

# Monoamine oxidase A gene (MAOA) predicts behavioral aggression following provocation

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**Monoamine oxidase A gene (MAOA) has earned the nickname “warrior gene” because it has been linked to aggression in observational and survey-based studies. However, no controlled experimental studies have tested whether the warrior gene actually drives behavioral manifestations of these tendencies. We report an experiment, synthesizing work in psychology and behavioral economics, which demonstrates that aggression occurs with greater intensity and frequency as provocation is experimentally manipulated upwards, especially among low activity MAOA (MAOA-L) subjects. In this study, subjects paid to punish those they believed had taken money from them by administering varying amounts of unpleasantly hot (spicy) sauce to their opponent. There is some evidence of a main effect for genotype and some evidence for a gene by environment interaction, such that MAOA is less associated with the occurrence of aggression in a low provocation condition, but significantly predicts such behavior in a high provocation situation. This new evidence for genetic influences on aggression and punishment behavior complicates characterizations of humans as “altruistic” punishers and supports theories of cooperation that propose mixed strategies in the population. It also suggests important implications for the role of individual variance in genetic factors contributing to everyday behaviors and decisions.**

warrior gene | genetics | punishment | power-to-take game | hot sauce paradigm

One of the common assumptions of rational choice theory is that individuals are purely self-interested utility maximizers. However, research in economics and other social sciences has found that individual preferences can also include other-regarding factors, such as altruism, status, and fairness. In addition, individuals are often willing to incur nontrivial costs to influence others' behavior, even when such behavior can confer no direct or strategic personal benefit. In particular, humans readily try to harm others who have hurt them or their group, despite the fact that such behavior may not generate any future individual benefit (1). Because in many cases those who punish do not end up better off overall, it remains a puzzle as to why such behavior survives if it does not improve prospects for cooperation (2, 3). An additional puzzle arises in the face of cross-cultural data suggesting that individuals in some societies do not engage in costly punishment as much as those in Western industrialized societies (4, 5).

Although it varies somewhat across societies, real life cooperation and punishment behavior does not always follow the predictions of rational choice theory (4, 6). Various models have tried to address the reasons for this discrepancy (7–11). Here, we suggest a possible genetic source of individual variation in this behavior. This is the first study to investigate a genetic basis for punishment and the first to provide some evidence for a gene-environment interaction in the context of a behavioral economics experiment. Our results support previous work using financial incentives, which indicates that the experimental punishment literature may in fact reflect a broad based tendency to punish.

In this study, we examine conditions under which individuals pay money to cause physical pain to others who have taken money from them in a previous interaction. This study not only replicates previous experimental work demonstrating a willingness to engage in costly punishment, but also tests the influence of the monoamine oxidase A (MAOA) gene, which has been linked to aggression.

We draw on two separate but overlapping literatures: One from behavioral genetics, examining the effect of a genetic polymorphism on propensity for aggression; and a second from economics, addressing an individual's willingness to pay to punish others. In so doing, we combine a behavioral measure of aggression from psychology with a clear and simple economic game to investigate the conditions under which people will aggress against others despite incurring a financial cost to themselves. Previous literature suggests that one of those conditions may be individual variability in genetic alleles, such that individuals with a low activity form of the gene that encodes monoamine oxidase A (MAOA-L) will be more likely to react with aggression to challenge (12). Recent work in behavioral genetics has stressed the importance of interactions between genetic predispositions and environmental contingencies (13).

We build on an emerging literature examining genetic influences within experimental economics, including twin studies demonstrating the influence of heritability in trust game (14) and ultimatum game play (15) among a sample of both American and Swedish subjects. Although there is mounting evidence that behavior in experimental economic games has a heritable component, implying an influence from some unidentified part of the whole genome, there have been few studies explicitly testing for a relationship between economic behavior and a single gene. One recent experimental study suggests a link between a common human polymorphism in vasopressin (AVPR1a RS3) and monetary allocations in a Dictator Game (16), but there have not been any studies of genes related to aggression in such games, and none of this previous work has looked for a link between genetic and environmental interactions. Ours is the first study to look at any gene explicitly in two-player interactions, and the first to examine genetic correlates of behavioral punishment. We conduct this test examining the role of MAOA in aggressive behavior toward others in the context of an economic power-to-take game.

The MAOA gene codes for the enzyme monoamine oxidase A that plays a key role in the catabolism of neurotransmitters,

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including dopamine, norepinephrine, and serotonin (12, 17). Earlier studies found that mice with MAOA knockouts were more aggressive than their normal counterparts or mice with MAOB knockouts (which regulates different neurotransmitters) (17). A Dutch family with a repeated incidence of violent criminal behavior among males across several generations was also found to have an abnormality in MAOA (a missense mutation of a single nucleotide of the gene) (18). Recent imaging work in a large sample demonstrates that during emotional arousal, MAOA-L men show greater reactivity in the amygdala and lower activity in the regulatory prefrontal areas. Such work suggests the emotional and cognitive channels that link MAOA-L to impulsive forms of aggression (12, 19).

Among humans, a functional polymorphism in the MAOA gene can mediate the impact of traumatic early life events on the propensity to engage in violence as an adult. Specifically, children who had suffered abuse and who had the low activity form of MAOA were much more likely to develop antisocial problems as adults (20, 21). Later studies replicated the risk of low activity MAOA, in combination with traumatic early life events, for both psychiatric patients and healthy adults' predispositions toward physical aggression (22). A major gap in the literature remains, however, in that it is unclear whether self-report measures of aggression actually reflect behavioral aggression (i.e., actions rather than words).

We combine this work with a second literature drawn from economics. Our experiment also taps into the growing literature on punishment behavior, in which subjects voluntarily incur costs to punish others. A key result from experimental economics is that even where individuals are not the beneficiary of any postpunishment change in behavior (because partners are anonymous and never meet again in subsequent rounds), they often pay to punish those who violate social norms such as cooperation or reciprocity (23–27). However, the link between punishment and aggression is not always clear. Fehr and Gächter's famous experiment included post game self-reports, which suggested that punishers were motivated by anger at free-riders (1). Later replication demonstrated this same association between anger and punishment where free-riding was not allowed. This link was exacerbated with the increasing presence of monetary inequality. These findings indicate that the motivation to punish others may not derive simply from the desire to punish free-riders but also (or instead) from egalitarian motives (26). This literature in economics has not, however, examined whether individuals would be willing to harm someone *physically* in response to economic harm. Therefore, it seems important to examine the underlying causes of aggression and punishment—in themselves—given that they appear to be linked even in widely different social scenarios. Our experiment aims to do this, by examining a genetic proclivity for engaging in costly aggressive behavior in the context of environmental provocation. In short, we deploy hypotheses from behavioral genetics, by using a psychological paradigm, to address an economic puzzle. In this way, we seek to test the extent to which these differences relate to variance in MAOA allele expression.

**Methods Primer.** In 2008 we collected genetic samples from 78 male subjects, who were assigned to 2 groups: those carrying the high activity (MAOA-H) or low activity (MAOA-L) allele (*SI Text* Section S1 and [Table S1](#)). Women were excluded because of the difficulty of assigning levels of MAOA enzymatic activity in heterozygous females and to minimize potential confounding factors.

We used the “hot sauce” paradigm (28) from the psychological literature, in which subjects have the opportunity to administer unpleasantly hot (spicy) sauce to an opponent who is known to not like its taste. How much hot sauce the subject administers constitutes the behavioral measure of their aggression (*SI Text* Section S2). The punishment elicitation experiment comprised 4 rounds and had a structure similar to a “power-to-take” game (29, 30) (*SI Text* Section S3). In each round, subjects had an experimentally

manipulated portion of their earnings from a vocabulary task stripped by an anonymous — and unknown to them, fictional — person. Subjects then were given the opportunity to punish this player through the forced administration of hot sauce.

The exact procedure was repeated for 3 subsequent rounds with a (purportedly) new partner each time. Subjects were told that in each round they would have a new supply of hot sauce that they could either administer to their partner or trade in for money. After the experiment, subjects were fully debriefed.

We focus on an individual's willingness to pay to harm someone who had just taken money from them (money that they had worked to earn). The experiment manipulated the amount taken at two levels: 80% and 20%. We label the 80% decision a “high take” outcome and the 20% decision “low take”. We hypothesized that subjects who had 80% taken from them would be more likely to administer hot sauce, and administer more of it, than those who had only 20% taken.

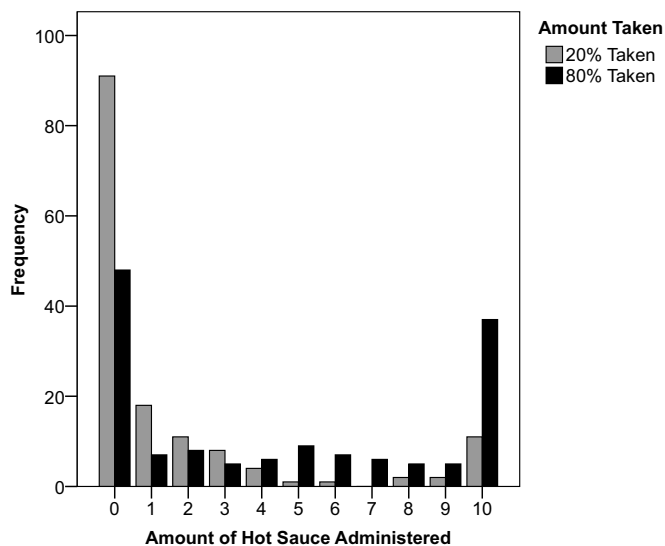
Further, if MAOA has an effect on behavior, we predict that it will depend on the nature of the stimulus. From previous brain imaging work demonstrating greater sensitivity to social rejection among MAOA-L types (31), we hypothesized that MAOA-L subjects should behave no differently than other subjects with low takes, because this represents little provocation to the individual irrespective of genotype. Conversely, MAOA-L subjects are predicted to respond more aggressively than MAOA-H subjects when 80% is taken. Thus, subjects with MAOA-L were hypothesized to behave more aggressively, but only when significantly provoked (i.e., a gene by environment interaction).

## Results

Our analysis drops 8 subjects that in a postexperiment survey expressed disbelief in whether they were actually forcing other subjects to eat hot sauce [although our results change little whether we include these subjects or not (*SI Text* Section S5 and [Table S2](#))]. We find clear support for our initial hypothesis that high take subjects behave in an aggressive manner toward their opponent more frequently, and with greater intensity, than low take ones. Because our design randomized the amount taken in each round, approximately half the people in each round had 80% of their earnings taken whereas the other half had 20% taken. Because of the nonnormal distributions of our data we use Wilcoxon rank-sum tests. We also report one-tailed test statistics in keeping with our directional hypotheses. Pooling across all 4 rounds of the experiment, subjects demonstrated higher levels of behavioral aggression when 80% was taken than when 20% was taken ( $n = 292$ ,  $Z = -6.16$ ,  $P < 0.001$ , Fig. 1). A test of proportions also revealed that subjects were more likely to try to harm their adversary by some amount (rather than nothing) when 80% was taken than 20% (66% versus 39%,  $n = 292$ ,  $Z = -4.70$ ,  $P < 0.001$ ). We also observed highly significant differences in the same direction when analyzing each of the rounds separately and when we include observations only of the *first* time someone experienced 20% or 80% taken from them (*SI Text* Section S6).

Furthermore, a battery of emotional surveys administered after the first round showed that those having 80% taken were more likely to report being ‘mad’ and ‘angry’ than those who had 20% taken (*SI Text* Section S7).

For our subsequent hypotheses we divide subjects by MAOA genotype. Fig. 2 pools observations across rounds and shows a significant difference between MAOA types when 80% was taken ( $n = 139$ ,  $Z = 2.33$ ,  $P < 0.01$ ) but no difference when 20% was taken ( $n = 141$ ,  $Z = .87$ ,  $P = 0.19$ ). The proportion of observations in which subjects meted out any aggression (rather than none) when 80% was taken was also higher among MAOA-L types (75%) versus MAOA-H types (62%) ( $n = 139$ ,  $Z = 1.40$ ,  $P = 0.08$ ). When 20% was taken there was no difference between MAOA types (40% versus 34%,  $n = 141$ ,  $Z = .71$ ,  $P = 0.24$ ). Ignoring the amount taken, MAOA-L types had higher levels of aggression ( $n = 280$ ,



**Fig. 1.** Amount of hot sauce subjects chose to administer (our measure of aggression) after having 20% (gray bars) or 80% (black bars) of their winnings taken by their (supposed) interaction partner. Wilcoxon ranksum test:  $n = 292$ ,  $Z = -6.16$ ,  $P < 0.001$ .

$Z < 1.98$ ,  $P < 0.05$ ). This suggests that the influence of genetic differences may be moderated by the environmental stimulus, in this case the amount taken.

Because our subjects participated in the task several times, in the next part of the analysis we (a) check for interference across rounds (e.g., did those who received the 80% treatment in round 1 behave differently in round 2 from those who received the 20% treatment?), and (b) analyze each round separately. First, although we did not observe any significant interference between rounds (SI Text Section S8), we also compare behavior across MAOA types for the first time someone had 80% taken and the first time someone had 20% taken (to eliminate any confound or noise arising from past experience). MAOA-L subjects were slightly more likely to be aggressive the first time 20% was taken ( $n = 67$ ,  $Z = 1.39$ ,  $P = 0.08$ ), but we see a larger, and significant, difference across MAOA types when 80% was taken ( $n = 66$ ,  $Z = 1.85$ ,  $P = 0.032$ ; see Fig. 3). This suggests a main effect influence of MAOA but this effect is

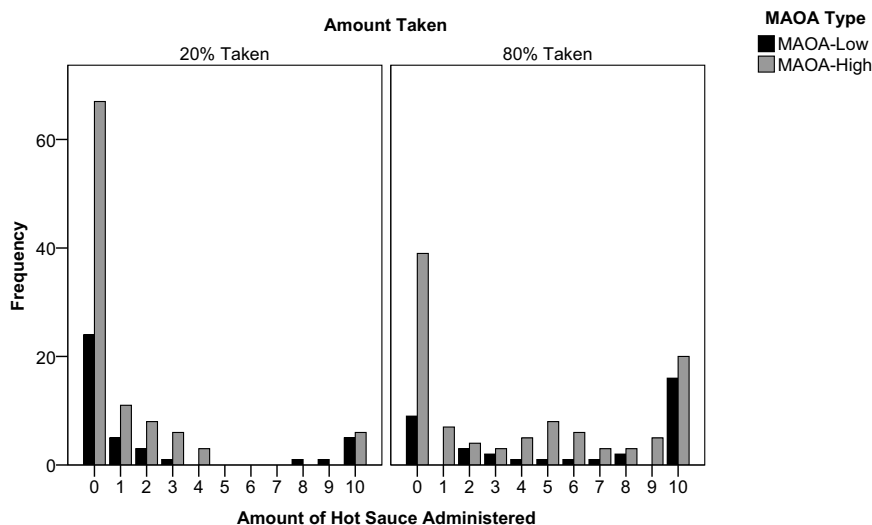
moderated by the environmental stimulus (take amount). Furthermore, if we ignore the amount taken, but directly incorporate the repeated measure nature of our design (using a repeated measure ANOVA), we find a main effect for MAOA (SI Text Section S9 and Fig. S1).

Second, we considered each round separately (Fig. 4). In round 1 there was a significant difference between high and low MAOA types among those who had 80% taken ( $n = 33$ ,  $Z = 3.09$ ,  $P < 0.001$ ), whereas there was no discernible difference in genetic types between those who had 20% taken ( $n = 37$ ,  $Z = .58$ ,  $P = 0.28$ ). Rounds 2 and 3 show no statistical difference and round 4 shows a slight difference (see Fig. 4 legend for full statistics).

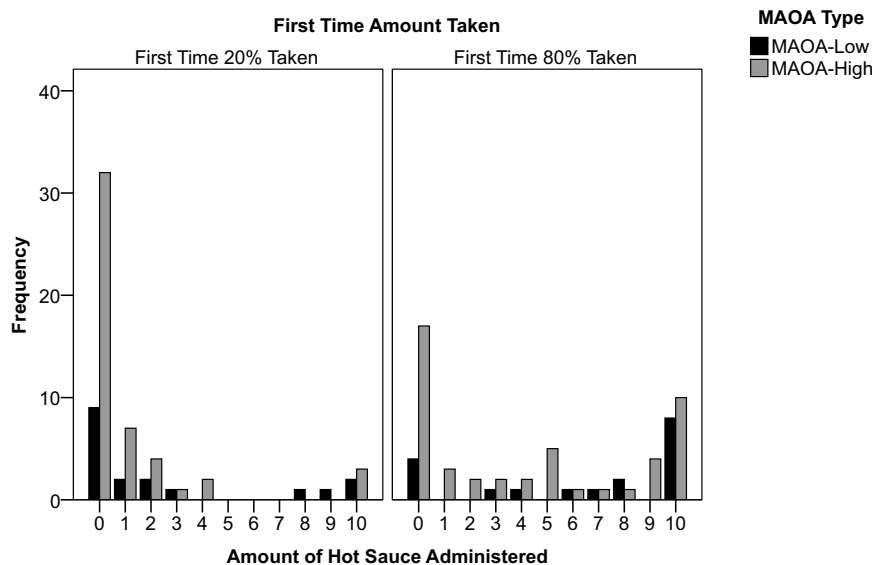
In our experiment we bounded the amount of hot sauce that someone could use to try to hurt their partner. In reality, subjects may have wished to give up more points and behave even more aggressively toward their partner with a larger amount of hot sauce. Thus, our data are artificially censored from above by our experimental design.

To address this we compare the proportion of observations that administered the maximal amount by MAOA and experimental treatment (i.e., subjects that used all possible \$3 to punish). When 80% was taken, 44% of observations from MAOA-L administered the maximal amount of hot sauce, whereas only 19% of observations from MAOA-H types did ( $n = 139$ ,  $Z = 2.95$ ,  $P < 0.01$ ). When subjects had 20% taken from them, 12% of observations from MAOA-L types and 6% from MAOA-H types administered the maximal amount ( $n = 141$ ,  $Z = 1.31$ ,  $P = 0.1$ ). We obtain similar results if we look at the first time someone had 80% or 20% taken. We also obtain similar results if we examine rates of aggression depending on whether more or less than half the amount of hot sauce was assigned. The message from this exercise is that comparing those who administered the highest amount of hot sauce to those who administered less than this amount reveals the same patterns as our other analyses above.

Finally, Table 1 reports results from a tobit regression model to account for this censoring. Our dependent variable is the amount of behavioral aggression. We include a dummy variable for whether 80% was taken ( $80\%Take$ ), a variable equal to one if the subject had the low activity form of the gene ( $MAOA-L$ ), and an interaction between these variables ( $MAOA-L \times 80\%Take$ ). In the results that follow, we estimate models for each round separately, and when pooling all observations. In the first round the interaction term is positive and highly significant ( $t = 3.51$ ,  $P < 0.001$ ). For the second round the interaction term is negative ( $t = -1.39$ ,  $P = 0.085$ )



**Fig. 2.** Amount of hot sauce administered by high activity MAOA subjects (gray bars) and low activity MAOA subjects (black bars), after having 20% (Left;  $n = 141$ ,  $Z = .865$ ,  $P = 0.19$ ) or 80% (Right;  $n = 139$ ,  $Z = 2.33$ ,  $P < 0.01$ ) of their winnings taken by their (supposed) interaction partner.



**Fig. 3.** Amount of hot sauce administered by high activity MAOA subjects (gray bars) and low activity MAOA subjects (black bars), the *first time* they experienced having 20% (Left;  $n = 67$ ,  $Z = 1.39$ ,  $P = 0.081$ ) or the *first time* they experienced having 80% (Right;  $n = 66$ ,  $Z = 1.85$ ,  $P = 0.032$ ) of their winnings taken by their (supposed) interaction partner.

whereas it is positive in the third ( $t = 1.14$ ,  $P = 0.12$ ) and fourth ( $t = .87$ ,  $P = 0.19$ ) rounds. Recall that round 1 offers the best test of these relationships, because the first reactions remain uncontaminated by any other possible effects as the experiment proceeds. When we pool observations the interaction term is positive and approaches statistical significance at the .05 level ( $t = 1.37$ ,  $P = 0.08$ ). Incorporating the censoring created by our design, we see that aggression is generally increasing in the interaction between MAOA type and amount taken and this is most apparent in the first round.

### Discussion

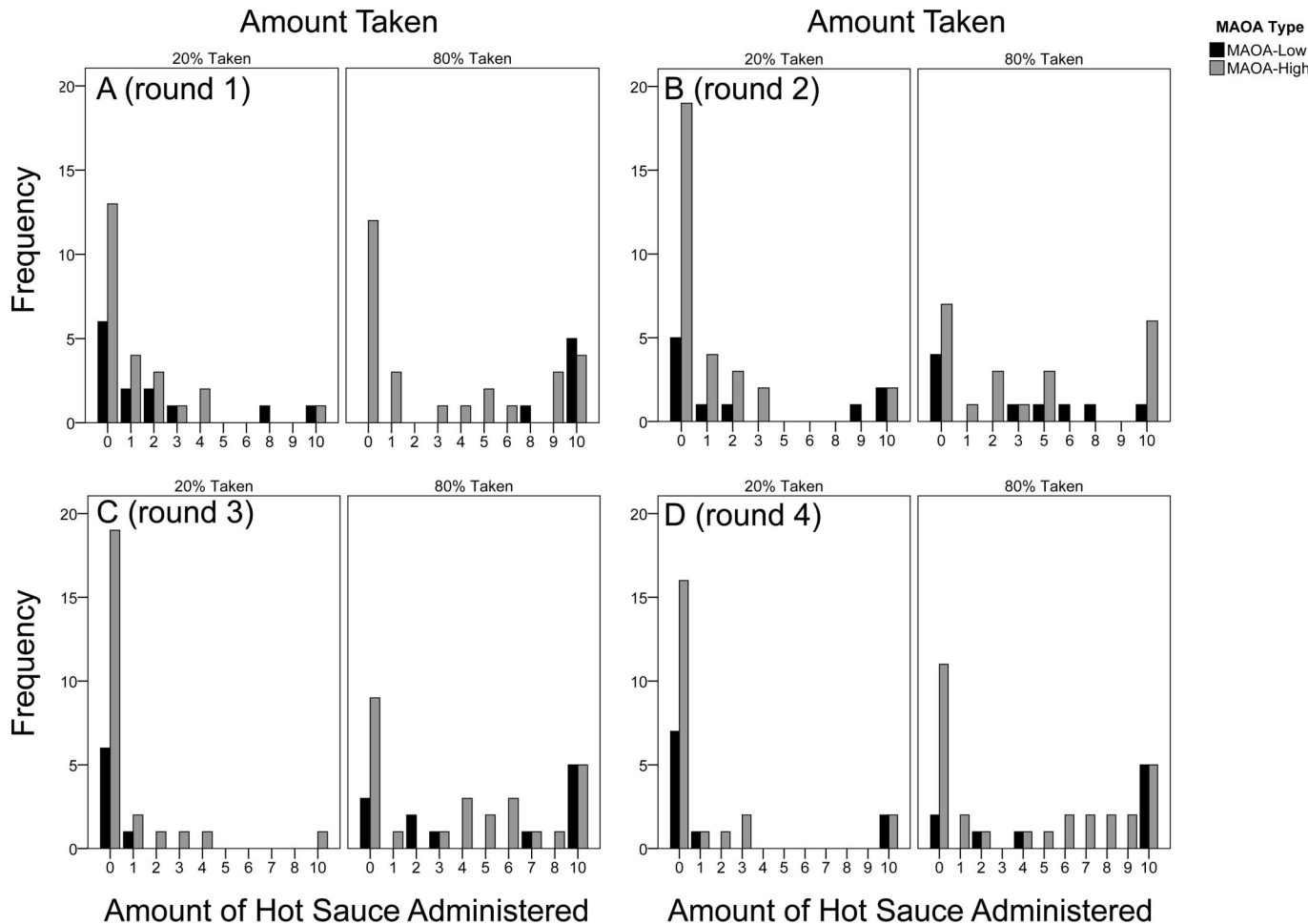
In this study, we applied a behavioral measure of aggression—the willingness of subjects to pay to administer hot sauce to someone they believed had taken money from them—within the context of a simple economic power-to-take game. We take their allocation of hot sauce to their opponent as a behavioral measure of aggression in reaction to the challenge of having money taken from them. Because of previous genotyping of subjects, we were able to investigate the relationship between genetic variance in our subject population and their willingness to engage in physical aggression toward another.

Subjects who had 80% of their money taken were more likely to aggress against the person responsible for their loss, and proved significantly more likely to administer hot sauce, and more of it, to their purported opponent than those who had 20% of their money taken. We also find evidence for a gene by environment interaction, such that individuals with the low activity form of MAOA proved more likely to administer hot sauce to their opponent when 80% of their earnings were taken than those with the more active version of the gene. There were lower differences between genetic groups when only 20% of subjects' money was taken, demonstrating an interaction between the degree of threat or challenge and aggressive response. We note, however, that overall smaller punishment rates when 20% was taken and our smaller proportion of MAOA-L subjects mean that our statistical tests in this treatment have lower power. For present purposes we felt it better to probe both a main effect difference and an interactive effect by varying the take amount. Experimentalists with similar research interests as ours might consider designs using collected genetic information and treatment assignment probabilities that optimize efficiency and

power, especially in experiments including treatments with low hypothesized effect sizes. This can be of particular importance for studies of MAOA given that the low activity form is carried by only  $\approx 1/3$  of the population in Western societies (32). Future studies that vary the size of the affront in a more fine grained manner (not just 20% or 80%) might also better calibrate the functional relationship between provocation and aggression across genetic populations, and better test hypotheses about the gene-environment interaction we consider.

This behavioral demonstration of the impact of MAOA-L on aggression documents its activity beyond previous survey results. Specifically, it expands on previous work in behavioral genetics which found a relationship between MAOA-L and self-reported aggression (20–22) by providing a clear demonstration of the relationship between MAOA-L and actual behavioral aggression (and in controlled experimental conditions). In so doing, this experiment suggests a potential genetic contribution to the findings from behavioral economics demonstrating individuals' willingness to pay to behave aggressively toward their opponent (23, 26, 27).

Although our results suggest MAOA plays a role in aggression, a major question remains as to how and why individual genetic differences cause different behavioral outcomes. In other words, what might be the underlying *psychological* phenomena at work? In a previous study, Eisenberger *et al.* showed how MAOA related to a negative socio-emotional experience (31). Although MAOA-L individuals are more aggressive, the psychological mechanisms by which this occurs have been unclear. They may be more aggressive because they are *hyposensitive*, and care less about harming others, or because they are *hypersensitive* and overreact. Eisenberger *et al.*'s research examined the relationship between MAOA and trait forms of both aggression and interpersonal hypersensitivity, and neural responses in brain areas associated with rejection-related distress. They found that individuals with MAOA-L demonstrated higher trait aggression and higher trait interpersonal hypersensitivity than those with MAOA-H. In addition, such MAOA-L individuals showed greater activity in the dorsal anterior cingulate cortex (dACC), an area that has been associated with distress related to rejection or status challenges. Because the relationship between MAOA and aggression was mediated by the dACC activity, the authors suggest that MAOA might produce aggression



**Fig. 4.** Amount of hot sauce administered by high activity MAOA subjects (gray bars) and low activity MAOA subjects (black bars) for each of the 4 rounds of the experiment (A–D). Each round had subjects who faced the 20% take treatment (Left), or faced the 80% take (Right). Wilcoxon tests of aggression by MAOA type: Round 1: 20% ( $n = 37$ ,  $Z = .58$ ,  $P = 0.28$ ), 80% ( $n = 33$ ,  $Z = 3.09$ ,  $P < 0.001$ ); Round 2: 20% ( $n = 40$ ,  $Z = 1.03$ ,  $P = 0.153$ ), 80% ( $n = 30$ ,  $Z = -.35$ ,  $P = 0.637$ ); Round 3: 20% ( $n = 32$ ,  $Z = -.66$ ,  $P = 0.746$ ), 80% ( $n = 38$ ,  $Z = .81$ ,  $P = 0.2$ ); Round 4: 20% ( $n = 32$ ,  $Z = .23$ ,  $P = 0.41$ ) 80% ( $n = 38$ ,  $Z = 1.45$ ,  $P = 0.073$ ).

through heightened, rather than reduced, sensitivity to social rejection.

Our findings confirm a role for MAOA genotype in response to provocation, and in particular extend this link to aggressive behavior in response to financial loss. The demonstration of a gene-environment interaction also helps establish the importance of examining genetic variance within particular ecologically valid contexts. Replication across additional populations and within diverse environmental contexts now appears warranted.

A final remaining question involves the evolutionary implications of these results. Subjects proved willing to incur private financial cost to punish others in an actual physical way, even

when such actions did not provide any return on their investment. This suggests that a primary puzzle of human economic behavior is “spite” (behavior that is costly to self and others), not “altruistic punishment” (costly to self but beneficial to others) (33). Indeed, recent behavioral games suggest that, since spite is costly, winners do not punish (2). Although spite has been the “neglected ugly sister of altruism” (34), there is good reason to expect it may have played a significant role in the evolution of social behavior.

The influence of genotypic variation among individuals also complicates the notion that humans are “altruistic” punishers because it raises questions about whether one behavioral strat-

**Table 1. Effect of MAO-L and take interaction**

	Rnd1	Rnd2	Rnd3	Rnd4	AllRnds
80%Take	2.315 [1.005]	3.291 [1.177]	3.620 [1.050]	3.151 [1.312]	3.101 [0.583]
MAOA-L	0.833 [1.231]	2.249 [1.503]	-0.746 [1.589]	1.004 [1.773]	0.978 [0.775]
MAOA-L × 80%Take	8.922 [2.544]	-3.086 [2.227]	2.372 [2.081]	2.250 [2.585]	1.567 [1.140]
Constant	1.341 [0.729]	1.297 [0.745]	0.889 [0.745]	1.481 [0.985]	1.247 [0.412]
Sigma constant	3.563 [0.340]	4.067 [0.395]	3.716 [0.360]	4.594 [0.462]	4.124 [0.202]
Observations	70	70	70	70	280

Tobit regression for each round of the experiment and pooling across all rounds. Dependent variable: punishment amount (censoring at 10). SE in brackets.

egy is really common to a majority of people. Models of the evolution of cooperation might usefully be revisited with this in mind, especially because recent game theoretic treatments find that punishment may evolve in some subsections of the population but not others (raising the possibility of frequency dependence or mixed strategies) (8). Our study suggests that there may be genetic bases for such a hypothesis. Indeed, our results beg the question of why the MAOA-L allele has been maintained in the population [typically 1/3 in western populations, although approaching 2/3 are reported in Maori populations (32)] if it promotes aggressive behavior. There is some evidence of an underlying positive selection sweep in human population structures in some areas (32). One possibility for why MAOA-L has not become universal lies in frequency dependent selection; if everyone were MAOA-L, its advantages would disappear. If everyone were MAOA-H, there may be a niche for more aggressive individuals to exploit (8). Another possibility is that genetic variation is preserved because it is linked to other genes or has a mix of positive and negative characteristics. Finally, genetic differences in aggression may be an example of the adaptive logic of “moralistic aggression” in promoting effective reciprocal bargaining or cooperative relationships (35, 36). All of these hypotheses are ripe for investigation.

## Methods

Genotype frequencies among our group of college subjects (27% MAOA-L) did not significantly deviate from those reported from other western populations (20, 22) (*SI Text* Section S1). Basic demographic breakdowns are given in *SI Text* Section S10.

We asked subjects whether they wanted to first try the hot sauce themselves. We inquired as to whether subjects tasted and liked the hot sauce. There was no significant difference in behavior based on those who liked and did not like hot sauce (*SI Text* Section S11).

In each round, subjects were told they were being paired up with an anonymous person in a separate laboratory located across campus but connected through the internet. Then subjects took a quiz where they completed five multiple choice vocabulary questions. Subjects were told that each vocabulary question they answered correctly was worth 20 points, with an

exchange rate of 10 points to 1 US dollar. After submitting their answers they were told how many points they had earned (\$10 maximum). In reality, we fixed the number of points earned in each round (to either 100 or 80) to allow across subject control in subsequent rounds. Such control is desirable both because different earnings might have translated into substantial differences in the amount of money taken, and because people may make their decisions in response to the amount taken, or the percentages taken. Thus, within any given round, subjects earned the same amount, but we varied this amount across rounds to ensure realism. Subjects were then forced to wait a small, randomly determined period during which their partner “decided” whether they would take 0%, 20%, or 80% of the money earned by the subject. In reality, each subject was paired up with a computer that randomly chose to take either 20% or 80%. Although deception is forbidden in many economic laboratories, it remains a common and effective (often essential) methodology in experimental psychology (37). In this case, we had to deceive subjects because ethical considerations ruled out inflicting potential pain on subjects by making them actually ingest hot sauce. Because we knew from previous work that subjects respond differently to people than to computers, we needed to deceive them about the true nature of their opponent (27). Our subjects were told that their partners had also completed a similar vocabulary task for money. Subjects then were shown a screen telling them the percentage taken by the other player and given the opportunity to behave aggressively toward the other person who had just taken their earnings.

Subjects were told that they had been given a fixed endowment of 10 1/8 teaspoon doses of hot sauce. They could choose to force the other person to drink an amount of the hot sauce, or trade the hot sauce in for money, at a rate of 3 points per dose and 10 points to a dollar (i.e., they could keep a total of \$3 or use some or all of it to hurt their partner; they could not use their prior earnings to buy or administer extra hot sauce). They were told that the other person would have to drink the hot sauce to keep the money they took. Otherwise, the stolen points were returned to the experimenters. In either case, the subject would lose the points taken by their partner. We told subjects their partner’s supposed rating of how much they liked the hot sauce. This value was fixed for all subjects within a round to a low level (either 1, 2, or 3 of 10) of enjoyment (varied for realism). We found no difference in behavior as a function of partner rating across rounds (*SI Text* Section S4).

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