

How learning shapes the empathic brain

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Deficits in empathy enhance conflicts and human suffering. Thus, it is crucial to understand how empathy can be learned and how learning experiences shape empathy-related processes in the human brain. As a model of empathy deficits, we used the well-established suppression of empathy-related brain responses for the suffering of out-groups and tested whether and how out-group empathy is boosted by a learning intervention. During this intervention, participants received costly help equally often from an out-group member (experimental group) or an in-group member (control group). We show that receiving help from an out-group member elicits a classical learning signal (prediction error) in the anterior insular cortex. This signal in turn predicts a subsequent increase of empathy for a different out-group member (generalization). The enhancement of empathy-related insula responses by the neural prediction error signal was mediated by an establishment of positive emotions toward the out-group member. Finally, we show that surprisingly few positive learning experiences are sufficient to increase empathy. Our results specify the neural and psychological mechanisms through which learning interacts with empathy, and thus provide a neurobiological account for the plasticity of empathic reactions.

empathy | in-group | learning | prediction error | fMRI

Empathy deficits have detrimental social effects (1). When they concern out-groups, these deficits are particularly pervasive (2) and expressed in brain regions that are related to empathy processing (3–9), for example the anterior insular cortex (AI) (7–9). Given the multicultural nature of our societies, scholars from various disciplines have aimed to increase empathy for out-groups. They report evidence that positive intergroup contact, for example broadcasted in a radio drama (10) or experienced in an intergroup workshop (11), can increase empathy for out-group members. However, the mechanisms underlying such changes in empathy are poorly understood, which impedes the development of principled interventions to foster empathy.

Based on the finding that positive intergroup contact is beneficial, one can plausibly assume that increases in empathy toward another person can be achieved via the establishment of positive associations with that person (12). The mechanisms that drive the establishment of positive associations have been heavily studied in the domain of learning theory (13–18). A learning-theoretical framework predicts that the establishment of positive associations toward a person is most efficient, if the actions of that person result in unexpected positive outcomes. This is because unexpected positive outcomes elicit “positive prediction errors” (18–20), that is, a large difference between the learner’s prior (low) expectation and the positivity of the actual outcome. Based on this rationale, empathy for a person is learned if the person’s actions elicit a prediction error signal (i.e., yields an unexpected positive outcome) that drives the establishment of positive associations. This increased positivity toward the other person, in turn, should raise empathy (12).

These assumptions provide a clear and testable mechanism how learning can increase empathy. However, so far little is known about the interplay between classical learning mechanisms and empathic processes, and how learning experiences shape empathy-related processes in the human brain. Here, we use functional MRI

(fMRI), combined with formal learning theory and an intergroup conflict paradigm, to investigate whether and how classical learning mechanisms alter the empathy of males toward out-group members.

Rationale

Our study consisted of three parts, a preintervention part, a learning intervention, and a postintervention part. To investigate the interaction between learning and empathy, we exploited an ecologically valid intergroup conflict in our country (Switzerland). During all three parts of the study, the Swiss participants were paired with individuals of Swiss descent (in-group members) and individuals of Balkan descent (out-group members). The latter form a large minority in Switzerland whose presence is often portrayed as problematic.

The learning intervention was based on the principles of negative reinforcement, i.e., learning that arises from the absence of an expected negative outcome. The participant expected to receive painful shocks. However, he knew that one of the other individuals in the scanner room could give up money to save him from pain (Fig. 1). The name of the potential helper was revealed just before the intervention started, and was a typical Balkan name in the experimental, and a typical Swiss name in the control group. Apart from these differences in names, the intervention was identical in both treatment groups.

To measure empathy, we assessed participants’ brain responses while they were observing pain in the in-group and in the out-group member, which is a well-established procedure for assessing neural activation related to empathy for pain (9, 21). Pain stimulation on the back of the in-group or out-group member’s hand was indicated by visual cues (*Methods*). Importantly, before and after the intervention, the in-group and the out-group members

Significance

Deficits in empathy for out-group members are pervasive, with negative societal impact. It is therefore important to ascertain whether empathy toward out-groups can be learned and how learning experiences change empathy-related brain responses. We used a learning intervention during which participants experienced help from a member of their own social group or of a generally depreciated out-group. Our results show that the intervention successfully increased empathy-related brain responses toward the out-group. These changes in out-group empathy were triggered by the learning signal (prediction error) elicited during the first two positive out-group experiences. Together, our results show that classical learning signals update empathic brain responses and that surprisingly few positive experiences with an out-group member are sufficient to increase out-group empathy.

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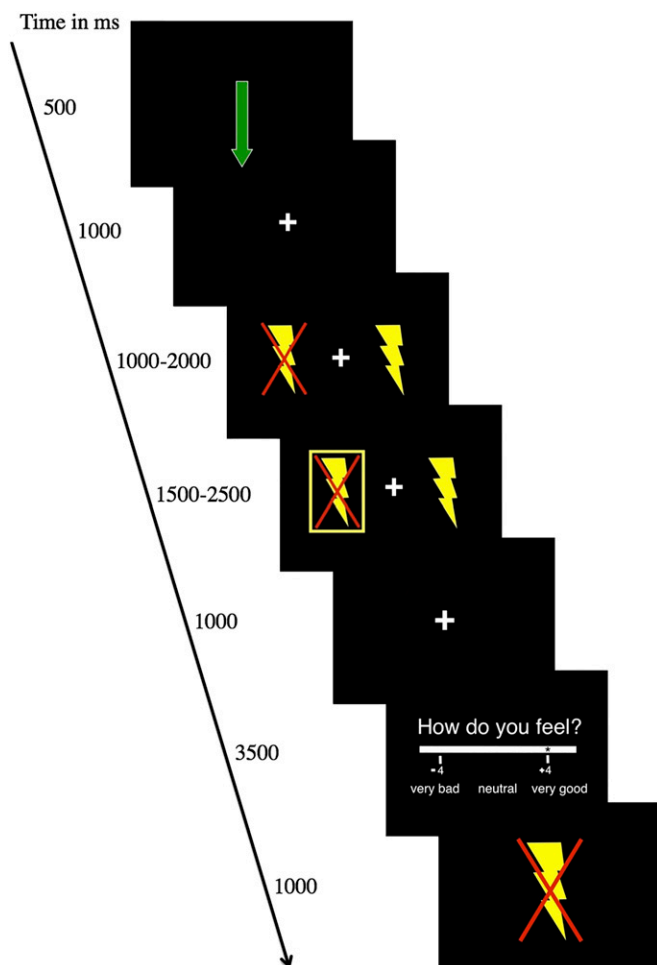


Fig. 1. Example trial of the learning intervention. The arrow cue indicated painful stimulation for the participant. Next, the options for the potential helper (out-group member in the experimental group, in-group member in the control group) were shown. By choosing the crossed-out lightning bolt symbol, the potential helper indicated his decision to give up five Swiss francs to cancel delivery of pain stimulation to the participant. By choosing the intact lightning bolt symbol, he indicated his decision to keep the money, which led to a painful shock for the participant at the end of the trial. The potential helper's decision was highlighted with a yellow square. The participant rated how he felt about the potential helper on an emotion rating scale. In this example, the potential helper's decision canceled delivery of the pain stimulation, indicated by a crossed-out lightning bolt at the end of the trial. Otherwise, the intact lightning bolt was presented, and the participant received a painful shock. To allow for successful positive conditioning, the participant was saved from pain in 75% of all trials (15 out of 20 trials).

were represented by different individuals. This setup allowed us to test whether and how the learning intervention affects the neural response to in-group and out-group pain and whether its potential impact generalizes to other individuals who were not present during the intervention but were members of the same respective groups.

Given that expectations concerning out-group members are typically more negative than those concerning in-group members (22), receiving help from an out-group member is an unexpected positive outcome that should elicit a strong positive prediction error. If so, then the participants of the experimental group should arguably use the prediction errors to establish positive associations with their out-group helper. This increase in out-group positivity should in turn increase empathy for the suffering of out-group members (12), reflected by an increase of activation

in empathy-related brain regions such as the AI (23, 24). In contrast, the participants of the control group are likely to expect help from the in-group member. Thus, even though the control group is saved from pain exactly as often as the experimental group, we predicted less learning and no significant change in empathy in the control condition.

Results

For the analyses of behavioral data, we focused on in-group vs. out-group differences in impression ratings and emotion ratings. Impression ratings (9) (*Supporting Information*) served as a manipulation check for the group manipulation and were collected before scanning. Participants had significantly more positive impressions of the in-group members, compared with the out-group members [$F_{(1,36)} = 12.5$, $P = 0.001$; experimental group, $t_{(1,19)} = 2.6$, $P = 0.018$; control group, $t_{(1,17)} = 2.5$, $P = 0.02$]. Emotion ratings served to determine whether the learning intervention had established positive out-group associations and were collected at the end of each intervention trial. We used a linear regression model to compare learning-related changes in emotion ratings in the experimental vs. the control group. To account for potential differences between early and late learning stages (25, 26), we also compared the effects in the first (trials 1–10) and the second half (trials 11–20) of the intervention. Emotions toward the out-group member (experimental group) became more positive than those toward the in-group member (control group), in particular in the first half of the intervention [Treatment (experimental/control group) \times Trial (trials 1–20) \times Half (first/second half), $T = 2.1$, $P = 0.03$; Treatment \times Trial interaction, first half, $T = -1.95$, $P = 0.05$ (Fig. S1), second half, $T = 1.29$, $P = 0.2$ (Table S1)]. These results show that in the experimental group, the learning intervention established positive associations toward the out-group member, with particularly strong effects in the first half.

Next, we tested whether the establishment of positive out-group associations in the experimental group had an impact on empathy-related brain responses after the intervention compared with before the intervention. Based on previous studies (7–9), we assumed that potential learning effects on out-group empathy should modulate the neural response in the AI. To test this assumption, we analyzed our data in bilateral anatomical masks of the insular cortex (27), using small-volume familywise error (SV FWE) correction (see Tables S2–S7 for whole-brain results). Before the intervention, participants' brain responses in the left AI were stronger when they saw the in-group member compared with the out-group member in pain (experimental group, $T = 6.16$, $Z = 4.14$, SV FWE-corrected; control group, $T = 5.71$, $Z = 3.9$, SV FWE-corrected) (Table S2). Confirming previous results (7–9), these findings show an out-group deficit in the AI when comparing observed in-group to out-group pain.

To investigate whether the learning intervention counteracted this out-group deficit, we calculated Group (in-group/out-group) \times Time (preintervention/postintervention) interactions for each participant and compared the average interaction contrasts between the experimental and the control group, using a two-sample t test. Note that this approach is testing a three-way interaction between Group (in-group/out-group), Time (preintervention/postintervention), and Treatment (experimental group/control group), while providing information about the direction of the effect at the same time (experimental group greater than control group). The main result was a significant intervention effect in bilateral AI, which was more substantial in the left hemisphere ($T = 4.72$, $Z = 4.13$, SV FWE-corrected) (Fig. 24 and Table S3).

In follow-up analyses, we tested for a Group (in-group/out-group) \times Time (preintervention/postintervention) interaction, for the experimental and the control group separately. For the experimental group, we again found activation in the left AI ($T = 4.41$, $Z = 4.15$, SV FWE-corrected). In contrast, in the control group, the interaction revealed no significant activations, even at

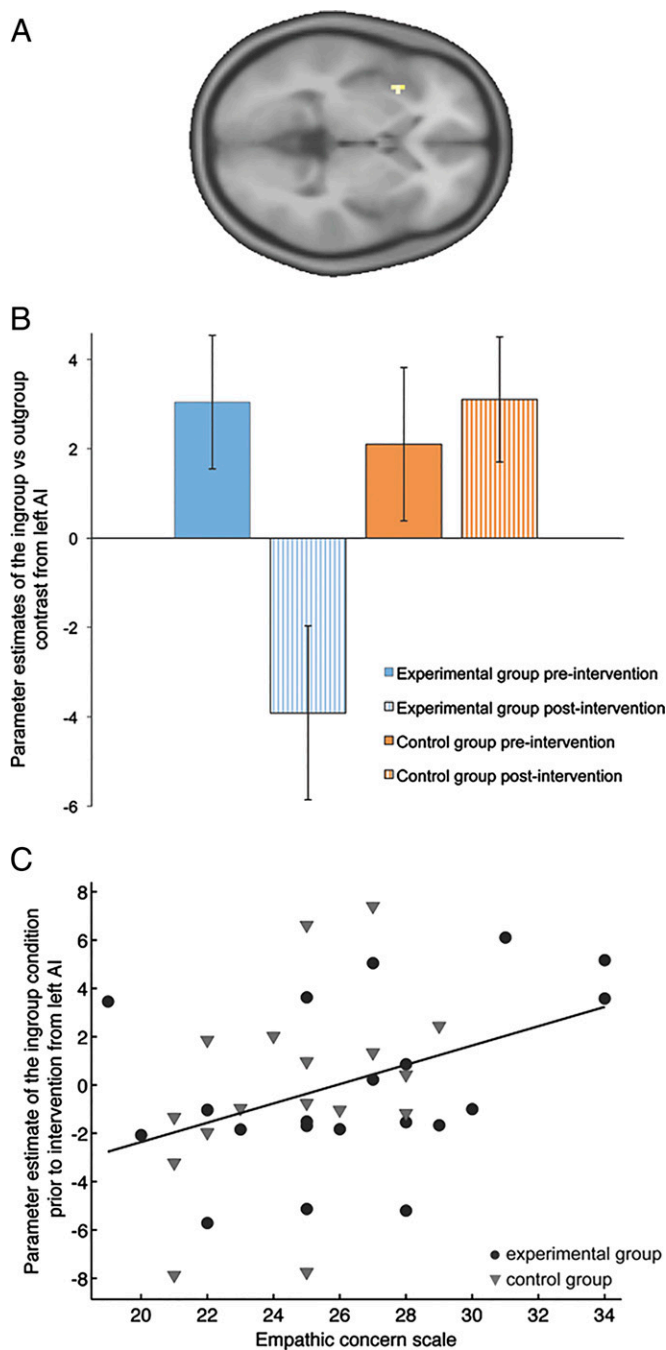


Fig. 2. Impact of the intervention on neural responses during observation of pain in the in-group and out-group member in the experimental and control group. (A) Significant activation in anterior insular cortex (AI), indicating stronger intervention-related effects in the experimental group compared with the control group (Table S3). (B) Average parameter estimates for the contrast between observing in-group pain vs. out-group pain in left AI. As a result of the intervention, empathy responses to out-group pain compared with in-group pain were elevated in the experimental group but remained biased for in-group pain in the control group. (C) Positive correlation between the individual ratings on the Empathic Concern Scale of the Interpersonal Reactivity Index (28) and the neural response in left AI to in-group pain before the intervention in the experimental group (black circles) and the control group (gray triangles). We chose the neural response in the in-group condition before the intervention because this measure is most likely to reflect participants' trait empathy (i.e., their tendency to empathize irrespective of the effects of the out-group manipulation or the intervention). Note that the extracted data are independent of the statistical analysis that defined the extraction region in left AI (i.e., the

a relaxed threshold of $P < 0.05$, uncorrected. Thus, the intervention effect in AI cortex is based on pre-to-post increases in AI activation in the experimental, but not in the control group (Fig. 2B). Moreover, AI activation increased primarily for out-group members in pain. This was evidenced by a significant difference in the out-group post vs. pre contrast between the experimental group and control group in left AI ($T = 4.25$, $Z = 3.8$, SV FWE-corrected). There was no difference between the groups with regard to the in-group conditions (in-group post vs. pre), even at $P < 0.05$ uncorrected. Together, these results demonstrate a significant intervention effect in left AI, which reflects an increase in the experimental group participants' neural response to the out-group member's pain.

Importantly, the neural response in the left AI region, which changed in response to the intervention, correlated with self-reported empathy, that is, the individual ratings on the Empathic Concern Scale (28), which we collected after scanning [$r(38) = 0.39$, $P = 0.015$] (Fig. 2C). This finding confirms that the observed neural effects in this region are related to empathy.

After establishing the success of our intervention, we investigated the mechanisms underlying it. We predicted that positive experiences, i.e., receiving help would elicit positive prediction errors, in particular in the case of an out-group helper. We computed the individual prediction errors based on a modified version of a reinforcement learning model (19), which allowed us to model learning as a function of individual prior expectations. The impression ratings reported above indicate that participants entered the learning intervention with more positive expectations toward the in-group compared with the out-group member. To capture the individual variability in expectations, our model included each participant's impression ratings for the out-group members (experimental group) and the in-group members (control group) as starting predictions for subsequent prediction error estimation (Methods). This approach extends classical prediction error models in which learning starts from a starting prediction of zero (the assumption being that participants have neutral prior expectations).

First, we identified brain regions that track individual prediction errors by regressing neural activity elicited by the decisions of the potential helper (out-group member in the experimental group and in-group member in the control group) against trial-by-trial estimates of prediction errors. The results revealed activation in the AI (Fig. 3A, red), which was more substantial in the right AI ($T = 4.56$, $Z = 4.01$, SV FWE-corrected) (Table S4).

Second, we tested whether the individual prediction error signal in right AI predicted the pre-to-post changes in empathy, that is, the neural intervention effect shown in Fig. 2A. For each individual, we extracted the prediction error-related beta values from right AI and regressed them against the individual magnitude of the intervention effect, as reflected by the Group (in-group/out-group) \times Time (preintervention/postintervention) interaction. The results showed significant activation in left AI ($T = 5.28$, $Z = 4.52$, SV FWE-corrected) (Fig. 3B, red, and Table S5), which overlapped with the observed intervention effect (Fig. 3B, orange; Fig. 2A for comparison). This result shows that the change in empathy after compared with before the intervention is linked to the magnitude of the learning signal during the intervention.

Third, we assumed that the prediction error signal affects empathy-related brain responses via the establishment of positive associations with the out-group member. Accordingly, the direct impact of the prediction error signal on the intervention effects should be mediated by the learning-related increase in

Group \times Time \times Treatment interaction). For additional correlation analyses, see Fig. S2. Error bars represent SEs. The imaging results are displayed at FWE-corrected < 0.05 (SV in bilateral anatomical masks of the insular cortex).

$P(\text{FDR-corrected}) = 0.4$; four positive experiences, $B = 0.36$, $T = -0.12$, $P(\text{FDR-corrected}) = 0.9$].

Discussion

Our findings show that empathy with an out-group member can be learned and generalizes to other out-group individuals. This learning is driven by classical prediction errors, whose impact on empathy signals is mediated by an increase in positivity toward the out-group member. Thus, our study provides a mechanistic account of how positive contacts with the out-group can counteract empathy deficits (10, 11).

The reductions in out-group deficits were predictable after surprisingly few (two) positive experiences with an out-group individual. Although the exact number of experiences should be taken with a grain of salt, it is unlikely that these findings reflect noise, for example induced by a specific constellation of helping and nonhelping trials in the beginning of the intervention. First, an analysis that was based on the full dataset (and therefore less prone to noise) independently identified the strongest learning effects in the first five trials (Table 1). Second, a detailed trial-by-trial analysis revealed that the average ratio of helping to nonhelping trials within the first two trials was not different from that ratio in later phases of the learning intervention, indicating that volatility does not change in early compared with late phases of the experiment (*SI Results*). Third, prediction error signals during the first two intervention trials were similar, irrespective of whether participants had experienced no or some nonhelping trials (*SI Results*). Fourth, in the analysis that revealed a predictive relation between the AI activity between the first two helping trials and subsequent empathy change, we controlled for the number of nonhelping trials experienced by each subject.

So far, efficient learning based on a very few events has mainly been shown in the domain of punishment. For example, in animals, aversive reactions can be learned based on a single aversive event (31, 32), and humans require only two to three negative experiences (33). Going beyond this work, our findings show that people can learn very efficiently from positive social experiences that prevent them from harm, and that this type of learning has a strong impact on complex internal states, such as empathy for a person in pain. Thus, we provide insights into the efficiency of negative reinforcement learning in humans and its potential to serve as an intervention to counteract deficits in out-group empathy.

Moreover, we found that the learning experience, which was initiated by one representative of the out-group, resulted in an increase in empathy for another representative of the out-group who was in pain. The generalization of the learning effect is important, because it shows the robustness of the learning intervention and its potential relevance for society (1). The complex experimental setup (including cover story and confederates) did not allow for repeating the empathy measure for a second time to test for long-term effects. However, it has been shown that negative reinforcement can induce robust and long-lasting learning effects, for example in therapeutic settings (34). In the light of such results, it is conceivable that learning interventions like ours might elicit lasting intervention effects.

Our results reveal how learning underpins the dynamics of empathy. Unexpected positive outcomes resulting from help of another person elicit prediction error signals, that is, signals that resemble the ones known to drive reinforcement learning in the monkey brain (17, 18). Our results indicate that basic learning mechanisms are also used during complex social learning, which is in line with previous studies (13, 35–38). Going further, our findings show how classical learning mechanisms shape other-regarding motivational states such as empathy.

We find that learning about another person and experiencing empathy for a person in pain recruit a common neural structure, namely, the AI cortex (see *Supporting Information* for a discussion of lateralization). Different fields of research have independently

accumulated evidence for the important role of the AI in the processing of empathy (23) and the encoding of prediction errors during learning (15) (see Table S7 and *SI Discussion* for less significant striatal effects). Our results integrate these two domains. We show that, during learning, the AI is involved in updating predictions about future outcomes as well as in implementing the resulting emotional states. Interestingly, the updated information is used to modulate the empathic reaction to another person. Based on these results, an empathy-learning model would propose that empathy-related processes in AI are altered by a person's individual learning history.

According to the empathy-learning model, empathic responses are altered by any information that elicits prediction errors and thereby results in an update of predictions about others. Thus, it makes the clear prediction that empathy learning should be the stronger, the more positive and unexpected the information revealed about another person. These predictions of the empathy-learning model provide a plausible mechanism for the effect of positive intergroup contact (10, 11) and can inspire interventions to foster empathy. On a conceptual level, our results uncover the neural interplay between empathy and learning, and thus provide a neurobiological mechanism for the profound plasticity of empathic reactions, which has been widely documented (39, 40), but so far not explained.

Methods

Participants. Forty healthy men (mean age, 22.7; SE, 0.41) participated in the study. They were randomly assigned to the experimental and the control group with no age difference between the groups [$t_{(38)} = -0.34$, $P = 0.73$]. We chose a male instead of a gender-mixed participant group because it allowed us to also choose male confederates and avoided the potential complications of gender-mixed pairing of participants and confederates. Moreover, testing the modulation of empathy in males is more conservative than in females, because males are less likely to simulate others' emotional state on the neural level (41). Two datasets of the control group had to be excluded because of technical problems during fMRI data collection. Participants gave informed consent, and the study was approved by the Research Ethics Committee of the Canton of Zurich.

Prescanning Procedure. We used a well-established priming procedure (42) to induce the in-group/out-group manipulation and to activate the relevant stereotype. Details about the prescanning procedure and the cover story are provided in *Supporting Information*.

Scanning Procedure. During the "preintervention empathy session," the participant in the scanner observed the in-group or the out-group confederate receive painful stimulation (18 trials each). Each trial started with an arrow cue (500 ms), whose color indicated the recipient of the pain (in-group/out-group member). After a fixation period (1,500 ms), a lightning bolt was presented whose color matched the color of the arrow cue (1,000 ms). Next, the color of the bolt changed to yellow, which indicated the delivery of the painful stimulation to the respective person (1,000 ms). After a fixation period (1,000–3,000 ms), the next trial was presented. The colors indicating the in-group and out-group condition were counterbalanced across participants. The trials were presented in pseudorandomized order (no more than two consecutive trials of the same condition). The "intervention session" consisted of 20 trials, 15 trials in which the participant received help from the other person, and 5 in which he did not receive help and was thus subjected to pain. Helping and nonhelping trials were presented in random order (for details, see Fig. 1). The "postintervention session" was identical to the preintervention session, except that the participant observed the painful stimulation of a new in-group and a new out-group member. The participants (and confederates) were informed that they would not meet after the study and had separate visual displays to keep emotion ratings anonymous.

Prediction Error Model. Prediction errors were computed according to $\delta_t = \alpha(\lambda_t - V_t)$, where V_t corresponds to the value V predicted by all stimuli presented in trial t , λ_t corresponds to the value of the outcome in trial t , and α corresponds to the learning rate. The learning rate determines how much weight is given to recent experience as captured by the prediction error. We assumed a learning rate of 0.3, which is most commonly reported in

reinforcement learning paradigms (43). We found that the prediction error-related effects were very similar for other learning rates (0.2 and 0.4). Additional prediction error estimates based on the emotion ratings confirmed the applied learning rate (*SI Results*). To capture individual variability in prior expectations, V_1 was equal to each participant's average out-group score (experimental group) or in-group score (control group) on the impression scale. In contrast to our approach, traditional learning theory assumes no prior expectations ($V_1 = 0$), which is unlikely in our social setting. The boundary outcome values were set according to the maximum (54 points) and minimum (6 points) score of the impression scale. Accordingly, $\lambda_t = 54$ in helping trials and $\lambda_t = 6$ in nonhelping trials.

Imaging Analyses. We conducted standard preprocessing, first- and second-level analyses (*Supporting Information*). Second-level results were corrected

for multiple comparisons by using FWE correction within bilateral anatomical masks of the entire insular cortex, as defined by the Automated Anatomical Labeling atlas (27). For data extraction, we used the entire cluster of the respective activations. The extracted beta values reflect the average activation of all voxels within the cluster. Details about the multiple-regression analyses and the mediation analysis are provided in *Supporting Information*.

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