Pathogen disgust sensitivity protects against infection in a high pathogen environment

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Disgust is hypothesized to be an evolved emotion that functions to regulate the avoidance of pathogen-related stimuli and behaviors. Individuals with higher pathogen disgust sensitivity (PDS) are predicted to be exposed to and thus infected by fewer pathogens, though no studies have tested this directly. Furthermore, PDS is hypothesized to be locally calibrated to the types of pathogens normally encountered and the fitness-related costs and benefits of infection and avoidance. Market integration (the degree of production for and consumption from market-based economies) influences the relative costs/benefits of pathogen exposure and avoidance through sanitation, hygiene, and lifestyle changes, and is thus predicted to affect PDS. Here, we examine the function of PDS in disease avoidance, its environmental calibration, and its socioecological variation by examining associations among PDS, market-related lifestyle factors, and measures of bacterial, viral, and macroparasitic infection at the individual, household, and community levels. Data were collected among 75 participants (ages 5 to 59 y) from 28 households in three Ecuadorian Shuar communities characterized by subsistence-based lifestyles and high pathogen burden, but experiencing rapid market integration. As predicted, we found strong negative associations between PDS and biomarkers of immune response to viral/bacterial infection, and weaker associations between PDS and measures of macroparasite infection, apparently mediated by market integration-related differences. We provide support for the previously untested hypothesis that PDS is negatively associated with infection, and document variation in PDS indicative of calibration to local socioeconomic conditions. More broadly, findings highlight the importance of evolved psychological mechanisms in human health outcomes.

disgust | pathogen avoidance | behavioral immune system | market integration | Shuar

Darwin first recognized disgust as an evolved human emotion, hypothesizing that it aided in avoidance or expulsion of "tainted" food (1). Since then, many studies have supported the hypothesis that disgust is a universal human emotional response that evolved to motivate avoidance of certain kinds of fitness-reducing substances, activities, or individuals, particularly those associated with infection (2–9). What constitutes "fitness-reducing," however, varies depending on individual and environmental circumstances (8–12). To function adaptively, how sensitive someone is to pathogen-related cues (i.e., pathogen disgust sensitivity [PDS]) should be context-specific, calibrated to the local costs and benefits associated with infection risk and avoidance behaviors, and influenced by endemic parasites, lifestyle, nutritional status, phenotypic qualities, and life-history parameters (8–13). Additionally, PDS to certain pathogen-related stimuli—and the resultant disease-avoidance behaviors—show cross-cultural variation. Disgust-related variation in food preferences and taboos (14, 15), disease-limiting behaviors (e.g., hand washing, wearing of surgical masks) (16), and even political ideology (17) exists cross-culturally, although the degree to which environmental triggers (18) and socially transmitted information (19) account for these differences remains unclear.

Despite general acceptance of the hypothesis that pathogen disgust functions to regulate pathogen exposure, evidence has been largely indirect. Research shows that pathogen disgust is activated in response to hypothesized cues of potential pathogen-harboring stimuli (2, 5, 7, 20, 21) and seems to function to provide answers to the adaptive problems of what to touch, what to eat, and with whom to interact (13). Higher PDS is predicted to motivate greater avoidance of cues recurrently associated with pathogens, and therefore be associated with lower prevalence and intensity of infections (8, 21). While some research suggests that frequent past infections may up-regulate PDS and fear of contamination (22), no studies have directly tested whether greater PDS is associated with fewer current infections.

In addition to environmental cues, individuals are expected to use social information and individual experience to identify local environment-specific sources of contagion or toxicity (8, 23). While prior studies have attempted to examine how disgust is calibrated to local environmental (e.g., hygiene practices, sanitation infrastructure, subsistence strategies) and individual phenotypic conditions (e.g., sex, age, reproductive and health status) (24–28), no studies have explored this among an Indigenous subsistence-based population living in high-pathogen

Significance

Disgust likely evolved to regulate exposure to pathogen-related stimuli and behaviors. One key prediction, that individuals with greater pathogen disgust sensitivity (PDS) will be exposed to fewer pathogens and thus suffer fewer infections, has never been tested directly. To function adaptively, PDS must respond to the local cost/benefit context of avoidance, but this too has been undertested. We provide a test of these predictions among an Indigenous population with notable variation in PDS, infection, and infrastructure. We document predicted negative associations between PDS and pathogen exposure, while illuminating complex, multidirectional relationships among disgust, infection, and environmental variation. Our findings support the hypothesis that disgust functions to regulate pathogen exposure, demonstrating the importance of evolved psychological mechanisms in disease avoidance.

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environments, conditions more like the selective environments hypothesized to have shaped disgust psychology than those studied to date.

Evolutionarily Relevant Pathogens and Cues

Pathogens have imposed important selective pressures throughout evolutionary history, at least since the origin of eukaryotic organisms (29–33). In response, hosts evolved several physiological, psychological, and behavioral defense mechanisms (10, 34). Specifically, the disgust response likely evolved because it motivated strategic avoidance of cues associated with the presence of evolutionarily relevant pathogens (13), including viruses, bacteria, and macroparasites.

To adaptively regulate avoidance of pathogen-containing substances, PDS must result in selective avoidance of evolutionarily relevant cues associated with substances for which the costs of contact or ingestion outweigh the potential benefits (13). Selection is expected to have primarily targeted cues with high signal value, including clear cues of pathogen infection in other people (e.g., vomit, coughing, sputum, diarrhea, mucus/blood in feces), food spoilage (e.g., decay, rotten scent), substances that often harbor infectious agents (e.g., blood, feces), and animal vectors (e.g., maggots, flies, cockroaches, rats) (2, 7, 13, 23).

Calibration of PDS

Furthermore, to function adaptively, pathogen disgust is predicted to include regulatory mechanisms that calibrate PDS based on trade-offs among the local costs and benefits of avoidance, including the likelihood of encountering a pathogen, types of pathogens encountered, and probable fitness-reducing costs of both exposure and avoidance (8, 13, 35, 36). For example, in subsistence-based populations, potential pathogenic substances that must regularly be encountered to acquire food or shelter are relatively costly to avoid (e.g., direct exposure to soil, animal feces, dirt floors, possibly contaminated natural water sources). As disgust-motivated avoidance becomes relatively more costly, PDS is predicted to be down-regulated in response (7, 8, 13). When the costs of avoidance are lower (e.g., in areas with reliably clean water and cooking surfaces that are easily cleaned), PDS is predicted to be up-regulated (7, 8, 13). To date, however, these hypotheses have seldom been tested (9).

In addition, disgust in humans likely evolved to utilize information acquired through both individual experience and social transmission (13). Food aversion based on sickness following ingestion of a particular food is the clearest example of individual experience-induced avoidance, and is present across animal taxa (37, 38). However, since individual experience with a pathogenic contaminant can be costly (even deadly), socially acquired information can be used to reduce these costs when cues are absent, ambiguous, or evolutionarily novel. Thus, PDS is likely to be influenced by social transmission, including via observation of others' sickness experience, instruction and observation of appropriate foods and food-preparation techniques, and hygiene and disease-avoidance measures. Additionally, within-family variation in PDS may highlight a heritable component (39). Thus, studies are needed to track social influences within households and communities, especially as cultural practices change with time, and partition these influences from environmental calibration and possible heritable variance.

Market Integration and Disgust

Societies that traditionally relied on small-scale hunting, gathering, and horticulture are increasingly producing for and purchasing from the market economy, a process frequently referred to as market integration. Market integration affects many factors related to pathogen exposure and the costs of avoidance, and should consequently also shape PDS. For example, market integration is frequently accompanied by increased sanitation infrastructure (e.g., latrines, clean water sources, more hygienic cooking surfaces/practices) and greater access to Western medical care (40–42). It is therefore likely to affect not only disease barriers, but social expectations of cleanliness and hygiene, as well as education about biomedical ideas of disease transmission (42). Market integration is also associated with changes in food production, acquisition, and availability (42), thus likely altering the relative costs and benefits of exposure, expectations about contact with and ability to avoid disgust-eliciting substances, and norms about appropriate and taboo foods and practices. The context of market integration therefore presents a natural experiment for studying how disgust and infection vary with changing ecological and social circumstances.

Disgust and Market Integration among the Indigenous Shuar

In this study, we test: 1) If PDS is associated with lower infection prevalence and intensity; 2) if PDS is calibrated to the local environment; and 3) how relationships among PDS, infection, and environment vary at individual, household, and community levels among Ecuadorian Shuar. In so doing, we also examine the applicability of disgust domains originally defined in high-income populations.

The Shuar are a large Indigenous Amazonian population (>100,000 individuals, according to the Consejo de Desarrollo de las Nacionalidades y Pueblos del Ecuador, https://latinno.net/es/case/8080/#:~:text=El%20Consejo%20de%20Desarrollo%20de,de%20Ecuador%20y%20el%20Estado, in 2012). To capture important lifestyle and infrastructural variation necessary to test relationships among PDS, market integration, and pathogen exposure, participants were recruited from three communities: One in the Upano Valley and two east of the Cordillera de Cutucú (i.e., "Cross Cutucú"). Shuar in both regions rely heavily on traditional cultigens, engage in subsistence horticulture, and interact regularly with domesticated animals (43–49). Cross Cutucú communities are relatively isolated, and subsistence continues to be based primarily on traditional horticulture, hunting, and fishing (43–48). However, in the Upano Valley, hunting and fishing have declined with increasing population size and economic development. Market access, agricultural sales, and wage labor are more pronounced in Upano Valley communities (46–49). At the time of this study, Upano Valley participants could access a regional market center and medical facility within 60 min by bus or truck. Cross Cutucú participants could only access this center after 7 to 12 h by motorized canoe then bus, although more limited services were available 1.5 to 3 h away by motorized canoe.

Among the Shuar, regional- and household-level market integration is linked to differences in both soil-transmitted helminth (STH) exposure and intestinal microbial diversity. Individuals living in the Cross Cutucú region tend to have higher prevalence and intensity of STH infection (50, 51). More market-integrated housing, particularly floor type (i.e., wood versus dirt) and water access (i.e., piped/well versus river/stream), are associated with reduced STH burden (52) and are more common in the Upano Valley. Additionally, more modern housing and reduced engagement in traditional subsistence activities are linked to less-diverse intestinal microbiota (53).

Data were collected as part of the Shuar Health and Life History Project (SHLHP, http://www.shuarproject.org/). To test relationships between PDS, socioecological factors, market integration, and infection, we administered a 19-item disgust questionnaire (*SI Appendix*, Table S1) modified for use with the Shuar from previous disgust scales (7, 54, 55) and a material style-of-life (SOL) interview used previously by the SHLHP (47), and collected stool and finger-prick dried blood spot samples from 75 Shuar participants (ages 5 to 59 y) from 28 households in three communities. This distribution allows us to examine clustering of PDS and infection at the individual, community, and household levels to identify patterns more

closely related to environmental calibration. We used three markers of parasite infection: 1) *Ascaris lumbricoides* (large roundworm) and 2) *Trichuris trichiura* (whipworm) eggs per gram (EPG; indicative of infection presence and intensity) measured from stool samples, and 3) immunoglobulin E (IgE; a long-term marker of macroparasite infection) (56–58) measured from dried blood spot samples. We also analyzed two dried blood spot biomarkers of acute inflammatory response to immediate viral and bacterial infection (interleukin-6 [IL-6] and C-reactive protein [CRP]) (58–61). Material SOL variables were calculated to assess market-integrated SOL (MSOL; indicative of greater ownership of market-purchased items [e.g., propane stove, cellphone, refrigerator]), household SOL (HSOL; indicative of degree of market-integrated household construction and infrastructure [e.g., cinder block, lumber or palmwood walls, cement, wood or dirt floors, water from river, well or spring-fed water system, access to electricity]), and traditional SOL (TSOL; indicative of higher number of items owned that are associated with traditional subsistence and cultural activities [e.g., fishing nets, blowguns]) (47).

We predicted that among individual participants, higher PDS would be associated with lower levels of current infection indicators. Beyond the individual level, we expected to see associations between PDS and infection levels at both the family and community level. Because the costs of pathogen cue avoidance will be higher and the ability to successfully mitigate contamination will be lower among less market-integrated individuals, households, and communities, we predicted that these participants would have lower PDS and higher pathogen exposure.

Results

We used principal components analysis (PCA) to reduce the disgust questionnaire to a single factor (Disgust) (*SI Appendix*, Table S1). Infection and biomarker data were reduced to two factors using PCA (*SI Appendix*, Table S2). Factor 1 (Parasites) was associated with indicators of parasitic infection (e.g., IgE, species-specific EPG). Factor 2 (Inflammation) was associated with inflammatory indicators (CRP and IL-6), consistent with immune responses to other kinds of infection. We used Bayesian models to analyze associations, and report posterior parameter means, 95% credible intervals (CIs), and the proportion of the posterior greater than zero ($P_{>0}$, values close to zero or one indicate that most of the posterior probability suggests a nonzero effect).

Disgust, infection measures, and SOL data clustered strongly at the household and community levels (Fig. 1 and *SI Appendix*, Figs. S1 and S2 and Tables S3 and S4). Specifically, one community had a high level of market integration (i.e., high MSOL/HSOL; low TSOL), and was characterized by high levels of Disgust and low levels of Inflammation and Parasites. A second community had intermediate levels for all variables. The third community had lower levels of market integration (i.e., low MSOL/HSOL; high TSOL), low levels of Disgust, and elevated levels of Inflammation and Parasites.

We analyzed variance components for each variable (Fig. 1*A* and Table S4) at the community, household, and individual levels, and found high degrees of variance attributable to community for Disgust (65%), Parasites (69%), and market-integration variables (HSOL 91%; MSOL: 60%). Additionally, household accounted for 13% of the variance in both Inflammation and Parasites, and 35% of the variance in MSOL and TSOL. Residual variance, attributable to the individual, was highest for Inflammation (62%) and Disgust (31%), with little individual level variation in the other variables.

We first adjusted for this clustering with random effects (*SI Appendix*, Table S5). However, to explicitly model the connections between household and community members, we calculated two variables for each individual: 1) The mean disgust and infection levels for all household members, excluding the individual in question, and 2) the mean disgust and infection values for all community members outside of the individuals' household. Using these variables, we constructed path models, fit simultaneously as multivariate models (Fig. 2 and *SI Appendix*, Tables S6 and S7) and as partial models, to examine how household and community effects mediate associations between disgust and infection (see *SI Appendix*, Fig. S3 for partial model results).

The models revealed strong negative associations between Disgust and Inflammation, at both the individual (standardized β: −0.31, CI: −0.56, −0.06; $P_{>0} < 0.01$) and household levels (β: −0.34, CI: −0.73, 0.06; $P_{>0} = 0.05$). This relationship was also present in each of the three communities when examined individually (Fig. 3). The models also revealed a weaker effect with a wider credibility

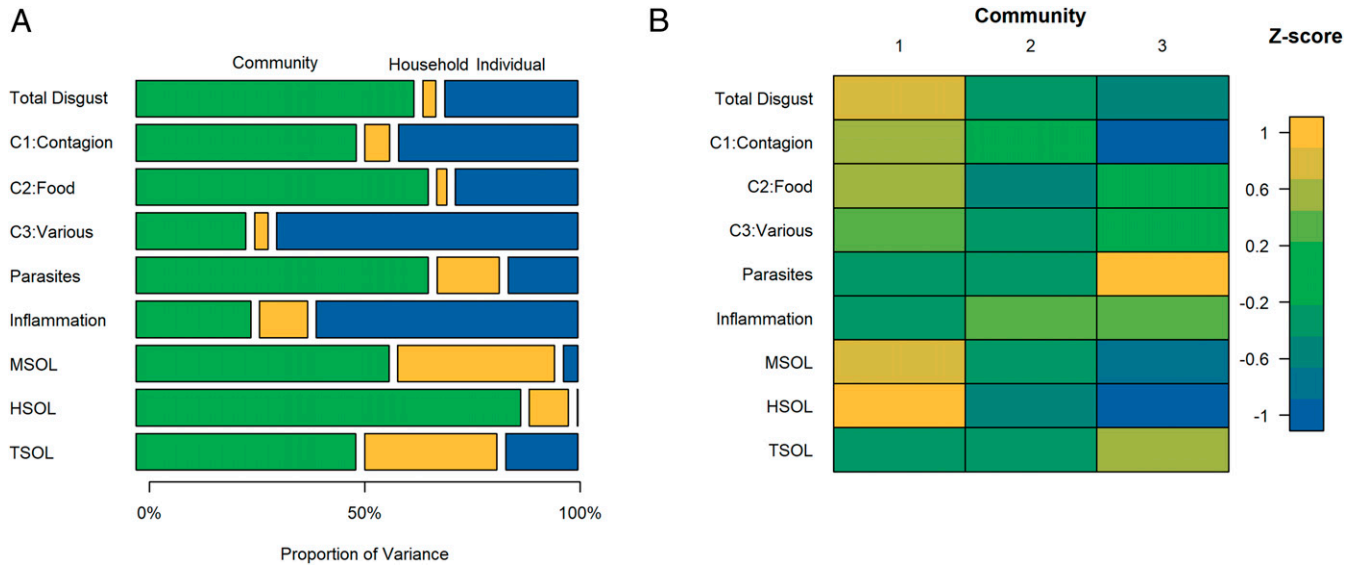

Fig. 1. (*A*) The proportion of variance in each variable attributable to the community, household, and individual level. C1 to C3 are scores from the three components extracted from the disgust scale by PCA. See *SI Appendix*, Table S4. (*B*) Mean standardized values by community (yellow = high, blue = low). See *SI Appendix*, Table S3.

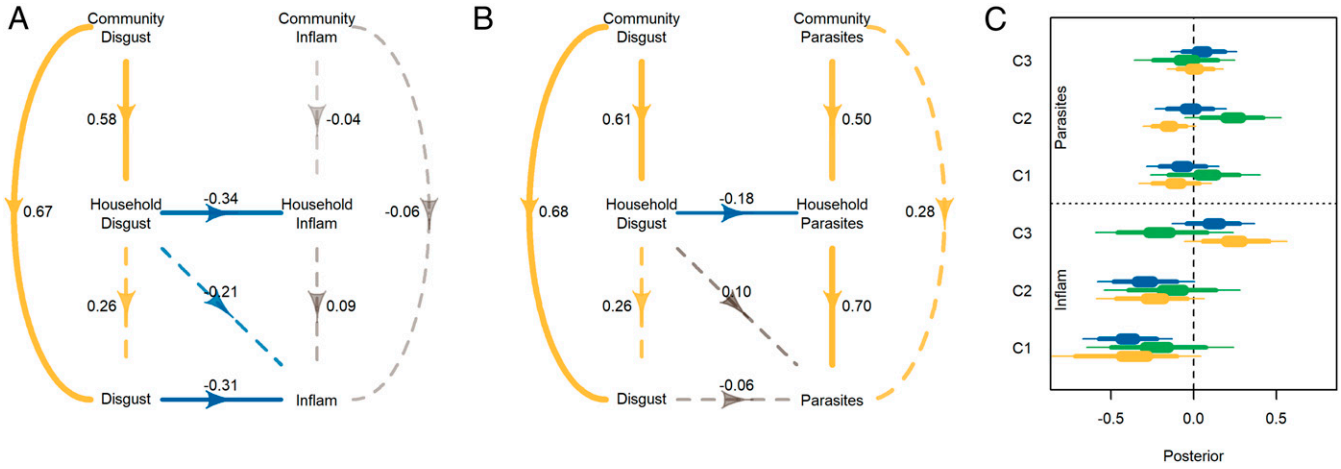

Fig. 2. Complete models showing relationships between disgust and infection at the individual, household, and community levels. Models show relationships between (*A*) Disgust and Inflammation (Inflam) and (*B*) Disgust and Parasites. Line thickness is proportional to the mean posterior effect size. Line type indicates the posterior certainty: Solid line = more than 95% of the posterior is on one side of zero; long dashes = <95% of the posterior is on one side of zero. Color indicates direction of effect: blue = negative, yellow = positive. Effects with less than 80% of the posterior on one side of zero are shaded gray. See *SI Appendix*, Table S6. (*C*) Parameter values for models with disgust components and Inflammation or Parasites (*SI Appendix*, Tables S8 and S9). Blue = individual disgust on individual infection, green = household disgust on individual infection, yellow = household disgust on household infection. Line thickness indicates 25%, 80%, and 95% highest posterior density interval.

interval of other household members' Disgust on an individual's Inflammation (β: −0.21, CI: −0.58, 0.16; $P_{>0} = 0.13$).

When examined without accounting for village-level clustering, there was a negative association between Disgust and Parasites (Fig. 3 and *SI Appendix*, Fig. S3). However, once village-level clustering was accounted for, there was very little evidence for associations between Disgust and Parasites at the individual level (β: −0.06, CI: −0.26, 0.15; $P_{>0} = 0.29$), and within individual villages there was almost no relationship (Fig. 3). However, at the household level, there was evidence for an association, although weaker than that seen for Inflammation (β: −0.18, CI: −0.39, 0.02; $P_{>0} = 0.04$).

Community-level clustering in Disgust was reiterated in complete models as there were strong associations between community-level disgust and individual- and household-level disgust (Fig. 2 and *SI Appendix*, Tables S6 and S7). Since disgust and infection might be jointly influenced by individual and local circumstances, we added market-integration variables (MSOL, HSOL, and TSOL) to the models (Fig. 4 and *SI Appendix*, Fig. S4). The strongest evidence for their effect was a positive association between MSOL and Disgust, evident at both the individual and household levels (β: 0.26, CI: −0.01, 0.53; $P_{>0} = 0.97$; β: 0.15, CI: −0.09, 0.37; $P_{>0} = 0.90$) (Fig. 4 and *SI Appendix*, Fig. S4). HSOL was also positively associated with Disgust at the household level, albeit with a relatively broad posterior (β: 0.28, CI: −0.10, 0.61; $P_{>0} = 0.94$). Also notable was a strong negative association between HSOL and Parasites at the household level (β: −0.41; CI: −0.96, 0.11; $P_{>0} = 0.06$). Other associations with market-integration variables were relatively weak and uncertain.

Due to the cultural inappropriateness of some items for use with the Shuar, we did not replicate the full disgust surveys used in past studies (7, 54, 55). We therefore used PCA to examine components of disgust based on the questions included in our questionnaire. Parallel analysis and scree plots suggested three factors (*SI Appendix*, Table S1). The first factor (C1: contagion) was most closely associated with direct contact with potential pathogens from other individuals (e.g., vomiting, coughing, sharing food with an ill individual), as well as bodily contamination (e.g., not bathing) or contamination of water and food (e.g., worms in food, dirty water). The second factor (C2: food)

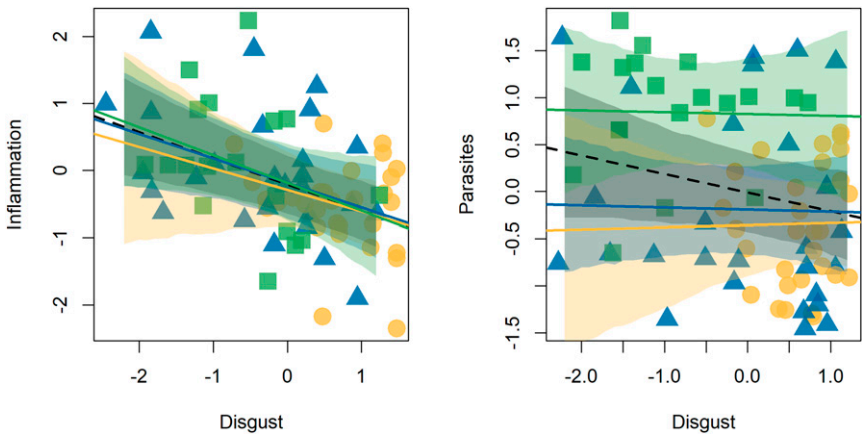

Fig. 3. The relationship between Disgust and infection variables across and within communities. Dashed line shows the linear fit line across all communities. Colored lines and points show the linear fit within each of the three communities in a model with random slopes (yellow circle = community 1; blue triangle = community 2; green square = community 3). Shading represents the 95% CI for each fit line. Points are marginal values corrected for age and sex.

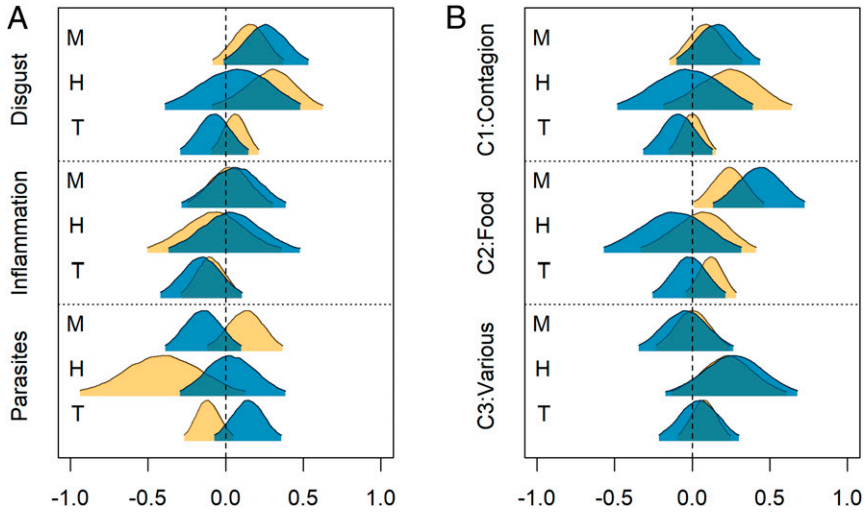

Fig. 4. Market integration, disgust, and infection. (*A*) Posterior parameter distributions for effects of market integration on infection and disgust. (*B*) Effects of market integration on disgust components. Parameters are from full multivariate path models (*SI Appendix*, Fig. S4 and Tables S7–S9), with each plotted posterior distribution representing the combined distribution from the two models with that pathway. Yellow distributions are effects on households. Blue distributions represent effects at the individual level, controlling for levels of other household members. Shaded area is the 95% highest posterior density interval. The abbreviations M, H, and T represent market-integrated, household, and traditional SOL, respectively.

was closely associated with eating spoiled or raw food or touching a dead animal. In this context, touching a dead animal was interpreted to mean hunted meat and was thus perceived as food related. The third factor was comprised of disparate elements not easily classified, and so is referred to simply as C3. As with total Disgust, a high proportion of the variance in contagion and food components clustered at the community level (Fig. 1), whereas C3 was dominated by individual variance. The contagion component showed the strongest association with lower Inflammation (Fig. 2*B* and *SI Appendix*, Table S8) (β: −0.39; CI: −0.66, −0.12; $P_{>0} = 0.003$), followed by the food component (β: −0.29; CI: −0.57, 0.01; $P_{>0} = 0.03$). Food Disgust was associated with lower Parasites, but only at the household level (*SI Appendix*, Table S9) (β: −0.15; CI: −0.31, 0.01; $P_{>0} = 0.03$). Food Disgust also showed a strong positive association with MSOL (Fig. 4 and *SI Appendix*, Tables S8 and S9), whereas other components were not clearly associated with market-integration variables.

Discussion

The present paper is unique in directly testing hypothesized relationships between PDS and various types of pathogen infection among an Indigenous subsistence-based population living in a high-pathogen environment. Significant intracultural environmental and lifestyle variability in this population, due to rapid market integration, also allowed us to test predictions about the calibration of disgust psychology to local socioecological conditions. This research is important for several reasons. First, we provide evidence that higher PDS is associated with lower levels of pathogen infection, as predicted if it functions as a disease-avoidance mechanism. Second, we show that PDS varies at the household and community levels due to factors associated with market integration, consistent with theories of environmental and social calibration of disgust. Third, we provide some support for the applicability of disgust categories first documented in high-income nations to a sample from a subsistence-based population.

Pathogen Disgust Reduces Exposure to Relevant Pathogens. Our results indicate a robust relationship between Disgust and Inflammation, with higher Disgust associated with lower levels of Inflammation. Here, CRP and IL-6 were used as continuous variables to indicate acute immune response to pathogens (59–61). In healthy individuals, CRP and IL-6 levels are usually negligible, but during infections they increase up to 1,000-fold (60). Past research among the Shuar shows no evidence of chronic low-grade inflammation (62), so elevation in these markers is likely capturing acute inflammatory responses associated with infection at the time of study. Specifically, Shuar with high inflammation scores are experiencing varying degrees of inflammatory immune responses to pathogens at the time of study, while individuals with lower Inflammation scores are likely not experiencing an acute infection (60). Intermediate values indicate a recent infection and thus serve as a graded signal of the likelihood of recent infection. Our results (*SI Appendix*, Figs. S6–S9) with simulated biomarkers suggest that measures of acute inflammation are likely to capture associations between disgust and infection.

Disgust predicted lower Inflammation, consistent with the hypothesis that disgust functions to lower pathogen exposure. This relationship was robust when analyzed within individual communities, and varied little between communities (Fig. 3). Negative relationships between Disgust and Inflammation were also observed at the household level. Furthermore, models provided evidence that the Disgust of household members can protect others in the household; when other household members had higher Disgust scores, individuals in those households also benefited by having lower Inflammation scores. These effects were partially mediated through shared PDS in the household (*SI Appendix*, Fig. S3), but suggest that household members can provide both direct and indirect protection from infection, likely through hygiene/sanitation-related pathogen avoidance practices and overall reduced pathogen exposure via household transmission.

The lack of strong relationships between Disgust and Parasites at the individual level, though not originally predicted, is not surprising. The parasitic infections identified in the Shuar and used in these analyses (i.e., *A. lumbricoides*, *T. trichiura*) are generally less virulent, but can incur major fitness-reducing costs including diarrhea, cognitive deficiencies, stunted growth, and altered fertility (29, 63–66). These types of parasite infections are not directly foodborne and instead occur through contact with or consumption of fecal-contaminated soil containing embryonated

eggs that may become infectious after obvious signs of fecal contamination have faded. Therefore, the variables included in our Disgust questionnaire are not primary pathways of STH transmission. Analyses of larger datasets collected after this study also show that parasite load varies by region and community, likely due to variation in market integration-related hygiene/sanitation, infrastructure, and environmental factors, like water source (50–52). The present study supports this finding, with Parasites clustering strongly at the community level.

In fact, we might expect PDS to function differently with respect to STH infection prevention. Unlike viruses and bacteria, STHs cannot replicate in the host and frequency of exposure determines infectious load (64). Exposure to only a few eggs or larvae leads to less-intense infections, while exposure to many eggs or larvae leads to more-intense infections. Immunological pathways keep infection intensities in check while reducing the costs associated with damage to host tissue that would occur from a more aggressive inflammatory response (67). Thus exposure-level and immune response drive infection intensity. With STHs, there is relatively little cost to light initial exposures, thus PDS may be less sensitive to STH-specific vectors (e.g., soil and soil-contaminated foods). These vectors may be extremely difficult to avoid in contexts where exposure to soil is needed for subsistence (e.g., agriculture, hunting, foraging) or where infrastructure does not allow efficient avoidance (e.g., dirt floors, lack of running water/sewage systems), all factors associated with STH infection among the Shuar (52), and characteristic of the evolutionary environment that shaped the disgust response. In these cases, heightened disgust sensitivity related to these vectors may be maladaptive.

Since parasite load is dependent on repeated exposure, simulation results suggest that if PDS were protective against STHs, the effect should be easily detected with the current approach (*SI Appendix*, Figs. S6–S9). The fact that we find little effect likely indicates that PDS, as measured here, is negligibly protective against these kinds of parasites. On the other hand, microparasites, like viruses and bacteria, replicate in the host and can cause intense infections even with light initial exposure to limited numbers of bacteria or viral elements. For this reason, it makes sense for the disgust response system to treat vectors or cues of microparasites as contaminants (i.e., substances to be avoided even in small amounts and which are capable of spreading serious disease with even limited contact) (68–70) and a robust response to microparasite-related vectors, which are relatively more easily avoided in these settings, would be more adaptive.

While we have assumed that PDS influences infection, infection might also be capable of influencing PDS. Sickness behavior, for example, is characterized by loss of appetite (71–75) and as changes in depression and anxiety (75–77), and could therefore relate to PDS, though we know of no evidence for this. Indeed, over longer time periods, disgust is expected to be calibrated by environmental cues and thus to be influenced by infection. Based on our results, however, reversed causality for infection and PDS (*SI Appendix*, Fig. S5) seems less logically plausible, since we would expect Inflammation to increase Disgust, given greater vulnerability to other infections during illness (75).

The finding that infection, Disgust, and SOL variables clustered differently at the community, household, and individual level (Fig. 1) is also important. Most of the variance in Inflammation was at the individual level, demonstrating the importance of individual variation in exposure to viral and bacterial pathogens, and immune system development and function. Despite PDS being moderately heritable genetically (39), in our study Disgust varied considerably at the individual level with little clustering by household, suggesting that PDS is calibrated to individual experience, perhaps based on factors such as life-history stage, immune system development, and overall health.

Environmental and Social Calibration of Disgust. Beyond direct relationships between individual PDS and infection, community explained a large portion of the variance in most variables (Fig. 1*A* and *SI Appendix*, Table S4). Consistent with expectations, Disgust was highest in the most market-integrated community, where access to sanitation and clean water is greater and the costs of disgust-motivated avoidance likely lowest (Fig. 1*B* and *SI Appendix*, Fig. S2). Conversely, Disgust was lowest in the least market-integrated community, where there is greater engagement in traditional subsistence activities of hunting and fishing, coupled with living conditions that make avoidance more costly (i.e., dirt floors, cooking fires on floors, no plumbing/latrines, lack of piped spring water, possibly contaminated water sources). In these conditions, PDS may be down-regulated in response to the relatively high costs of pathogen-avoidance behaviors. In more market-integrated communities, sanitation and improved water infrastructure may reduce costs of avoiding disgust-eliciting stimuli and effective preventative behaviors (e.g., more effective hand and dish washing). Due to greater ease of avoidance, PDS may be up-regulated to further motivate greater avoidance behaviors.

The patterns described here are perhaps contrary to some hypotheses that predict disease-avoidance behaviors should be highest in high pathogen areas (36). However, these hypotheses are largely based on cross-country comparisons, rather than individual-level data, and generally overlook the relative costs of avoidance under different circumstances (9). When we look at individual-level data, high PDS is associated with lower levels of infection and with more market-integrated conditions, which likely lower the relative costs of pathogen avoidance. This suggests a "Simpson's paradox" (78): When we compare individuals within the same culture, we see that individuals with higher PDS have lower pathogen loads; but when whole regions or countries are compared, countries with higher parasite loads have higher PDS. It may be that the highest disgust sensitivity is found in individuals in high pathogen countries who can afford to avoid pathogens. Studies biased toward sampling more affluent individuals in countries with high parasite loads might therefore suggest a positive association between pathogens and pathogen avoidance, even though at an individual level the opposite might be observed.

Cross-Cultural Evidence for Pathogen Disgust Domains. Early research grouped disgust-eliciting stimuli into five categories, the first three of which are specific to pathogen disgust: 1) Bodily excretions and body parts, 2) decayed and spoiled food, and 3) particular types of living creatures (e.g., rats, worms, maggots, cockroaches, lice) (2). More recent research suggests that disgust-regulating cues are linked to the specific evolutionary problems of what to touch, what to eat, and with whom to interact or have sex (13). Until now, these categorizations have not been validated among populations living in high-pathogen environments. We provide some preliminary support for these categorizations.

The two disgust components (i.e., contagion disgust and food disgust) identified by factor analysis in this study appear to address the evolutionary problems of "what to touch" and "what to eat," respectively, and the variables included in each appear to factor into similar categories to bodily excretions and body parts, as well as decayed and spoiled food. Furthermore, the third factor (C3) did include exposure to animals like rats and spiders, which demonstrates some overlap with the third category (*SI Appendix*, Table S1). Although our PCA is somewhat informal in terms of validating domains, these findings provide some limited support for these basic psychological disgust categories among an Indigenous subsistence-based population. It would be useful for future studies to assess whether pathogen disgust stimuli fall into even more specific domains and the role of each in disease avoidance (2, 6, 13). Additional disgust domains (i.e., sexual and

moral disgust) (7, 79) are also important; we did not assess them here for reasons of cultural appropriateness, but future studies may find ways to investigate these in a broader cross-cultural context.

These more specific pathogen-related disgust domains may be important because they each respond to different stimuli, are shaped by distinct environmental factors, likely protect against different pathogens, and thus entail different context-sensitive costs and benefits of avoidance. For example, while higher contagion and food disgust scores were both associated with lower levels of Inflammation, contagion disgust had a stronger relationship. Further work to understand the kinds of pathogens contributing to elevated inflammation could shed light on nuances in these variables. It is possible, for example, that contagion disgust protects from viral and bacterial pathogens that spread via person-to-person contact through bodily fluids (i.e., what to touch), while food disgust protects from foodborne pathogens and toxins and is an important component in deciding what to eat (13).

In fact, food disgust, but not contagion disgust, was associated with one of our indicators of market integration, specifically MSOL (Fig. 3 and *SI Appendix*, Fig. S4). Generally, market integration affects subsistence strategies, including how foods are obtained, processed, and stored. While traditional cultigens formed the subsistence base in all study communities, the more market-integrated community engaged in less hunting and fishing. Furthermore, income from market-based activities and proximity to market centers allowed greater purchase of market foods, some of which are preprocessed or prepackaged (e.g., noodles, sardines, rice). Tellingly, MSOL included specific items directly related to food preparation (e.g., how food is cooked) and storage (e.g., whether refrigeration is available). To take one example, cooking on a propane stove—compared to an open dirt-floor fire—enhances food preparation hygiene with relatively little individual effort. Similarly, refrigeration facilitates easy and safe food storage, allowing both fresh and cooked foods to be kept longer without spoilage. Without refrigeration, Shuar food storage entails either leaving root crops growing in the ground, processing manioc into a fermented beverage (*niahamanche* in Shuar, *chicha* in Spanish), smoking excess game or fish over the fire, or simply leaving leftover food at ambient temperature until the next meal. However, even smoked meat spoils rapidly in tropical heat and humidity. This may increase the relative value of consuming marginally spoiled foods—rather than letting them go to waste—under these conditions, which likely lowers food disgust sensitivity. Based on these findings, it may be useful to further explore how particular features of different disgust domains relate to specific forms of pathogen avoidance and environmental calibration.

Limitations

Although simulations suggest that the sample size in this study is sufficient to credibly detect the associations tested here with a low probability of a false positive (*SI Appendix*, Figs. S6–S9), the sample of 75 individuals is not sufficient to test additional individual phenotypic factors that might influence PDS. Although we did not detect appreciable effects of age or sex (*SI Appendix*, Tables S6–S9), many other factors (e.g., reproductive status) might interact with these variables.

This study is also relatively limited in the scope of market integration sampled. All participants were Shuar horticulturalists living in rural communities. Approximately half the sampled individuals resided in an Upano Valley community, while the other half lived in Cross Cutucú communities. While the sampling strategy was intentionally designed to capture variation in market integration, these communities are still much less market integrated than individuals living in the regional market center, a group not included in this study.

Furthermore, this study adapted preexisting disgust scales (7, 54, 55) created in high-income countries for use with an Indigenous population living in a high-pathogen environment whose diet remains primarily based on subsistence horticulture. The scale may not have been effective at measuring stimuli relevant to transmission of STHs and other macroparasites, which are less common than microparasites in high-income regions. Future research should incorporate disgust elicitors associated with contaminated soil and oral-fecal contamination (e.g., consumption of soil or soil-covered produce, eating with soil-covered hands) to test relationships between PDS and macroparasite exposure more directly.

Besides STH infection intensity, measured using EPG of feces, other measures of infection were indirect and based on immune biomarkers. The biomarkers CRP and IL-6 are related to increased inflammatory immune activity associated with acute infection with microparasites (59–61), but they cannot tell us about the type, intensity, or duration of the infection, or about individual variation in immune response. Furthermore, IgE is associated with long-term immune response to macroparasites over several years (56). While IgE increases based on infection intensity (24) and does eventually diminish after an infection clears (56, 80, 81), it provides only a snapshot of the immune response to macroparasites and cannot be used to deduce how long individuals have been infected, when they were first exposed, and how regularly they are being reexposed. We also cannot dissect individual variation in IgE based on genetics, immune-priming, and age. If PDS is calibrated to current conditions, its measure at time of study may be temporally out of sync with the IgE measure of macroparasitic infection.

The study is also limited in its ability to parse environmental calibration and evoked culture (18) from factors related to cultural transmission. While these are difficult to fully assess among human samples, future studies could incorporate longitudinal data or data that look explicitly at individual behavior, knowledge, and beliefs to better separate these effects. Longitudinal data would also allow for more direct tests of causality between environmental calibration and PDS as it occurs throughout an individual's lifetime. It is worth noting, however, that market integration among the Shuar has increased so rapidly that adults in the most market-integrated community grew up in conditions similar to or even less market-integrated than the Cross Cutucú communities sampled at the time of this study, suggesting that environmental calibration continues throughout life.

Conclusions

This study provides support for the hypothesis that disgust is an evolved human emotion that functions to limit infection by regulating pathogen exposure in response to the local costs and benefits of avoidance and infection. It is unique in its findings that higher PDS is associated with lower levels of pathogen infection among an Indigenous subsistence-based population living in a high pathogen environment, conditions that are, in important ways, more similar to those experienced throughout human evolutionary history than those tested to date. Furthermore, it shows that market integration is associated with higher PDS, as is predicted if disgust sensitivity is calibrated to the local costs/benefit structure of avoidance and infection. Finally, although we did not test the idea that pathogen disgust evolved to solve the adaptive problem of with whom to interact, PCA suggests that two domains of pathogen disgust exhibited by the Shuar may be consistent with those first described in high income, industrialized populations, providing cross-cultural support for the hypothesis that pathogen disgust functions to solve adaptive problems related to what to touch and what to eat.

Materials and Methods

Participants and Sampling. Cross-sectional data were collected over two field seasons (August to September 2011, August to September 2012) in one Upano Valley community (population ∼350 individuals) and two Cross Cutucú communities (combined population ∼360 individuals). Seventy-five

participants (Upano Valley $n = 30$; Cross Cutucú $n = 45$; ages 5 to 59 y) completed the disgust questionnaire, SOL interview, and provided finger-prick blood and stool samples. The study was approved by the Institutional Review Board at the University of Oregon and all participants provided informed consent.

Disgust Sensitivity Measures. Commonly used disgust scales (7, 54, 55) were adapted for relevance to Shuar culture and translated to Spanish. Most Shuar speak Spanish fluently, but a bilingual (Spanish/Shuar) assistant translated as necessary. The questionnaire measured PDS to 19 items (*SI Appendix*, Table S1 provides English translation for all questionnaire items) using a 5-point Likert scale, with higher values indicating greater disgust (1, "not disgusting" to 5, "very disgusting").

Lifestyle Measures. Structured interviews were administered in Spanish to collect basic demographic and lifestyle information from adult household members. Participants were asked questions from a material SOL index (82, 83) modified for use with the Shuar (47, 49). Three SOL variables were calculated: HSOL, TSOL, MSOL. See *SI Appendix* for more details. Participants could be high on all scales, low on all, or any combination, so these variables were not further combined.

Dried Blood Spot Collection and Analyses. Capillary whole blood samples were collected via finger prick and preserved on protein saver cards (Whatman #903, GE Healthcare) following standard methods (84). Samples were dried, stored at −20 °C, and shipped frozen to the Global Health Biomarker Laboratory at the University of Oregon. They were analyzed using commercially available ELISA for IgE and IL-6, and commercially available antibodies for CRP, based on previously established dried blood spot protocols (56, 57, 65, 85–87). See *SI Appendix* for assay details.

Stool Collection and Analysis. Fresh stool samples were collected and processed based on methods previously described (50–52). For each specimen, a single Kato-Katz thick smear (Vestergaard Frandsen) (88) was examined microscopically by a trained observer (T.J.C.-R.). Species-specific EPG of feces were calculated by multiplying the total number of eggs of each STH species on a single slide by 24 (89). Higher EPG represent higher-intensity infections (89).

Statistical Analyses. All analyses were conducted in R 4.0.3 (https://cran.r-project.org/). We first reduced the disgust questionnaire to a single principal component and extracted scores via regression. In secondary analyses, we extracted three components as suggested by parallel analysis and scrutiny of scree plots (90) (*SI Appendix*, Table S1). Infection variables (CRP, IL-6, IgE, *A. lumbricoides* and *T. trichiura* EPG counts) were log-transformed and standardized prior to analysis. Missing values for IgE, CRP, and IL-6 ($n = 15$, 11, and 19, respectively) were imputed with multivariate imputation by chained equations [mice (91)] and a PCA of infection variables was performed on the combined 10 imputed datasets, yielding two oblimin-rotated components as suggested by parallel analysis and scrutiny of scree plots (*SI Appendix*, Table S2). Individual scores were extracted by regression and labeled Parasites and Inflammation.

For each individual, we calculated the mean disgust, infection, and market-integration value for all household members excluding the target individual, and for all community members excluding the household. Modeling using these variables explicitly modeled the contribution of other household and community members to the variance in the dependent variable. Some models included random effects to control for lack of independence in repeat measures, as appropriate. Models with imputed values were fit using brm_multiple in the brms package (92). Components of multivariate path models were fit simultaneously in the same model. Reported values are the mean posterior estimate and 95% CI. Further details on statistical methods and model specifications are given in *SI Appendix*.

Data Availability. The deidentified dataset for this article is available upon request on the SHLHP website (https://www.shuarproject.org/data-sharing) in accordance with data use agreements to ensure compliance with relevant Institutional Review Board, participant expectations, and authorship conditions. Code for all analyses is available at http://doi.org/10.5281/zenodo.4487336 (93).

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Supplemental Analyses

In examining associations between Disgust and infection variables, we first used simple models and then added random effects to account for non-independence. However, using random effects to adjust for covariance by household and community ignores the fact that this covariance is also of interest because it can reveal community and household patterns in infection and Disgust, which might result from shared environments or cultural transmission. Thus, our final models include explicit parameters for community and household effects, as described in the main text. We went through these steps so we could explicitly examine how accounting for clustering affected estimates.

Analyzed across all communities and households, with no adjustment for the non-independence of household and community members (**Figure S3**), strong negative associations were seen between both Disgust and Inflammation (Standardized β: -0.39, CI: -0.62, -0.16) and Disgust and Parasites (β: -0.20, CI: -0.43, 0.03).

Controlling for community and household with random effects terms (**Table S5**), the negative association between Disgust and Inflammation was largely unchanged (β: -0.34, CI: -0.60, -0.07), while the association between Disgust and Parasites was largely eliminated (β: -0.03, CI: -0.24, 0.18).

In building path models, we first examined the effects of household clustering before accounting for community (**Figure S3C-F**). In these models we found associations between an individual's infection levels and the disgust sensitivity of his or her household members. However, these effects were partially indirect and mediated through other pathways. For example, considered alone, the Disgust of an individual's other household members was associated with that individual's Inflammation (β: -0.41, CI: -0.74, -0.09), but when controlling for that individual's Disgust, this direct effect was reduced (β: -0.24, CI: -0.58, 0.10). However, household member Disgust was associated with individual Disgust (β: 0.54, CI: 0.15, 0.25), which was in turn associated with Inflammation (β: -0.32, CI: -0.56, -0.07).

A more dramatic example of this mediation is seen with Parasites. The association between Disgust and Parasites was greatly reduced when household and community level clustering of Parasites was considered (**Figure S3**).

We hypothesized that PDS would protect against infection, and our models were set up with this prediction in mind. However, it is worth cautioning that our models cannot conclusively establish causality or the direction of effects. As a final analysis step, we also constructed models with reversed causality, i.e. with infection predicting disgust. We ran models similar to **Figure 2**, but with PDS dependent on infection rather than vice-versa (**Figure S5**). Overall, these models yielded similar associations. Model fits (assessed with 10-fold cross validation) with reverse causality were not distinguishably better or worse than those in **Figure 2**.

Validation of Analysis Assumptions

The current analysis assumes that a cross-sectional sample of biomarkers can be used to assess whether disgust has protected someone against infection. Biomarkers are indirect measures in the sense that they measure a downstream consequence of infection, rather than infection itself. In our assays we measured two types of biomarker responses: markers of an inflammatory response (CRP and IL-6) typical of short-term infections [1-4], and a composite measure of parasite load/exposure, composed of fecal EPG and IgE. Apart from the biomarkers used to distinguish them, three primary features

differentiate these two measures. The first is the time course of infection: inflammatory responses are typical of short-term infections lasting a week or two [2-4], while parasitic infections may last months or years [1, 5-6]. The second is the way infections progress. Infections caused by viral, bacterial, or other single-celled organisms can become systemic as the pathogen replicates in the host. This causes a rapid increase in inflammatory biomarkers, followed by a relatively rapid drop after the infection resolves [3]. Infections caused by macroparasites, such as helminths, do not behave in this way. These parasites cannot replicate in the host, so parasite load is dependent on continued exposure to new infections. Helminth infections may last for months or years [7].

To examine whether the type of infection might affect our power to detect protective effects of disgust, we created a simple model which simulates each type of infection over time. Complete simulation code is available at https://github.com/adblackwell/shuardisgust. In the simulation each individual has a disgust value which modifies their daily probability of contracting a new infection. Disgust does not affect duration or intensity of infection once acquired. While infected, individuals experience elevated biomarkers. Biomarkers decline gradually after the infection ends. By varying the duration of infection and whether individuals are able to contract multiple infections we can simulate biomarker responses to either inflammatory or parasitic infections (**Figure S6**). Parameters for the models reflect the effect of disgust, the daily likelihood of infection, the distribution of infection durations, and the rate of decline of biomarkers when infection load ends or decreases.

We simulated biomarker responses for 750 individuals under a range of circumstances, varying the effect of disgust on infection risk (expressed as an odds-ratio per 1 standard deviation change in disgust) as well as the baseline infection risk at average disgust levels (**Figure S7**). From these simulated biomarker progressions, we sampled 75 individuals once each at random time points, to match the sample size used in the empirical study. We repeated this sampling 50 times, and each time tested whether we detected the association between disgust and infection that was used to generate the simulated infection progression.

The results suggest that for inflammatory infections that occur with 20% or higher probability per month, a sample size of 75 is sufficient to obtain reliable posterior estimates >80% of the time when a 1 standard deviation difference in disgust causes a reduction in infection risk of ~30%. Weaker effects of disgust should still be detected, but with less certainty. For effects on parasitic infections, the study should be even more sensitive (**Figure S7C-D**). This is because differences in disgust should result in long-term differences in biomarker levels, which are more readily measured by a sampling a random time point. This is illustrated by examining the interaction between infection duration and infection prevalence on power to detect an effect (**Figure S8**). Figure S8 shows that an infection must either be sufficiently prevalent or sufficiently long for an effect of disgust to be picked up in this kind of cross-sectional sampling.

Perhaps more relevant to evaluating our current results is the probability of detecting an effect when no effect is present. To this end, we ran a simulation in which Disgust had no effect on Inflammation and examined the proportion of trials in which we recovered a parameter estimate of -0.25 or lower. Out of 300 simulated trials, a false effect of this magnitude or stronger was detected in only 2% of trials.

Comparing the simulation results to our empirical data, 14% of our sample had elevated CRP or IL-6 in the cross-sectional sample. If we assume this is indicative of a recent or current infection and that an infection lasts 1-2 weeks, then this is likely equivalent to a monthly risk of about 30-50%. It is more

difficult to estimate monthly exposure to helminths, but in the sample roughly 60% were positive for helminth infections, and about 40% had high levels of infection, so likely this is relatively high as well.

The standardized parameter values obtained from the empirical results (**Figure 2**) are around -0.30 for inflammatory biomarkers, comparable to the results produced from an initial reduction in infection odds of about 30% (**Figure S9**). Given the cumulative nature of parasite infections, however, the biomarker is potentially more sensitive to disgust. The low parameter estimates we obtained (around -0.10 after correcting for clustering) are suggestive of a very small effect of disgust. Even larger values, as high as the -0.35 obtained for contagion disgust on parasites when with no cluster correction is applied (**Figure S3C**), would not be indicative of much more than a 10% reduction in infection risk.

Additional Methods Information

Style of Life Interviews and Variables. The selection of items used in the Shuar SOL scales was based on extensive ethnographic observations and pilot testing by one author (LSS). These SOL scales have been used in previous research among the Shuar [8-10]. Two scales were created from the MSL index: Traditional Style of Life (TSOL) and Market-Integrated Style of Life (MSOL). The final TSOL scale contained six items reflecting investment in a foraging lifestyle (fishing hook/line, hunting dogs, blowgun, firearm, fishing net, canoe), while the MSOL scale included 12 items reflecting investment in a market economy (radio, propane stove, mobile phone, television, chainsaw, bicycle, refrigerator, computer, outboard motor, motorcycle, car, truck). Individual scores on each of the MSOL and TSOL were calculated as the fraction of list items owned (range 0-1).

Seven household measures were also incorporated in the SOL questionnaire to capture household construction, access to water and electricity, market participation, and risk of pathogen exposure. These included, in order of increasing market integration, floor (0: dirt, 1: palmwood, 2: milled lumber, 3: concrete; 4: tile), wall (0: palmwood, 1: mixed, 2: milled lumber, 3: cinder block), latrine type (0: none, 1: pit toilet, 2: indoor toilet without water, 3: outdoor toilet with water, 4: indoor toilet with water), water source (0: river/stream/spring pond, 1: well or outdoor pipe, 2: indoor pipe), electricity (0: none, 1: lights only, 2: outlets), number of rooms in house (total number), and number of houses owned (total number). A Household Style of Life (HSOL) value for each household was computed based on a summation of the scores [8-9].

Biomarker Assays. Biomarkers were analyzed using commercially-available enzyme-linked immunosorbent assays (ELISAs) for IgE (E80-108; Bethyl Laboratories, Montgomery, TX) and IL-6 (HS600B; R&D Systems, Minneapolis, MN), and commercially-available antibodies for CRP (M86005M [coating], M86264M [detection]; Biodesign, Memphis, TN) based on previously established dried blood spot protocols [5-6, 11-13]. Immunoglobulin-E and CRP were run in duplicate and cases where CVs were over 12% were rerun. The average sample intra-assay CVs for IgE and CRP were 2.89% and 4.74%, respectively. Interleukin-6 was only run in single due the large amount of sample needed per assay and limited sample availability. Six samples yielded IL-6 levels below the limit of detection. These were set to the lower level of detection of the assay (0.006 pg/mL).

Statistical Analyses. For the 19 disgust questions, we used `principal` in the `psych` package [14] to first reduce the disgust scale to a single component. Scores were extracted via regression. In later analyses we extracted three rotated components, as suggested by parallel analysis and scrutiny of scree plots. An oblimin rotation was chosen to improve interpretability without assuming components to be

uncorrelated, since theoretically dimensions of disgust should covary. Overall, the three components were marginally correlated (r between 0.16 and 0.33).

Because the second component included three items related to consumption of raw animal products (**Table S1**), we repeated the factor analysis but replaced these three items with a single item representing the mean of these three questions. Components extracted with this single raw animal products score were nearly identical, suggesting the second factor was not purely dependent on the replication of these similar questions.

Infection variables (CRP, IL-6, *A. lumbricoides* and *T. trichiura* egg counts) were log transformed and standardized prior to analysis. Out of 75 cases, there were missing values for IgE, CRP, and IL-6 (n = 15, 11, and 19, respectively). Cases were missing due to insufficient blood spots available on DBS cards. To avoid excluding these cases and introducing bias, we used multivariate imputation by chained equations (`mice` [15]) to generate 10 imputed datasets, using random forest imputation. The 10 imputed datasets were merged, and a principal components analysis of infection variables was performed. Parallel analysis suggested two components (**Table S3**). These were clearly identifiable as Parasites and Inflammation and were labeled as such. Component scores were extracted for each individual in each of the ten imputed datasets via regression. Component scores were extracted for each individual in each of the ten imputed datasets. Mean correlations between component scores for each of the 10 imputed datasets were r=0.84±0.05 for Inflammation and r=0.95±0.02 for Parasites, reflecting the fact that only some of the variables contributing to overall scores were imputed.

For each individual, we calculated the mean disgust, infection, and market integration value for all household members excluding the target individual. We then calculated these values for all community members, excluding the household. In this way, each individual had a unique value for other household members excluding themselves, and for other community members excluding themselves and their household. This ensured that values for individual, household, and community were independent, since for each individual, the household was not a component of the value for other community members, and the individual is not a component of either the score of their neighbors or other household members. Modeling using these variables explicitly modeled the contribution of other household and community members to the variance in the dependent variable, an approach that differs from using random effects to control for covariance within hierarchical groupings. However, both approaches control for covariance by making it part of the model. Some models did include random effects to control for lack of independence in repeat measures, as appropriate.

Models were fit using `brm_multiple` in the `brms` package [16], which fits models based on multiple imputed datasets and then combines posterior estimates. All models used default non-informative priors except the models in Tables S4 and S5, which included regularizing priors for the effects of community-level inflammation. These were included since the posterior in a few (but not most) models suggested unlikely negative associations between inflammation at the community and at the household and individual levels. Components of multivariate path models were fit simultaneously in the same model. Inspection of individual model Rhats within the `brm_multiple` output was used to assess model convergence. Reported values are the mean posterior estimate and 95% credibility interval.

Code for all analyses is posted at http://doi.org/10.5281/zenodo.4487336.

Table S1. Disgust questionnaire principal components

	Single Factor	Three Factor		
		1: Contagion	2: Food	3: Various
Finding a worm in your food	0.590	0.855		-0.227
Stepping in feces with bare feet	0.746	0.772		
Drinking *chicha* made by someone who has no teeth	0.686	0.766		
Someone vomiting on your shoes	0.725	0.715	0.156	
Drinking *chicha* made by someone who is ill	0.753	0.713		0.199
Finding a cockroach in your food	0.524	0.635		-0.128
Someone coughing in your face	0.655	0.570		0.297
Knowing someone has not bathed in three days	0.495	0.549	-0.102	0.147
A dog licking your face	0.567	0.473		0.224
Drinking brown, dirty water	0.696	0.419	0.144	0.418
Eating raw fish	0.550	-0.111	0.928	
Eating raw chicken	0.576		0.923	
Eating raw beef	0.679	0.209	0.835	-0.108
Eating meat that has gone bad	0.512		0.716	
Picking up a dead animal with your hands	0.711	0.295	0.500	0.215
Not washing your hands before eating	0.514		0.351	0.522
Seeing a rat in your kitchen	0.627	0.150	0.316	0.513
Coming into contact with someone else's blood	0.442	0.236	-0.232	0.698
Finding a spider in your house	0.391	-0.106		0.860

Table S2. Infection data principal components

	1: Parasites	2: Inflammation
Ascaris EPG	0.847	
Trichuris EPG	0.472	-0.118
IgE	0.731	0.333
CRP	-0.534	0.583
IL-6	0.146	0.821

All variables were natural log transformed prior to analysis.

Table S3. Summary statistics by community

Community	1 (n=30)	2 (n=27)	3 (n=18)	$F_{2,72}$	p
Total Disgust	0.68 (0.63)	-0.35 (1.01)	-0.61 (0.77)	17.28	<0.01
C1: Contagion	0.60 (0.47)	-0.08 (1.06)	-0.89 (0.82)	18.58	<0.01
C2: Food	0.53 (0.49)	-0.53 (1.17)	-0.08 (0.88)	9.96	<0.01
C3: Various	0.27 (0.90)	-0.30 (0.94)	0.00 (1.10)	2.40	0.10
Parasites	-0.40 (0.67)	-0.19 (1.10)	0.95 (0.63)	10.59	<0.01
Inflammation	-0.29 (0.83)	0.21 (1.01)	0.18 (1.12)	1.27	0.29
MSOL	0.64 (1.06)	-0.16 (0.67)	-0.82 (0.45)	18.27	<0.01
HSOL	1.07 (0.55)	-0.56 (0.44)	-0.94 (0.28)	134.69	<0.01
TSOL	-0.23 (1.00)	-0.13 (0.69)	0.57 (1.15)	4.33	0.02
Age	20.2 (15.8)	19.7 (13.1)	19.3 (15.5)	0.02	0.98
Sex (% male)	33%	41%	56%	$X^2 = 2.30$	0.32

Values are means (standard deviations) except for sex. All values except sex and age are standardized and centered.

Table S4. Variance components estimated by random effects

Variable	Community	Household	Individual
Total Disgust	0.65	0.03	0.31
C1: Contagion	0.52	0.06	0.42
C2: Food	0.69	0.02	0.29
C3: Various	0.26	0.03	0.71
Parasites	0.69	0.15	0.16
Inflammation	0.27	0.11	0.62
MSOL	0.60	0.37	0.03
HSOL	0.91	0.09	0.00
TSOL	0.52	0.31	0.17

Table S5. Simplified models with household and community level random intercepts

Dependent	Independent	Estimate	l-95% CI	u-95% CI
Inflammation	Intercept	-0.20	-0.99	0.58
	Age	0.01	-0.00	0.03
	Sex	-0.16	-0.62	0.31
	Total Disgust	-0.34	-0.60	-0.07
	sd(Household)	0.29	0.01	0.71
	sd(Community)	0.41	0.01	1.87
Parasites	Intercept	0.04	-1.58	1.61
	Age	-0.00	-0.02	0.01
	Sex	0.23	-0.14	0.59
	Total Disgust	-0.03	-0.24	0.18
	sd(Household)	0.57	0.34	0.85
	sd(Community)	1.25	0.30	3.64

Table S6. Disgust and Inflammation or Parasites

		Model	
Dependent	**Independent**	**Inflammation**	**Parasites**
Infection	Intercept	-0.16 (-0.59, 0.28)	0.00 (-0.34,0.33)
Disgust	Intercept	0.11 (-0.27, 0.49)	0.11 (-0.27,0.49)
HHInfection	Intercept	-0.01 (-0.25, 0.23)	0.01 (-0.28,0.29)
HHDisgust	Intercept	-0.01 (-0.24, 0.23)	-0.01 (-0.23,0.22)
Infection	Age	0.01 (0.00, 0.03)	0.00 (-0.02,0.01)
Infection	Sex	-0.20 (-0.68, 0.28)	0.22 (-0.16,0.61)
Infection	Disgust	-0.31 (-0.56,-0.06)	-0.06 (-0.26,0.15)
Infection	HHDisgust	-0.21 (-0.58, 0.16)	0.10 (-0.18,0.38)
Infection	HHInfection	0.09 (-0.30, 0.45)	0.70 (0.46,0.93)
Infection	ViInfection	-0.06 (-0.35, 0.22)	0.28 (-0.09,0.66)
Disgust	Age	-0.01 (-0.02, 0.01)	-0.01 (-0.02,0.01)
Disgust	Sex	-0.04 (-0.46, 0.38)	-0.04 (-0.46,0.38)
Disgust	HHDisgust	0.26 (-0.06, 0.59)	0.26 (-0.06,0.59)
Disgust	ViDisgust	0.67 (0.23, 1.11)	0.68 (0.24,1.12)
HHInfection	HHDisgust	-0.34 (-0.73, 0.06)	-0.18 (-0.39,0.02)
HHInfection	ViInfection	-0.04 (-0.33, 0.24)	0.50 (0.00,0.99)
HHDisgust	ViDisgust	0.58 (0.19, 0.96)	0.61 (0.22,1.00)
HHInfection	sd(Household)	0.50 (0.25, 0.80)	0.73 (0.54,1.00)
HHDisgust	sd(Household)	0.53 (0.37, 0.75)	0.53 (0.36,0.76)
	cor(Household)	-0.34 (-0.82, 0.37)	0.03 (-0.42,0.47)

Model Formula:
Infection ~ Age + Sex + PDSTotal + HHPDSTotal + HHInfection + ViInfection
PDSTotal ~ Age + Sex + HHPDSTotal + ViPDSTotal
HHInfection ~ HHPDSTotal + ViInfection + (1 | p | Household)
HHPDSTotal ~ ViPDSTotal + (1 | p | Household)
Infection = Inflammation or Parasites, as indicated. Disgust = Total Disgust
HH = Household mean, excluding target individual. Vi = Village mean, excluding target household. Items below the grey bar are group level effects for Household

Table S7. Disgust and Inflammation or Parasites with Market Integration Variables

		Model	
Dependent	**Independent**	**Inflammation**	**Parasites**
Infection	Intercept	-0.17 (-1.19, 0.81)	0.04 (-1.02,1.06)
Disgust	Intercept	0.06 (-1.23, 1.44)	0.06 (-1.26,1.44)
HHInfection	Intercept	0.00 (-0.90, 0.90)	0.03 (-1.38,1.48)
HHDisgust	Intercept	-0.01 (-0.93, 0.82)	0.00 (-0.87,0.86)
Infection	Age	0.01 (-0.01, 0.03)	-0.01 (-0.02,0.01)
Infection	Sex	-0.19 (-0.66, 0.29)	0.20 (-0.19,0.58)
Infection	Disgust	-0.34 (-0.62,-0.06)	-0.02 (-0.23,0.20)
Infection	HHDisgust	-0.25 (-0.70, 0.21)	0.12 (-0.19,0.43)
Infection	HHInfection	0.03 (-0.36, 0.41)	0.66 (0.41,0.91)
Infection	MSOL	0.06 (-0.28, 0.39)	-0.15 (-0.39,0.10)
Infection	TSOL	-0.16 (-0.42, 0.11)	0.14 (-0.07,0.36)
Infection	HOUSE	0.04 (-0.37, 0.48)	0.04 (-0.29,0.40)
Disgust	Age	0.00 (-0.02, 0.01)	0.00 (-0.02,0.01)
Disgust	Sex	-0.04 (-0.46, 0.37)	-0.04 (-0.46,0.37)
Disgust	HHDisgust	0.06 (-0.30, 0.43)	0.07 (-0.30,0.43)
Disgust	MSOL	0.26 (-0.01, 0.53)	0.26 (-0.01,0.54)
Disgust	TSOL	-0.07 (-0.30, 0.15)	-0.07 (-0.29,0.15)
Disgust	HOUSE	0.05 (-0.41, 0.47)	0.05 (-0.40,0.47)
HHInfection	HHDisgust	-0.31 (-0.75, 0.14)	-0.13 (-0.34,0.07)
HHInfection	MSOL	0.03 (-0.25, 0.30)	0.13 (-0.12,0.37)
HHInfection	TSOL	-0.09 (-0.29, 0.11)	-0.11 (-0.26,0.05)
HHInfection	HOUSE	-0.08 (-0.51, 0.35)	-0.41 (-0.96,0.11)
HHDisgust	MSOL	0.15 (-0.09, 0.37)	0.15 (-0.08,0.37)
HHDisgust	TSOL	0.06 (-0.10, 0.20)	0.07 (-0.08,0.22)
HHDisgust	HOUSE	0.28 (-0.10, 0.61)	0.28 (-0.11,0.62)
HHInfection	sd(Household)	0.52 (0.25, 0.83)	0.75 (0.54,1.05)
HHDisgust	sd(Household)	0.46 (0.29, 0.67)	0.46 (0.29,0.68)
Infection	sd(Community)	0.59 (0.02, 2.47)	0.63 (0.02,2.58)
Disgust	sd(Community)	0.91 (0.03, 3.25)	0.91 (0.04,3.25)
HHInfection	sd(Community)	0.54 (0.01, 2.30)	1.06 (0.08,3.39)
HHDisgust	sd(Community)	0.54 (0.01, 2.33)	0.54 (0.01,2.32)

Model Formula:

Infection ~ Age + Sex + PDSTotal + HHPDSTotal + HHInfection + +(1 | q | Village) + MSOL + TSOL + HOUSE

PDSTotal ~ Age + Sex + HHPDSTotal + (1 | q | Village) + MSOL + TSOL + HOUSE

HHInfection ~ HHPDSTotal + (1 | q | Village) + (1 | p | Household) + MSOL + TSOL + HOUSE

HHPDSTotal ~ (1 | q | Village) + (1 | p | Household) + MSOL + TSOL + HOUSE

Infection = Inflammation or Parasites, as indicated. Disgust = Total Disgust

HH = Household mean, excluding target individual. Vi = Village mean, excluding target household

Items below the grey bar are group level effects for Household

Table S8. Three Disgust Components and Inflammation with Market Integration Variables

		Model		
Dependent	**Independent**	**C1:Contagion**	**C2:Food**	**C3:Other**
Inflam	Intercept	-0.29 (-2.28, 1.68)	-0.31 (-2.65,1.94)	-0.24 (-1.54,1.12)
Disgust	Intercept	-0.06 (-3.07, 2.83)	-0.10 (-3.32,3.10)	0.39 (-0.72,1.52)
HHInflam	Intercept	0.00 (-1.63, 1.72)	0.05 (-1.58,1.89)	0.01 (-1.14,1.16)
HHDisgust	Intercept	-0.08 (-2.50, 2.38)	0.04 (-1.92,1.83)	0.01 (-1.06,1.07)
Inflam	Age	0.02 (0.00, 0.03)	0.02 (0.00,0.04)	0.02 (0.00,0.03)
Inflam	Sex	-0.16 (-0.64, 0.32)	-0.13 (-0.62,0.35)	-0.08 (-0.55,0.40)
Inflam	Disgust	-0.39 (-0.66,-0.12)	-0.29 (-0.57,0.01)	0.11 (-0.14,0.36)
Inflam	HHDisgust	-0.21 (-0.66, 0.23)	-0.13 (-0.55,0.27)	-0.19 (-0.60,0.23)
Inflam	HHInflam	0.00 (-0.42, 0.41)	0.05 (-0.40,0.49)	0.20 (-0.20,0.56)
Inflam	MSOL	0.00 (-0.29, 0.29)	0.05 (-0.29,0.39)	-0.09 (-0.40,0.21)
Inflam	TSOL	-0.17 (-0.43, 0.11)	-0.10 (-0.37,0.18)	-0.10 (-0.38,0.18)
Inflam	HOUSE	0.11 (-0.33, 0.61)	0.05 (-0.41,0.58)	0.06 (-0.40,0.63)
Disgust	Age	0.00 (-0.02, 0.01)	0.00 (-0.01,0.02)	-0.01 (-0.03,0.00)
Disgust	Sex	0.12 (-0.30, 0.54)	0.01 (-0.42,0.43)	-0.35 (-0.83,0.12)
Disgust	HHDisgust	0.17 (-0.20, 0.55)	-0.16 (-0.51,0.20)	-0.17 (-0.57,0.23)
Disgust	MSOL	0.16 (-0.11, 0.44)	0.44 (0.14,0.73)	-0.05 (-0.35,0.26)
Disgust	TSOL	-0.09 (-0.32, 0.13)	-0.02 (-0.26,0.22)	0.05 (-0.22,0.31)
Disgust	HOUSE	-0.04 (-0.50, 0.39)	-0.12 (-0.57,0.32)	0.27 (-0.18,0.69)
HHInflam	HHDisgust	-0.40 (-0.86, 0.04)	-0.26 (-0.60,0.06)	0.26 (-0.05,0.57)
HHInflam	MSOL	-0.01 (-0.28, 0.26)	0.01 (-0.28,0.30)	-0.01 (-0.32,0.33)
HHInflam	TSOL	-0.12 (-0.31, 0.07)	-0.08 (-0.27,0.12)	-0.12 (-0.33,0.08)
HHInflam	HOUSE	-0.01 (-0.45, 0.43)	-0.07 (-0.50,0.40)	-0.16 (-0.62,0.37)
HHDisgust	MSOL	0.09 (-0.15, 0.32)	0.23 (-0.01,0.45)	-0.02 (-0.25,0.22)
HHDisgust	TSOL	0.00 (-0.15, 0.15)	0.12 (-0.04,0.28)	0.08 (-0.09,0.24)
HHDisgust	HOUSE	0.20 (-0.21, 0.60)	0.08 (-0.30,0.42)	0.27 (-0.09,0.64)
HHInflam	sd(Household)	0.49 (0.20, 0.82)	0.53 (0.24,0.86)	0.65 (0.39,0.98)
HHDisgust	sd(Household)	0.51 (0.33, 0.74)	0.36 (0.19,0.57)	0.42 (0.21,0.67)
Inflam	sd(Community)	1.12 (0.02, 5.84)	1.30 (0.03,6.63)	0.83 (0.02,3.17)
Disgust	sd(Community)	1.83 (0.07, 8.16)	2.07 (0.28,8.56)	0.70 (0.03,2.70)
HHInflam	sd(Community)	0.94 (0.02, 5.18)	0.97 (0.02,5.31)	0.72 (0.02,2.89)
HHDisgust	sd(Community)	1.48 (0.07, 6.80)	1.09 (0.03,5.59)	0.69 (0.03,2.68)

Model Formula:

Inflam ~ Age + Sex + Disgust + HHDisgust + HHInflam + (1 | q | Village) + MSOL + TSOL + HOUSE

Disgust ~ Age + Sex + Disgust + (1 | q | Village) + MSOL + TSOL + HOUSE

HHInflam ~ HHDisgust + (1 | q | Village) + (1 | p | Household) + MSOL + TSOL + HOUSE

HHDisgust ~ (1 | q | Village) + (1 | p | Household) + MSOL + TSOL + HOUSE

Disgust = The component indicated for each model (C1-C3)

HH = Household mean, excluding target individual. Vi = Village mean, excluding target household

Items below the grey bar are group level effects for Household

Table S9. Three Disgust Components and Parasites with Market Integration Variables

		Model		
Dependent	**Independent**	**C1:Contagion**	**C2:Food**	**C3:Other**
Parasites	Intercept	0.04 (-1.92,1.99)	0.03 (-2.11,2.07)	0.05 (-0.94,1.05)
Disgust	Intercept	-0.06 (-3.12,2.92)	-0.11 (-3.34,3.05)	0.39 (-0.70,1.54)
HHParasites	Intercept	0.04 (-2.76,2.87)	0.05 (-3.03,3.10)	0.04 (-1.36,1.45)
HHDisgust	Intercept	-0.06 (-2.27,2.14)	0.04 (-2.04,2.06)	-0.01 (-1.01,1.00)
Parasites	Age	0.00 (-0.02,0.01)	0.00 (-0.02,0.01)	-0.01 (-0.02,0.01)
Parasites	Sex	0.14 (-0.24,0.52)	0.13 (-0.24,0.51)	0.17 (-0.21,0.56)
Parasites	Disgust	-0.07 (-0.28,0.16)	-0.02 (-0.23,0.20)	0.06 (-0.14,0.26)
Parasites	HHDisgust	0.07 (-0.26,0.40)	0.23 (-0.06,0.53)	-0.05 (-0.35,0.26)
Parasites	HHParasites	0.66 (0.39,0.93)	0.64 (0.36,0.90)	0.68 (0.43,0.92)
Parasites	MSOL	-0.12 (-0.36,0.13)	-0.18 (-0.44,0.08)	-0.13 (-0.37,0.10)
Parasites	TSOL	0.13 (-0.08,0.34)	0.10 (-0.10,0.31)	0.15 (-0.06,0.36)
Parasites	HOUSE	0.08 (-0.26,0.44)	0.08 (-0.26,0.46)	0.08 (-0.25,0.45)
Disgust	Age	0.00 (-0.02,0.01)	0.00 (-0.01,0.02)	-0.01 (-0.03,0.00)
Disgust	Sex	0.12 (-0.30,0.54)	0.01 (-0.41,0.43)	-0.35 (-0.82,0.12)
Disgust	HHDisgust	0.17 (-0.20,0.54)	-0.16 (-0.51,0.20)	-0.17 (-0.57,0.22)
Disgust	MSOL	0.16 (-0.11,0.43)	0.44 (0.14,0.74)	-0.04 (-0.34,0.27)
Disgust	TSOL	-0.09 (-0.31,0.13)	-0.02 (-0.25,0.22)	0.04 (-0.21,0.30)
Disgust	HOUSE	-0.04 (-0.49,0.38)	-0.12 (-0.58,0.31)	0.26 (-0.16,0.68)
HHParasites	HHDisgust	-0.11 (-0.33,0.11)	-0.15 (-0.31,0.01)	0.01 (-0.16,0.17)
HHParasites	MSOL	0.15 (-0.09,0.39)	0.13 (-0.12,0.37)	0.13 (-0.11,0.37)
HHParasites	TSOL	-0.11 (-0.25,0.03)	-0.10 (-0.23,0.04)	-0.11 (-0.24,0.03)
HHParasites	HOUSE	-0.39 (-0.92,0.12)	-0.39 (-0.94,0.13)	-0.43 (-0.93,0.06)
HHDisgust	MSOL	0.09 (-0.15,0.32)	0.24 (0.01,0.46)	0.02 (-0.21,0.25)
HHDisgust	TSOL	0.00 (-0.15,0.15)	0.12 (-0.04,0.29)	0.08 (-0.09,0.25)
HHDisgust	HOUSE	0.26 (-0.17,0.67)	0.02 (-0.37,0.38)	0.19 (-0.18,0.55)
HHParasites	sd(Household)	0.73 (0.51,1.04)	0.75 (0.54,1.06)	0.76 (0.55,1.07)
HHDisgust	sd(Household)	0.52 (0.34,0.76)	0.35 (0.18,0.56)	0.43 (0.19,0.67)
Parasites	sd(Community)	1.08 (0.02,5.95)	1.16 (0.02,6.07)	0.61 (0.02,2.51)
Disgust	sd(Community)	1.87 (0.08,8.28)	2.04 (0.27,8.29)	0.70 (0.03,2.68)
HHParasites	sd(Community)	1.66 (0.08,7.29)	1.88 (0.13,8.02)	1.03 (0.08,3.35)
HHDisgust	sd(Community)	1.34 (0.05,6.34)	1.21 (0.04,6.12)	0.63 (0.02,2.60)

Model Formula:

Parasites ~ Age + Sex + Disgust + HHDisgust + HHParasites + +(1 | q | Village) + MSOL + TSOL + HOUSE

Disgust ~ Age + Sex + HHDisgust + (1 | q | Village) + MSOL + TSOL + HOUSE

HHParasites ~ HHDisgust + (1 | q | Village) + (1 | p | Household) + MSOL + TSOL + HOUSE

HHDisgust ~ (1 | q | Village) + (1 | p | Household) + MSOL + TSOL + HOUSE

Disgust = The component indicated for each model (C1-C3)

HH = Household mean, excluding target individual. Vi = Village mean, excluding target household

Items below the grey bar are group level effects for Household

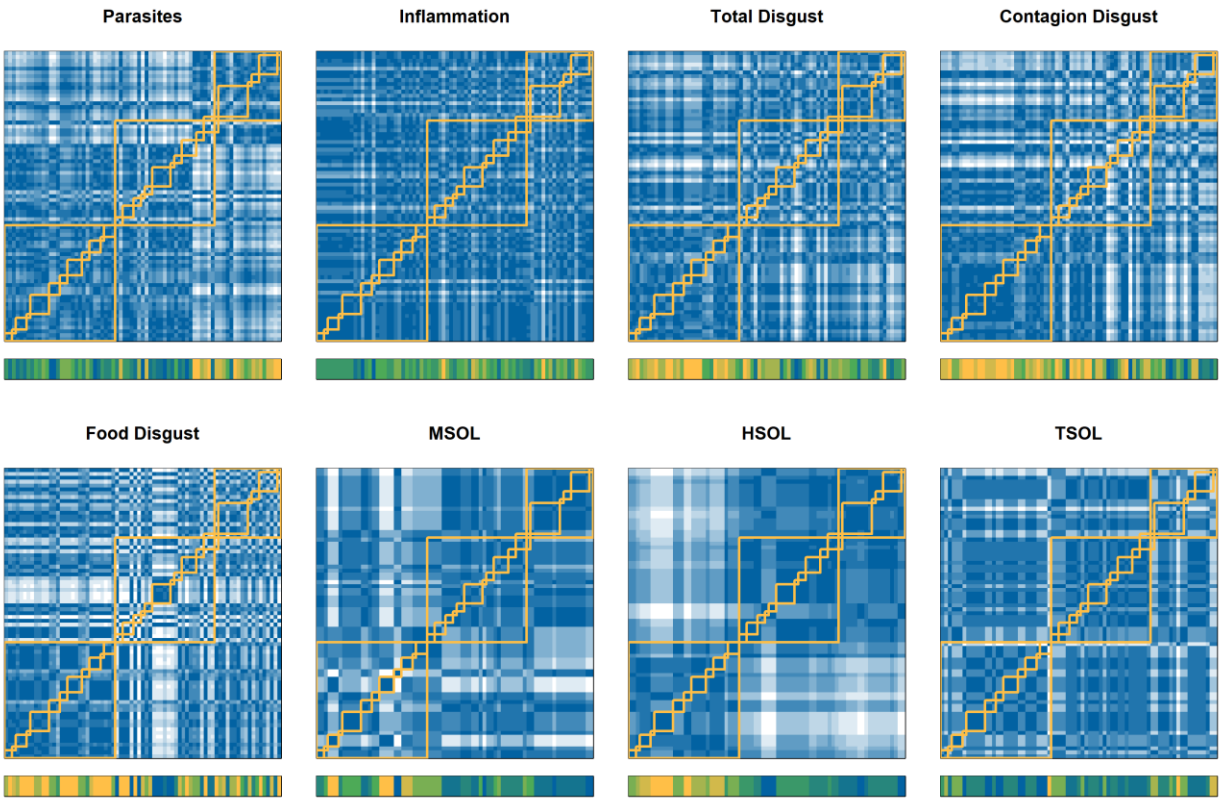

Figure S1. Similarity matrix plots for all individuals in the dataset showing household and community clustering. Each x or y line indicates an individual. Each cell represents the similarity between two individuals, with darker blue cells indicating more similarity and yellow indicating more divergence. Individuals are ordered by household and community, and households and communities are outlined in yellow. Squares of blue indicate clusters of similarity, while disordered patterns indicate independence. Wider bands in SOL measures indicate that these measures were largely collected at the household level. The colored bars below the plots show the relative absolute value of the measure for the individual in that column (yellow=high, green=intermediate, blue=low).

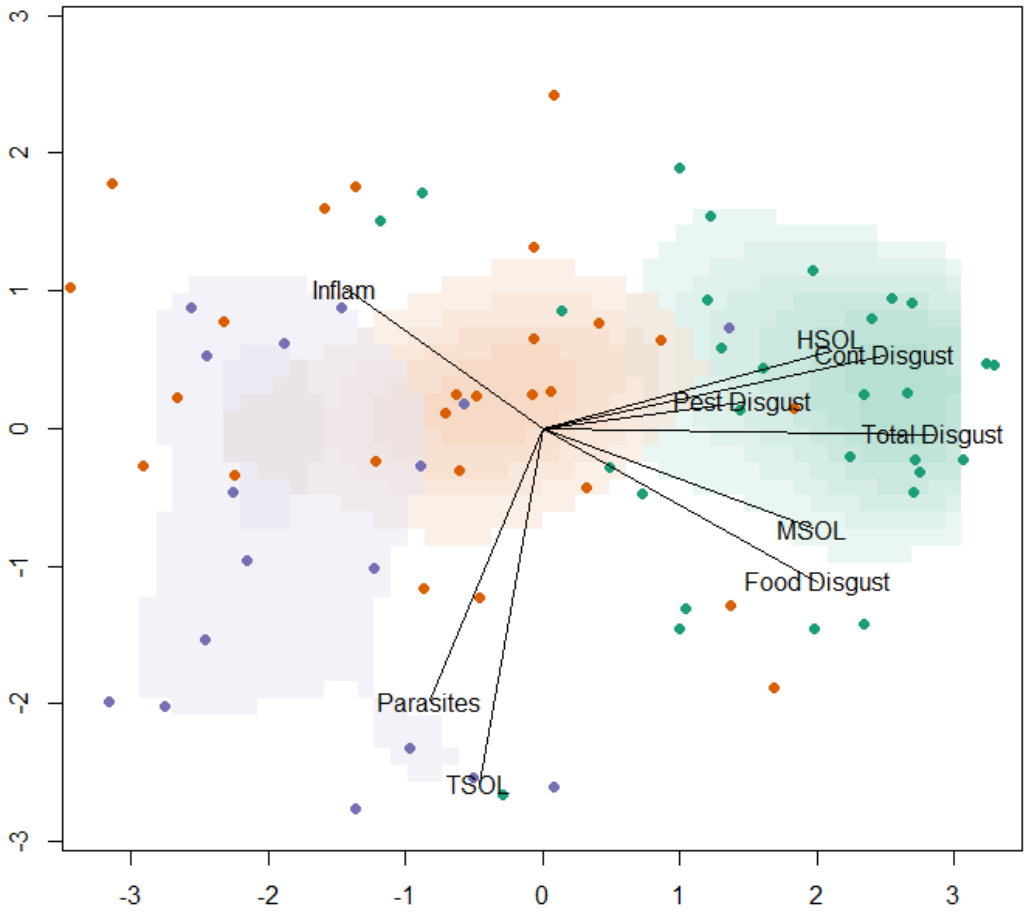

Figure S2. Multidimensional scaling based on variables of interest. Points are individual participants. Colors indicate the three communities, with shading indicating the density function for that community (green=community 1, orange=community 2, purple=community 3). Note, figure does not control for age, sex, or household clustering.

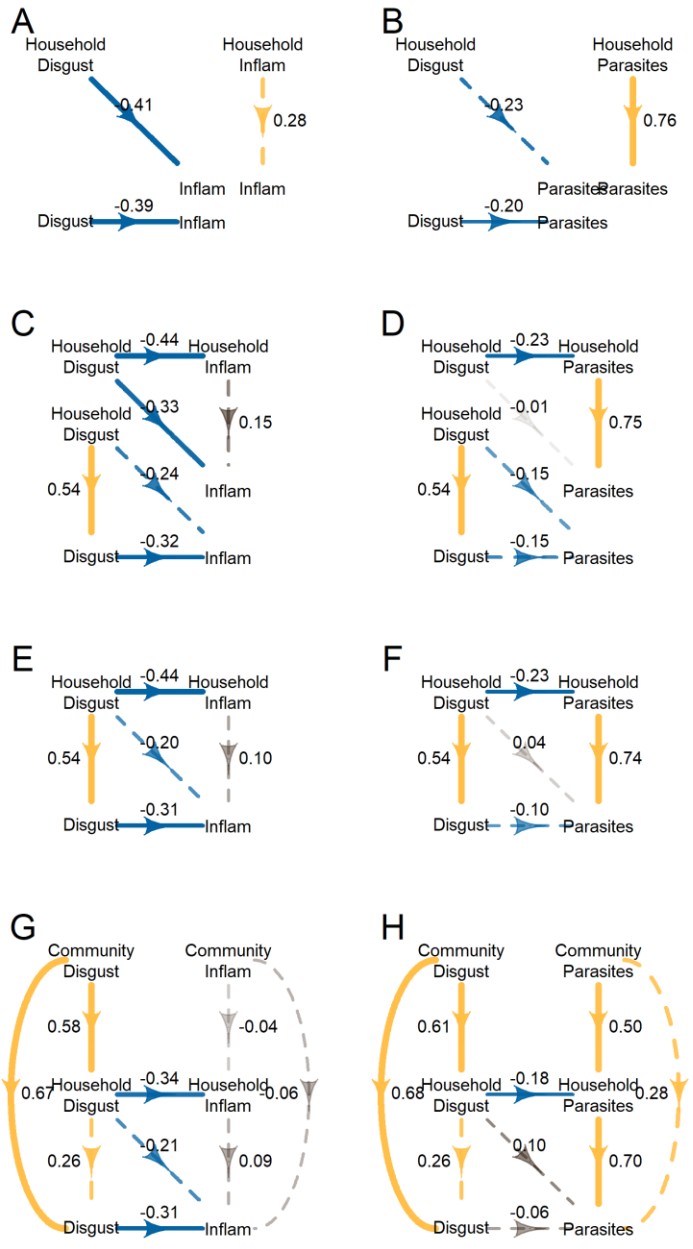

Figure S3. Associations between infection and disgust variables, A-D) Simple associations. E-H) Tests of whether family infection level or individual disgust mediate associations between family disgust and infection. I-L) Combined models showing associations between family level and individual level variables. M-P) Complete models with community level variables. Line type indicates the posterior certainty: solid line, more than 95% of the posterior is on one side of zero; long dashes, <95% of the posterior is on one side of zero. Color indicates the direction of the effect: blue=negative, yellow=positive. Effects with less than 80% of the posterior on one side of zero are shaded grey-white, proportional to the credibility intervals.

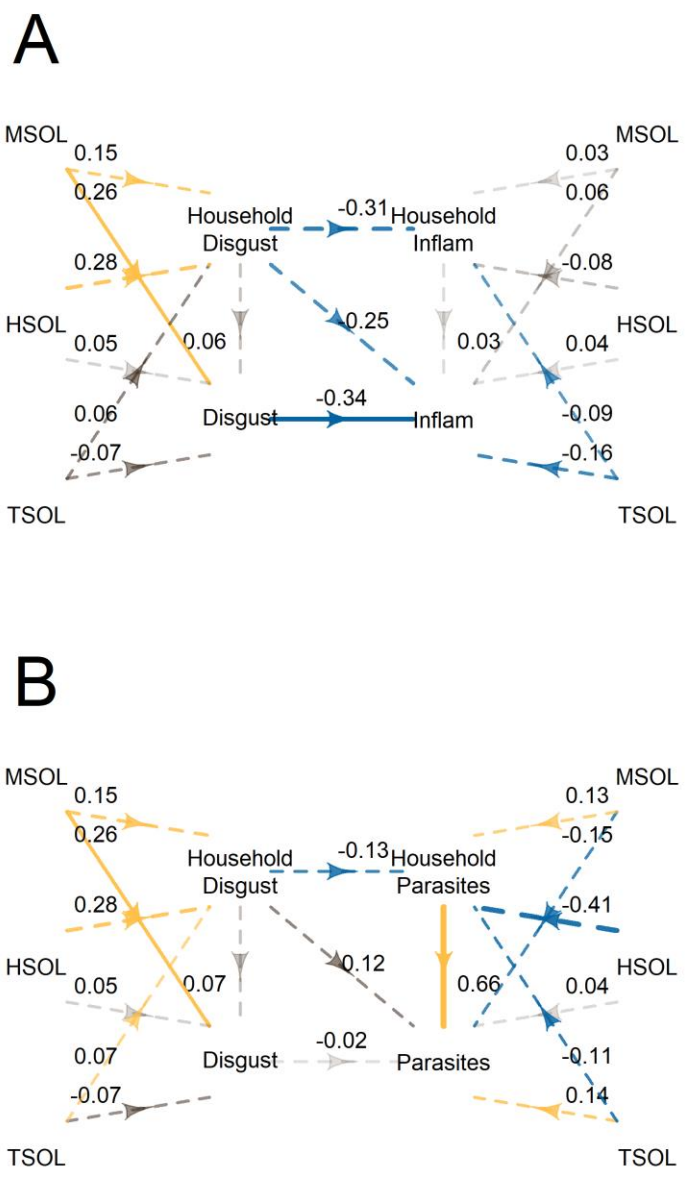

Figure S4. Associations between style of life variables, infection, and disgust. Note, style of life variables are shown twice to improve graph organization. Models control for Community with a random effect term. Multivariate models were specified as: Infection ~ Age + Sex + Disgust + HHDisgust + HHInfection + MSOL + TSOL + HOUSE + (1|q|Community); Disgust ~ Age + Sex + HHDisgust + MSOL + TSOL + HOUSE +(1|q|Community); HH Infection ~ HH Disgust + MSOL + TSOL + HOUSE) + (1|p|Family) + (1|q|Community); HH Disgust ~ MSOL + TSOL + HOUSE + (1|q|Community) + (1|p|Family). Line type indicates the posterior certainty: solid line, more than 95% of the posterior is on one side of zero; long dashes, <95% of the posterior is on one side of zero. Color indicates the direction of the effect: blue=negative, yellow=positive. Effects with less than 80% of the posterior on one side of zero are shaded grey-white, proportional to the credibility intervals.

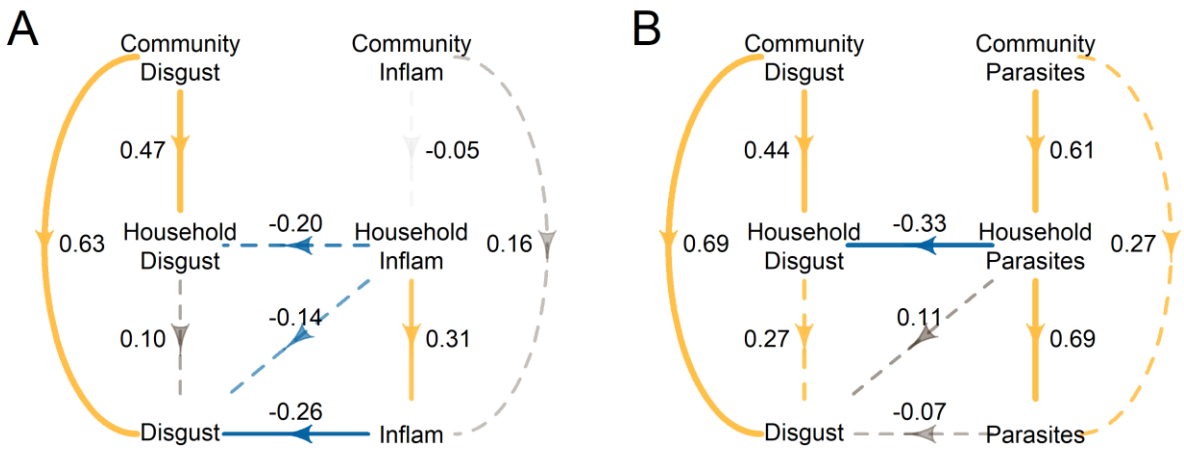

Figure S5. Models comparable to Figure 2, but with a reversed relationship between infection and disgust. Line thickness is proportional to the mean posterior effect size. Line type indicates the posterior certainty: solid line, more than 95% of the posterior is on one side of zero; long dashes, <95% of the posterior is on one side of zero. Color indicates the direction of the effect: blue=negative, yellow=positive. Effects with less than 80% of the posterior on one side of zero are shaded grey-white, proportional to the credibility intervals.

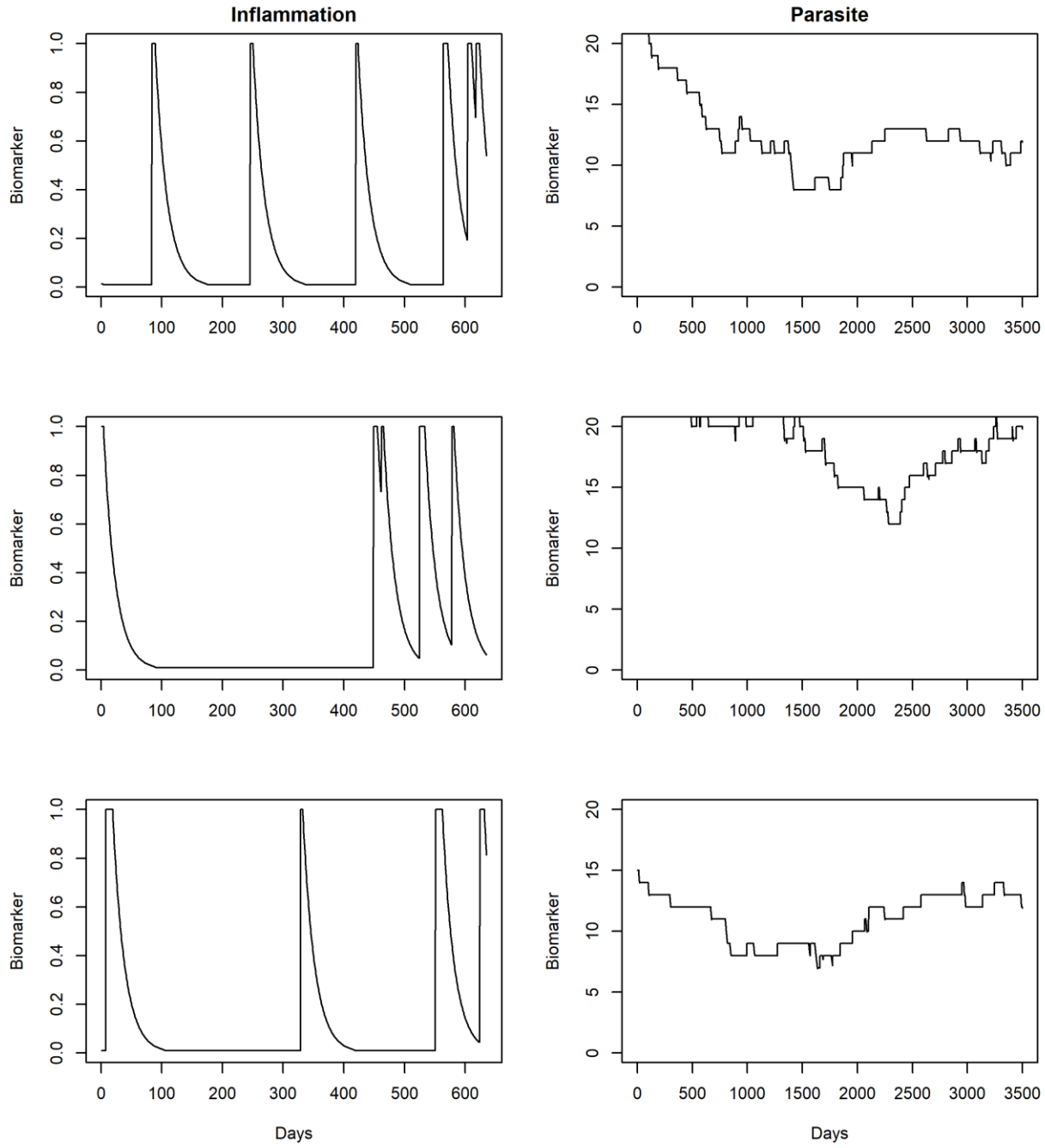

Figure S6. Example simulated biomarker responses to short-term inflammatory infections (left) and long-term microparasite infections (right). Units on the y-axis are arbitrary. Note that the long-term infection is simulated for a longer period of time, and that the simulation was run for 365 days (short-term) or 3500 days (long term) before recording values (not shown of the graph), in order to equilibrate baseline biomarker levels.

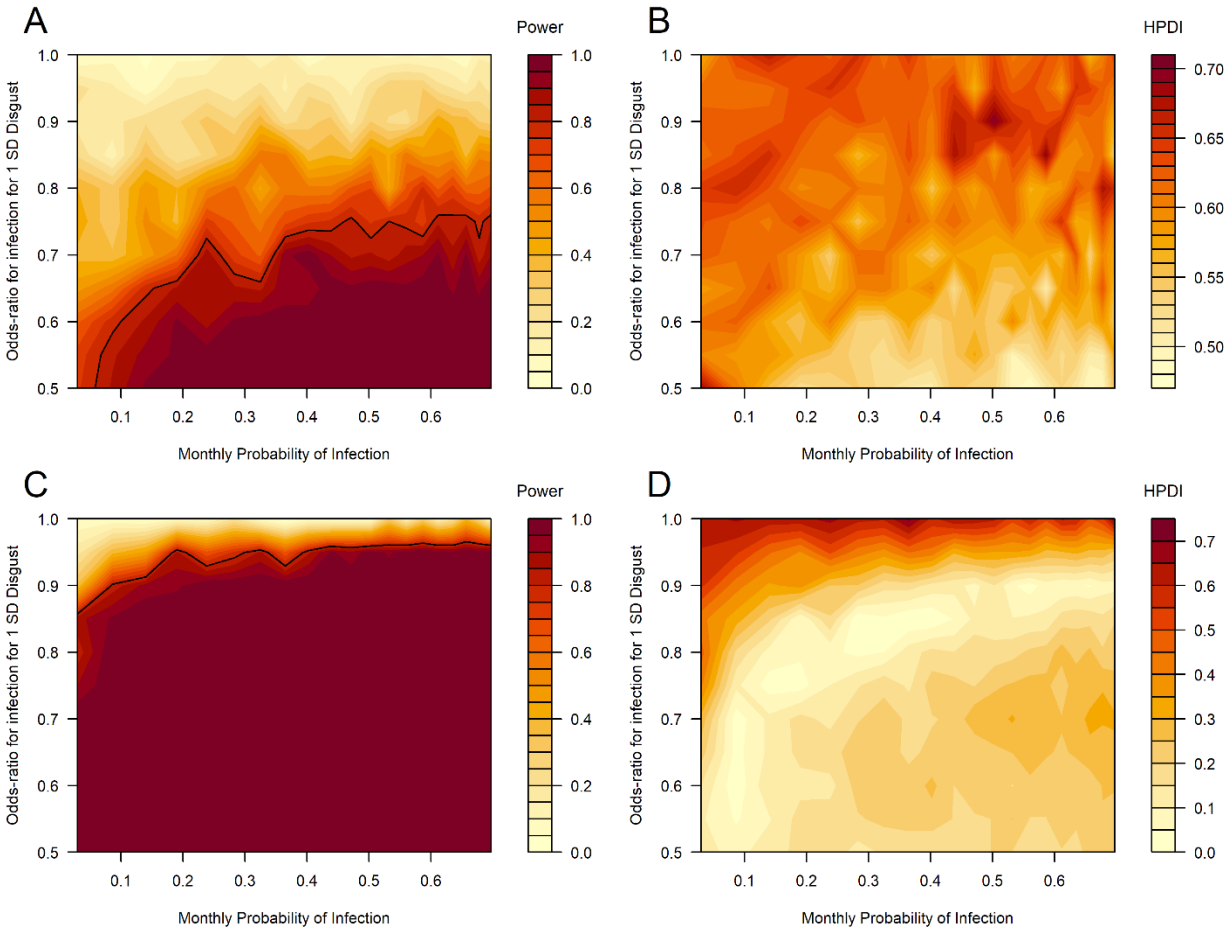

Figure S7. Power to credibly detect an effect given different probabilities of infection and different effect sizes for disgust. In the simulation, 75 individuals were sampled from a pool of 750, with sampling repeated 50 times. A and C show the proportion of simulated samples that produced a posterior estimate in which ≥90% of the posterior distribution showed a protective effect of disgust. B and D show the average range of the 95% highest posterior density interval, with lower values indicating more certainty in the posterior. A and B show an "inflammatory" infection with an average duration of 7 days, as in the left of Figure SX. C and D show a "parasitic" infection with an average duration of 2000 days, stacking infections (i.e. additional exposure increases parasitic load) and a slow return of the biomarker to baseline (right of Figure SX).

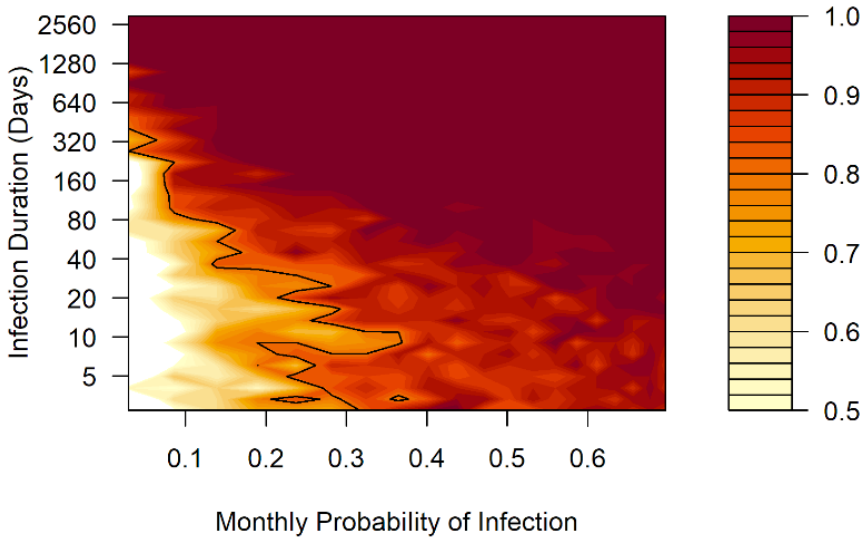

Figure S8. Simulated effect of infection duration and risk of infection on the power to detect a credible effect with a disgust odds-ratio of infection of 0.7 / SD Disgust. Black line shows 80% of trials resulted in posterior estimates with ≥90% of the posterior distribution showing a protective effect of disgust.

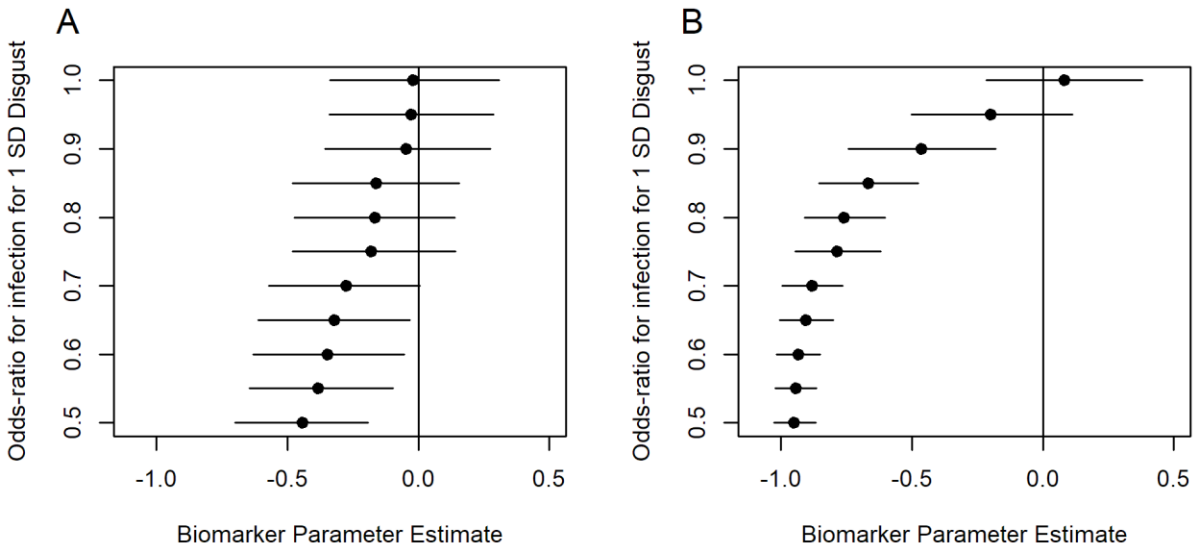

Figure S9. Simulated relationship between the initial effect of disgust on infection and the posterior parameter estimate for the effect of disgust on biomarker level. A) Parameter for a short-term "inflammatory" infection. B) Parameter for a long-term "parasite" infection. Lines show the 95% highest posterior density interval. Simulated biomarkers were logged and standardized before model fitting. and shown for daily infection risk = 0.015 (36% infection probability per month).

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