game.

the experiment. Two variants in each gene were genotyped, and haplotypes for *SLC6A4* and *HTR2A* were inferred for 177 and 174 participants, respectively. Haplotypes were classified as *SLC6A4* 1 (*SH1*) or *SLC6A4* 2 (*SH2*) and *HTR2A* 1 (*HH1*) or *HTR2A* 2 (*HH2*) (Table S1 and *SI Text: Haplotype Classification*).

To investigate the effects of the haplotypes on the number of tokens contributed to the group fund, we constructed a base regression model with game variables that previous studies have shown to be important predictors of contributions (17, 19) (Table 1) and varying intercepts for individuals. Analyzing each gene separately, we then introduced haplotype into the models, investigating different relationships between haplotype and phenotype as well as interactions between haplotype and the game variables. Model selection was conducted via Akaike Information Criterion (AIC) (20). Results from the best model for each gene, as well as the model that combines the best model for each gene, are presented in Table 1.

Results and Discussion

The effect of *SLC6A4* and *HTR2A* haplotypes on contribution behavior depends on the game played. *SH2* homozygotes (33.90% of participants) contributed less in the No Punishment game (Fig. 1, Table 1, Fig. S1, and Table S2). The predicted contribution for *SH2* homozygotes in round 1 is 7.65 (7.05, 8.31) tokens, compared with 8.96 (8.41, 9.43) tokens for *SH1* homozygotes and heterozygotes. (Parentheses contain 95% confidence intervals for predictions.) By the final round of the No Punishment game, predicted contributions are 1.94 (1.50, 2.40) and 4.90 (4.43, 5.30) tokens for *SH2* homozygotes and those with *SH1*, respectively. In the Punishment game, this effect was diminished. The presence of punishment opportunities stemmed the decay in contributions of *SH2* homozygotes (Fig. 1, Table 1, Fig. S1, and Table S2). This game-dependent behavior is consistent with the interpretation that *SH1* homozygotes and heterozygotes experience greater norm internalization. It is also concordant with the explanation that they are more averse to harming others, an outcome that has been experimentally influenced via manipulation of serotonin levels (7).

In the No Punishment game, the contribution behavior of *HH1* homozygotes and heterozygotes (82.76% of participants) is not discernible from that of *HH2* homozygotes (Fig. 2, Table 1, Fig. S1, and Table S2). However, in the Punishment game, participants with *HH1* contributed more than *HH2* homozygotes. For the final round of the Punishment game, the predicted contribution for those with *HH1* is 12.31 (11.86, 12.78) tokens, compared with 9.47 (8.73, 10.26) tokens for *HH2* homozygotes. The higher contributions in the Punishment game for participants with *HH1* did not depend upon the number of negative tokens received by participants in the previous round. The mere introduction of explicit punishment opportunities induced higher contributions in those

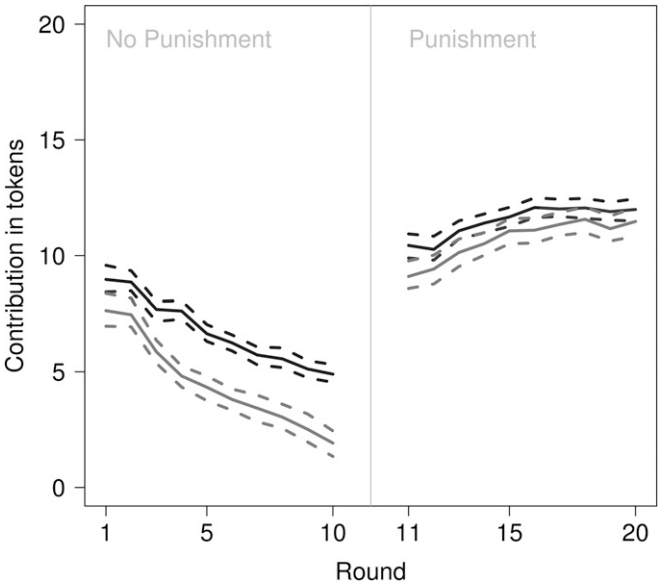

Fig. 1. Predicted contributions from model 1. *SH1* homozygotes and heterozygotes are dark gray and *SH2* homozygotes are light gray. Dotted lines illustrate 95% confidence intervals.

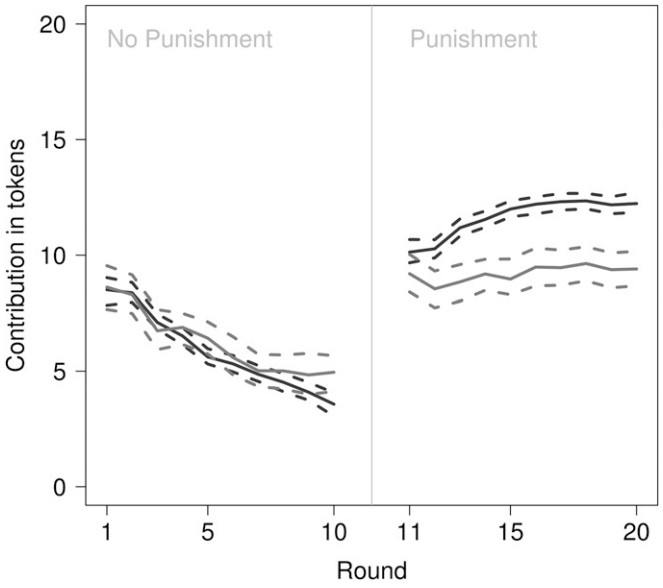

Fig. 2. Predicted contributions from model 2. *HH1* homozygotes and heterozygotes are dark gray and *HH2* homozygotes are light gray. Dotted lines illustrate 95% confidence intervals.

with *HH1* relative to those homozygous for *HH2*. This is in agreement with the interpretation that individuals with *HH1* are more averse to imagined punishment or have a higher expectation of being punished. Because the Punishment game always followed the No Punishment game, the role of serotonin and the 2A receptor in reversal learning (21) is also relevant.

In our experiment, groups were formed without prior knowledge of genetic variation. However, there are still detectable effects of the groups' haplotypic composition on contributions. Groups with three or four participants with one or two copies of *SH1* had substantially higher mean contributions compared with groups with zero to two participants with *SH1* (Fig. 3). This group-level difference was erased in the Punishment game (Fig. 3). This result complements previous work that has demonstrated the possibility for group-level outcomes to be influenced by individual variation in cooperative behavior (22, 23).

We do not observe an association between our measures of neuroticism or depression and variation at *SLC6A4* or *HTR2A*. However, individuals homozygous for *HH1* (27.01% of participants) felt worse as a result of participating in the games (Fig. S2 and Table S3). Negative affect (NA) for participants with two copies of *HH1* began to increase relative to that for other participants after the introduction of the No Punishment PGG. Predicted NA by the end of the experiment is 8.04 (7.16, 9.02) for those homozygous for *HH1* and 7.01 (6.50, 7.52) for those with one or two copies of *HH2*. Predicted NA is not, however, higher for *HH1* homozygotes before the games (Fig. S2 and Table S3). A relationship between *HH1* and a more stressful experience of the games is also indicated by higher cortisol secretion during the Punishment game for nondepressed females with *HH1* (Table S4). Predicted cortisol secretion during the Punishment game for females in the lowest quartile of the sex-specific distribution of depression scores is 140.87 nmol/L (114.11, 172.29 nmol/L) for females with *HH1*, compared with 56.74 nmol/L (34.96, 93.06 nmol/L) for females without *HH1*. Although mild, these effects demonstrate the potential psychological cost of an aversion to or expectation of punishment and are relevant to the hypothesized complex interaction of the serotonergic system, hypothalamic pituitary adrenal axis, and stress exposure in the development of depression (24, 25).

A defining characteristic of human evolutionary history is the diversity of institutions and norms that shape cooperation and punishment (18, 26). Thus, selective pressures for punishment avoidance may vary with cultural environments. For example, in a corrupt society, the probability of being punished for a violation of a law may be unpredictable and depend little on the actor's behavior. Such an outcome is similar to that observed by (18). Herrman et al. (18) demonstrated that in countries with a weak rule of law, punishment in the PGG is not strongly biased toward those who give less than the punisher, unlike in countries with a strong rule of law, in which punishment is heavily biased toward those who give less than the punisher. When punishment is highly unpredictable, the evolutionary costs and benefits of a psychology that is more averse to punishment may be altered.

However, mechanisms upon which selection may act to drive behavioral adaptation to local norms of cooperation and punishment remain largely unknown. Our results suggest that the effect of *SLC6A4* and *HTR2A* variation on cooperative behavior may vary depending upon aspects of the social context, including opportunities for behavior to be punished. Thus, substantial population-level variation in frequencies of *SH1* and *HH1*, as well as evidence of potential selection at *SLC6A4* and *HTR2A* and a very recent estimated age (19,000 ± 4,000 y ago) for *SH1* (16) are provocative. (Here, we refer to a subset of *SH1* further characterized by the derived allele at reference single nucleotide polymorphism 1042173 (rs1042173). See *SI Text*: *Robustness of Contribution Inferences*: *Characterization of haplotypes* and ref. 16.)

Of equal interest is recent molecular evidence for genotype- or haplotype-specific epigenetic regulation at *HTR2A* (27, 28) and *SLC6A4* (29, 30). These results, in concert with those from gene by environment studies (31, 32), lend increasing support to the hypothesis that, rather than conferring susceptibility to psychopathology, polymorphisms at *SLC6A4* and *HTR2A* enable increased plasticity of the serotonergic system in response to the social environment (31, 32) (*SI Text: Haplotype Classification*). This precludes a universal assumption of how *SH1*, *SH2*, *HH1*, and *HH2* affect serotonergic functioning. Moreover, it suggests an additional route by which cross-cultural variation in

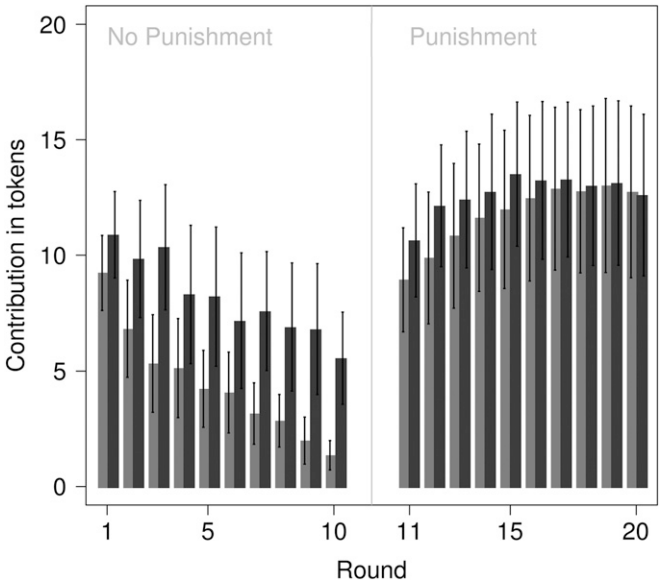

Fig. 3. Mean and 95% confidence intervals of mean contributions for groups characterized by the number of participants with *SH1*. Groups with three or four participants with *SH1* are dark gray, and those with zero, one, or two participants with *SH1* are light gray.

cooperation could be produced. Whereas the ability of cultural environments to shape selective pressures has received attention (33, 34), and has even been implicated in the global distribution of the short allele at 5-HTTLPR (35), the possible role of epigenetic regulation in this scenario has not. Genetic variation that enables behavioral plasticity could facilitate the rapid evolution of behavior in response to changing environments (36), including sanctioning institutions and other aspects of the sociocultural environment.

Methods

Experimental Sessions. Eleven sessions were conducted from November 2010 to March 2011 in computer clusters on the Newcastle University campus. The number of participants per session ranged from eight to 28. Participants were spaced such that there was either an empty computer or wall immediately adjacent to both sides of each participant. They were instructed not to communicate with each other in any way, including eye contact or body language. A purpose-built website was used to communicate all instructions to participants, administer questionnaires, and conduct the PGG.

Participants. One hundred and eighty four participants (77 males; mean age, 20.8 y) were recruited through the university psychology student mailing list, advertisements on the Web site of Newcastle University, a participant pool maintained by the Newcastle University Institute of Neuroscience, and flyers posted on campus. The study received approval from the Newcastle University Medical School Board of Ethics before commencement. All participants gave their written consent to participate in the study. Participation criteria included the following: fluency in written English, a minimum age of 18 y, and no psychiatric or steroid medications. Subjects received either a show-up fee or course credit (the latter option for psychology students only). A show-up fee of £3 was increased to £5 for the last six sessions to motivate participation.

Public Goods Game. The PGG structure used closely follows that of ref. 18. After reading instructions for the No Punishment game, participants had to correctly answer a set of questions designed to assess their understanding before proceeding. Participants were told only that they would be introduced to a different version of the game after playing the current game for 10 rounds. The marginal per capita return on the public good (the sum of tokens contributed by all group members to the project) was set at 0.4 tokens. Following the contribution stage of each round, each player was shown the contribution and income of all players in her group. Cumulative income for the game was summarized at the end of each round. Player identity could not be tracked from round to round.

Participants were then introduced to the Punishment game. They had to correctly answer questions designed to assess their understanding of the new version of the game before proceeding to 10 rounds of the Punishment game. After assigning 0 to 10 negative tokens to each player in a given round, each player saw a summary screen that included the number and cost of negative tokens given and received and income adjusted for the cost of negative tokens. Participants were immediately paid their earnings and show-up fee in cash after completing the experiment.

Genotyping. DNA was extracted from buccal samples at Newcastle University with the Isohelix DNA Isolation kit (trademarked product of Cell Projects Ltd.). Samples were genotyped by NewGene for the length polymorphism in the promoter region (5-HTTLPR) and variable number of tandem repeats in intron 2 (serotonin transporter intron 2 variable number of tandem repeats, STin2 VNTR) of *SLC6A4* as well as two single nucleotide polymorphisms (SNPs) in *HTR2A*, rs6311, and rs6313 (positions −1438 in the promoter and 102 in exon 1, respectively).

Cortisol. Participants were instructed to refrain from strenuous exercise and alcohol the day of the experiment and from having a meal or caffeine within two hours of the start of the experiment. Sessions all commenced at 1430 hours. Saliva was collected with the Sarstedt Salivette at three time points during the experiment: the beginning of the experiment (T1), 15 min after the end of the No Punishment game (T2), and 15 min after the end of the Punishment game (T3). For the first 8 min of each 15-min waiting period after the No Punishment and Punishment games, participants completed a self-assessment of positive affect (PA) and NA and then watched nature videos. The remaining final seven minutes of each waiting period were spent either reading the instructions to the Punishment game, answering questions that tested understanding of the game, and completing an additional self-assessment of positive and negative affect (T2) or being debriefed about the experiment (T3). Cortisol was assayed in duplicate for each sample at the laboratory of C. Kirschbaum (University of Dresden, Dresden, Germany). The average of each pair of measurements was calculated and used as the measurement for that sample. Salivary cortisol levels generally decreased over the duration of the experiment, possibly attributable to time of day (all experiments started at 1430 hours).

Self-Reported Assessments and Background Information. Before the PGG, participants completed a self-reported personality assessment, major depression inventory, and baseline assessment of PA and NA. Personality was assessed with a 120-item version of the International Personality Item Pool version of the NEO-PI-R.* Depression was assessed with the 10-item Major Depression Inventory (MDI) (37). PA and NA were assessed at five different times with the 10-item International Positive and Negative Affect Schedule short form (38): PA1 and NA1, beginning of the experiment; PA2 and NA2, after reading the PGG instructions (No Punishment version) and before commencing the game; PA3 and NA3, immediately after the No Punishment version of the PGG; PA4 and NA4, after reading instructions and before commencing the Punishment game; and PA5 and NA5, immediately after the Punishment game. The following information was also collected for each participant: age, sex, use of hormonal contraceptives, and "biological ancestry" (options were: Subsaharan African, Northern African, Southern European, Northern European, Eastern European, West Asian, Central Asian, East Asian, Southeast Asian, multiple origins, and no response).

Haplotype Phasing and Classifications. Haplotype phase was estimated separately for each locus with the software PHASE Version 2.1.1 (39–41). The only imputed genotype included was for one individual at rs6313. The most likely haplotype pairs for each individual as estimated by PHASE were used in downstream analyses (Table S1). Our schema for grouping haplotypes were similar to that of ref. 16, which classified haplotypes at *HTR2A* and *SLC6A4* as predicted high or low expression based on published molecular studies. Our schema differs from ref. 16 in that we do not use data on rs6312 (position −783 relative to the start of transcription) in *HTR2A*. Also, because of increasing evidence that the effect of these variants on expression is under epigenetic regulation (*SI Text: Haplotype Classification*), we note that "differential expression" may be a more appropriate description than high or low expression. *SH1* is characterized by the short allele at 5-HTTLPR and the 12-repeat allele at STin2 VNTR. *HH1* is characterized by rs6311G and rs6313C alleles (referred to in ref. 16 as −1438G and 102C). We investigate the robustness of our results to the haplotype classifications used (Fig. S3 and *SI Text: Robustness of Contribution Inferences: Characterization of haplotypes*).

Data Analysis. We analyzed the data and created all figures in the R statistical and computing environment (42–47).

Contributions. To predict contributions, we constructed a base model that includes the following game variables: *P game* (binary; whether Punishment game), *Round*, *First round* (first round of either game), *Lag contribution* (lagged contribution of ego; lagged refers to the previous round), *Lag MCO* (lagged mean contribution of group members, excluding ego), and *Lag punished* (lagged number of negative tokens received). These predictors are consistent with important predictors of contributions from previous studies (17, 19). More complex models that include interactions among these game variables were not used as they resulted in little change in predicted contributions. Random intercepts for groups were also considered but not included. For the Gaussian model, the among-group variance estimate decreases from 17.77 ($\sigma = 4.22$) to 0.39 ($\sigma = 0.63$) when the six game variables above are included in the base model. This largely results from the inclusion of *Lag contribution* and *Lag MCO*. Predicted contributions from this base model are consistent with classic PGG outcomes (e.g., ref. 17) (Fig. S1).

In assessing the effect of the *SLC6A4* and *HTR2A* on contributions, we considered three possible relationships between *SH1* or *HH1* and phenotype: dominant (Hd), recessive (Hr), and incomplete dominant (Hi). For each possible phenotype, we iterated over the base model, first including phenotype as a main effect and then interacting it with each of the control variables. Models with AIC (20) weights greater than 0.05 were combined until more

*Johnson JA, *Development of a short form of the IPIP-NEO Personality Inventory*. Second Biennial Meeting of the Association for Research in Personality, June 16–18, 2011, Riverside, CA.

complex models were not supported. The best models for each phenotype were then compared.

For *SLC6A4* Hd and Hi, the best models each include an interaction between phenotype and *Lag MCO*, as well as phenotype and *Lag punished*. The best Hd model receives far greater support than the best Hi and Hr models (predictions plotted in Fig. 1). For *HTR2A*, the best Hd and Hi models for each include an interaction between phenotype and *P game*. The *HTR2*A Hd model receives most of the support (predictions plotted in Fig. 2), although there is some support for Hi and Hr as well. The *SLC6A4* and *HTR2A* models that received the most support were then combined. Individuals with one or two copies of *SH1* are not more or less likely to have one or two copies of *HH1* ($\chi^2 = 2.29$; df = 1; $P = 0.13$). Predictions from the combined model, which receives far more support than either model alone, are plotted in Fig. S1, and the coefficients are presented in Table 1 (model 3). Henceforth, we refer to this model as the "best candidate model."

Predicted contributions were generated from samples from the posterior density of the model, assuming a multivariate normal density. This was done 100 times for each combination of round, game, and haplotype. The mean and 2.5 and 97.5 percentiles of the predicted values are plotted.

Robustness of the results to different model families, ethnic composition, sex, and haplotype classification was confirmed (Figs. S1, S3, and S4; Tables S2 and S5; and *SI Text: Robustness of Contribution Inferences*).

Neuroticism and depression. Regression analyses were used to separately assess the effect of *SLC6A4* and *HTR2A* haplotypes on neuroticism. Three specifications of the relationship between haplotype and phenotype were used for each locus: dominant, recessive, and incomplete dominant. AIC (20) was then used to select among these models for each locus. For each locus, models without any genetic information receive the least support, and models specifying a dominant relationship between *SHI* or *HH1* and phenotype receive the most support. However, the 95% confidence intervals for the coefficients for SHI and *HH1* overlap substantially with zero.

An effect of *SLC6A4* and *HTR2A* haplotypes on MDI (square root of Major Depression Inventory Score) was assessed in the same manner as neuroticism. Results were similar in that the least favored models are those that do not include any genetic information, and the models that receive the most support specify a dominant relationship between *SH1*/*HH1* for each locus and phenotype. However, as with neuroticism, the 95% confidence intervals for the coefficients for *SH1* and *HH1* overlap extensively with zero.

NA. Mixed model Poisson regression was used to assess the effect of *SLC6A4* and *HTR2A* on NA. Poisson regression was used because distributions of NA are right-skewed for all sampling periods and NA scores are discrete. Normally distributed varying intercepts for participants were included because of the repeated nature of the sampling. NA generally increased during the experiment and decreased at the final sampling period, NA5 (i.e., after the No Punishment game and at the end of the experimental session). Consistent with this observation, comparison of AIC weights indicates vastly more support for a model that specifies a parabolic relationship between sampling period and NA.

The effect of each locus on NA was explored separately. Three different specifications of the relationship between haplotype and phenotype (dominant, recessive, incomplete dominant) were considered. Interactions between haplotype and sampling period were considered as well. Model selection was conducted via AIC. For *SLC6A4*, the model specifying a main effect of *SH1*, with a recessive relationship between haplotype and phenotype, receives the greatest support. However, the 95% confidence intervals for the coefficient for *SH1* overlap considerably with zero.

For *HTR2A*, the model specifying a interaction between sampling period and *HH1*, with a recessive relationship between haplotype and phenotype, receives the greatest support. Inspection of the fixed effects coefficients for this model (Table S3) and predictions for NA from the fitted model (Fig. S2) reveals a growing trend for higher NA for *HH1* homozygotes following introduction of the No Punishment game (sampling period NA2).

Cortisol. Area under the curve with respect to ground (AUC_G) provides a measure of total cortisol secretion over a given time interval. AUC_G was calculated using equation 1 in ref. 48 for interval 1 (from T1 to T2, 15 min after the end of the No Punishment game) and for interval 2 (from T2 to T3, 15 min after the end of the Punishment game). The effect of variation at *HTR2A* and *SLC6A4* on AUC_G over intervals 1 and 2 was assessed with regression analyses. The natural logarithm of AUC_G was used in all analyses because distributions for this measurement were positively skewed. *HTR2A* and *SLC6A4* were analyzed separately, as were intervals 1 and 2. Cortisol response to a psychosocial stressor may be affected by sex, hormonal contraceptive use (49), and depression (50), and the effect of depression on cortisol reactivity may be particularly strong in the afternoon (50). Thus, a base model was constructed with the variables *Sex contraceptive* and *MDI*. *Sex contraceptive* consists of three categories: male, female, and female using hormonal contraceptive. *MDI* is the square root of the participant's score on the Major Depression Inventory. Haplotypes were added to this base model. All possible models allowing for interactions among these three variables (*Sex contraceptive*, *MDI*, and haplotype) were considered. This was done for three specifications of the relationship between haplotype and phenotype (dominant, recessive, incomplete dominant). AIC was used to select the best model for each specification of the relationship between haplotype and phenotype and then to select the best model among these three specifications.

For interval 1, the base model receives far more support than any of the models that include either *SLC6A4* or *HTR2A*. That is, there is no evidence that variation at *SLC6A4* or *HTR2A* affected total cortisol secretion during the No Punishment game. However, for interval 2, models that include *SLC6A4* or *HTR2A* outperform the base model. The best candidate models specify dominance of *SH1* and *HH1*. The best candidate model for *SLC6A4* does not specify interactions among any of the variables and indicates a negative effect of *SH1* on total cortisol secretion. Although AIC supports inclusion of *SLC6A4*, the SE of the coefficient for *SH1* is quite large relative to the point estimate and the 95% confidence interval overlaps with zero.

Comparison of the best candidate models for *SLC6A4* and *HTR2A* for total cortisol secretion over interval 2 indicates far more support for the model with *HTR2A*. The best candidate model for *HTR2A* includes a three-way interaction among *Sex contraceptive*, *MDI*, and *HH1* (Table S4). Total cortisol secretion during the Punishment game was greater for females with one or two copies of *HH1* but not for those with high scores on the Major Depression Inventory (Table S4).

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Supporting Information

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SI Text

Data Collection

There were two instances of technical malfunction that lead to error in data collection. In the first session, nine players, in three different groups, saw a phantom additional "other player" when contributions for round 1 of the No Punishment game were summarized. For these nine players, the lagged mean group contributions for round 1 of the No Punishment game were adjusted for analyses according to what they saw. Three players, one in each of the same groups, saw a phantom additional "self." For two of these players, no contribution was entered and the phantom contribution was 0. For one player, he or she reentered his/her initial contribution (10 tokens). These three players were informed by the researcher that a technical error had occurred and so their "phantom self" contributions were disregarded in all analyses.

In the Punishment game of a later session, negative tokens for four players, in four different groups, were not applied, because of a technical error (i.e., if/when other players assigned negative tokens to one of these four, the recipient did not receive the information, nor was his or her income reduced). The negative tokens these four players assigned to others remained uncorrupted and were included in analyses. The contributions of these four players were also included in calculations of the group mean contribution, but contributions these four individuals made during the Punishment game were excluded from analyses.

Data Analysis

Haplotype Classification. Our schema for grouping haplotypes is similar to that of ref. 1, which classified haplotypes at the serotonin 2A receptor gene [*5-hydroxytryptamine* (*serotonin*) *receptor 2A*, *G protein-coupled*, or *HTR2A*] and the serotonin transporter gene [*solute carrier family 6* (*neurotransmitter transporter*, *serotonin*), *member 4*, or *SLC6A4*] as predicted high or low expression based on the results of previously published molecular studies. We note that a more appropriate description may be differential expression haplotypes instead of high and low expression haplotypes. This is because of increasing evidence that the effect of these variants on expression is under epigenetic regulation. For *SLC6A4*, we characterize haplotypes that include the short allele at 5-HTTLPR and the 12-repeat allele at serotonin transporter intron 2 variable number of tandem repeats (STin2 VNTR) as *SLC6A4* 1 (*SH1*) and haplotypes with the long allele at 5-HTTLPR or the short allele at 5-HTTLPR and the 10-repeat allele at the STin2 VNTR as *SH2* (Table S1). Gene by environment studies show an association between the short allele and behavioral plasticity (2). Both the 5-HTTLPR and STin2 VNTR can regulate in vitro expression (3). Using a reporter gene assay (4) demonstrated that regulation of expression is dependent upon both the 5-HTTLPR and the STin2 VNTR alleles. The reporter gene construct with both the short allele at 5-HTTLPR and the 12-repeat allele at the STin2 VNTR supported the highest levels of activity in the absence of CCCTC-binding factor and the lowest levels of activity in its presence (4).

For *HTR2A,* we characterize haplotypes with the ancestral alleles at reference single nucleotide polymorphism 6311 (rs6311) and rs6313 (G and C) as *HTR2A* 1 (*HH1*) and those with the derived alleles at rs6311 and rs6313 (A and T) as *HH2* (Table S1). A potentially recombinant haplotype, represented by two chromosomes in our sample, with the ancestral allele at rs6311 (which is in the promoter) and the derived allele at rs6313, are included in this grouping. Gene by environment studies suggest that behavioral outcomes for individuals with the derived T allele at rs6313 are more susceptible to a positive environment (5). The derived alleles at rs6311 and rs6313 result in the loss of methylation sites (6). Methylation levels at both SNPs can affect *HTR2A* expression (6, 7). The derived allele at rs6311 also creates a transcription factor binding site, and a recent study suggests that regulation of *HTR2A* transcription is affected by a complex interaction among rs6311 genotype, promoter methylation, transcription factor binding, and cortisol levels (8).

The outcome of the reporter gene construct study cited above (4) and increased variability in expression for individuals with the C allele at rs6313 (6, 7) suggest that *SH1* and *HH1* may enable increased expression flexibility relative to *SH2* and *HH2*. However, this remains speculative until further work has been done. Below, we investigate the robustness of our results to the haplotype groupings used for *SLC6A4* and *HTR2A*.

Robustness of Contribution Inferences. ***Binomial model.*** The contribution data are discrete and censored, with contributions of multiples of five common, violating assumptions of normality. Thus, we refit the best candidate model (model 3 in Table 1) to the data, assuming a binomial distribution for the outcome variable, and checked for consistency with the Gaussian model. Coefficients for the binomial model are presented in Table S2 and are consistent with those for the Gaussian model. Inspection of a plot of predicted contributions from this model revealed little change from the predictions from the Gaussian model.

Ordered logit model. We refit the best candidate model to the data, this time modeling the number of tokens contributed as an ordinal variable, and checked for consistency with the Gaussian and binomial models. Coefficients for the ordered logit model are presented in Table S2 and are consistent with those for the Gaussian and binomial models. Inspection of a plot of predicted contributions from this model revealed little change from the predictions from the Gaussian and binomial models. Because all evidence indicates that the best candidate Gaussian model performs remarkably well despite the discrete and censored nature of the outcome variable, downstream analyses were conducted using this model as a starting point.

Sex. We assessed whether the inclusion of sex in the best candidate model altered the effect of *SH1* or *HH1* on contributions. We followed the same approach as for the genes; that is, we included *Sex* as a main effect and then iterated over the base model, interacting *Sex* with each of the six variables from the base model. Akaike Information Criterion (AIC) weights for the seven resulting models were assessed, and models with weights greater than 0.05 were combined until more complex models were not supported. The model that receives the most support includes interactions between *Sex* and *P game* and *Sex* and *Round*. The coefficients for *HH1* and *SH1* change little from those in Table 1: *SH1* = 1.323 (0.57, 2.08); *SH1* × *Lag MCO* = −0.087 (−0.14, −0.03); *SH1* × *Lag punished* = −0.231 (−0.38, −0.08); *HH1* = 0.009 (−0.86, 0.88); *HH1* × *P game* = 1.011 (0.30, 1.72).

Population structure. A number of participants in later sessions were foreign students, primarily from Asian countries. One concern is that the genetic effects on contributions that we observe in our study are actually cultural. That is, the observed variation in contribution behavior could be largely driven by differences in cultural norms within our participant pool. If these cultural norms are associated with differences in haplotype frequency, then we

could erroneously infer an effect of haplotype on contribution behavior.

Self-reported biological ancestry was collected during the experiment. One hundred twenty-one individuals self-identified as Northern European. No other category has more than 13 individuals (13 participants self-identified as East Asian), so participants were combined into the following categories: European (136), Asian (33), and African (7). Regional ancestry is unidentifiable for eight participants.

Haplotype frequencies for participants grouped by regional ancestry were estimated in PHASE. Consistent with the observations of ref. 1 haplotype frequencies at both loci vary among participants by regional ancestry. Claw et al. show that the 5-HTTLPR short allele and *SH1* further characterized by a transversion at rs1042173 are higher in frequency among individuals sampled from Asian populations compared with those sampled from European and African populations (1). There are 169 individuals for which we have *SLC6A4* data and self-reported biological ancestry. In our study, *SH1* is higher in frequency among individuals who claim Asian ancestry (0.53; $\sigma = 0.02$) than among those who claim European (0.34; $\sigma = 0.01$) or African (0.06; $\sigma = 0.04$) ancestry. There are 166 individuals for whom we have *HTR2A* data and self-reported biological ancestry. Similar to the observation of ref. 1, the frequency of *HH1* (referred to as the −1438G/102C haplotype by ref. 1) is higher among individuals in our study who claim European ancestry (0.57; $\sigma = 0.00004$) or African ancestry (0.60; $\sigma = 0.00063$) than those who claim Asian ancestry (0.47; $\sigma = 0.00011$).

To assess whether a correlation between haplotype frequency and cultural norms could underlie our results, we checked whether the observed relationship between haplotype and contribution behavior can be recovered in a subset of the data defined by regional ancestry. We fit the best candidate (Gaussian) model to two subsets of the data, European and Asian. We have *SLC6A4* and *HTR2A* data for all 33 individuals claiming Asian biological ancestry and *SLC6A4* and *HTR2A* data for 130 and 128 individuals, respectively, claiming European ancestry. Predicted contributions from this model fit to the European and Asian subsets of the data are shown in Fig. S4.

The major results from the full dataset are clearly replicated with the European subset of the data. That is, individuals with one or two copies of *SH1* contribute more in the No Punishment game (Fig. S4*A*), and individuals with one or two copies of *HH1* contribute more in the Punishment game (Fig. S4*B*). Table S5 shows that the results are replicated even with the small number of individuals in the Asian subset of the data; individuals with *SH1* contribute more in the No Punishment game, and those with *HH1* contribute more in the Punishment game. Predicted contributions are plotted in Fig. S4 *C* and *D*.

Thus, the effects of *SH1* and *HH1* on contributions persist when only participants with European ancestry are considered. Although not all of the participants with European ancestry grew up in the United Kingdom (we can confirm that 12 did not; countries of origin include France, Romania, and the United States), there is no evidence that the European subset of the data contains a substantial number of participants sharing a common biological and cultural origin outside of the United Kingdom (or a distinct biological and cultural origin within the United Kingdom), as would be required for a spurious association between haplotype and contribution behavior.

We note that although similar results are achieved when the subset of participants with Asian ancestry are considered separately, this should not be considered as a replication of the study in a culturally and biologically distinct population. Aside from the very small sample size (33 individuals), this subset of the data represents both individuals of South Asian ancestry who have grown up in the United Kingdom and individuals who recently arrived in the United Kingdom.

Characterization of haplotypes. SLC6A4. Our grouping of *SLC6A4* haplotypes (i.e., haplotypes with the short allele at 5-HTTLPR and the 10-repeat allele at STin2 VNTR are classed as *SH1* with all haplotypes with the 5-HTTLPR long allele, as shown in Table S1) is based on results from a reporter gene assay (4). However, a large number of studies have considered the 5-HTTLPR alone. In our sample, over 93% of chromosomes with the short allele at 5-HTTLPR also have the 12-repeat allele at STin2 VNTR. The effect of *SLC6A4* on contributions is similar when only 5-HTTLPR genotype is considered; participants with one or two copies of the short allele contribute more in the No Punishment game, and this difference is attenuated in the Punishment game. Fig. S3*A* shows predicted contributions for the best candidate model fit to data for 5-HTTLPR genotypes instead of haplotypes.

Claw et al. (1) observed the strongest linkage disequilibrium at *SLC6A4* for a haplotype characterized by the short allele at 5-HTTLPR, the 12-repeat allele at STin2 VNTR, and the derived allele, G, at rs1042173 [position 23966 relative to the start of transcription, exon 14 (3′ untranslated region)]. They referred to this haplotype as "S/12/G." The frequency of this haplotype varies dramatically across world regions; without even considering sub-Saharan Africa, it ranges from 10 to 75% (1). Despite its overall high frequency, this haplotype has very little background variation (1). This pattern does not appear to be explained solely by demography and may be partly attributable to directional selection (1). Moreover, the estimated most recent common ancestor of this haplotype is 19,000 ± 4,000 y ago (1).

The S/12/G haplotype corresponds to the subset of *SH1* with a G at rs1042173. We genotyped our participants for this SNP as well but have no expectation for its function and, so, for our main analyses did not differentiate participants with *SH1* on the basis of this SNP. To determine whether the result we observe with *SH1* is still apparent with the S/12/G haplotype, we fit the best candidate model to the subset of data that includes only participants of European ancestry and substituted the S/12/G haplotype for *SH1*. Twenty-four of the 136 participants in this dataset have the S/12/G haplotype. Predicted contributions are shown in Fig. S3*B*. As with the full dataset and the *SH1*, participants with the S/12/G haplotype contribute more in the No Punishment game, and this difference is attenuated with the introduction of punishment. A similar result is observed when the entire dataset is considered (i.e., all participants are included irrespective of ancestry).

HTR2A. Over 98% of chromosomes that we sampled have one of two *HTR2A* haplotypes. In most studies of *HTR2A* variation, only one of the SNPs in these haplotypes is genotyped, and complete linkage disequilibrium is assumed. We observe four haplotypes that are potential recombinants. We note that because of our grouping of these four potential recombinants (Table S1), our *HTR2A* data and results are exactly what we would have achieved had we only genotyped the commonly studied promoter polymorphism rs6311 (position −1438 relative to the start of transcription). We tried excluding data for the four individuals with potentially recombinant haplotypes. The resulting fixed effect regression coefficients [*HH1* = −0.03 (−0.93, 1.23); *HH1* × *P game* = 0.89 (0.16, 1.61)] can be compared with those for model 3 in Table 1. The effect of *HH1* on contributions in the Punishment game does not change substantially with exclusion of the four individuals with potentially recombinant haplotypes.

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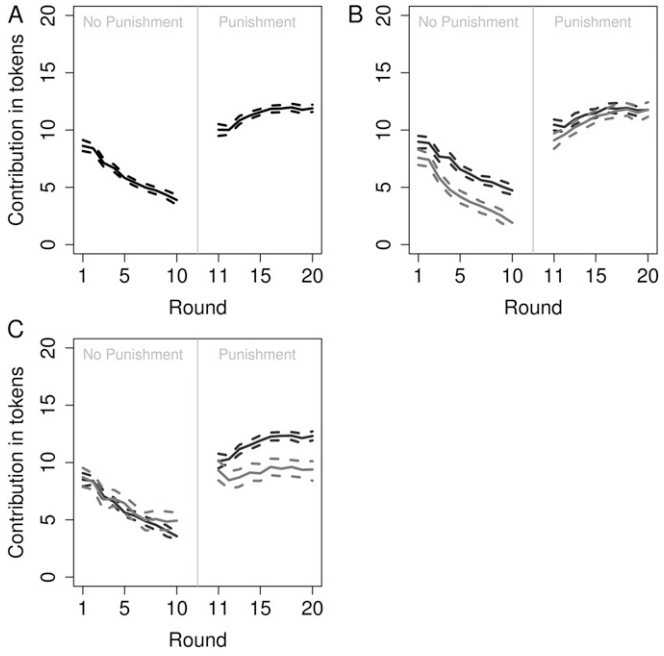

Fig. S1. Predicted contributions for Gaussian base model and best candidate model with genetic data. Dotted lines illustrate 95% confidence intervals. (*A*) Base model. (*B*) *SLC6A4*. (*C*) *HTR2A*. For *B*, *SH1* homozygotes and heterozygotes are in dark gray and *SH2* homozygotes are in light gray. For *C*, *HH1* homozygotes and heterozygotes are in dark gray and *HH2* homozygotes are in light gray.

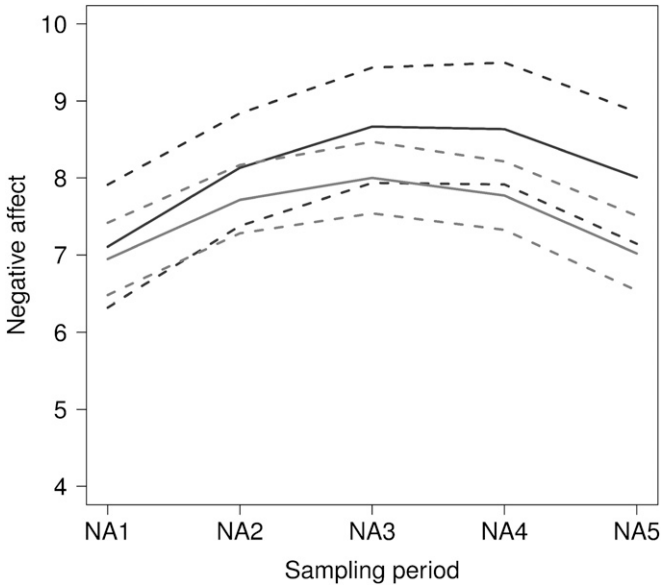

Fig. S2. Effect of *HTR2A* on NA. Model specifies a recessive relationship between *HH1* and phenotype, with an interaction between phenotype and sampling period. Dotted lines illustrate 95% confidence intervals. *HH1* homozygotes are in dark gray and heterozygotes and *HH2* homozygotes are in light gray. Sampling periods: NA1, beginning of the experiment; NA2, after reading the PGG instructions (No Punishment version) and before commencing the No Punishment game; NA3, immediately after the No Punishment game; NA4, after reading instructions and before commencing the Punishment game; and NA5, immediately after the Punishment game.

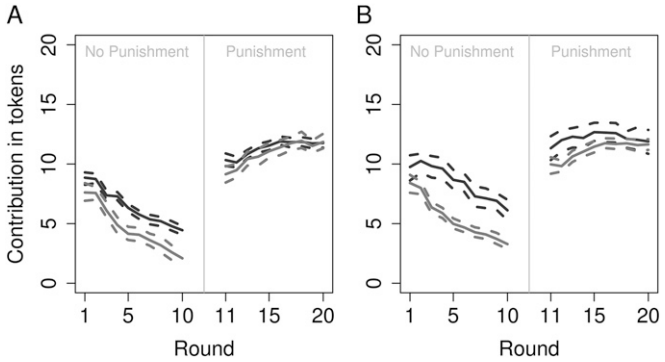

Fig. S3. For both *A* and *B*, dotted lines illustrate 95% confidence intervals. (*A*) Predicted contributions for best candidate model fit to data for 5-HTTLPR genotypes instead of *SLC6A4* haplotypes. Participants with one or two copies of the 5-HTTLPR short allele in dark gray and two copies of the long allele in light gray. (*B*) Predicted contributions for best candidate model fit to the S/12/G haplotype. Only participants of European ancestry were included. Participants with one or two copies of the S/12/G haplotype are in dark gray and those without the haplotype are in light gray.

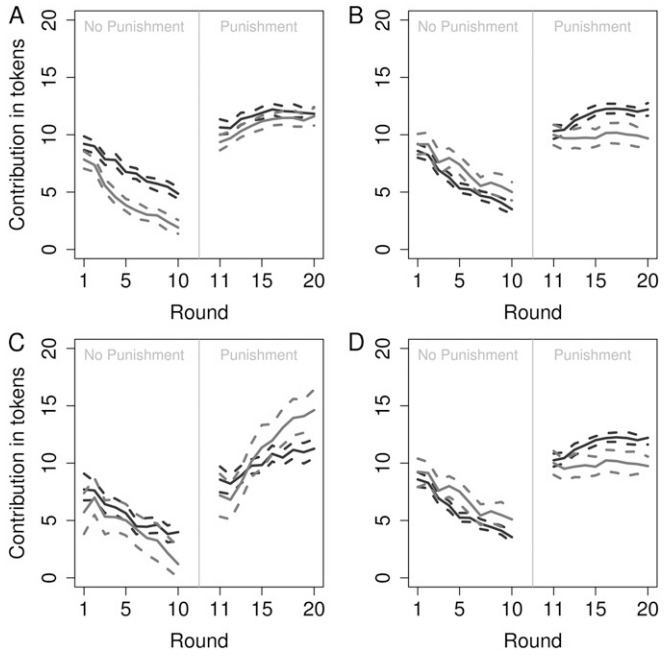

Fig. S4. Predicted contributions for best candidate model fit to subsets of the data. Dotted lines illustrate 95% confidence intervals. (*A*) *SLC6A4*, participants with European ancestry only. (*B*) *HTR2A*, participants with European ancestry only. (*C*) *SLC6A4*, participants with Asian ancestry only. (*D*) *HTR2A*, participants with Asian ancestry only. For *A* and *C*, *SH1* homozygotes and heterozygotes are in dark gray, and *SH2* homozygotes are in light gray. For *B* and *D*, *HH1* homozygotes and heterozygotes are in dark gray and *HH2* homozygotes are in light gray.

Table S1. *SLC6A4* and *HTR2A* haplotype classification and sample sizes

N	Gene	Haplotype	Differential expression haplotype	Frequency
144	*SLC6A4*	5HTTLPR-S / STin2 VNTR.12	*SH1*	0.407
24	*SLC6A4*	5HTTLPR-S / STin2 VNTR.10	*SH2*	0.068
4	*SLC6A4*	5HTTLPR-L / STin2 VNTR.9	*SH2*	0.011
95	*SLC6A4*	5HTTLPR-L / STin2 VNTR.10	*SH2*	0.268
87	*SLC6A4*	5HTTLPR-L / STin2 VNTR.12	*SH2*	0.246
189	*HTR2A*	rs6311G / rs6313C	*HH1*	0.543
2	*HTR2A*	rs6311G / rs6313T	*HH1*	0.006
155	*HTR2A*	rs6311A / rs6313T	*HH2*	0.445
2	*HTR2A*	rs6311A / rs6313C	*HH2*	0.006

Table S2. Fixed-effects regression coefficients and variance components for Gaussian, binomial, and ordered logit models of the number of tokens contributed to the group fund

	Gaussian model			Binomial model			Ordered logit model		
Parameter	Estimate	2.5%	97.5%	Estimate	2.5%	97.5%	Estimate	2.5%	97.5%
Fixed effects									
Intercept	0.01 (0.54)	−1.046	1.075	−2.85 (0.183)	−3.21	−2.49			
SH1	1.38 (0.39)	0.619	2.132	0.48 (0.132)	0.22	0.74	0.57 (0.19)	0.18	0.938
SH1 × *Lag MCO*	−0.09 (0.03)	−0.143	−0.034	−0.03 (0.005)	−0.04	−0.02	−0.04 (0.01)	−0.06	−0.009
SH1 × *Lag punished*	−0.23 (0.08)	−0.382	−0.081	−0.06 (0.011)	−0.08	−0.04	−0.11 (0.04)	−0.18	−0.035
HH1	0.02 (0.45)	−0.855	0.897	0.07 (0.162)	−0.25	0.39	0.09 (0.22)	−0.34	0.531
*HH1 * P game*	1.01 (0.36)	0.298	1.719	0.21 (0.055)	0.10	0.32	0.27 (0.17)	−0.05	0.607
P game	0.66 (0.34)	−0.004	1.316	0.37 (0.050)	0.27	0.47	0.53 (0.16)	0.22	0.831
Round	−0.06 (0.03)	−0.113	−0.003	−0.02 (0.004)	−0.03	−0.01	−0.03 (0.01)	−0.06	−0.004
First round	7.61 (0.32)	6.995	8.232	2.06 (0.045)	1.98	2.15	3.46 (0.16)	3.15	3.777
Lag contribution	0.32 (0.02)	0.286	0.355	0.07 (0.002)	0.06	0.07	0.16 (0.01)	0.14	0.174
Lag MCO	0.56 (0.03)	0.502	0.613	0.18 (0.004)	0.17	0.19	0.26 (0.02)	0.23	0.290
Lag punished	0.25 (0.07)	0.122	0.379	0.06 (0.009)	0.04	0.08	0.11 (0.03)	0.04	0.171
Variance components									
Participant	3.27 (1.81)			0.60 (0.775)			0.92 (0.07)		
Residual	16.04 (4.01)								

Based on results of the model selection process, dominant relationships between both *SH1* and *HH1* and phenotype are assumed. Parentheses contain SEs or, for the variance components and for the fixed effects estimates for the ordered logit model, SDs of the estimates. Lag refers to the previous round. *First round*, the initial round of either game (i.e., round 1 or round 11); *Lag contribution*, the lagged contribution of ego; *Lag MCO*, the lagged mean contribution of the group, excluding ego; *Lag punished*, the lagged number of negative tokens ego received; *P game*, the punishment game.

Table S3. Fixed-effects regression coefficients and variance component for the effect of *HH1* on NA

Parameter	Estimate	2.5%	97.5%
Fixed effects			
Intercept	1.754 (0.084)	1.59	1.92
Sampling period	0.240 (0.047)	0.15	0.33
HH1 Hr	0.007 (0.077)	−0.14	0.16
Sampling period^2	−0.035 (0.007)	−0.05	−0.02
Sampling period × *HH1 Hr*	−0.028 (0.019)	−0.07	0.01
Variance component			
Participant	0.057 (0.240)		

SEs (SD for the variance component) are in parentheses. *Hr*, a recessive relationship between *HH1* and phenotype.

Table S4. Regression coefficients for the effect of *HH1* on total cortisol secretion during interval 2

Parameter	Estimate	2.5%	97.5%
Intercept	5.67 (0.70)	4.30	7.04
HH1 Hd	−0.90 (0.78)	−2.43	0.63
Female	−3.48 (1.00)	−5.43	−1.53
Male	−0.82 (0.83)	−2.45	0.81
MDI	−0.14 (0.18)	−0.50	0.22
HH1 Hd × *Female*	3.68 (1.09)	1.54	5.82
HH1 Hd × *Male*	0.76 (0.92)	−1.05	2.57
HH1 Hd × *MDI*	0.16 (0.20)	−0.23	0.56
Female × *MDI*	0.79 (0.26)	0.29	1.30
Male × *MDI*	0.20 (0.23)	−0.25	0.64
HH1 Hd × *Female* × *MDI*	−0.83 (0.28)	−1.38	−0.28
HH1 Hd × *Male* × *MDI*	−0.14 (0.25)	−0.62	0.35

SEs are in parentheses. *Female*, females who were not taking hormonal contraceptives; *Hd*, a dominant relationship between *HH1* and phenotype; *MDI*, the square root of the Major Depression Inventory.

Table S5. Fixed effects coefficients and variance components for the best candidate Gaussian model of the number of tokens contributed to the group fund, fit to subsets of the data

	Model fit to subset of data*			Model fit to subset of data†		
Parameter	Estimate	2.5%	97.5%	Estimate	2.5%	97.5%
Fixed effects						
Intercept	0.53 (0.62)	−0.68	1.75	−2.41 (1.24)	−4.85	0.02
SH1	1.33 (0.44)	0.47	2.19	2.03 (0.98)	0.10	3.96
*SH1 * Lag MCO*	−0.07 (0.03)	−0.14	−0.02	−0.18 (0.08)	−0.33	−0.03
*SH1 * Lag punished*	−0.28 (0.09)	−0.45	−0.11	0.02 (0.22)	−0.41	0.44
HH1	−0.44 (0.53)	−1.48	0.60	0.69 (0.81)	−0.89	2.28
*HH1 * P game*	0.85 (0.42)	0.03	1.68	1.39 (0.69)	0.05	2.74
P game	0.81 (0.39)	0.05	1.57	0.01 (0.65)	−1.26	1.28
Round	−0.07 (0.03)	−0.14	−0.01	0.05 (0.06)	−0.07	0.17
First round	7.77 (0.36)	7.06	8.47	7.43 (0.66)	6.14	8.71
Lag contribution	0.32 (0.02)	0.28	0.36	0.39 (0.04)	0.32	0.47
Lag MCO	0.55 (0.03)	0.49	0.61	0.62 (0.08)	0.46	0.78
Lag punished	0.26 (0.07)	0.11	0.40	0.10 (0.21)	−0.30	0.51
Variance components						
Participant	3.38 (1.84)			2.32 (1.52)		
Residual	15.31 (3.91)			13.41 (3.66)		

Dominant relationships between both *SH1* and *HH1* and phenotype are assumed. Lag refers to the previous round. *First round*, the initial round of either game (i.e., round 1 or round 11); *Lag contribution*, the lagged contribution of ego; *Lag MCO*, the lagged mean contribution of the group, excluding ego; *Lag punished*, the lagged number of negative tokens ego received; *P game*, the punishment game. Parentheses contain SEs or, for the variance components, SDs of the estimates.

*Data from 128 participants who claim European ancestry.

†Data from 33 participants who claim Asian ancestry.