

Problems With Predicting IQ From Pathogen Prevalence:

A Group-Level Re-Analysis in the USA

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## Abstract

Analyzing aggregate data may produce inaccurate inferences if the research question is about phenomena at a non-aggregate level. Previous research has shown this misaggregation to have occurred in Fincher and Thornhill's (2012a) use of Parasite Stress USA, an index ostensibly of pathogen prevalence but whose variation results mostly from the percent of state residents who are black (Hackman & Hruschka, 2013; Koenig, van Leeuwen, & Park, 2017). The current paper highlights how misaggregation can be problematic by re-evaluating the use of this index in Eppig, Fincher, & Thornhill's (2011) reported association ( $r = -.67$ ) between Parasite Stress USA and state IQ. We re-analyzed the state-level data controlling for racial/ethnic subgroups using group-mean-centered variables. We found that (a) the association between pathogen prevalence and IQ is misaggregated across racial/ethnic subgroups, (b) when predicting IQ, racial/ethnic subgroups alone explained slightly more variance (adj.  $R^2 = .79$ ) than pathogen prevalence alone (adj.  $R^2 = .75$ ), (c) while controlling for racial/ethnic subgroups, the strength of association between pathogen prevalence and IQ (std.  $\beta = -.33$ ) is substantially weaker than that reported by Eppig and colleagues (2011), and (d) after accounting for the proportion of variance explained by racial/ethnic subgroup, wealth, and life expectancy, pathogen prevalence uniquely explained only 7% of the variance in IQ. Given the apparent trend of systematic overestimation of effect sizes in publications reporting associations at the aggregate level, we advise researchers, editors, and reviewers to be skeptical of effect sizes resulting from correlations at the incorrect level of analysis.

### Problems With Predicting IQ From Pathogen Prevalence:

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Misaggregation occurs when analyses with aggregated data lead to invalid conclusions with respect to individual-level data (Koenig, van Leeuwen, & Park, 2017). Aggregation can simplify data collection and analyses, but the inherent loss of information may undermine the validity of resulting claims. We use the term *misaggregation* to refer to erroneous aggregation of non-collapsible data. A measure of association is non-collapsible if the aggregated measure of association differs from the weighted average of the stratified measures of association (Huitfeldt, Stensrud, & Suzuki, 2019). Non-collapsibility causes several related phenomena, including the widely known Simpson's paradox (Simpson, 1951) and the ecological fallacy (Robinson, 1950). In application, non-collapsibility becomes troublesome when researchers make inaccurate claims based on analyses of aggregated data. The presence of misaggregation in any situation depends on whether the association demonstrates non-collapsibility and whether aggregated analysis is the correct representation of the purported association. The scope of misaggregation includes a full reversal in direction of an association, an elimination of an association, or a diminishment/enhancement of an association's strength.

A well-known example of misaggregation is when UC Berkeley graduate admissions data was misinterpreted to show a gender bias. In 1973, 44% of male applicants gained admission schoolwide, whereas only 35% of female applicants gained admission. Bickel, Hammel, and O'Connell (1975) found no evidence of discrimination when the data was analyzed at the department-level because women tended to apply to more competitive departments than men. By doing so, fewer women overall gained admission while men were more readily accepted into less competitive departments. In this case, UC Berkeley's graduate admissions data was

misaggregated across departments. Other scenarios may demonstrate non-collapsibility, but not be misaggregated if the aggregated data is the correct level of analysis. For example, it is valid to say that UC Berkeley admitted a higher percentage of male applicants than female applicants to their graduate programs in 1973. The current paper highlights how misaggregation can be problematic for researchers. It does so by re-analyzing a previous finding based on aggregated data.

### **Parasite Stress Theory**

Parasite stress theory is a compelling example for which analyses of aggregated data have been frequently published. This theory posits that historical trends of parasite stress have broadly impacted human cultures, values, and behaviors (Fincher, Thornhill, Murray, & Schaller, 2008; Schaller & Murray, 2008; Thornhill, Fincher, Murray, & Schaller, 2010). Indeed, parasite stress theory has been proposed as a general theory of culture and sociality (Fincher & Thornhill, 2012b; Thornhill & Fincher, 2014, 2015). However, scholars have leveled numerous criticisms on the theoretical foundation of parasite stress theory and its empirical support, such as failure to adequately investigate alternative hypotheses (Figueredo, Gladden, & Black, 2012; Hackman & Hruschka, 2013). Other criticisms point to statistical concerns, with variation in cultural traits accounted for by factors other than parasite stress such as non-independence in the data, non-equivalence of measurement, non-representative samples, misaggregation, clustered data, and confounding/covariation (Bromham, Hua, Cardillo, Schneemann, & Greenhill, 2018; Currie & Mace, 2012; Hackman & Hruschka, 2013; Hruschka & Hackman, 2014; Koenig, van Leeuwen, & Park, 2017; Pollet, Tybur, Frankenhuys, & Rickard, 2014; Van de Vliert, & Postmes, 2012). Each of these criticisms could affect the validity of parasite stress theory's research if not properly addressed.

*Parasite Stress USA*

Fincher and Thornhill (2012a) created an index of state-level pathogen prevalence in the United States—Parasite Stress USA—which has been used to support parasite stress theory. However, Hackman and Hruschka (2013) found that black people in the United States have substantially different rates of pathogen prevalence within states as compared with white people, so the percentage of black residents within a state (hereafter “percent black”) disproportionally affects that state’s aggregate pathogen rate. Koenig and colleagues (2017) further demonstrated that Parasite Stress USA is misaggregated via the use of four red-flag checks. Parasite Stress USA failed all four red-flag checks, suggesting that the literature may contain erroneous results from publications using Parasite Stress USA or similar measures. One candidate is the predicted negative association between parasite stress and intelligence (Eppig, Fincher, & Thornhill, 2011; hereafter “EFT”). For that association, the researchers reasoned that since the human brain is metabolically expensive to develop and maintain, fending off pathogens leaves fewer metabolic resources available for brain development and maintenance. Geographic areas with high pathogen prevalence are thus predicted to have worse cognitive ability.

Supporting this prediction, EFT reported a strong negative association between Parasite Stress USA and state-level IQ, even when controlling for wealth and education. The researchers did not control for racial/ethnic subgroups in their analyses, citing three reasons. First, they discount the importance of percent black because its distribution closely follows climate-related variation in pathogen prevalence. Second, percent black correlated more with Parasite Stress USA ( $r = .90, p < .001, N = 50$ ) than with state IQ ( $r = -.51, p < .001, N = 50$ ). Finally, percent black added little additional explanatory power ( $R^2$ -change) in a multiple regression with Parasite Stress USA and percent black predicting state IQ.

We found EFT's conceptual arguments inconclusive due to the strong correlations of percent black with both Parasite Stress USA and state IQ, which suggest the possibility of misaggregation across racial/ethnic subgroups. Therefore, we wanted to *empirically* test the association when controlling for racial/ethnic subgroup differences. If parasite stress indeed reduces cognitive ability, we could still see a strong correlation between pathogen prevalence and IQ at the subgroup-level. We therefore investigated possible misaggregation across racial/ethnic subgroups in EFT's finding that pathogen prevalence is a strong predictor of IQ at the state-level.

### Method

Parasite Stress USA does not have available data that is also stratified by racial/ethnic subgroups, so Hackman and Hruschka (2013) created statistically equivalent race-stratified indices based on rates of chlamydia and gonorrhea obtained from the U.S. Department of Health and Human Services (2011). Koenig and colleagues (2017) recreated those indices, which we used for measures of pathogen prevalence at both the aggregate level ( $CG_{\text{aggregate}}$ ) and racial/ethnic subgroup level ( $CG_{\text{white}}$ ,  $CG_{\text{black}}$ ,  $CG_{\text{hispanic}}$ ,  $CG_{\text{asian}}$ ,  $CG_{\text{native}}$ ). We also calculated measures of percent population for each racial/ethnic subgroup (%White, %Black, %Hispanic, %Asian, %Native). EFT used estimates created by McDaniel (2006) as a measure of state IQ ( $IQ_{\text{McDaniel}}$ ). We recreated this at both the aggregate level ( $IQ_{\text{aggregate}}$ ) and racial/ethnic subgroup level ( $IQ_{\text{white}}$ ,  $IQ_{\text{black}}$ ,  $IQ_{\text{hispanic}}$ ,  $IQ_{\text{asian}}$ ,  $IQ_{\text{native}}$ ) using data from the National Assessment of Educational Progress (U.S. Department of Education). We validated these measures by comparing their correlations with other variables to those previously published.

### *Analysis Strategy*

We first tested whether state IQ failed the four red-flag checks for misaggregation put forth by Koenig and colleagues (2017). Next, for state-level pathogen prevalence and IQ, we compared the aggregated correlation with correlations from each racial/ethnic subgroup both alone and weighted by each subgroup's proportion to the overall population. After affirming the need for further analyses (see results), we attempted multilevel modeling (not reported). However, there were too few level-two groups (racial/ethnic subgroups) to estimate the random effect of the grouping variable on the level-one model (Garson, 2020). We identified the fixed effects approach to multiple regression as an alternative when analyzing hierarchical data with a small number of level-two groups (McNeish & Stapleton, 2016; Möhring, 2012). Our fixed effects model used variables centered on national means of racial/ethnic subgroups to control for subgroup differences. To use as control variables, we recreated EFT's measure of wealth (U.S. Census Bureau, 2000) and obtained a measure of life expectancy (Lewis & Burd-Sharps, 2014), each stratified at the racial/ethnic subgroup level. In selecting these control variables, we wanted to recreate the variables used by Eppig, Fincher, and Thornhill (2011) as closely as possible. We also made an effort to be theoretically comprehensive by considering several other constructs as potential control variables. We were unable to obtain a measure of educational quality that was similar to those used by EFT and stratified by racial/ethnic subgroup.

## Results

Koenig and colleagues (2017) demonstrated that Parasite Stress USA failed all four red-flag checks for misaggregation: "Aggregated rates that correlate strongly with the relative size of one or more subgroup(s), unequal sample sizes across subgroups, unequal rates or mean values across subgroups, and aggregated rates that do not correlate with subgroup rates" (p. 16). We likewise found that  $IQ_{McDaniel}$  failed all four red-flag checks. Check 1:  $IQ_{McDaniel}$  correlated

strongly with %White ( $r = .71, p < .001, N = 50$ ), moderately with %Black ( $r = -.51, p < .001, N = 50$ ), and weakly with %Hispanic ( $r = -.32, p = .023, N = 50$ ). Check 2: The percent population of each state's racial/ethnic subgroups are unequal in size (Friedman Test,  $\chi^2 = 143.02, p < .001, N = 50$ ). Check 3: For the 10 states that had IQ data for all racial/ethnic subgroups, the IQ scores for state's racial/ethnic subgroups have unequal mean values (Friedman Test,  $\chi^2 = 30.69, p < .001, N = 10$ ). Check 4:  $IQ_{McDaniel}$  is not strongly or significantly correlated with  $IQ_{hispanic}$  ( $r = .30, p = .052, N = 42$ ),  $IQ_{asian}$  ( $r = .30, p = .112, N = 29$ ), and  $IQ_{native}$  ( $r = .35, p = .169, N = 17$ ). Since both Parasite Stress USA and  $IQ_{McDaniel}$  each fail all four red-flag checks, there is a strong risk of misaggregation for analyses including these measures.

We predicted IQ from pathogen prevalence separately for each racial/ethnic subgroup (see Table 1, Models 2-6). All standardized betas (correlations) were negative. However, all but one correlation had a substantially weaker association than the  $r = -.67$  reported by EFT. The weighted average (across racial/ethnic groups, weighted by %population) association strength ( $r = -.36$ ) is also much smaller than EFT's aggregated association strength ( $r = -.67$ ), meaning that the association between pathogen prevalence and IQ is non-collapsible.

Table 1. *Regression Models Predicting IQ*

Model	Predictors	adj. $R^2$	std. $\beta$	N
1	Parasite Stress USA <sup>1</sup>	.43***	-.67***	50
2	$CG_{white}^2$	.11*	-.35*	50
3	$CG_{black}^2$	.05	-.26	42
4	$CG_{hispanic}^2$	.18**	-.44**	42
5	$CG_{asian}^2$	.41***	-.65***	29
6	$CG_{native}^2$	.00	-.22	17
7	Dummy race variables <sup>3</sup>	.79***		180
8	$CG_{LOG\_uncentered}^3$	.75***	-.87***	180
9	$CG_{LOG\_centered}^4$	.23***	-.49***	180



10	Wealth <sub>centered</sub> <sup>4</sup>	.18***	.43***	180
11	Life <sub>centered</sub> <sup>4</sup>	.19***	.44***	148
*** $p < .001$ ** $p < .01$ * $p < .05$				

<sup>1</sup> Predicting IQ<sub>McDaniel</sub>. See Eppig and colleagues (2011).

<sup>2</sup> Predicting IQ<sub>race</sub>.

<sup>3</sup> Predicting IQ<sub>uncentered</sub>. Including 4 dummy coded variables with whites as the reference group.

<sup>4</sup> Predicting IQ<sub>centered</sub>.

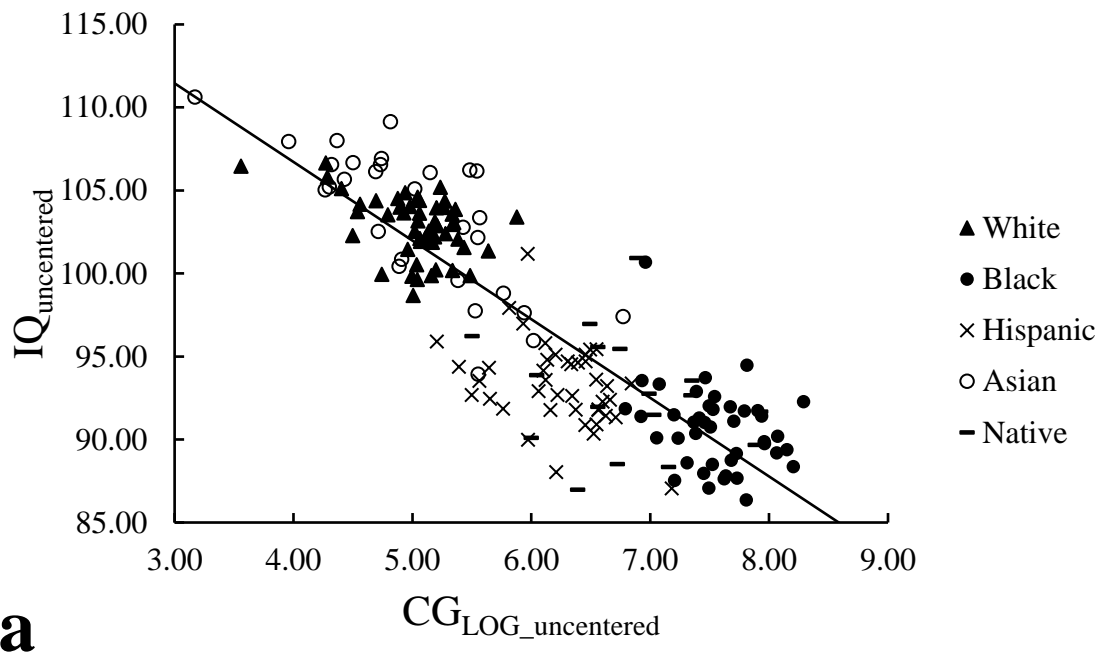
We used group-mean centering<sup>1</sup> to control for differences across racial/ethnic subgroups, creating long-form (non-hierarchical) measures of pathogen prevalence (CG<sub>centered</sub>), IQ (IQ<sub>centered</sub>), wealth (Wealth<sub>centered</sub>), and life expectancy (Life<sub>centered</sub>). We chose this method over including dummy variables for N-1 subgroups as it provided more interpretable collinearity diagnostics and more degrees of freedom. A scatterplot with CG and IQ stratified by racial/ethnic subgroup demonstrated a logarithmic association, thus we log-transformed the long-form measures of CG prior to performing the following analyses.

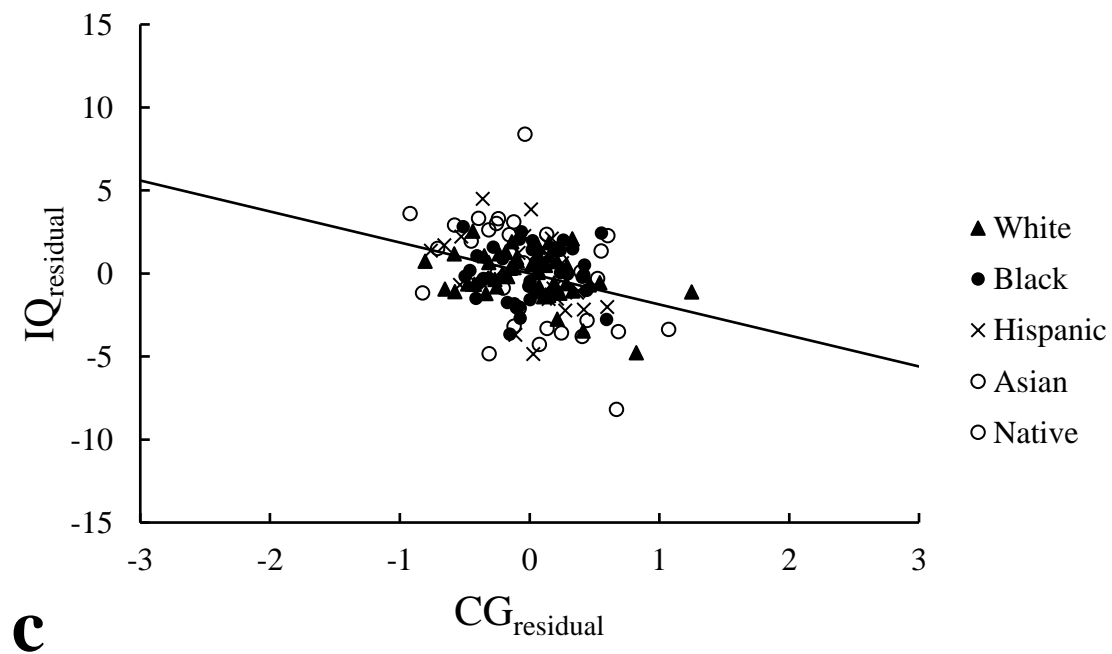
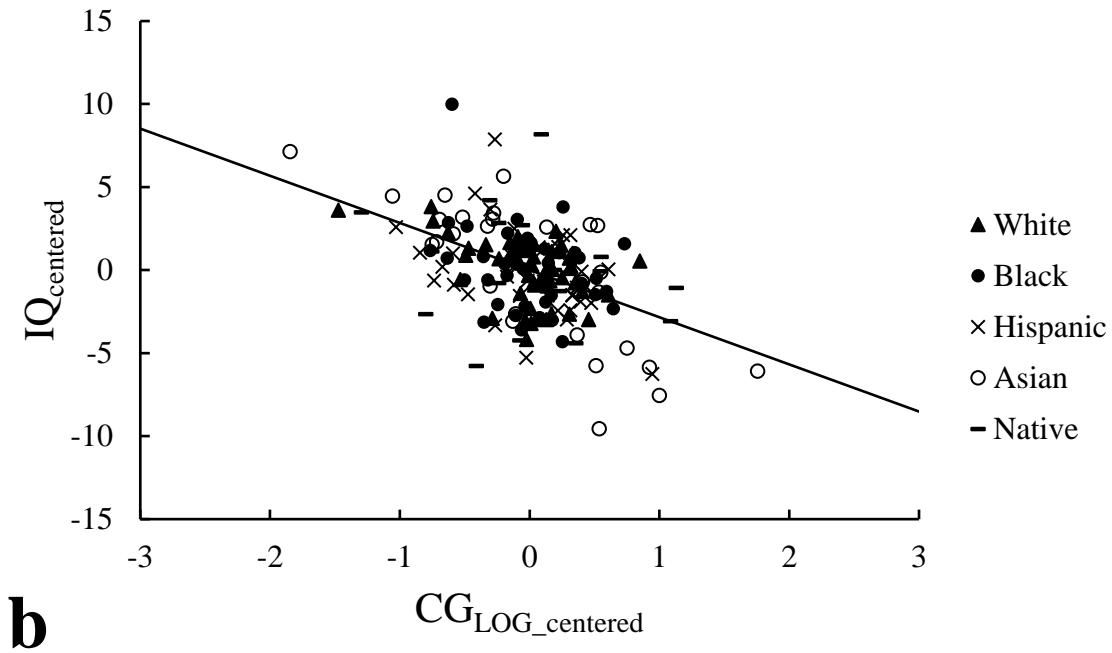
Notably, before group-mean centering, predicting IQ<sub>uncentered</sub> from only dummy variables for racial/ethnic subgroups explained slightly more variance than did CG<sub>LOG\_uncentered</sub> alone (see Table 1, Models 7-8). This is due to clustering by racial/ethnic subgroup (see Figure 1a). When controlling for racial/ethnic subgroup clustering with group-mean centering, the regression predicting IQ<sub>centered</sub> from CG<sub>LOG\_centered</sub> yielded a substantially smaller effect size compared to the same regression prior to group-mean centering (see Table 1, Models 8-9, and Figure 1b). Further, group-mean-centered wealth and life expectancy alone each had an effect size nearly equal in magnitude to that of pathogen prevalence (see Table 1, Models 9-11).

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<sup>1</sup> This was accomplished by subtracting the national mean for each subgroup from each score for members in that subgroup. To maximize the sample size for each analysis, variables were centered based off the corresponding analysis' available list-wise sample (i.e., analyses including both IQ and life expectancy have N = 148 whereas analyses without life expectancy have N = 180). National means were recalculated for the final sample for each analysis.

Figure 1. Scatterplot of racial/ethnic subgroup stratified CG and IQ for (a) uncentered variables, (b) group-mean-centered variables, and (c) group-mean-centered variables controlling for  $Wealth_{centered}$  and  $Life_{centered}$ .





Including covariates in a multiple regression with racial/ethnic mean differences removed revealed pathogen prevalence's association with IQ to be even further diminished in our final

model predicting  $IQ_{\text{centered}}$  from  $CG_{\text{LOG\_centered}}$ ,  $Wealth_{\text{centered}}$ , and  $Life_{\text{centered}}$  (adj.  $R^2 = .35$ ,  $p < .001$ ; see Table 2). Moreover, life expectancy predicted IQ nearly as strongly as pathogen prevalence (see Table 2). This differs from EFT's final model, in which pathogen prevalence was by far the strongest predictor of IQ (see Table 2). Additionally, EFT found that in their final multiple regression model, "adding these covariates [pathogen prevalence, wealth, highly qualified teachers, and student-teacher ratio] in a different order did not appreciably change the additive  $R^2$  of each iteration" (p. 4). In other words, the covariates had just as much explanatory power in predicting IQ as pathogen prevalence. Indeed, pathogen prevalence had an additive adjusted  $R^2 = .07$ , meaning that pathogen prevalence only uniquely explained 7% of the variance in IQ for our final model. To visualize the unique relationship between  $IQ_{\text{centered}}$  and  $CG_{\text{LOG\_centered}}$ , Figure 1c shows the residuals resulting from predicting  $IQ_{\text{centered}}$  and  $CG_{\text{centered}}$  each separately from  $Wealth_{\text{centered}}$  and  $Life_{\text{centered}}$ .

Table 2. *Final Multiple Regression Models Predicting IQ*

	std. $\beta$	N	VIF
Eppig and colleagues' (2011) Model <sup>1</sup>		50	
Parasite Stress USA	-.62***		1.02
Wealth	.30***		1.00
Highly Qualified Teachers <sup>2</sup>	.29**		1.16
Student-Teacher Ratio <sup>2</sup>	-.22*		1.16
Our model <sup>3</sup>		148	
$CG_{\text{LOG\_centered}}$	-.33***		1.48
$Wealth_{\text{centered}}$	.17		1.58
$Life_{\text{centered}}$	.26**		1.24

\*\*\*  $p < .001$     \*\*  $p < .01$     \*  $p < .05$

<sup>1</sup> Predicting  $IQ_{\text{McDaniel}}$ .

<sup>2</sup> See Eppig and colleagues (2011).

<sup>3</sup> Predicting  $IQ_{\text{centered}}$ .

## Discussion

Our findings suggest that the association between pathogen prevalence and IQ previously reported by EFT is misaggregated. Once detected, misaggregation still requires interpretation. The data alone provides little to no indication of whether the aggregate or stratified analyses are correct. However, attending to the (hypothesized) causal context reveals the correct level of analysis (Hernán, Clayton, & Keiding, 2011). In the case of pathogen prevalence and IQ, EFT hypothesized a developmental trade-off between pathogens and intelligence occurring in individuals. As such, our racial/ethnic subgroup stratified analyses—though still flawed by the usage of state-level rather than individual-level data—present a more accurate representation of the association between pathogen prevalence and IQ.

The limitations to this study highlight how several other concerns may further impact both EFT's and our own analyses. Our data may be misaggregated further at the state level and our analyses fail to account for potential spatial autocorrelation (a state's data may not be independent due to similarities to other nearby states). We also did not attempt to account for other statistical concerns mentioned earlier such as non-equivalence of measurement, non-representative samples, and confounding/covariation. Other minor limitations include missing data points in the measures of IQ and life expectancy, difficulty in finding control variables stratified by both state and racial/ethnic subgroup, and slight differences in the definitions of racial/ethnic subgroups across each variable.

By accounting for a single problem, misaggregation across racial/ethnic subgroups, we found a dramatically different association between pathogen prevalence and IQ. It may be that accounting for other concerns further changes the results, or not. Substantial effort would be needed to account for all of these statistical concerns using aggregated data. Therefore, we highly

recommended that researchers design studies and collect data at the correct level for their analyses (relative to the research question). Individual-level data can always be aggregated across groups and is therefore optimal. In cases where individual-level or stratified data is unavailable or difficult to obtain, we strongly advise that researchers attend to these issues when conducting analyses and interpreting their results.

In the case of parasite stress theory, much of the current research relies on aggregated data and correlational analyses. This often (and perhaps incorrectly) resulted in causal relationships being inferred about individuals. Further, other researchers have pointed out how these studies fail to account for other concerns we have mentioned (e.g., Bromham, Hua, Cardillo, Schneemann, & Greenhill, 2018; Currie & Mace, 2012). Misaggregation is just one such concern, but we have demonstrated that only one of these issues may dramatically alter a study's results. This calls into question the validity of much of the research on parasite stress theory. To know for sure, parasite stress theory's findings should be investigated with more robust empirical methods using data at the correct level of analysis.

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