The human behavioural immune system is a product of cultural evolution

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Abstract
Disgust is a basic emotion that serves to avoid contaminants, and is central to the behavioural immune system. While disgust-motivated avoidance occurs in bonobos and chimpanzees, humans show uniquely high levels of contamination sensitivity. Current theory postulates that human disgust is primarily a genetic adaptation, side-lining social transmission and denying parental modelling. Here, we test whether this strong view is warranted by simulating 100,000 years of cultural and biological evolution in various scenarios. We modelled disgust as a trait that governed the extent to which individuals forewent potentially contaminated nutrition. This lowered their risk of gastrointestinal illness, at the cost of increasing starvation risk and birth-interval. As environmental levels and costs of contamination rose, so did relative fitness of high-disgust individuals. Evolutionary shifts in disgust as polygenic trait occurred as a consequence of germ-cell mutations, but were most prominent in populations with high initial genetic variance. Crucially, cultural transmission between generations operated at a higher rate, even if parental modelling was eliminated. This study serves not only as evidence of cultural evolution shaping the behavioural immune system, but also as an illustration of emerging theories that paint affective and cognitive mechanisms as socially transmitted rather than biologically determined functions.
Background

Disgust is a basic emotion (1) evoked by “something revolting” through taste, smell, touch, or sight (2). Because of its role in avoiding potential contaminants (3–6), disgust is an important part of the \textit{behavioural immune system} (7,8). This system promotes disease avoidance not only by distancing from bodily effluvia (9), but also through more complex behavioural patterns in response to e.g. the threat of COVID-19 (10). Crucially, disgust helps to prevent oral incorporation of potential contaminants (4,6).

While our closest living relatives (11) show some disgust-motivated avoidance (12,13), 30-50\% of bonobos still feed on banana slices placed upon or adjacent to faeces (12), and around 50\% of chimpanzees consume food off faecal replicas (13). By contrast, humans are reluctant to interact even with known replicas (e.g. fudge moulded as dog faeces) (14). While originally observed in industrialised populations, similar contamination sensitivity is found in hunter-gatherers (15). Thus, high levels of disgust appear uniquely human (3).

Contemporary theories disagree on how uniquely sensitive disgust developed in humans. One leading theory argues for both biological and cultural evolution (3,7,16), the latter through parents socially transmitting disgust to their offspring. Another theory postulates that disgust is primarily a genetic adaptation (6,17), side-lining social transmission (18) and denying parental modelling (19).

Evidence for disgust socialisation comes from its surprising absence in early childhood (3,20,21), resemblance between family members (21,22), and maternal moderation of children’s disgust (23); although these findings could be reconciled with a genetic account (6,18). Evidence for heritability comes from twin studies that found no shared-environmental influences (24–26). However, these are predicated upon the implausible assumptions that siblings share the same environment (27,28) and that environment and heritability are unrelated (29); and indeed generally overestimate genetic heritability (30–32). In sum, no conclusive evidence exists towards either evolutionary account of the behavioural immune system’s disgust-driven pathogen avoidance.

Here, we directly compare genetic and cultural transmission in agent-based simulations of 100 000 years of evolution. In our model, high levels of disgust reduced mortality associated with eating contaminated food. However, it did so at the cost of reducing nutritional intake, which in turn increased malnutrition-related mortality and the duration of postpartum amenorrhoea.

We modelled disgust as a polygenic trait passed down through sexual reproduction, and as a cultural trait imperfectly copied from previous generations (33–35). In simulated environments, we systematically varied the available nutrition, and the level and cost of contamination. Parameters in our simulation were informed by research on contemporary hunter-gatherer societies.
Evolution of the behavioural immune system

Methods

Experimental design
We tested how culturally and genetically transmitted traits behaved under different mechanisms and in different environments. Due to the difficulties associated with the experimental manipulation of human evolution, we employed agent-based simulations. Each simulation comprised a combination of specific parameters that governed the initial genetic variance, the parental influences in cultural transmission, nutritional availability, environmental contamination, and the deadliness of contaminated nutrition. The effect of these parameters was assessed in reference to control conditions without selection pressures. In such control environments, there was no environmental contamination or mortality cost to contamination.

Trait inheritance
Disgust was modelled as polygenic trait (mimicking other psychological traits, e.g. (36)), and transmitted through sexual reproduction. The genotype was initialised with 60% avoidance of contaminated nutrition (mirroring bonobo (12)); and a variance of $2n\mu S=0.0132$ (37), with $n=10$ loci contributing equally to the trait, mutation rate per locus $\mu=6.6e^{-5}$, and stabilising selection $S=0.1$ (38). Germ cell mutation probability was $1.1e^{-8}$ per position per haploid genome (39). Assuming ~6000 bases (size of AURKB, implicated in polygenic anxiety (36)), this translated to $1-(1-1.1e^{-8})^{6000}=6.6e^{-5}$ per gene. This is likely a high estimate, because de novo mutations are not equiprobable across the genome (40), so we ran controls with lower initial variance ($1.32e^{-3}$).

When germ cell mutations occurred, allele deviations were sampled from a normal distribution with $\mu=0$ and $\sigma=0.025$. Beneficial and detrimental mutations were thus equally likely, and smaller were more likely than larger fitness effects. For context, in a monogenic scenario with 11088 kJ daily nutrition, 0.1 contamination probability, $5.1114e^{-9}$ probability of death per contaminated kJ, and alleles $D=1.0$ and $d=0.975$ ($=1-\sigma_{\text{mutation}}$), selection coefficients were roughly in the middle of the typical range (41): $s_{DD}=1.62e^{-3}$, $s_{Dd}=7.51e^{-4}$, and $s_{dd}=0$ (best relative fitness). This approximated typical distributions of mutational effects, which are wide and unpredictable (42).

Disgust was also modelled as culturally transmitted trait, using established models (33–35) in which individuals in generation $t+1$ imperfectly copy trait $s$ from their parents with error $\varepsilon$, or from prior generations $t$ with probability $\lambda$ (Equation 1). The trait started at barely any culturally transmitted disgust avoidance, at $s(t)=0.95$ with a variance of $6.25e^{-4}$. Parameter values were based on empirical findings, with $\varepsilon=0.025$ (35,43) and $\lambda=0.38$ (35).

\begin{equation}
(1) \ s(t+1) = \lambda \bar{s}(t)\left[1+\varepsilon(t)\right] + \left(1-\lambda\right)s(t)\left[1+\varepsilon(t)\right]
\end{equation}
Environments

Environmental nutrition \( \nu \) was set at 6650, 8866, 11083, 13300, or 15516 kilo-Joule (kJ) per day; 60-140% of typical adult male intake ((44), Table 1). The level of contamination \( \eta \) varied from 0 to 50% of available nutrition; and the cost of contamination \( \kappa \) was implemented as independent probability of death per ingested contaminated kJ of 0, 2.0883e-9, 5.1114e-9, 8.2622e-9, 1.1551e-8, or 1.4993e-8. With an intake of 11083 kJ/day and 10% contamination, this corresponded to a probability of death-by-contamination of 0%, 2.75%, 6.6%, and 10.45%, 14.3%, and 18.15% over the average lifespan of 33 years (not counting child mortality). In reality, gastrointestinal illness and fever caused 13.6% (5.5+8.1) and 34.9% (13.2+21.7) of deaths among hunter-gatherer and settled Ache, respectively ((45), Table 5).

Nutritional intake

Individual nutritional intake \( i \) (Equation 2) was the sum of consumed contaminated and uncontaminated nutrition, but never more than daily requirement \( \rho_{\text{male}}=11083 \text{ or } \rho_{\text{female}}=7853 \text{ kJ} \) (44). Uncontaminated intake (Equation 3) was governed by environmental contamination \( \eta \), and daily nutritional availability \( \nu \). Crucially, contaminated intake (Equation 4) was not only determined by \( \eta \) and \( \nu \), but also impacted by trait \( \tau \): the unweighted average of genetic and cultural disgust. (The trait determined the proportion of contaminated nutrition individuals consumed, so lower values indicate higher levels of disgust-motivated avoidance.)

\[
(2) \quad i = \min \left( \rho, i_{\text{contaminated}} + i_{\text{uncontaminated}} \right)
\]

\[
(3) \quad i_{\text{uncontaminated}} = (1 - \eta) \nu
\]

\[
(4) \quad i_{\text{contaminated}} = \tau \eta \nu
\]

Mortality

Mortality (Equation 5) at age \( x \) was computed through the Siler hazard function (Equation 6) (46), disgust-related mortality (Equation 8) weighted by proportion \( \gamma \) (Equation 7); and nutrition-related mortality (Equation 9).

\[
(5) \quad m(x, i) = \gamma f(x, i) + (1 - \gamma) h(x) + n(i)
\]

Siler parameters were based on those found in Hadza, Ache, Hiwi, !Kung, and Agta ((45), Table 2): initial infant mortality \( \alpha_1=0.422 \), infant mortality decline \( \beta_1=1.131 \), general mortality \( \alpha_2=0.013 \), initial adult mortality \( \alpha_3=1.47e-4 \), and adult mortality increase \( \beta_3=0.086 \).

\[
(6) \quad h(x) = \alpha_1 \exp\left(-\beta_1 x\right) + \alpha_2 + \alpha_3 \exp\left(\beta_3 x\right)
\]
Evolution of the behavioural immune system

The proportion of mortality affected by disgust is computed from cost of contamination $\kappa$, environmental contamination $\eta$, and the lowest of daily nutrition $\nu$ or male requirement $\rho_{\text{male}}$.

\begin{equation}
\gamma = 1 - \left( 1 - \kappa \right)^{365.25 \eta \min(\nu, \rho_{\text{male}})}
\end{equation}

Disgust-associated mortality was directly impacted by disgust trait $\tau$. Its gradual emergence over childhood was normally distributed with $\mu=5$ and $\sigma=3$; because children in industrialised nations develop disgust between ages 3 and 5 years (3,21), and because children in hunter-gatherer societies function as “helpers at the nest” (47), with Hadza children engaging in food acquisition and processing from as early as 3.5 years (48).

\begin{equation}
f(x, i) = \Phi\left( \frac{x - \mu}{\sigma} \right) \left( 1 - \left( 1 - \kappa^{365.25 \eta \tau} \right) \right)
\end{equation}

Nutrition-based mortality was computed with nutrition-specific $\alpha_2$, set at -0.001 for male and -0.001 ($\rho_{\text{male}}/\rho_{\text{female}}$) for female individuals.

\begin{equation}
n(i) = \exp\left( \alpha_2, \text{nutrition} \cdot i \right)
\end{equation}

Fertility

While the relationship between dietary restrictions and female fertility is complex, under-nutrition increases birth intervals in hunter-gatherers (49) and forager-horticulturalists (50). Thus, in addition to mortality, nutritional intake impacted postpartum amenorrhoea: The yearly cumulative probability of menstruation recovery was 0, 0.49, 0.59, 0.72, 0.86, and 1 in mothers with sufficient nutrition, and weighted down by insufficient nutrition in years 2 and 3 (50).

Fertility onset was normally distributed with $\mu=16.0$ and $\sigma=2.5$ for female, and $\mu=19.5$ and $\sigma=3.4$ for male individuals (Savannah Pumé, (51), Table 1); and offset with $\mu=45.0$ and $\sigma=2.5$ for female (52,53), and $\mu=55.0$ and $\sigma=3.4$ for male individuals (!Kung and Hadza, (53), Table 1). Fertile individuals were randomly paired, reflecting high marriage rates among e.g. !Kung (54). We did not model polygeny, because of its low prevalence in many hunter-gatherer societies (53–55).

The probability of a baby being born to fertile (and menstruating) pairs was initially set at 0.42, and allowed to vary between 0.32 and 0.52 with yearly steps of 0.002 (up if population size decreased, down if it increased or surpassed 5000). This resulted in about 7 children per mother at completed fertility, mirroring Savannah Pumé (51), Table 1). Sex ratio at birth was kept at 1.076 (Savannah Pumé, (51), Figure 1c); slightly more skewed towards male than in industrialised nations, where birth sex ratios range from 1.031 to 1.067 (56).
**Statistical Analysis**
Each unique combination of simulation parameters was applied in 10 independent runs. In each run, populations were initialised with N=1000, and allowed to grow to N≈5000. Averages and pooled standard deviations were computed over all 10 runs within each simulation, or the subset of runs that did not end in population collapse. Statistical tests were not employed, as they would result in trivial significance due to the high number of total individuals in each simulation (N≈50000). Effect sizes for evolutionary shifts in traits were computed as changes in disgust-approach proportions compared to chance level drift. Chance level was defined as the average change in traits in control conditions without contamination (η=0) and with no cost to contamination (κ=0).

**Data and code availability**
Simulation code and data is available from a public GitHub repository: [https://github.com/esdalmaijer/2020_disgust_evolution](https://github.com/esdalmaijer/2020_disgust_evolution).

**Results**
Over evolutionary time, in an environment with sufficient pressure (environmental nutrition ν=11083 kJ/day, level of contamination η=0.1, cost of contamination κ=1.1551e-8), selection occurred for disgust avoidance (Figure 1A). In a control environment without pressure (ν=11083 kJ/day, η=0, κ=0), lower drift occurred (Figure 1B). The magnitude of drift exceeded chance levels to a greater extent for the culturally than the genetically transmitted trait (Figure 1C). While the degree of selective pressure increased with contamination danger, it was always higher for cultural (Figure 2A) than polygenic (Figure 2B) disgust avoidance (Figure 2C shows the difference).

The increase in disgust-avoidant genotypes was driven by better fitness of individuals already in the more contamination-avoidant tail at population initialisation. Populations initialised with high initial variance (Figure 2C) showed greater genetic adaptation than populations with lower initial variance (Figure 2D). Hence, when genetic evolution was dependent on mutations, cultural evolution rapidly outpaced it.

Additional controls were run to simulate the unlikely scenario of absent parental modelling (conformist bias λ=1). When initialised with high genetic variance, changes (compared to random drift) in culturally and genetically transmitted traits were almost equally matched (Figure 2E), but not when initial genetic variance was less high (Figure 2F). In sum, the rate of cultural evolution was stunted when parents did not directly transmit disgust to their offspring, but the mechanism still drove change more quickly than mutation-driven genetic adaptation.
Evolution of the behavioural immune system

**Fig. 1** – Results of 100,000 year simulations of disgust, a core trait in the human behavioural immune system. Higher disgust-motivated avoidance reduced intake of potential contaminants, and thus reduced mortality due to gastrointestinal illness; at the cost of reducing total nutritional intake, and thus increased birth interval and mortality due to malnourishment. **A)** Probability density of the polygenic and cultural trait components in an environment with selective pressure ($\nu = 11083 \text{ kJ/day}, \eta = 0.1, \kappa = 1.1551 \times 10^{-8}$), each averaged across 10 simulation runs (brighter colours reflect later years). The distribution moves towards better fitness at more disgust-motivated avoidance, at higher magnitude for the cultural compared to the genetic trait. **B)** Probability density for the trait components in a control simulation ($\nu = 11083 \text{ kJ/day}, \eta = 0, \kappa = 0$), averaged across 10 simulation runs each. While modelled as a Markov process with no inherent direction, culture drifted towards improved disgust-motivated avoidance, likely because its starting point near the maximum value prevented upward drift. **C)** Averages (line) and pooled standard deviations (shading) over 10 simulation runs each for traits passed down through genetic (top) or cultural transmission (bottom) under varying cost of contamination ($\kappa$). **D)** Changes in allele frequency over time in a population with a high initial genetic variance. Alleles that contribute to increasing disgust-motivated avoidance became more prevalent.
Evolution of the behavioural immune system

**Fig. 2** (previous page) – Results of all simulated environments, with each square representing an average of 10 simulation runs. Each block represents environments with different nutritional availability (in kilo-Joule per day). Within each block, the proportion of contaminated nutrition ($\eta$) increases over the x-axis, and the probability of death per contaminated kJ ($\kappa$) increases along the y-axis. The origin ($\eta=0, \kappa=0$) is a control condition, as it presents no contamination danger, and thus no selection pressure. Skulls indicate total population collapse, with higher opacity indicating more runs ended in collapse, and grey squares indicating no populations survived. **A)** Total shift in average cultural trait over 100 000 years. Shifts are always towards more disgust-motivated avoidance, even in the control condition, because the trait’s starting point is to approach all the contaminated nutrition. **B)** Total shift in average polygenic trait over 100 000 years. **C-F)** Magnitudes of evolutionary shifts compared to their control condition (no selection pressure at $\eta=0, \kappa=0$), split between cultural (upper-left triangles) and genetic (lower-right triangles) inheritance. Simulated scenarios implemented high (C and E) or less high (D and F) initial genetic variance, and did (C and D) or did not (E and F) implement parental modelling.

**Discussion**

Inspired by disagreements on the role of cultural evolution between prominent theories of the behavioural immune system’s development, we simulated 100 000 years of human life. We focussed on disgust because of its central role in pathogen avoidance (3–5,7,8) and its uniquely prominent profile in humans (3,14,15). Our results showed that cultural disgust shifted further than the genetic trait, and that this was particularly so when initial genetic variance was low and adaptation thus relied on rare mutations. This aligns with theories that propose human disgust emerged through selective pressures on both genes and learned behaviours (3,7,16).

The emphasis on genetic inheritance in other theories of disgust development is frequently supported by the idea that parents do not socially transfer the trait to their offspring (18,19). Twin studies have indeed estimated that heritability drives half of the variance in questionnaire responses on disgust sensitivity, and that influences outside of siblings’ shared environment (e.g. societal influences and measurement error) explain the remainder (24–26). These findings seem to contrast with ours, as we found that social transmission, particularly through parental modelling, is a plausible mechanism of inheritance.

While the lack of estimated shared-environmental influences (e.g. parental modelling and home environment) appears to be evidence against parental influences on children’s disgust, it rests on the implausible assumptions that siblings share the exact same environment (27,28), and that environment and heritability are unrelated (29). Violations of these assumptions cause twin studies to generally overestimate genetic heritability (30–32). In the probable case that individual differences in children’s disgust-approach tendencies inspire differences in parenting, twin studies on disgust would have been biased because of evocative gene-environment correlation (57). Regardless, even when parental influences were eliminated in control simulations, cultural adaptation was on par with or faster than genetic adaptation (depending on the initial genetic...
variance). These findings suggest that cultural transmission should have a central place in theory regardless of whether parenting impacts offspring’s disgust.

As highlighted in the introduction, avoidance of potential contaminants is much stronger in humans (14,15) compared to bonobos (12) or chimpanzees (13). Thus, while disgust-motivated avoidance is not unique to humans, the high degree to which humans display it is unique. This is mirrored in cultural evolution: while some have suggested it is uniquely human (58), plenty of evidence suggest that it is not. The type of cumulative culture that relies on form-copying social learning (as it is modelled here) occurs in non-human primates too (59–64), and cannot be explained through (re)innovation (65). As with disgust itself, it is not the existence of cumulative culture that is unique to humans, but rather the degree to which it is supported by a range or social and cognitive skills (66,67). Hence, we think humans developed their uniquely strong propensity for disgust due to their uniquely strong ability to socially transmit traits between generations.

Limitations
Disgust is theorised to serve other adaptive functions not addressed in our model, including mate selection and coordinating moral condemnation (17). However, most theories view pathogen or “core” disgust as the original form, which was then co-opted to serve other functions (3,16,17). In addition, whether these functions build on core disgust, or only share superficial overlap grounded in common parlance (68), remains an open question (69).

Because of the above, our model did not incorporate assortative mating. This, and other types of dependencies between genotype and phenotype through cultural expressions, have been the focus of previous population-level modelling efforts (70). We modelled genetic and cultural inheritance as two independent underlying processes that acted on the same phenotype, which is defensible as the most conservative approach in the absence of strong data on potential sources of gene-culture coevolution in the context of disgust. However, future work could incorporate different types of coevolution scenarios to evaluate their relative likelihood.

Another simplification in our model was in the lack of niche construction. We did not incorporate in the idea that type and probability of cultural transmission could differ as a function of individuals’ cultural background (71), or the population’s demographic structure (72,73).

Conclusion
Our agent-based simulations suggest that under selective pressure of pathogens, cultural evolution outpaced genetics in shaping human’s unique propensity for disgust. Our results support the hypothesis that the behavioural immune system evolved, at least in part, as a “cognitive gadget” (74): a mental mechanism passed down generations through cultural transmission.
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Author contributions
ESD conceptualised the study, developed the methodology and software, conducted statistical analyses, visualised the data, and drafted and edited the manuscript. TA assisted in conceptualising the study, and reviewed and edited the manuscript.

Competing interests
The authors declare that they have no competing interests (financial or other) that could have influenced or appeared to influence the work reported here.

Preprint version history
ArXiv v4, 2022-02-14
• Limitations. Improved Discussion section on limitations, which now includes the aspects of disgust that remain unaddressed in our model (e.g. sexual and moral disgust). Additionally, the section addresses evolutionary processes that were not modelled, including gene-culture coevolution and demographic factors that could affect social transmission.
• Methodological overview. The Methods section now opens with an overview of the study, and includes a new section on the (lack of) statistical testing.

ArXiv v3, 2021-12-16
• Scholarship. An anonymous reviewer of this manuscript pointed out that the model of cultural evolution attributed to Eerkens & Lipo (2005) is highly similar to that proposed by Cavalli-Sforza & Feldman (1973). We now appropriately cite them.
• Nutrition-illness trade-off. In previous versions, disgust was modelled as a dampening factor on gastrointestinal illness due to its effect on the avoidance of contaminated food. The flip side of this, missed nutrition, was not incorporated. The current model does incorporate missed nutrition, which now impacts on survival and on postpartum amenorrhoea.
• Phylogenetic starting point. The genetic starting point for disgust avoidance is now set at an average of 40% feeding of visibly contaminated food. This is based on work by Sarabian and colleagues (2018) who show that about 30-50% of studied bonobos ate banana slices that were atop or directly adjacent to faeces; and in chimpanzees who avoid about 50% of
olfactory and tactile faecal replicas (Sarabian et al., 2017). The cultural starting point was set near 100% of feeding on contaminated food, i.e. near 0% disgust-motivated avoidance.

ArXiv v2, 2020-09-06

- Corrected reference numbering. (We forgot to hit Zotero’s refresh button after changing the order of several sentences. Oops!)

ArXiv v1, 2020-08-30

- Original submission.

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Evolution of the behavioural immune system

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