## University of New Mexico UNM Digital Repository

Biology ETDs

**Electronic Theses and Dissertations** 

12-1-2011

# Infectious disease and the worldwide distribution of IQ

Christopher Eppig

Follow this and additional works at: https://digitalrepository.unm.edu/biol\_etds

#### **Recommended Citation**

Eppig, Christopher. "Infectious disease and the worldwide distribution of IQ." (2011). https://digitalrepository.unm.edu/biol\_etds/ 32

This Dissertation is brought to you for free and open access by the Electronic Theses and Dissertations at UNM Digital Repository. It has been accepted for inclusion in Biology ETDs by an authorized administrator of UNM Digital Repository. For more information, please contact disc@unm.edu.

Christopher Guiles Eppig Candidate
Biology Department
This dissertation is approved, and it is acceptable in quality and form for publication
Approved by the Dissertation Committee:
Randy Thornhill, Chairperson
Paul Watson
Steve Gangestad
Astrid Kodric-Brown

### INFECTIOUS DISEASE AND THE WORLDWIDE DISTRIBUTION OF IQ

by

#### **CHRISTOPHER GUILES EPPIG**

B. A., Biology, Earlham College, 2004

#### DISSERTATION

Submitted in Partial Fulfillment of the Requirements for the Degree of

## **Doctor of Philosophy**

## **Biology**

The University of New Mexico Albuquerque, New Mexico

December, 2011

## **DEDICATION**

I dedicate this work to my friends and family who have supported me over the years.

#### **ACKNOWLEDGEMENTS**

I would first like to thank my coauthors, Corey Fincher and Randy Thornhill, for their contributions to the research contained herein.

My committee members, Randy Thornhill, Paul Watson, Steve Gangestad, and Astrid Kodric-Brown provided invaluable guidance during my time as a graduate student.

Several members of the behavioral ecology and evolutionary psychology programs at UNM provided feedback and comments on earlier drafts of my research: Rachael Falcon, Angela Hung, Kenneth Letendre, and Rhiannon West.

by

#### **Christopher Guiles Eppig**

## B. A., BIOLOGY, EARLHAM COLLEGE, 2004 PH. D., BIOLOGY, UNIVERSITY OF NEW MEXICO, 2011

#### Abstract

We show that infectious disease is a major contributor of the worldwide distribution of human cognitive ability, as measured by psychometric IQ. In areas where infectious disease is high, average human intelligence tends to be lower, and in areas where infectious disease is low, average human intelligence tends to be higher. In separate studies, we tested this across both world nations (chapter 2) and across states of the USA (chapter 3).

In efforts to disseminate our research to wider audience, I reviewed the findings contained chapters 2 and 3 using language that is accessible non-biologists (chapter 4). Although it contains no original research, this chapter makes our research more readily available to people in non-biological fields, such as economics and political science, who may be interested in our findings.

An early prediction we made, based on our first analysis, was that infectious disease could account for the apparent link between IQ and rates of asthma, that other research has discovered (chapter 5). Additionally, this chapter attempts to reconcile the predictions made by several related hypotheses.

## **TABLE OF CONTENTS**

LIST OF FIGURES.	.viii
LIST OF TABLES.	ix
CHAPTER 1 – INTRODUCTION.	1
CHAPTER 2 – PARASITE PREVALENCE AND THE WORLDWIDE	
DISTRIBUTION OF COGNITIVE ABILITY	7
CHAPTER 3 – PARASITE PREVALENCE AND THE DISTRIBUTION OF	
INTELLIGENCE AMONG THE STATES OF THE	
USA	39
CHAPTER 4 – INFECTIOUS DISEASE PREVALENCE PREDICTS IQ ACROSS	
WORLD NATIONS AND STATES OF THE USA	65
CHAPTER 5 – INFECTIOUS DISEASE AND THE RELATIONSHIP OF THE	
HYGIENE HYPOTHESIS TO IQ	81

## LIST OF FIGURES

2-1	35
3-1	60
3-2	61
5-1	98

## LIST OF TABLES

2-1	32
2-2	33
2-3	34
2-4 (electronic supplementary table 1)	36
2-5 (electronic supplementary table 2)	37
2-6 (electronic supplementary table 3)	38
3-1	62
3-2	63
3-3	64
5-1	97

Chapter 1. Introduction

Christopher G. Eppig

Humans possess exceptionally high intelligence that appears to be unique among animals. The brain, which is the source of this intelligence, requires an immense amount of energy both to build and to operate — it is not just an extremely expensive trait, but it is, arguably, the most expensive trait in the human body. Energy does not, of course, come easily to any animal, and humans are no exception. For such a costly trait to evolve and be maintained by selection, its importance to survival and reproduction must necessarily be paramount. For this reason, the importance of the study of human intelligence cannot be overstated. The study of variation in this trait across the world is one important area of research.

In the following chapters, I present research conducted with my colleagues supporting the parasite-stress hypothesis applied to the global variation of human intelligence. Exposure to infectious disease during periods of childhood development when the brain is most energetically expensive, we argue, leads to reduced intelligence. We begin by demonstrating this effect across world nations (chapter 2) — nations with a higher burden of infectious disease tend to have lower average IQ, and nations with a lower burden of infectious disease tend to have a higher average IQ. Our second study continues to find support for our hypothesis using states of the USA as the level of analysis rather than world nations (chapter 3). This study provides much finer geographic resolution, and makes it easier to control for certain variables. Again, we find that areas with higher infectious disease stress have lower average IQs than areas with lower infectious disease stress.

The study of human intelligence is of great interest to non-biologists, as well.

Most research on human intelligence has implications in many fields, including

economics, political science, medicine, criminology, and sociology. We go on to review our findings from chapters 2 and 3 using language that is accessible to people in these fields (chapter 4).

Our final study diverges slightly from determining the causes of the worldwide variation in human intelligence, and investigates the relationship among intelligence, asthma and other, related factors (chapter 5). As we had previously predicted (chapter 2), the correlation between average IQ and rates of asthma appear to be mediated entirely by infectious disease, which is a common cause of variation in both IQ and asthma. By developing this larger image of the relationships among several variables, we make efforts to reconcile the various hypotheses that each describe the relationships among fewer variables.

There is a great deal of work still to be done in this research avenue. We have presented evidence that infectious disease reduces intelligence, but the impact of the various infections, as well as the duration and timing of these infections, is still unknown. Many scientists, both critics and supporters, have expressed concern that the experimental methods we have used have not adequately established that causation exists between IQ and infectious disease, and that its direction is the one we predict. The use of longitudinal studies could answer all of these questions. Such studies would track children from as early an age as possible, documenting the intensity, duration, timing, and types of infections they acquire, and track their cognitive development through the use of culturally-appropriate IQ tests. While the central nervous system is extremely expensive, and therefore readily subject to developmental insult when sufficient energy is not available for its development, other traits in the body are theoretically subject to

reduced quality when they are limited by available energy. The theoretical framework we have developed can therefore inform the study of the effects of infectious disease stress on other organs, as well.

The following are the abstracts for each chapter.

Chapter 2: Parasite prevalence and the worldwide distribution of cognitive ability.

(Christopher Eppig, Corey L. Fincher and Randy Thornhill)

In this study, we hypothesize that the worldwide distribution of cognitive ability is determined in part by variation in the intensity of infectious diseases. From an energetics standpoint, a developing human will have difficulty building a brain and fighting off infectious diseases at the same time, as both are very metabolically costly tasks. Using three measures of average national IQ, we found that the zero-order correlation between average IQ and parasite stress ranges from r = -0.76 to r = -0.82 (p < 0.0001). These correlations are robust worldwide, as well as within five of six world regions. Infectious disease remains the most powerful predictor of average national IQ when temperature, distance from Africa, gross domestic product per capita, and several measures of education are controlled for. These findings suggest that the Flynn Effect may be caused in part by the decrease in the intensity of infectious diseases as nations develop.

Chapter 3: Parasite prevalence and the distribution of intelligence among the states of the USA. (Christopher Eppig, Corey L. Fincher and Randy Thornhill)

In this study, we tested the parasite-stress hypothesis for the distribution of intelligence among the USA states: the hypothesis proposes that intelligence emerges from a developmental trade-off between maximizing brain vs. immune function. From this we predicted that among the USA states where infectious disease stress was high, average intelligence would be low and where infectious disease stress was low, average intelligence would be high. As predicted, we found that the correlation between average state IQ and infectious disease stress was -0.67 (p < 0.0001) across the 50 states. Furthermore, when controlling the effects of wealth and educational variation among states, infectious disease stress was the best predictor of average state IQ.

## Chapter 4: Infectious disease predicts IQ across world nations and states of the USA. (Christopher G. Eppig)

In two studies, we predicted that intelligence emerges partly from an energetic tradeoff maximizing brain development vs. immune system function. Levels of infectious disease predict average IQ across world nations (r = -0.82) and across USA states (r = -0.67). This relationship is robust when education, wealth and other factors are controlled. On both levels of analysis, infectious disease stress was the best predictor of average IQ.

Chapter 5: Infectious disease and the relationship of the hygiene hypothesis to IQ. (Christopher G. Eppig, Corey L. Fincher and Randy Thornhill)

In this study, we investigate the previously documented positive relationship between asthma and IQ. Given that infectious disease has been implicated as a cause of variation in both IQ and asthma rates, we propose that the link between asthma and IQ is not directly causal, but the result of both being affected by the common cause of infectious disease. We attempt to reconcile the effects of both infectious disease and vitamin D deficiency on asthma. Across the 50 USA states, we found that both ambient light intensity (which contributes to vitamin D production) and infectious disease contribute to regional variation in rates of asthma (r = -0.28 and r = -0.35, respectively). Average IQ across the states and rates of asthma correlated positively (r = 0.25), but this relationship dropped close to zero when the effects of infectious disease were removed (r = 0.016).

Chapter 2: Parasite prevalence and the worldwide distribution of cognitive ability.

Christopher Eppig, Corey L. Fincher and Randy Thornhill

This chapter was published in substantially similar form as Eppig, C., Fincher, C. L. and Thornhill, R. (2010). Parasite prevalence and the worldwide distribution of cognitive ability. *Proceedings of the Royal Society B*, 277, 3801-3808.

Parasite prevalence and the worldwide distribution of cognitive ability

Christopher Eppig, Corey L. Fincher, Randy Thornhill

Biology Department, University of New Mexico, Albuquerque, NM 87131, USA

Contact Information for Corresponding Author:

UNM Biology Department MSC03 2020

1 University of New Mexico

Albuquerque, NM 87131

(505)277-4999

e-mail: <a href="mailto:ceppig@unm.edu">ceppig@unm.edu</a>

#### Summary

In this study, we hypothesize that the worldwide distribution of cognitive ability is determined in part by variation in the intensity of infectious diseases. From an energetics standpoint, a developing human will have difficulty building a brain and fighting off infectious diseases at the same time, as both are very metabolically costly tasks. Using three measures of average national IQ, we found that the zero-order correlation between average IQ and parasite stress ranges from r = -0.76 to r = -0.82 (p < 0.0001). These correlations are robust worldwide, as well as within five of six world regions. Infectious disease remains the most powerful predictor of average national IQ when temperature, distance from Africa, gross domestic product per capita, and several measures of education are controlled for. These findings suggest that the Flynn Effect may be caused in part by the decrease in the intensity of infectious diseases as nations develop.

Keywords: Brain Growth, Developmental Stability, Evolution, Flynn Effect, Infectious Disease, Life History

#### 1. INTRODUCTION

Since the first publication of quantitative data on average national IQ scores (Lynn & Vanhanen 2001, 2002, 2006), five empirical studies have attempted to explain the global distribution of variation in intelligence. Barber (2005) hypothesized that IQ —

like many other psychological traits — is a highly plastic trait that may increase ontogenetically as the rewards of higher intelligence increase and with exposure to education and other cognitively demanding environments such as non-agricultural labor. He reported that, across 81 nations, average national IQ correlated with enrollment in secondary school (r = 0.72), illiteracy (r = -0.71), agricultural labor (r = -0.70), and gross national product (r = 0.54). He also proposed that health and nutrition may affect intelligence, and found that average national IQ correlated negatively with rates of low birth weight (r = -0.48) and with infant mortality (r = -0.34). While it is plausible that formal education increases intelligence, Barber (2005) admits that it is not possible to determine from the data he used whether the correlation between education and intelligence is due to education increasing intelligence or whether more intelligent individuals seek more education. Research has shown this relationship to be intractable (reviewed in Ceci 1991). The same direction-of-causation ambiguity is true for agricultural labor (Barber 2005). We agree with Barber's (2005) assertions that health and nutrition may affect intelligence, although the variables he studied — low birth weight and infant mortality — are probably rather incomplete measures of these factors.

Lynn (1991) and Rushton (1995, 2000) proposed that temperature and climate provide important Darwinian selective pressures for intelligence, with cold climates selecting for higher intelligence, because low temperatures provide more fitness-related problems for humans that must be solved through cognitively demanding means, and through more complex social organization. Templer & Arikawa (2006) tested and supported predictions of this proposal in a cross-national study and found that average IQ correlated significantly with winter high temperature (r = -0.76), winter low temperature

(r = -0.66), summer high temperature (r = -0.31), and summer low temperature (r = -0.41). Templer & Arikawa (2006) also found that average IQ correlated significantly with average skin darkness (r = -0.92). The authors offered little explanation of why this trend exists, except that they believed skin colour was related to exposure to certain climates over evolutionary time.

Kanazawa (2004) hypothesized that intelligence evolved as a domain-specific psychological adaptation to deal with environments that are evolutionarily novel. This hypothesis was tested and supported at the cross-national level (Kanazawa 2008). Results showed that distance from three points in or near central Africa — the evolutionary origin of humans — correlated positively with average national IQ (0°E, 0°N, r = 0.45; South Africa, r = 0.53; Ethiopia, r = 0.22). Kanazawa (2008) did not offer his findings as an alternative to those of Templer & Arikawa (2006), but, rather, as complementary to them. Wicherts *et al.* (2010a) and Borsboom & Dolan (2006) heavily criticized Kanazawa's hypothesis; for reasons they give in detail, we seriously question the ability of linear distance from sub-Saharan Africa to measure evolutionary novelty, undermining the foundation of Kanazawa's hypothesis.

Saadat (2008) and Woodley (2009) suggested that inbreeding depression and associated reduced phenotypic quality is a cause of the variation in cognitive ability across the world. They found cross-national correlations of r = -0.77 (n = 35, p < 0.0001) and r = -0.62 (n = 71, p < 0.01), respectively, between average IQ and measures of inbreeding. Woodley (2009), however, noted that rates of consanguineous marriage itself may not account for the magnitude of this variation because (1) the statistical significance of the effect disappears when education and gross domestic product are controlled for,

and (2) the effect of inbreeding on intelligence had previously been shown to be relatively small.

Here, we offer a new hypothesis — the parasite-stress hypothesis — to explain the worldwide distribution of intelligence. The brain is the most complex and costly organ in the human body. In human newborns, the brain demands 87% of the body's metabolic budget, 44% at age five, 34% at age ten, and 23% and 27% for adult males and females, respectively (Holliday 1986). Presumably, if an individual cannot meet these energetic demands while the brain is growing and developing, the brain's growth and developmental stability will suffer. Lynn (e.g. 1990, 1993) has argued that nutrition is vital to high degrees of mental development. Lynn (1990) suggested that nutrition may account for the Flynn Effect (large increases in IQ over short periods of time as nations develop; Flynn 1987), and later (Lynn 1993) reviewed evidence showing that undernourished children have smaller heads, smaller brains, and lower psychometric intelligence than sufficiently nourished children.

Parasitic infection affects the body, and hence the brain, energetically in four ways: (1) some parasitic organisms feed on the host's tissues: the loss must be replaced at energetic cost to the host. Such organisms notably include flukes and many kinds of bacteria. (2) Some parasites inhabit the intestinal tract or cause diarrhea, limiting the host's intake of otherwise available nutrients. These notably include tapeworms, bacteria, giardia, and amoebae. (3) Viruses use the host's cellular machinery and macromolecules to reproduce themselves, at the energetic expense of the host. (4) The host must activate its immune system to fight off the infection, at energetic expense. Of these, diarrheal diseases may impose the most serious cost on their hosts' energy budget. First, diarrheal

diseases are the most common category of disease on every continent, and are one of the two top killers of children under five, accounting for 16-17% of all of these deaths worldwide (WHO 2004a). Second, diarrhea can prevent the body from accessing any nutrients at all. While at highest risk of death from diarrheal diseases, children under five years old require between 44% and 87% of their metabolic budget to build and maintain their brain (Holliday 1986). If exposed to this type of disease during this time, individuals may experience lifelong detrimental effects to their brain development and thus intelligence. Parasites may negatively affect cognitive function in other ways, such as by infecting the brain directly, but we focus only on energetic costs.

The worldwide distribution of parasites is well known. Disease-causing organisms of humans are more prevalent in equatorial regions of the world and become less prevalent as latitude increases. Ecological factors contributing to this distribution include mean annual temperature, monthly temperature range, and precipitation (e.g. Guernier *et al.* 2004). Similar trends of parasite distribution have been shown in other host species (e.g. Møller 1998).

Many studies have shown a negative relationship between intestinal helminth infection and cognitive ability (reviewed in Watkins & Pollitt 1997; see also Dickson *et al.* 2007). Although several hypotheses have been proposed to explain this phenomenon, none have considered intestinal worms in the larger context of all parasitic infection, nor have they considered fully the energetic cost of infection and its consequences on the brain. Other studies have shown relationships between helminth infection and economic and education factors, that be related to intelligence. For example, Bleakley (2007) studied the effects of eradication of hookworm in the US South during the early 20<sup>th</sup>

century, and found that areas where hookworm infections had been greatly reduced had higher average incomes after treatment than areas that had not received treatment.

Jardim-Botelho *et al.* (2008) found that Brazilian children infected with hookworm performed more poorly on cognitive tests than uninfected children, and that children infected with more than one type of intestinal helminth performed more poorly than children infected with only one.

Thus, from the parasite-stress hypothesis, we predict that average national intelligence will correlate significantly and negatively with rates of infectious disease and that infectious disease will remain an important predictor of average national intelligence when other variables are controlled for. It is the purpose of this study to introduce this hypothesis to describe the worldwide variation in intelligence and to provide some supportive evidence using correlations and linear modeling techniques.

#### 2. METHODS

National average intelligence was taken from Lynn & Vanhanen (2006), who present their methods in detail. IQ was measured directly in 113 nations, and estimated for 79 more nations by averaging the IQs of nearby nations with known IQ. These estimates were validated by Lynn & Vanhanen (2006) by comparing them to actual measurements of IQ in the same nations. At least two studies have presented evidence of validation for these data (Lynn & Mikk 2007; Rindermann 2007), by showing strong positive correlations between Lynn & Vanhanen's (2006) national IQ scores and other measures of cognitive ability. Wicherts *et al.* (2010b) have criticized Lynn & Vanhanen's

(2006) estimates of IQ in sub-Saharan African nations on the grounds that the selection criteria used to include studies in these estimations did not produce national IQ scores that were representative. Using selection criteria that they argue are more appropriate, Wicherts *et al.* (2010b) proposed new average IQ values for 17 nations in Sub-Saharan Africa (but see also Lynn & Meisenberg 2010). Analyses will be performed using three data sets: Lynn & Vanhanen's (2006) original data, including estimates (LVE; mean = 84, median = 84.2, SD = 11.8); Lynn & Vanhanen's (2006) data using collected data only (LVCD; mean = 86.7, median = 87.5, SD = 11.9); and Wicherts *et al.*'s (2010b) revisions of Lynn & Vanhanen's (2006) data with estimates (WEAM; mean = 85.0, median = 85, SD = 11.0).

As a measure of infectious disease levels for each nation, disability-adjusted life years lost (DALY) due to infectious disease was used (WHO 2004b). This measure combines years of life lost and years spent disabled due to 28 representative and important human diseases including tetanus, malaria, tuberculosis, hepatitis, syphilis, and leishmaniasis, such that one 'DALY' equals one healthy year of life lost per 100,000 people. Although other cross-national measures of disease exist, we believe this to be the best for our study because (1) data exist for most countries of the world (n = 192), and (2) this variable is a reasonable measure of the physiological costs of infectious disease, which concerns the parasite-stress hypothesis applied to cognitive ability. The DALY infectious-disease measure correlates strongly with other measures of human infectious disease (e.g. Thornhill *et al.* 2009). This variable was log transformed due to an extreme skew to the right (mean = 3.36, median = 3.28, SD = 0.761).

As an independent measure of nutrient stress, DALY due to nutritional deficiencies (WHO 2004b) was used. This calculation includes mortality and healthy years lost due to protein-energy malnutrition, iodine deficiency, vitamin A deficiency, and iron-deficiency anemia. This variable was log-transformed due to an extreme skew to the right (mean = 2.59, median = 2.65, SD = 0.49).

Average winter high temperatures (mean = 15.6, median = 17, SD = 12.5) were taken from Templer & Arikawa (2006). Although they used four intercorrelated temperature variables in their analysis, they reported that average winter high temperature was the best predictor of IQ of the four, so we used it.

Although Templer & Arikawa (2006) found a positive relationship between IQ and skin darkness, we will not use skin darkness in our analyses for three reasons: (1) although evidence suggests that skin darkness is a measure of historical infectious disease intensity over evolutionary time, it is unclear exactly what kind of infectious diseases it is indicative of (see discussion), (2) Templer and Arikawa (2006) argued that the relationship between skin darkness and IQ is not causal, and (3) Templer and Arikawa (2006) did not sufficiently explain why the association between intelligence and skin darkness exists. Without a reasonable theoretical framework for this association, we did not feel it was appropriate to compare it to other variables for which there is a better theoretical rational.

Literacy rates (mean = 87.6, median = 96, SD = 15.9) were taken from the World Bank (World Bank 2008). This variable is defined as the percent of the population 15 years old and older who have at least a basic proficiency at reading. Data were from the most recent year available for each nation between 1990 and 2007. Barber (2005) used

data from 1976 for his analysis of literacy because that was the average year in which IQ data were collected for all countries. We felt that a more recent date was more appropriate, however, because the IQ scores for each country we used had been modified based on recorded trends of the Flynn Effect to reflect the expected modern IQ score (Lynn & Vanhanen, 2006).

Enrollment in secondary school (mean = 29.0, median = 28.5, SD = 16.8), completion of secondary school (mean = 12.0, median = 10.5, SD = 9.25), and average years of education (AVED; mean = 6.17, median = 5.81, SD = 2.89) were taken from Barro & Lee (2001). These numbers represent the percent of the population age 25 and older who have attended some or all of secondary school, and the average number of years of schooling in the population. Data was used for the most recent years available after 1990. Data that were only available for years prior to 1990 were omitted.

Data of gross domestic product per capita in US dollars (GDP) was taken from the World Factbook (CIA 2007), and were log-transformed for normality (mean = 3.81, median = 3.85, SD = 0.53).

Distance from central Africa, or the human environment of evolutionary adaptedness (EEA), was calculated using the Pythagorean Theorem, as done by Kanazawa (2008). Kanazawa used three points at the corners of Sub-Saharan Africa, so theoretically any point within this triangle should be a valid centre point from which to calculate distance. We selected -5° latitude, -25° longitude, which is in the approximate centre of this area, and calculated distance from this point using the same methods as Kanazawa. Despite our own criticism of this variable and that of others (Wicherts *et al.*)

2010a) we included this variable in the interest of thoroughness. This variable was log-transformed for normality (mean = 4.0, median = 3.97, SD = 0.710).

Percent consanguineous marriages was not used in this study because it was the conclusion of Woodley (2009) that this variable is unlikely to account for the worldwide variation in intelligence. Additionally, consanguineous marriage and associated inbreeding may be a strategy for maintaining coadapted gene complexes that defend against local infectious diseases (Denic & Nicholls 2007; Fincher & Thornhill 2008).

All analyses were performed using JMP! 8.0.2 statistical software.

#### 3. RESULTS

Log DALY infectious disease and average national IQ correlated at r = -0.82 (n = 107, p < 0.0001) using LVCD, r = -0.82 (n = 184, p < 0.0001; figure 1) using LVE, and r = -0.76 (n = 184, p < 0.0001; figure 1) using WEAM. Zero-order correlations were also performed for each of Murdock's (1949) six world regions (see table 2). A hierarchical linear model (HLM) was also performed to determine whether this relationship is consistent across the six regions. It is (LVE:  $R^2 = 0.78$ , p < 0.0001, n = 184; LVCD:  $R^2 = 0.77$ , p < 0.0001, n = 107; WEAM:  $R^2 = 0.68$ , p < 0.0001, n = 184).

Log DALY nutritional deficiencies and IQ correlated at r = -0.72 (n = 184, p < 0.0001). Log DALY infectious disease and Log DALY nutritional deficiencies correlated at r = 0.89 (n = 192, p < 0.0001). The partial correlation between IQ and DALY nutritional deficiencies with the effects of DALY infectious disease removed was near zero, r = 0.028 (n = 184, p = 0.71), while the partial correlation between IQ and DALY

infectious disease with the effects of DALY nutritional deficiencies removed remained strong, r = -0.56 (n = 184, p < 0.0001). See table 1 for other zero-order correlations. The correlations between these variables and IQ are very similar across the three measures of IQ (see electronic supplementary tables 1 and 2).

To select which, if any, education and wealth variables to include in a multiple regression analysis, partial correlations were performed independently between literacy, enrollment in secondary school, completion of secondary school, and AVED, and average national IQ, with the effects of infectious disease removed. If a variable was no longer significant when the effects of infectious disease were removed, it was not included in the multiple regression. Only WEAM and LVE IQ measures were used for these multivariate analyses in order to have a sample size large enough to make inferences about the individual contributions of each variable. When the effects of log DALY infectious disease were removed, the correlation between IQ and literacy was r = 0.15 (n = 113, p = 0.094) using LVE and r = 0.16 (n = 113, p = 0.1087) using WEAM; IQ and some secondary education was r = 0.093 (n = 120, p = 0.32) using LVE, and r = 0.23(n = 120, p = 0.049) using WEAM; IQ and completion of secondary education was r =0.17 (n = 110, p = 0.08) using LVE and r = 0.23 (n = 110, p = 0.030) using WEAM; IQ and AVED was r = 0.23 (n = 127, p = 0.0084) using LVE and r = 0.30 (n = 127, p = 0.0084) 0.0005) using WEAM; and IQ and GDP was r = 0.054 (n = 184, p = 0.46) using LVE and r = 0.036 (n = 184, p = 0.61) using WEAM. AVED was the best predictor of IQ when the effects of infectious disease were removed for both measures of IQ, so this education variable was used in regressions. As such, AVED will have the best chance of all of the education variables at being significant in the multiple regression. Although GDP was not a statistically significant predictor of IQ when the effects of infectious disease were removed, and the partial correlation coefficients were well below .1, we included this variable in some models at the request of a reviewer.

In a multiple linear regression, average national IQ (LVE and WEAM) was predicted using infectious disease, average winter high temperature, distance from sub-Saharan Africa, AVED, and GDP (see table 3 for model details). Significant predictors in this model were infectious disease, distance from Africa, and winter high temperature. AVED was not significant. When GDP was removed from this model, virtually identical patterns emerged (see electronic supplementary table 3 for model details).

#### 4. DISCUSSION

The negative relationship between infectious disease and IQ was statistically significant at the national level both worldwide and within five of Murdock's (1949) six world regions. All analyses showed that infectious disease was a significant predictor of average national IQ, whether using either of Lynn & Vanhanen's (2006) two data sets or Wicherts *et al.*'s (2010b) data. The zero order correlation between DALY infectious disease and average national IQ was higher than that of any other variable for which there is a previously proposed causal explanation. The world-regions analysis showed that the international pattern is repeated within five out of six regions despite a region's generally similar cultural history. The only world region in which this relationship was not significant is South America. This exception may be due to the presence of several outliers. The group of conspicuous outliers in which IQ was much lower than the

expected in the worldwide trend (figure 1) are all Caribbean countries (St. Lucia, Dominica, St. Kitts and Nevis, Antigua and Barbuda, Grenada, St. Vincent and Grenadines, and Jamaica), which represent 4 of 23 nations in the South America analysis (Saint Lucia, Dominica, Grenada, and Saint Vincent and the Grenadines). Because these outliers are in the same geographical location, it is possible that local parasites which are not included in the DALY infectious disease variable are causing these outliers. Hierarchical Linear Model analysis shows that, despite the nonsignificance of the correlation between IQ and infectious disease within South American nations, this trend is significant overall across Murdock's (1949) six world areas.

Nutritional stress correlated with average national IQ (r = -0.72), but this relationship was not significant when the effects of infectious disease were removed. This supports the suggested link between intelligence and nutrition. Given the energetic cost of infectious disease, individuals who are burdened with parasites may be more likely to be affected by nutritional deficiencies. Likewise, individuals who are suffering from nutritional deficiencies may be less able to mount an effective immune response.

Multiple regression shows that, of infectious disease, temperature, evolutionary novelty, and average years of education, infectious disease is the best predictor of intelligence by a large margin. The effects of years of education are not significant, while temperature and evolutionary novelty seem to have distinct predictive power beyond infectious disease. Although this model cannot rule out the independent effect of distance from central Africa, this effect is difficult to interpret because of the doubt cast on the theory underlying this variable (Wicherts *et al.* 2010a). Although the effects of education and GDP per capita are not statistically significant when other factors are controlled for,

this is not to say that these factors are not involved. A nation of more intelligent individuals is likely to produce a higher GDP, but a wealthier nation is also more able to pay for public education as well as public medical and sanitation services. An indirect link between education and intelligence may also exist, as a better-educated population may be more interested in public health measures — leading to increased IQ by reducing parasite stress — provided that education includes information about germ theory and hygiene. These sources of endogeneity must be considered when interpreting our findings (and see below). It should also be mentioned that we are not arguing that global variation in intelligence is only caused by parasite stress. Rather, variation in intelligence is likely caused by a variety of factors, including those we have mentioned here as well as factors that are yet unknown.

If the general pathway we propose is correct, there are two plausible mechanisms by which a tradeoff in allocation to immune vs. brain development and maintenance may occur. First, parasitic infection may intermittently cause the redirection of energy away from brain development. In this case, during periods of infection, the brain receives fewer energetic resources, but this allocation to brain function will return to pre-infection levels during healthy periods. During periods of infection, whatever aspects of the brain that are growing and developing will suffer reduced phenotypic quality. Second, exposure to infectious agents may cause a developmental pathway that permanently invests more energy into immune function at the expense of brain growth. In this scenario, large amounts of energy would be allocated into immune function during periods of health, as opposed to only redirecting energy during periods of infection. This could operate through a variety of mechanisms. A plausible mechanism is that higher investment in

immune system is triggered by individual exposure to infectious disease at some point during ontogeny. This may include triggering from exposure to maternal antibodies while in utero.

We also propose a complementary hypothesis that may explain some of the effects of infectious disease on intelligence. As we mentioned, it is possible that a conditional developmental pathway exists that invests more energy into the immune system at the expense of brain development. In an environment where there has consistently been a high metabolic cost associated with parasitic infection, selection would not favor the maintenance of a phenotypically plastic trait. That is, the conditional strategy of allocating more energy into brain development during periods of health would be lost, evolutionarily, if periods of health were rare. Peoples living in areas of consistently high prevalence of infectious disease over evolutionary time thus may possess adaptations that favor high obligatory investment in immune function at the expense of other metabolically expensive traits such as intelligence. Data does not currently exist on temporal variation of the severity of infectious disease across the world over human history. For genetically distinct adaptations in intelligence to exist based on this principle, parasite levels must be quite consistent over evolutionary time. If this is not the case, then selection would maintain investment in the immune system and in the brain as a plastic — as opposed to static — trait. The Flynn Effect (Flynn 1987) indicates that conditional developmental causes must be at work at least in part. Large increases in intelligence across a few generations cannot be attributed to genetic differences caused by evolutionary processes. Hence, it does not seem likely that region-specific genetic adaptations are the primary cause of the worldwide variation in intelligence.

Our findings suggest that the heritable variation in intelligence may come from two sources: brain structure and immune system quality. Thus, two individuals may possess identical genes for brain structure, but have different IQ due to differences in immune system quality reflecting their personal allocation of energy into brain versus immunity.

Our findings are consistent with a number of other findings in the literature. In particular, the Flynn Effect (Flynn 1987) demands that any hypothesis regarding the worldwide variation and distribution of intelligence must be able to account for some factor that allows for large IQ gains over time spans seemingly too short to be attributed to evolution by natural selection. The parasite-stress hypothesis allows for such a factor in the form of reduced parasitic infection. As societies become modernized, decreased parasite stress may occur through multiple pathways. As national wealth increases, medicine, vaccinations, and potable water can be purchased by both the government and by individuals. Moreover, there is cross-national evidence that, as democratization increases, there are corresponding increases in public health legislation and infrastructure. Democratization also increases levels of education, better allowing individuals to seek out and understand information that reduce parasitic infection (Thornhill *et al.* 2009). This source of endogeneity is not a flaw, but a prediction of our hypothesis.

Mackintosh (2001) presented comprehensive evidence that skin darkness and the associated cellular components (e.g. melanocytes) have an important role in defending against infectious disease. Moreover, Manning *et al.* (2003) found that, in sub-Saharan Africa, rates of HIV infection were negatively associated with skin darkness. Manning *et al.* (2003) attributed this relationship in part to lower infection rates of other parasites,

especially bacteria and fungi, that lead to tissue damage in the genital tract and hence increased opportunity for contracting HIV. Templer & Arikawa (2006) concluded that, despite the strong negative correlation between skin colour and average national IQ, there must be an unknown mediating factor accounting for both because there is no obvious reason for skin darkness to reduce IQ. Given the previous research linking skin colour to infectious disease (Mackintosh 2001; Manning *et al.* 2003), the unknown factor linking skin colour and IQ may be infectious disease.

Several studies have shown a positive relationship between IQ and body symmetry (e.g. Furlow *et al.* 1997; Prokosch *et al.* 2005; Bates 2007; Penke *et al.* 2009; but see also Johnson *et al.* 2008). There is evidence that body symmetry is a measure of developmental stability, an important component of which is due to reduced contact with infectious disease (Thornhill & Møller 1997). Our study suggests that IQ and body symmetry correlate because they are both affected negatively by exposure to high infectious disease. Individuals who are exposed to infectious disease may have many aspects of their body develop imperfectly, including the brain, negatively affecting both their body symmetry and cognitive ability. Indeed, recent research indicates that there is a positive relationship between body asymmetry and atypical brain asymmetries (Yeo *et al.* 2007).

The hygiene hypothesis proposes that some autoimmune diseases may be caused by low exposure to pathogens during ontogeny (e.g. Strachan 1989). Previous studies of individual differences have shown that intelligence correlated positively with the frequency of asthma and allergies (reviewed in Jensen & Sinha 1993). According to the parasite-stress hypothesis, high intelligence is allowed in part by low exposure to

infectious disease. Thus the relationship between intelligence and autoimmune diseases, such as asthma and allergies (reviewed in Gangal & Chowgule 2009), is likely mediated through exposure to infectious disease. We predict that this positive relationship between IQ and autoimmune diseases will also be robust across nations, and that it will be mediated by infectious disease.

Although our results support our predictions, further studies must be done to establish causation. Longitudinal methods could be used to test this hypothesis on the individual level. Children's IQ could be measured at an early age and IQ re-measured later in life, while monitoring for infectious diseases throughout childhood. This would not only provide another test of our hypothesis, but may be able to determine the affects of individual infectious diseases on cognitive development. Additionally, it could be determined which, if either, tradeoff mechanism we discussed is responsible for the detrimental effects of infectious disease on intelligence. Both may operate but with geographical differences based on the consistency of infectious disease over time. As nations develop, they could be monitored for declining rates of parasitic infection to determine (1) whether this corresponds with elevated IQ and (2) whether any IQ gain is sufficient to account for the Flynn Effect.

The authors thank Rachael Falcon, Steve Gangestad, Angela Hung, Kenneth Letendre, Paul Watson, and Rhiannon West for their comments and suggestions. Three anonymous reviewers provided helpful and polite criticism.

#### REFERENCES

- Barber, N. 2005. Educational and ecological correlates of IQ: a cross-national investigation. *Intelligence* **33**, 273-284.
- Barro, R. J. & Lee, J. 2001. International data on educational attainment: updates and implications. *Oxford Econ. Pap.* **53**, 541-563.
- Bates, T. C. 2007. Fluctuating asymmetry and intelligence. *Intelligence* **35**, 41-46.
- Bleakley, H. 2007. Disease and development: evidence from hookworm eradication in the American south. *Q. J. Econ.* **122**, 73-117.
- Borsboom, D. & Dolan, C. V. (2006). Why *g* is not an adaptation: a comment on Kanazawa (2004). *Psychol. Rev.* **113**, 433-437.
- Ceci, S. J. (1991). How much does schooling influence general intelligence and its cognitive components? A reassessment of the evidence. *Dev. Psychol.* **27**, 703-722.
- CIA World Factbook. 2007. https://www.cia.gov/library/publications/the-world-factbook/
- Denic, S. & Nicholls, M. G. 2007. Genetic benefits of consanguinity through selection of genotypes protective against malaria. *Hum. Biol.* **79**, 145-158.
- Dickson, R., Awasthi, S., Williamson, P., Demellweek, C. & Garner, P. 1997. Effects of treatment for intestinal helminth infection on growth and cognitive performance in children: systematic review of randomized trials. *Brit. Med. J.* **320**, 1697-1701.
- Fincher, C. L. & Thornhill, R. 2008. A parasite-driven wedge: infectious diseases may explain language and other biodiversity. *Oikos* **117**, 1289-1297.

- Flynn, J. R. 1987. Massive IQ gains in 14 nations: what IQ tests really measure. *Psychol. Bull.* **101**, 171-191.
- Furlow, F. B., Armijo-Prewitt, T., Gangestad, S. W. & Thornhill, R. 1997. Fluctuating asymmetry and psychometric intelligence. *P. Roy. Soc. Lond. B Biol.* **264**, 823-829.
- Gangal, S. V. & Ghowgule, R. 2009. Infections in early life and susceptibility to allergic diseases: relevance of hygiene hypothesis. *Curr. Sci. India* **96**, 784-793.
- Guernier, V., Hochberg, M. E. & Guégan, J. 2004. Ecology drives the worldwide distribution of human diseases. *PLOS Biol.* **2**, 740-746.
- Holliday, M. A. 1986. Body composition and energy needs during growth. In *Human Growth: A Comprehensive Treatise, Volume 2* (ed. F. Falkner, J. M. Tanner), pp. 101-117. New York, NY; Plenum.
- Jardin-Botelho, A., Raff, S., Rodrigues, R. A., Hoffman, H. J., Diemert, J. H., Correa-Oliviera, R., Bethony, J. M., & Gazzinelli, M. F. (2008). Hookworm, *Ascaris lumbricoides* infection and polyparasitism associated with poor cognitive performance in Brazilian schoolchildren. *Trop. Med. Int. Health.* **13**, 994-1004.
- Jensen, A. R. & Sinha, S. N. 1993. Physical correlates of human intelligence. In Biological Approaches to the Study of Human Intelligence (ed. P. A. Vernon), pp 139-242. Norwood, NJ; Ablex.
- Johnson, W., Segal, N. L. & Bouchard Jr., T. J. 2008. Fluctuating asymmetry and general intelligence: no genetic of phenotypic association. *Intelligence* **36**, 279-288.
- Kanazawa, S. 2004. General intelligence as a domain-specific adaptation. *Psychol. Rev.* **111**, 512-523.

- Kanazawa, S. 2008. Temperature and evolutionary novelty as forces behind the evolution of general intelligence. *Intelligence* **36**, 99-108.
- Lynn, R. 1990. The role of nutrition in secular increases in intelligence. *Person. Indiv. Differ.* **11**, 263-285.
- Lynn, R. 1991. The evolution of racial differences in intelligence. *Mankind Quart.* **32**, 99-121.
- Lynn, R. 1993. Nutrition and Intelligence. *In Biological Approaches to the Study of Human Intelligence* (ed. P. A. Vernon), pp. 243-258, Norwood, NJ; Ablex.
- Lynn, R. & Meisenberg, G. (2010). The average IQ of sub-Saharan Africans: Comments of Wicherts, Dolan, and van der Maas. *Intelligence* **38**, 21-29.
- Lynn, R. & Mikk, J. 2007. National differences in intelligence and educational attainment. *Intelligence* **35**, 115-121.
- Lynn, R. & Vanhanen, T. 2001. National IQ and economic development: a study of eighty-one nations. *Mankind Quart.* **41**, 415-435.
- Lynn, R. & Vanhanen, T. 2002. IQ and the Wealth of Nations. Westport, CT; Praeger.
- Lynn, R. & Vanhanen, T. 2006. *IQ and Global Inequality*. Augusta, GA; Washington Summit.
- Mackintosh, J. A. 2001. The antimicrobial properties of malanocytes, melanosomes and melanin and the evolution of black skin. *J. Theor. Biol.* **211**, 101-113.
- Manning, J. T., Bundred, P. E. & Henzi, P. 2003. Melanin and HIV in sub-Saharan Africa. *J. Theor. Biol.* **223**, 131-133.

- Møller, A. P. 1998. Evidence of larger impact of parasites on hosts in the tropics: investment in immune function within and outside the tropics. *OIKOS*, **82**, 265-270.
- Murdock, G. P. 1949. Social Structure. New York, NY; MacMillan.
- Penke, L., Bates, T. C., Gow, A. J., Pattie, A., Starr, J. M., Jones, B. C., Perrett, D. I., & Deary, I. J. (2009). Symmetric faces are a sign of successful cognitive aging. *Evol. Hum. Behav.* **30**, 429-437.
- Prokosch, M. D., Yeo, R.A. & Miller, G.F. 2005. Intelligence tests with higher g-loadings show higher correlations with body symmetry: evidence for a general fitness factor mediated by developmental stability. *Intelligence* **33**, 203-213.
- Rindermann, H. 2007. The *g*-factor of international cognitive ability comparisons: the homogeneity of results in PISA, TIMSS, PIRLS and IQ-tests across nations. *Eur. J. Personality* **21**, 667-706.
- Rushton, J. P. 1995. *Race, Evolution, and Behavior: A Life History Perspective*. New Brunswick, NJ; Transaction.
- Rushton, J. P. 2000. *Race, Evolution, and Behavior: A Life History Perspective, 2<sup>nd</sup> Special Abridged Edition*. Port Huron, MI; Charles Darwin Research Institute.
- Saadat, M. 2008. Consanguinity and national IQ scores. *J. Epidemiol. Commun. H.* **62**, 566-567.
- Strachan, D. P. 1989. Hay-fever, hygiene, and household size. *Brit. Med. J.* **299**, 1259-1260.
- Templer, D. I. & Arikawa, H. 2006. Temperature, skin color, per capita income, and IQ: an international perspective. *Intelligence* **34**, 121-139.

- Thornhill, R., Fincher, C. L. & Aran, D. 2009. Parasites, democratization, and the liberalization of values across contemporary countries. *Biol. Rev.* **84**, 113-131.
- Thornhill, R. & Møller, A. P. 1997. Developmental stability, disease and medicine. *Biol. Rev.* **72**, 497-548.
- Watkins, W. E. & Pollitt, E. 1997. "Stupidity or Worms": do intestinal worms impair mental performance? *Psychol. Bull.* **121**, 171-191.
- Wicherts, J.M., Borsboom, D. & Dolan, C.V. (2010a). Why national IQs do not support evolutionary theories of intelligence. *Person. Indiv. Differ.* **48**, 91-96.
- Wicherts, J.M., Dolan, C.V. & van der Maas, H.L.J. (2010b). A systematic literature review of the average IQ of sub-Saharan Africans. *Intelligence* **38**, 1-20.
- Woodley, M. A. 2009. Inbreeding depression and IQ in a study of 72 countries. *Intelligence* **37**, 268-276.
- World Bank. 2008.
  - http://web.worldbank.org/WBSITE/EXTERNAL/DATASTATISTICS/0,,menuP K:232599~pagePK:64133170~piPK:64133498~theSitePK:239419,00.html
- World Health Organization. 2004a. *Global Burdon of Disease: 2004 Update*. Switzerland; World Health Organization.
- World Health Organization. 2004b. *World Health Report 2004*. Switzerland; World Health Organization.
- Yeo, R. A., Gangestad, S. W. & Thoma, R. J. 2007. Developmental instability and individual variation in brain development: implications for the origin of neurodevelopmental disorders. *Curr. Dir. Psychol. Sci.* **16**, 245-249.

	1	2	3	4	5	6	7	8	9
1. average IQ		-0.82	-0.72	0.48	0.61	0.74	0.64	0.36	0.67
2. DALY disease	184		0.71	-0.39	-0.66	-0.79	-0.67	-0.32*	* -0.79
3. winter high	124	122		-0.40	-0.57	-0.76	-0.76	-0.25*	-0.52
4. distance from EEA	190	192	124		0.40	0.36	0.25**	0.12ns	0.30
5. literacy	113	113	78	118		0.73	0.61	0.17ns	0.65
6. AVED	130	127	86	131	82		0.86	0.43	0.81
7. some 2° education	123	120	86	123	78	123		0.46	0.67
8. complete 2° education	114	112	73	126	68	95	91		0.37
9. GDP	190	192	124	226	117	130	123	120	

(Table 1: Zero-order correlations among average National IQ (LVE), log DALY infectious disease, average winter high temperature, distance from EEA, literacy, average years of education (AVED), % enrolling in secondary education, % completing all secondary education, and GDP. Values below the diagonal are sample sizes (number of countries), values above the diagonal are correlation coefficients. \* indicates p < 0.05, \*\* indicates p < 0.01, ns indicates p > 0.05. All others p < 0.0001.)

sample size

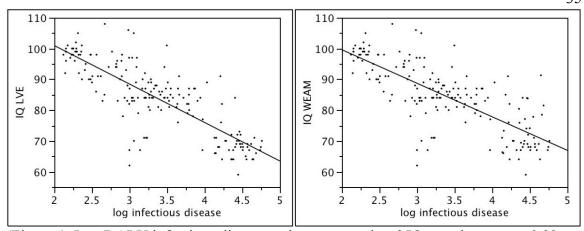
world area	correlation (r)	(countries)	p-value
Africa	-0.80 (-0.80) [-0.49]	53 (22) [53]	<0.0001 (<0.0001) [0.0002]
Eastern Eurasia	-0.62 (-0.70)	20 (11)	0.0033 (0.016)
Insular Pacific	-0.85 (-0.83)	17 (12)	<0.0001 (0.0009)
North America	-0.65 (-0.76)	12 (7)	0.022 (0.049)
South America	0.077 (0.043)	23 (16)	0.73 (0.88)
Western Eurasia	-0.65 (-0.73)	59 (39)	<0.0001 (<0.0001)

(Table 2: Zero-order correlations between average national intelligence and log DALY infectious disease within each of Murdock's (1949) six world regions. Values not in parentheses used LVE, values in parentheses used LVCD, and values in brackets used WEAM.)

term	estimate	STD error	STD beta	VIF	p
intercept	95.7 (95.4)	10.9 (13.6)	-	-	<0.0001
DALY disease	-8.30 (-6.50)	1.30 (1.61)	-0.597 (-0.51)	6.03	<0.0001 (0.0001)
distance from EEA	5.03 (3.91)	0.983 (1.22)	0.231 (0.20)	1.41	<0.0001 (0.0021)
winter high	-0.239 (-0.217)	0.0686 (0.0853)	-0.228 (-0.23)	2.97	0.0008 (0.013)
AVED	-0.0279 (0.394)	0.322 (0.40)	-0.00683 (0.10)	4.30	0.93 (0.33)
GDP	0.265 (-0.262)	0.854 (2.45)	0.0269 (-0.013)	5.22	0.76 (0.92)

whole model: n=83 countries, p<0.0001, R<sup>2</sup>=0.889 (0.796)

(Table 3: Multiple regression analyses predicting average national intelligence using LVE and WEAM (in parentheses where different) by log DALY infectious disease, log distance from EEA, average winter high temperature, average years of education (AVED), and log GDP.)



(Figure 1: Log DALY infectious disease and average national IQ correlate at r = -0.82 (LVE, left) and at r = -0.76 (WEAM, right) (n = 184, p < 0.0001). The line is the least-squares line through the points.)

	1	2	3	4	5	6	7	8	9
1. average IQ		-0.82	-0.72	0.48	0.59	0.75	0.50	0.50	0.73
2. DALY disease	107		0.71	-0.39	-0.66	-0.79	-0.67	-0.32**	* -0.79
3. winter high	71	122		-0.40	-0.57	-0.75	-0.76	-0.25*	-0.52
4. distance from EEA	112	192	124		0.40	0.36	0.25**	0.12ns	0.30
5. literacy	66	113	78	118		0.73	0.61	0.17ns	0.65
6. AVED	91	127	91	86	82		0.86	0.43	0.81
7. some 2° education	89	120	86	123	78	123		0.46	0.67
8. complete 2° education	73	112	73	126	68	95	91		0.37
9. GDP	112	192	124	226	117	130	123	120	

(Electronic Supplementary Table 1: Zero-order correlations among average National IQ (LVCD), log DALY infectious disease, average winter high temperature, distance from EEA, literacy, average years of education (AVED), % enrolling in secondary education, % completing all secondary education, and GDP. Values below the diagonal are sample sizes (number of countries), values above the diagonal are correlation coefficients. \* indicates p < 0.05, \*\* indicates p < 0.01, ns indicates p > 0.05. All others p < 0.0001)

	1	2	3	4	5	6	7	8	9
1. average IQ		-0.76	-0.70	0.40	0.57	0.73	0.65	0.38	0.62
2. DALY disease	184		0.71	-0.39	-0.66	-0.79	-0.67	-0.32**	* -0.79
3. winter high	124	122		-0.40	-0.57	-0.75	-0.76	-0.25*	-0.52
4. distance from EEA	190	192	124		0.40	0.36	0.25**	0.12ns	0.30
5. literacy	113	113	78	118		0.73	0.61	0.17ns	0.65
6. AVED	130	127	91	86	82		0.86	0.43	0.81
7. some 2° education	123	120	86	123	78	123		0.46	0.67
8. complete 2° education	114	112	73	126	68	95	91		0.37
9. GDP	190	192	124	226	117	130	123	120	

(Electronic Supplementary Table 2: Zero-order correlations among average National (WEAM), log DALY infectious disease, average winter high temperature, distance from EEA, literacy, average years of education (AVED), % enrolling in secondary education, % completing all secondary education, and GDP. Values below the diagonal are sample sizes (number of countries), values above the diagonal are correlation coefficients. \* indicates p < 0.05, \*\* indicates p < 0.01, ns indicates p > 0.05. All others p < 0.0001.)

term	estimate	STD error	STD beta	VIF	p
intercept	98.6 (94.2)	5.98 (7.45)	-	-	<0.0001
DALY disease	-8.56 (-6.39)	0.983 (1.22)	-0.616 (-0.50)	3.50	<0.0001
distance from EEA	5.05 (3.90)	1.00 (1.22)	0.232 (0.195)	1.41	<0.0001 (0.0019)
winter high	-0.235 (-0.219)	0.0666 (0.0828)	-0.224 (-0.228)	2.83	0.0007 (0.0099)
AVED	0.0116 (0.377)	0.294 (0.365)	0.00285 (0.10)	3.62	0.97 (0.31)

whole model: n = 83 countries, p < 0.0001,  $R^2 = 0.889$  (0.796)

(Electronic Supplementary Table 3: Multiple regression analyses predicting average national intelligence using LVE and WEAM (in parentheses when different) by log DALY infectious disease, log distance from EEA, average winter high temperature, and average years of education (AVED).)

Chapter 3: Parasite prevalence and the distribution of intelligence among the states of the USA.

Christopher G. Eppig, Corey L. Fincher and Randy Thornhill

This chapter was published in substantially similar form as Eppig, C., Fincher, C. L. and Thornhill, R. (2011). Parasite prevalence and the distribution of intelligence among the states of the USA. *Intelligence*, 39, 155-160.

Parasite prevalence and the distribution of intelligence among the states of the USA

Christopher Eppig<sup>a</sup>, Corey L. Fincher<sup>a</sup>, Randy Thornhill<sup>a</sup>

<sup>a</sup>Biology Department, MSC03 2020, 1 University of New Mexico, Albuquerque, NM 87131, USA

Contact Information for Corresponding Author:

Christopher Eppig

UNM Biology Department MSC03 2020

1 University of New Mexico

Albuquerque, NM 87131

Phone: 1(505)277-4999

e-mail: ceppig@unm.edu

## **ABSTRACT**

In this study, we tested the parasite-stress hypothesis for the distribution of intelligence among the USA states: the hypothesis proposes that intelligence emerges from a developmental trade-off between maximizing brain vs. immune function. From this we predicted that among the USA states where infectious disease stress was high, average intelligence would be low and where infectious disease stress was low, average intelligence would be high. As predicted, we found that the correlation between average state IQ and infectious disease stress was -0.67 (p < 0.0001) across the 50 states. Furthermore, when controlling the effects of wealth and educational variation among states, infectious disease stress was the best predictor of average state IQ.

Keywords: Brain Growth, Developmental Stability, Infectious Disease, Cognitive Ability, Biodiversity

#### Introduction

Several factors have been proposed to explain the global biodiversity of human intelligence on the cross-national level: education, wealth, agricultural labor, low birth weight, childhood mortality (Barber, 2005), temperature (Templer & Arikawa, 2006), evolutionary novelty of the environment (Kanazawa, 2008a), inbreeding depression (Saadat, 2008; Woodley, 2009), and parasite stress (a broad, ecological definition of 'parasite' was used here to mean any infectious organism; Eppig et al., 2010). Of these

factors, we found infectious disease stress was the best predictor of average national intelligence, with correlations between infectious disease stress and average national IQ ranging from r = -0.76 to r = -0.82 (Eppig et al., 2010). This relationship remained consistent in five of six world regions, and was significant overall across the six world regions when combined in a hierarchical linear model ( $R^2 = 0.77$  to 0.78, p < 0.0001, n = 184 countries). When considered with average temperature, distance from central Africa, average years of education, and gross domestic product per capita in a multiple regression analysis, infectious disease had the largest independent power to predict average IQ (Eppig et al., 2010).

The human brain is extremely metabolically expensive to produce (e.g. Holliday, 1986). Infectious disease may prevent a developing human from accessing sufficient energy to produce a high quality brain in four ways: (1) the body must replace tissue damaged or destroyed by parasites, (2) some parasites prevent access to ingested nutrients, through diarrhea, vomiting, or extraction from the host's digestive tract, (3) by using the host-body's macromolecular stores and cellular machinery to reproduce, and (4) by activating an immune response (Eppig et al., 2010). Given the cost of brains and the simultaneous cost imposed by disease, Eppig et al. (2010) argued that there is a developmental trade-off between the two, such that individuals who are exposed to energetically costly infectious diseases during the times when the brain is growing will suffer reduced phenotypic quality of the brain, leading to lower intelligence.

Several authors have argued that IQ negatively affects disease prevalence and positively affects other measures of health on the societal level. While it is almost certainly true that a population with a higher average intelligence will be more able to

cope with the difficulties of disease, and more able to afford medicine and other public health measures (as we have previously argued; Eppig et al., 2010), prior studies of these effects have not focused primarily on infectious disease. Instead, measures of health have included diseases that may be largely non-infectious (including obesity and heart disease), as well as health outcomes that are not necessarily directly or primarily related to infectious disease (including infant mortality, maternal mortality, life expectancy, and fertility rate). The only explicit measure of infectious disease was rates of HIV/AIDS, which is only one of the many human infectious diseases (Reeve, 2009; Reeve & Basalik, 2010; Rindermann & Meisenberg, 2009; Oesterdiekhoff & Rindermann, 2007). Furthermore, although there is a history of human intervention eradicating infectious disease in certain areas — such as the eradication of malaria, smallpox and polio from some or all regions of the earth — the distribution of human infectious disease is largely, if not primarily, exogenous of humans. Accordingly, it is dependent on climate and other local ecological factors (Dunn et al., 2010; Guernier et al., 2005) and shares a similar geographic pattern with infectious disease of other species (Calvete et al., 2004; Møller, 1998; Nunn et al., 2005).

If infectious disease is an important factor determining the biodiversity of human intelligence, infectious disease should predict intelligence on the cross-national level (as we have shown; Eppig et al., 2010), as well as on smaller geographic scales. Using regional variation within a nation, as opposed to between nations, avoids potential confounds produced by large, cross-national differences in culture. In this study, we will continue to test the parasite-stress hypothesis applied to cognitive ability. We test the prediction that, among USA States, levels of parasite-stress will negatively predict

average state intelligence, and that this relationship will be robust when other factors that have been proposed as causes of variation in intelligence are controlled.

### Methods

Four measures of average IQ among the states of the USA exist: (1) Kanazawa (2006) estimated average state IQ based on scores on the Scholastic Achievement Test (SAT), (2) McDaniel (2006a) estimated average state IQ based on scores on the ACT (not an acronym) test, (3) McDaniel (2006a) estimated average state IQ based on a composite of the SAT and ACT scores, and (4) McDaniel (2006b) estimated average state IQ based on scores on the National Assessment of Educational Progress test (NAEP), which, like the SAT and ACT, tests abilities in reading and math (see McDaniel, 2006b for more details). Of these four measures, those using either the SAT or ACT alone have shown to be incorrect. McDaniel (2006a) found that estimates of average state IQ derived from SAT scores correlated with average state IQ scores derived from the ACT at r = -0.84, and that these two measures seem actually to reflect only the percent of high school graduates who took one test or the other in a given state. The NAEP test is given to a sample of all public school students in all 50 states, and thus tests a sample of students in a state that is more representative than the SAT or ACT. The SAT and ACT are given to a highly non-random sample, as they are only taken by students who plan to attend colleges or universities that require one of these tests for admission, and the average state IQ scores that are based on these tests must reconstruct the rest of the IQ distribution for a state based on a highly nonrandom sample of people. These

estimates of average state IQ are therefore inherently less reliable than estimates that are based on more representative samples of the population. Indeed, McDaniel (2006a) found that average state IQ scores based on the SAT/ACT composite only correlate at r = 0.58 with average state IQ scores based on the NAEP, and noted that this correlation is not nearly high enough for two variables that ostensibly measure the same thing. McDaniel (2006a) suggests that the scores based on the NAEP are far more likely to be accurate, and we agree. For this reason, we will use only the NAEP-based IQ scores in these analyses (n = 50 states, mean = 100.3, median = 100.9, SD = 2.7).

We obtained the annual *Morbidity and Mortality Weekly Report's* "Summary of Notifiable Diseases, United States" from the Centers for Disease Control (CDC) for the years 1993 to 2007 (Center for Disease Control, 2008). For each year we adjusted the number of cases of all infectious diseases tracked by CDC for which there was information for all states for that year by the CDC-reported population size for each state (i.e., for some diseases, not all states reported whether cases occurred [termed 'non-notifiable' by CDC]; these diseases were not included in the tally). For each state, we determined the average z-score of this population adjusted disease incidence score for the 15 year time-span. This approach was necessary because the infectious diseases tracked by the CDC can vary between years, though there was often great similarity between years. The standardization allowed us to pinpoint each state's position along a parasite-stress gradient relative to the other states. Diseases in this measure include cholera, measles, meningitis, pertussis, rubella, tetanus, and tuberculosis (see Fincher & Thornhill, in press, for further details).

This index of parasite-stress,  $Parasite-Stress\ USA$ , is validated by the fact that it shows a negative correlation with latitude (-0.45, n = 50, p = 0.001; or after removing the latitudinal outliers Alaska and Hawaii, -0.71, n = 48, p < 0.0001) just as do global measures of parasite-stress (Cashdan, 2001; Guernier et al., 2004; Low, 1990). Furthermore,  $Parasite-Stress\ USA$  was correlated strongly and negatively across US states with the average lifespan expectancy at birth for both sexes in the year 2000 according to data we collected from www.census.gov (r = -0.67, n = 50, p < 0.0001). Similar strong relationships between infectious disease stress and lifespan expectancy are found in cross-national analyses (Thornhill et al., 2009). This variable was z-scored (mean = -0.0044, median = -0.023, SD = 0.91). See Fincher & Thornhill (in press) for further details and data.

Two measures of education quality were used: (1) student/teacher ratio (mean = 14.9, median = 14.1, SD = 2.6) and (2) percent of teachers in public schools teaching core classes who are 'highly qualified' (mean = 95.5, median = 97.4, SD = 5.08). Data on student/teacher ratio were taken from the National Center for Education Statistics, and are from the 2008-2009 school year (National Center for Education Statistics, 2009). 'Highly qualified' refers to teachers teaching core classes who are fully certified to teach in their state. Data on highly qualified teachers were taken from the US Department of Education and are from the 2008-2009 school year (US Department of Education, 2009). It should be noted that, because our measure of IQ was based on educational outcomes, the variables we selected to measure education quality cannot also be based on educational outcomes. To this end, the qualifications of the teachers and the student-teacher ratios are effective ways to measure the quality of the educational opportunities to

which students in each state have access. These two variables correlate with each other only moderately (r = -0.35, n = 50, p = 0.012), so it would not be appropriate to combine them.

Three measures of state wealth were used: (1) median household income (mean = 51966, median = 50170, SD = 8509), (2) income per capita (mean = 20767, median = 20566, SD = 2849), and (3) gross state product (mean = 0.0353, median = 0.0345, SD = 0.0065). Median household income and income per capita are in US dollars, and were taken from the US Census Bureau from the year 2000 census. Gross state product is in millions of US dollars, and is from McDaniel (2006b).

Our three measures of wealth (income per capita, gross state product, and median household income) correlate highly with one another (table 1) and are all measures of the same construct: state wealth. Using these three variables separately in our analysis could introduce multicolinearity into our model that could make our results unreliable. Hence, we reduced these variables into a single wealth variable by extracting the first principal component in a principal component analysis. This component accounts for 87% of the total variance in these three measures of wealth, and each measure of wealth loads on this component at 0.91 or higher. This constructed variable will hereafter be referred to as 'wealth.'

In addition to economic and education variables, Eppig et al.'s (2010) crossnational study examined variables that had previously been thought to predict average national intelligence because of influence over evolutionary time (e.g. geographical distance from deep-time evolutionary historical environments; Kanazawa, 2008a). Since the majority of populations living in US states have not been occupying those locations over any considerable length of evolutionary time, these variables are not appropriate for this analysis and were not used. It should be noted that temperature, which has previously been considered to be an evolutionary variable (e.g. Templer & Arikawa, 2006) has been found to correlate negatively with average state IQ (Ryan et al., 2010). We did not include this variable for two reasons: (1) Ryan et al. (2010) did not attempt to explain why this relationship exists, so we cannot treat temperature as a causal variable, and (2) temperature is known to positively influence infectious disease (e.g. Guernier et al., 2004), so by controlling for temperature we would, in part, be controlling for infectious disease.

Other studies examining the distribution of IQ across the United States have controlled for race, as it has been documented to be associated with IQ (e.g. Ryan et al., 2010). Currently, the southeastern states in the USA have higher percentages of Blacks than do states in other regions. Southeastern states also have higher rates of infectious disease, due largely to climate (see introduction). Thus controlling for race, the distribution of which closely follows the distribution of infectious disease, does not add meaningfully to our analysis. Furthermore, we found that the percent of a state population who are Black correlates much more strongly with our measure of infectious disease (r = 0.90, n = 50, p < 0.0001) than percent Black does with average IQ (r = -0.51, n = 50, p = 0.0001) (state data for percent Black from US Census Bureau, 2000). In a multiple regression with percent Black and infectious disease predicting average state IQ, percent Black is only marginally significant (p = 0.059), and has an additive  $R^2$  of only 0.045 over infectious disease. The high variance inflation factor (VIF = 5.2) further complicates

the use of this variable in our analyses. Thus, percent Black will not be used in our analyses for both theoretical and empirical reasons.

All statistical analyses were performed using JMP 8 statistical software.

## Results

Average state IQ and parasite stress correlated at r = -0.67 (n = 50, p < 0.0001; figure 1). Average IQ also correlated significantly with wealth (r = 0.32, n = 50, p = 0.025), percent teachers highly qualified (r = 0.42, n = 50, p = 0.0023), and student-teacher ratio (r = -0.31, n = 50, p = 0.031) (see table 1 for additional correlations).

Hierarchical regression was used to predict average state IQ using parasite stress, wealth, percent teachers highly qualified, and student/teacher ratio (table 2). Parasite stress was added in the first iteration of the model, resulting in a change in  $R^2$  of 0.445. Wealth was added in the second iteration of the model, resulting in a change in  $R^2$  of 0.075. Both education variables were added simultaneously in the third iteration of the model because they both measure the same theoretical construct, resulting in a change in  $R^2$  of 0.133. While these variables were added into the model in order of presumed causal priority, adding these variables in a different order did not appreciably change the additive  $R^2$  of each iteration. In the final model, parasite stress (Std Beta = -0.62, variance inflation factor (VIF) = 1.02, p < 0.0001), wealth (Std Beta = 0.30, VIF = 1.00, p = 0.0006), percent teachers highly qualified (Std Beta = 0.29, VIF = 1.16, p = 0.0019), and student/teacher ratio (Std Beta = -0.22, VIF = 1.15, p = 0.015) (table 3) were all significant predictors of average state IQ. The whole model  $R^2$  was 0.698 (p < 0.0001).

The VIF was well below 2 for all variables in all models, indicating that multicolinearity did not introduce significant error into these models, and that the standardized beta coefficients are interpretable (Fox, 1991).

#### **Discussion**

Across US states, there is a negative association between infectious disease stress and average intelligence (r = -0.67, n = 50, p < 0.0001). This relationship remains robust and significant when economic and education variables are controlled for in a hierarchical regression. In this regression, infectious disease, wealth, percent teachers highly qualified, and student/teacher ratio were significant. Of these variables, infectious disease had more independent predictive power (Std Beta = -0.62) than wealth (Std Beta = 0.30), percent teachers highly qualified (Std Beta = 0.29), or student/teacher ratio (Std Beta = -0.22). Infectious disease also had the highest additive  $R^2$  in our hierarchical regression (0.445 for infectious disease, compared to 0.075 for wealth and 0.113 for education).

In this analysis, infectious disease does not predict average IQ as well as it did in a similar analysis across nations, and education and economic variables have higher predictive power (Eppig et al., 2010). It is possible that this is an artifact of the way average IQ was measured across US states. Although the NAEP test, which was used to calculate average state IQ, is a valid measure of IQ (McDaniel, 2006b), it is likely influenced by education more than tests used cross-nationally which are designed to measure IQ more directly. It is also possible that the zero-order correlation between

infectious disease and average state IQ (r = -0.67) is lower than the correlation between infectious disease and average national IQ (r = -0.76 to -0.82; Eppig et al., 2010) because there is a wider range of IQ and of parasite stress on the cross-national level than there is on the cross-state level within the USA. Despite this, infectious disease is still a powerful predictor of average state IQ, and the best predictor of the variables we examined.

Some limitations exist in this study in addition to those we have previously mentioned. The measure of infectious disease that we used includes disease in both adults and in children, whereas our hypothesis primarily concerns disease in children. We do not consider this to be a significant problem, however, because infectious diseases that affect both adults and children tend to have a larger detrimental effect on children than on adults (e.g. World Health Organization, 2004). Secondly, our measure of infectious disease was based on incidence, rather than physiological cost (disability-adjusted life years) as we used in our previous study (Eppig et al., 2010). While our hypothesis concerns the latter, the prevalence of an infectious disease is a reasonable proxy for its impact. The error that this introduces into our study is more likely to diminish the apparent impact of infectious disease on intelligence than it is to increase it.

As we have previously argued (Eppig et al., 2010), while our model predicts that increased developmental insult as the result of infectious disease will lead to lower intelligence, it simultaneously predicts that reducing the metabolic cost of such diseases, especially during childhood, will lead to higher intelligence. Thus, it predicts that human interventions that lead to reduced infectious disease stress experienced during human development will also lead to increased intelligence. As discussed previously in this paper, others have suggested that a population of more intelligent individuals will be able

to reduce the negative effects of infectious disease compared to a less-intelligent population (e.g. Kanazawa, 2008b; Reeve, 2009; Reeve & Basalik, 2010; Rindermann & Meisenberg, 2009; Oesterdiekhoff & Rindermann, 2007), and our model also predicts that this should be true. The same is also probably true for a population of more highly educated individuals — and, indeed, a population of more intelligent individuals is more likely to have widespread education (e.g. Lynn & Mikk, 2007). Populations with higher average IQ and education are likely to make more money (e.g. Lynn & Vanhanen, 2006), and thus be more able to afford measures that will reduce the effects of infectious disease, although a wealthier society may not always choose to do so. Our model, depicted in figure 2, therefore predicts that IQ, education and wealth are endogenous factors that may be both the cause and result of variation in the burden of infectious disease stress. Climate, however, is an exogenous factor that, as we discussed earlier in this paper, has a very large independent effect on the geographic distribution of these diseases (Dunn et al., 2010; Guernier et al., 2005) (figure 2). Our model may therefore explain why the ability of education and wealth to predict intelligence is lower than that of infectious disease, and why the independent effects of education and wealth are so greatly diminished when infectious diseases are controlled for, insofar as it predicts that the effects of education and wealth are mediated through infectious disease.

The study of the biodiversity of human intelligence is of relevance to scientists studying both physical traits and social phenomena. IQ appears to be associated with many biometric traits, including body size, brain size, risk of obesity, vital capacity (reviewed in Jensen & Sinha, 1993), longevity (e.g. Gottfredson & Deary, 2004), and many other traits (Jensen & Sinha, 1993). IQ not only correlates positively with body

symmetry (developmental stability) (e.g. Banks et al., 2010; Bates, 2007; Furlow et al., 1997; Penke et al., 2009; Prokosch et al., 2005), but the correlation increases as the quality of the IQ test increases (Prokosch et al., 2005).

IQ also shows reliable correlations with many societal traits, including wealth (e.g. Kanazawa, 2006; Lynn & Vanhanen, 2004, 2006), several measures of societal inequality (e.g. Lynn & Vanhanen, 2006), and happiness (Lynn & Vanhanen, 2006; Pesta et al., 2010). Jensen & Sinha (1993) remarked that "The correlation between IQ and SES [socioeconomic status]...is so well established and well known as to scarcely call for documentation." Gottfredson (1997) reviewed comprehensive evidence that IQ predicts job performance, especially in highly complex jobs, the complexity of occupations that one may succeed at, ability in solving problems encountered frequently in everyday life, and may also be involved in dealing effectively with the complexities of everyday social life. These patterns are consistent with the 'social brain' hypothesis for the function of human-unique cognitive ability (e.g. Alexander, 1989; Jolly, 1966; Flinn et al., 2005).

Although more research needs to be done to establish causation, this analysis has provided further evidence for the parasite-stress hypothesis for the worldwide distribution of intelligence by examining the relationship between parasite stress and intelligence on a smaller geographical scale than has been studied previously. In addition to the research programs we have previously suggested for determining causation (Eppig et al., 2010), further studies are needed to determine whether the causal relationships we propose (figure 2) are correct.

# Acknowledgements

The authors thank Rachael Falcon and Steve Gangestad for advice on statistical analyses. Two anonymous reviewers provided useful criticism that improved the manuscript.

# References

- Alexander, R. D. (1989). Evolution of the human psyche. In P. Mellars, & C. Stringer (Eds.), *The Human Revolution* (pp. 455-513). Edinburgh: University of Edinburgh Press.
- Banks, G. C., Batchelor, J. H., & McDaniel, M. A. (2010). Smarter people are (a bit) more symmetrical: a meta-analysis of the relationship between intelligence and fluctuating asymmetry. *Intelligence*, *38*, 393-401.
- Barber, N. (2005). Educational and ecological correlates of IQ: a cross-national investigation. *Intelligence*, *33*, 273-284.
- Bates, T. C. (2007). Fluctuating asymmetry and intelligence. *Intelligence*, 35, 41-46.
- Calvete, C., Blanco-Aguiar, J. A., Virgós, E., Cabezas-Díaz, S., & Villafuerte, R. (2004). Spacial variation in helminth community structure in the red-legged partridge (*Alectoris rufa* L.): effects of definitive host density. *Parasitology*, 129, 101-113.
- Cashdan, E. (2001). Ethnic diversity and its environmental determinants: Effects of climate, pathogens, and habitat diversity. *American Anthropologist*, 103, 968-991.
- Center for Disease Control. (2008). *MMWR Summary of notifiable diseases*. Retrieved July, 2010, from <a href="http://www.cdc.gov/mmwr/mmwr\_nd/">http://www.cdc.gov/mmwr/mmwr\_nd/</a>.
- Dunn, R. R., Davies, T. J., Harris, N. C., & Gavin, M. C. (2010). Global drivers of human pathogen richness and prevalence. *Proceedings of the Royal Society, Series B*, 277, 2587-2595.

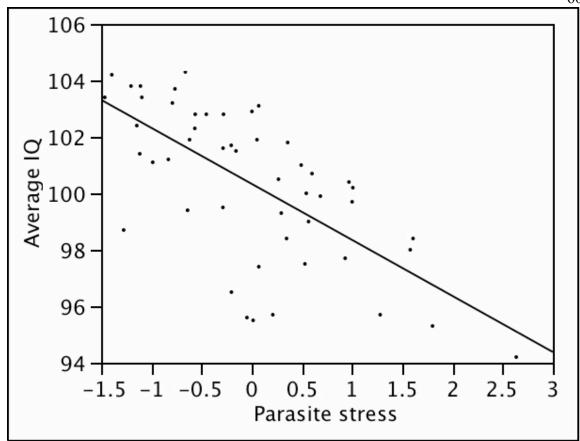
- Eppig, C., Fincher, C. L., & Thornhill, R. (2010). Parasite prevalence and the worldwide distribution of cognitive ability. *Proceedings of the Royal Society, Series B*, 277, 3801-3808.
- Fincher, C. L. & Thornhill, R. (in press). Parasite-stress promotes in-group assortative sociality: the case of strong family ties and heightened religiosity. *Behavioral and Brain Sciences*.
- Flinn, M. V., Geary, D. C., & Ward, C. V. (2005). Ecological dominance, social competition, and coalitionary arms races: why humans evolved extraordinary intelligence. *Evolution and Human Behavior*, *26*, 10-46.
- Fox, J. (1991). Regression diagnostics. Beverly Hills, CA: Sage.
- Furlow, F. B., Armijo-Prewitt, T., Gangestad, S. W., & Thornhill, R. (1997). Fluctuating asymmetry and psychometric intelligence. *Proceedings of the Royal Society,*Series B, 264, 823-829.
- Gottfredson, L. S. (1994). Why *g* matters: the complexity of everyday life. *Intelligence*, *24*, 79-132.
- Gottfredson, L. S. & Deary, I. J. (2004). Intelligence predicts health and longevity, but why? *Current Directions in Psychological Science*, 13, 1-4.
- Guernier, V., Hochberg, M. E. & Guégan, J. (2004). Ecology drives the worldwide distribution of human diseases. *PLOS Biology*, *2*, 740-746.
- Holliday, M. A. (1986). Body composition and energy needs during growth. In F. Falkner, & J. M. Tanner (Eds.), *Human growth: a comprehensive treatise*, vol. 2, (pp. 101-117). New York: Plenum.

- Jensen, A. R. & Sinha, S. N. (1993). Physical correlates of human intelligence. In P. A. Vernon (Ed.), *Biological Approaches to the Study of Human Intelligence*, (pp. 139-242). Norwood: Ablex.
- Jolly, A. (1966). Social behavior and primate intelligence. *Science*, 153, 501-506.
- Kanazawa, S. (2006). IQ and the wealth of states. *Intelligence*, 34, 593-600.
- Kanazawa, S. (2008a). Temperature and evolutionary novelty as forces behind the evolution of general intelligence. *Intelligence*, *36*, 99-108.
- Kanazawa, S. (2008b). IQ and the health of states. *Biodemography and Social Biology*, 54, 200-213.
- Low, B. S. (1990). Marriage systems and pathogen stress in human societies. *American Zool*ogist, *30*, 325-339.
- Lynn, R., & Mikk, J. (2007). National differences in intelligence and educational attainment. *Intelligence*, *35*, 115-121.
- McDaniel, M. A. (2006a). State preferences for the ACT versus SAT complicates inferences about SAT-derived state IQ estimates: A comment on Kanazawa (2006). *Intelligence*, *34*, 601-606.
- McDaniel, M. A. (2006b). Estimating state IQ: Measurement challenges and preliminary correlates. *Intelligence*, *34*, 607-619.
- Møller, A. P. (1998). Evidence of larger impact of parasites on hosts in the tropics: investment in immune function within and outside the tropics. *OIKOS*, *82*, 265-270.
- National Center for Education Statistics (2009). *State profiles home page*. Retrieved January 3, 2011, from http://nces.ed.gov/programs/stateprofiles/.

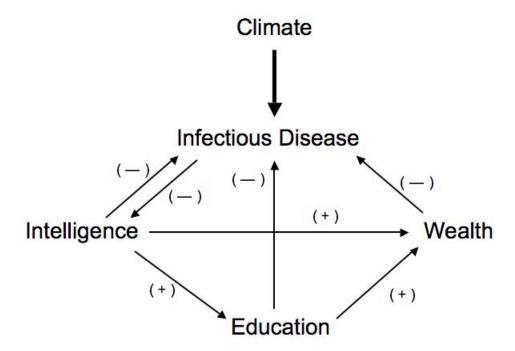
- Nunn, C. L., Altizer, S. M., Sechrest, W., & Cunningham, A. A. (2005). Latitudinal gradients of parasite species richness in primates. *Diversity and Distributions*, 11, 249-256.
- Oesterdiekhoff, G. W., & Rindermann, H. (2007). The spread of AIDS in developing countries: a psycho-cultural approach. *Journal of Social, Political and Economic Studies*, 32, 201-222.
- Penke, L., Bates, T. C., Gow, A. J., Pattie, A., Starr, J. M., Jones, B. C., Perrett, D. I., & Deary, I. J. (2009). Symmetric faces are a sign of successful cognitive aging. *Evolution and Human Behavior*, *30*, 429-437.
- Pesta, B. J., McDaniel, M. A., & Bertsch, S. (2010). Toward and index of well-being for the fifty U.S. states. *Intelligence*, *38*, 160-168.
- Prokosch, M. D., Yeo, R. A., & Miller, G. F. (2005). Intelligence tests with higher *g*-loadings show higher correlations with body symmetry: evidence for a general fitness factor mediated by developmental stability. *Intelligence*, *33*, 203-213.
- Reeve, C. L. 2009. Expanding the *g*-nexus: further evidence regarding the relations among national IQ, religiosity and national health outcomes. *Intelligence*, *37*, 495-505.
- Reeve, C. L., & Basalik, D. (2010). Average state IQ, state wealth and racial composition as predictors of state health statistics: partial support for 'g' as a fundamental cause of health disparities. *Intelligence*, *38*, 282-289.
- Rindermann, H., & Meisenberg, G. (2009). Relevance of education and intelligence at the national level for health: the case of HIV and AIDS. *Intelligence*, *37*, 383-395.

- Ryan, J. J., Bartels, J. M. & Townsend, J. M. (2010). Associations between climate and IQ in the United States of America. *Psychological Reports*, *107*, 251-254.
- Saadat, M. 2008. Consanguinity and national IQ scores. *Journal of Epidemiology and Community Health*, 62, 566-567.
- Templer, D. I., & Arikawa, H. (2006). Temperature, skin color, per capita income, and IQ: an international perspective. *Intelligence*, *34*, 121-139.
- U.S. Census Beareau. (2000). *State and country quickfacts*. Retrieved January 3, 2011, from http://quickfacts.census.gov/qfd/index.html.
- U.S. Department of Education. (2009). *State contacts and information*. Retrieved January 3, 2011, from http://www2.ed.gov/about/contacts/state/index.html.
- World Health Organization. (2004). *Global burden of disease: 2004 update*. Geneva: World Health Organization.
- Woodley, M. A. (2009). Inbreeding depression and IQ in a study of 72 countries. *Intelligence*, 37, 268-276.





(Figure 1: Bivariate relationship between average U.S. state IQ and infectious disease stress. Average state IQ and parasite stress correlated at r = -0.67 (n = 50, p < 0.0001). The line is the least-squares line through the points.)



(Figure 2: The directions of influences predicted by our hypothesis among climate, infectious disease, intelligence, education, and wealth.)

Table 1
Zero-order correlations among all variables.

1. average IQ	1.	2. -0.67**	3. -0.31*	4. 0.42*	5. 0.27†	6. 0.34*	7. 0.28*	8. 0.32*
		0.07			'			
2. parasite stress			-0.0069	-0.11	-0.15	-0.047	0.013	-0.065
3. student-teacher ratio				-0.35*	0.12	-0.0007	0.020	0.052
4. percent teachers highly qualified	S				-0.23	-0.07	0.029	-0.049
5. median househousehousehousehousehousehousehouse	old					0.88**	0.77**	0.95**
6. income per capita							0.80**	0.95**
7. gross state product								0.91**

# 8. wealth

†p < 0.1. \*p < 0.05. \*\*p < 0.001. All others p > 0.10. All n = 50.

Table 2 Hierarchical regression model predicting average state IQ.

Model	Term	p	$R^2$	change in R <sup>2</sup>
1		< 0.0001	0.445	0.445
	parasites	< 0.0001		
2		< 0.0001	0.520	0.075
	parasites	< 0.0001		
	wealth	0.0094		
3		< 0.0001	0.698	0.133
	parasites	< 0.0001		
	wealth	0.0006		
	HQT	0.0019		
	STR	0.015		

HQT = percent teachers highly qualified; STR = student/teacher ratio.

Table 3 Final multiple regression model predicting average state IQ.

Term	Estimate	Std Error	Std Beta	VIF	p
Intercept	88.99	5.14	-	-	< 0.0001
Parasite Stress	-1.84	0.250	-0.62	1.02	< 0.0001
Wealth	0.518	0.141	0.30	1.00	0.0006
HQT	0.155	0.0470	0.29	1.16	0.0019
STR	-0.229	0.091	-0.22	1.16	0.015

Whole model:  $R^2 = 0.70$ , n = 50, p < 0.0001. HQT = percent teachers highly qualified; STR = student/teacher ratio; VIF = variance inflation factor.

Chapter 4: Infectious disease prevalence predicts IQ across world nations and states of the USA.

Christopher G. Eppig

This chapter is formatted for submission by invitation to the journal World Economics.

Infectious disease prevalence predicts IQ across world nations and states of the USA

# Christopher G. Eppig

Biology Department, University of New Mexico, Albuquerque, New Mexico 87131

Information for Corresponding Author

UNM Biology Department MSC03 2020

1 University of New Mexico

Albuquerque, NM 87131

(505)277-4999

e-mail: ceppig@unm.edu

#### **Abstract**

In two studies, we predicted that intelligence emerges partly from an energetic tradeoff maximizing brain development vs. immune system function. Levels of infectious disease predict average IQ across world nations (r = -0.82) and across USA states (r = -0.67). This relationship is robust when education, wealth and other factors are controlled. On both levels of analysis, infectious disease stress was the best predictor of average IQ.

Keywords: infectious disease, ecology, cognitive ability, intelligence.

### Introduction

The topic of the worldwide distribution in cognitive ability has been of interest to scientists for quite some time, but it was not until Lynn & Vanhanen (2002, 2006) published data on average national IQ that empirical studies on the subject were possible. Since the publishing of these data, five hypotheses have been tested to explain the global variation in IQ. Barber (2005) hypothesized that intelligence is caused by education and employment prospects. He argued that the more education one receives, the higher his or her intelligence will be, and that intelligence will increase if one has access to jobs that require high intelligence to succeed. This hypothesis was supported by showing that average national IQ correlates positively with enrollment in secondary school (r = 0.72), ad negatively with illiteracy (r = -0.71) and frequency of employment in agricultural labor (r = -0.70).

Templer & Arikawa (2006) hypothesized that cold climates lead to evolutionary forces that favor higher IQ. Cold weather, they argue, is more difficult to survive in than warm weather, thus natural selection will favor individuals with higher IQ in cold areas. They supported this hypothesis by showing that average national IQ correlates with four measures of temperature: average winter low temperature (r = -0.66), average winter high temperature (r = -0.76), average summer low temperature (r = -0.41), and average summer high temperature (r = -0.31).

Kanazawa (2008) proposed that unfamiliar environments lead to evolutionary forces favoring higher IQ. Humans, he noted, evolved in sub-Saharan Africa. As humans radiated farther from this area of evolutionary origin, the environment became more and more different in terms of edible plants and animals, and climate. Thus it would become more difficult to survive in these increasingly unfamiliar environments as humans moved away from our geographical origin. He supported this hypothesis by showing that average national IQ correlates with linear distance from three locations in sub-Saharan Africa:  $0^{\circ}$ E,  $0^{\circ}$ N (r = 0.45), South Africa (r = 0.53) Ethiopia (r = 0.22).

Saadat (2008) and Woodley (2009) both argued that inbreeding reduces intelligence. Marrying one's close relative (consanguineous marriage), such as a cousin, is very common in some parts of the world, whereas it is very uncommon in other parts. While there may be good reasons for this behavior, it may have other negative consequences, such as reducing the quality of the brain. Rates of consanguineous marriage were found to correlate with average national IQ at r = -0.77 and r = -0.62 by Saadat (2008) and Woodley (2009), respectively. Woodley (2009) found, however, that

the correlation between consanguineous marriage and average national IQ was no longer significant when the effects of national wealth were removed.

The parasite-stress hypothesis was proposed by Eppig et al. (2010, 2011). In two publications, we argued that parasites are the primary cause of the worldwide distribution of human IQ. We used a broad, ecological definition of the word 'parasite' to mean any infectious agent that causes disease in humans, including viruses, bacteria, worms, amoebas, paramecium, fungi, etc. The human brain, which is the source of human intelligence, is among the most energetically costly organs in the human body. In a newborn human, the brain may require as much as 87% of the body's resting metabolic budget — that is, the number of calories required by a body at rest. This drops down to around 25% as adults (Holliday, 1986). Fundamentally, our argument rests on the assumption that a developing individual and their parasites will be in conflict over available energy.

Parasites may draw energy from the host's body in four ways: (1) viruses use the cellular machinery of the host to reproduce. These cellular resources would otherwise be used for growth and maintenance of the person. (2) Some parasites consume various tissues of the host's body. Liver flukes, for example, survive by attaching to and eating a host's liver. The liver is a very important organ, so the host must replace the tissue lost to the parasite. The energy required to rebuild the damaged liver, or cope with reduced liver function, may otherwise have been used for building or maintaining other body tissues.

(3) Some parasites absorb nutrients out of the host's intestines or cause diarrhea. Tape worms, for example, live in a host's intestines and absorb directly the already-digested nutrients therein. Some portion of the nutrients that the host has consumed recently will

thus go to feed the parasite instead of the host. Organisms that cause severe diarrhea can prevent the host from accessing any of the nutrients he or she has recently eaten. (4) The immune system will be activated in response to any parasitic infection. The production of immune cells and proteins, such as white blood cells and t-cells, requires energy. The cost may be small for any given unit of time, but when an individual's immune system is elevated for a period of months or years as the result of a chronic infection, the cost could be quite high. One or more of these factors could result in a large drain on the energy that would otherwise go to build energetically expensive tissues, such as the brain.

Presumably, if a developing human body does not have the available energy resources to

Presumably, if a developing human body does not have the available energy resources to devote to the brain, the quality of the brain will be reduced.

Some scientists have argued for the opposite direction of causation — that is, people with higher intelligence are better at fighting off infectious disease, such that areas of the world with higher average intelligence have lower levels of parasites as a result (e.g. Oesterdiekhoff & Rindermann, 2007; Reeve, 2009; Reeve & Basalik, 2010; Rindermann & Meisenberg, 2009). While we do not doubt that people with higher intelligence may be more able to avoid parasitic infection the distribution of infectious disease across the world appears to be largely independent of humans. First, climatic factors such as temperature and rainfall appear to be the major forces driving the worldwide distribution of infectious disease, because they create favorable conditions for parasites to live and infect humans in (e.g. Dunn et al., 2010; Guernier et al., 2004). Second, the distribution of infectious disease in non-human animals follows a similar pattern of distribution to infectious disease in humans (e.g. Calvete et al., 2004; Møller,

1998; Nunn et al., 2005). If the distribution of human diseases were based primarily on anthropic factors, one would not expect to see a similar distribution for wild animals.

# **Analyses**

In two published studies, we tested our hypothesis on two geographic scales: across nations of the world (Eppig et al., 2010) and across states of the USA (Eppig et al., 2011). To test our hypothesis on the cross-national level, we compiled existing data on average national IQ, parasite stress, education (literacy, percent enrolled in secondary school, and average years of education attained), and GDP per capita from a variety of sources. Average national IQ was compiled by Lynn & Vanhanen (2002, 2006). They compiled these data from many studies that had been carried out, including only those that had used culturally appropriate IQ tests and representative populations. In addition to using collected data, Lynn & Vanhanen (2002, 2006) estimated the average IQ of several nations for which there was not any direct data by averaging the IQs of nearby nations with known IQs. Both the collected data only (LVCD) and the combined collected data and estimates (LVE) were used in analyses. A third measure of IQ (WEAM) used was a modification of Lynn & Vanhanen's data by Wicherts et al. (2010), who claimed to have more accurate data for several African nations.

Data on parasite stress was taken from the World Health Organization (WHO, 2004). This variable, known as the 'disability-adjusted life years' (DALY), measures both life shortened by disease and time spent disabled by it, such that 1 DALY is equal to one healthy year of life lost due to parasites per 100,000 individuals in a nation. Data for

28 important human infectious diseases comprise this variable, including tuberculosis, tetanus, malaria, diarrheal diseases, and intestinal worms.

The zero-order correlation between average national IQ and parasite stress was -0.82 (p < 0.0001, n = 107) using LVCD, -0.82 (p < 0.0001, n = 184) using LVE, and -0.76 using (WEAM). Some have argued that, due to large cultural differences that may be influencing IQ scores, it is not appropriate to compare the IQ of people in distant parts of the world. While we see no reason why this should be true, we repeated this analysis within each of Murdock's (1949) world areas. These groupings correspond roughly to the continents, but are based primarily on cultural similarity. A significant negative correlation was found between average IQ and parasite stress in five out of six world areas: Africa (LVE: r = -0.80, n = 53, p < 0.0001; LVCD: r = -0.80, n = 22, p < 0.0001; WEAM: r = -0.49, n = 53, p = 0.0002), Eastern Eurasia (LVE: r = -0.62, n = 20, p = 0.0002) 0.0033; LVCD: r = -0.70, n = 11, p = 0.016), Insular Pacific (LVE: r = -0.85, n = 17, p < 0.0030.0001; LVCD: r = -0.83, n = 12, p = 0.0009), North America (LVE: r = -0.65, n = 12, p = 0.022; LVCD: r = -0.76, n = 7, p = 0.049), and Western Eurasia (LVE: r = -0.65, n = 0.022; LVCD: r = -0.65, n = 0.049). 59, p < 0.0001; LVCD: r = -0.73, n = 39, p < 0.0001). This relationship was not significant in South America (LVE: r = 0.077, n = 23, p = 0.73; LVCD: r = 0.043, n = 16, p = 0.88). Using a hierarchical linear model, we found that this relationship is robust within these world areas on average (LVE:  $R^2 = 0.78$ , p < 0.0001, n = 184; LVCD:  $R^2 =$ 0.77, n = 107, p < 0.0001; WEAM:  $R^2 = 0.68$ , p < 0.0001, n = 184).

Seven conspicuous outliers exist in the worldwide relationship: St Lucia,
Dominica, St Kitts and Nevis, Antigua and Barbuda, Grenada, St Vincent and the
Grenadines, and Jamaica. All of these nations have average IQ scores that are lower than

our model would predict, given their parasite scores. Four of these nations, St Lucia, Dominica, Grenada, and St Vincent and the Grenadines, were included in the South America world area due to cultural similarity. Because these nations are all in the same geographic region, we suspect that there may be local parasites that are affecting the populations that were not included in the DALY measure. When these four nations are removed, the correlation between average IQ and parasite stress in continental South America rises to -0.48 (LVE: p = 0.036, n = 19). These four nations were included in all other analyses.

Multiple regression analyses were used to determine the relative importance of infectious disease, education, wealth, temperature, and distance from sub-Saharan Africa in predicting average national IQ. In order to maintain a large enough sample size for these regressions, only LVE and WEAM IQ data were used. Using LVE IQ data, the final regression model included infectious disease, distance from sub-Saharan Africa, average winter high temperature, and average years of education. GDP per capita was removed in an earlier model selection procedure. In this model, infectious disease (p < 0.0001, Std Beta = -0.62), distance from sub-Saharan Africa (p < 0.0001, Std Beta = 0.23), and average winter high temperature (p = 0.0007, Std Beta = -0.22) were significant, and average years of education was not (p = 0.97, Std Beta = 0.0029). The whole model  $R^2$  was 0.889 (p < 0.0001, n = 83). Very similar results were found using WEAM IQ data ( $R^2 = 0.796$ , p < 0.0001).

In our cross-state analysis (Eppig et al., 2011), we compiled existing data on average state IQ, parasite stress, education (percent teachers highly qualified and student-teacher ratio) and wealth (gross state product, income per capita and median household

income). Average state IQ was taken from McDaniel (2006), who calculated scores based on state-wide data from the National Assessment of Educational Progress (NAEP) test. Although this is ostensibly a measure of education outcomes, it is also an accurate IQ test (McDaniel, 2006). Fincher and Thornhill (in press) calculated parasite-stress scores for each state using data on parasite incidence from the National Center for Disease Control. This measure includes cholera, measles, meningitis, pertussis, tetanus, tuberculosis, and many other infectious diseases that occur in the USA.

Across all 50 USA states, the correlation between parasite-stress and average IQ was -0.67 (p < 0.0001, n = 50). We used hierarchical regression to determine the relative effects of parasites, education and wealth. Because our three measures of wealth — income per capita, median household income, and gross state product — correlated highly among each other, we used principle component analysis to reduce them to a single variable: 'wealth.' Parasites were added in the first step of the model, yielding an  $R^2$  of 0.445 (p < 0.0001). Wealth was added in the second step, yielding a total  $R^2$  of 0.520 (p < 0.0001). The two education variables — percent of teachers highly qualified and student-teacher ratio — were added in the final step, yielding a total  $r^2$  of 0.698 (p < 0.0001). In the final model, parasite stress (Std Beta = -0.62, p < 0.0001), wealth (Std Beta = 0.30, p = 0.0006), percent teachers highly qualified (Std Beta = 0.29, p = 0.0019), and student-teacher ratio (Std Beta = -0.22, p = 0.015) were all significant.

#### **Discussion**

Across nations of the world and states of the USA, parasite stress was a significant predictor of average IQ. Parasite stress was not only a significant predictor when other factors were controlled, but it was the most important predictor in all analyses. To say that it is the best predictor, however, is not to say that other factors are not involved. While our hypothesis predicts that infectious disease reduces intelligence, it simultaneously predicts that any human interventions that reduce the stress of infectious disease will indirectly increase IQ.

Wealth may affect parasite stress in an area. A nation with more wealth will be more able to afford medical infrastructure, such as hospitals and sanitation, as well as medicine, both to prevent and to treat infectious disease. Wealth is not the same thing as willingness, however. If a nation is wealthy but the people who control the wealth are not interested in spending it on public health, then the money obviously will not contribute to the average IQ in the area.

Education may influence parasite stress directly. If a nation's people are more educated about the cause, transmission, prevention, and treatment of parasites, people may reduce the extent to which parasites negatively affect them. With more education, there may be more and better doctors and healthcare administrators, and politicians who are able to make better, more informed decisions about public health. Education may also influence parasite stress indirectly by influencing wealth. More education allows more people to enter into white-collar professions, which may increase both personal wealth and national wealth.

Intelligence may affect the incidence of parasites directly, as has been suggested by others (e.g. Oesterdiekhoff & Rindermann, 2007; Reeve, 2009; Reeve & Basalik,

2010; Rindermann & Meisenberg, 2009). Intelligence confers an ability to deal with cognitively complex problems (Gottfredson, 1997), and coping with parasites in the environment may be cognitively complex. People with higher IQ may be more successful at manipulating the world around them in a way that reduces the threat of parasites, such as through sanitation, personal hygiene and in the choice of where in the environment to live. People with higher IQ may also be better at identifying what parasites they are infected with and how and when to seek treatment. A nation with more intelligent people will be more able to solve local health problems and create more and better medicine.

There is evidence on both the individual level and the cross-national level (Lynn & Vanhanen, 2002, 2006) that higher intelligence leads to higher wealth. Making money can be cognitively demanding, especially in occupations that typically receive high salaries such as doctors, lawyers and bankers. Even in simpler professions, people with higher IQ tend to be more successful (Gottfredson, 1997). Thus higher IQ people will tend to make more money, and nations with higher average IQ will have higher national wealth.

People with higher intelligence benefit more from education (e.g. Gottfredson, 1997). Education is also cognitively demanding, so people with higher IQ will be more successful at it and likely seek more of it. Intelligence can therefore affect infectious disease by affecting education and wealth.

Climate appears to be the most important factor in determining the worldwide distribution of infectious disease. As discussed previously, climatic factors such as temperature and humidity create optimal environments for parasites to survive and infect new hosts (Guernier et al., 2004). It is not possible or wise to strategically alter these

factors in order to reduce the habitability of an environment for parasites. That is, although humans are certainly capable of altering the world climate, it is not possible to engineer local climates in a way that could reduce the burden of local parasites. Even if this were possible, it would be unwise to do so because of the catastrophic effects this would almost certainly have on the world. Thus, nations in locations that are very suitable for parasites would require more money and education to reduce the stress of parasites than would nations in areas that are less suitable for parasites. There are likely to be other variables and directions of influence that are affecting infectious disease, but this model serves to illustrate the primary driving forces as predicted by our hypothesis.

# Acknowledgements

The author thanks Corey Fincher and Randy Thornhill for their comments on this manuscript, and for their involvement in the research described herein.

# References

- Barber, N. (2005). Educational and ecological correlates of IQ; a cross-national investigation. *Intelligence*, 33, 273-284.
- Calvete, C., Blanco-Aguiar, J. A., Virgós, E., Cabezas-Díaz, S., & Cillafuerte, R. (2004).

  Spacial variation in helminth community structure in the red-legged partridge

  (*Alectoris rufa L.*): effects of definitive host density. *Parasitology*, 129, 101-113.
- Dunn, R. R., Davies, T. J., Harris, N. C., & Gavin, M. C. (2010). Global drivers of human pathogen richness and prevalence. *Proceedings of the Royal Society, Series B*, 277, 2587-2595.
- Eppig, C., Fincher, C. L. & Thornhill, R. (2010). Parasite prevalence and the worldwide distribution of cognitive ability. *Proceedings of the Royal Society, Series B*, 277, 3801-3808.
- Eppig, C., Fincher, C. L. & Thornhill, R. (2011). Parasite prevalence and the distribution of intelligence among the states of the USA. *Intelligence*, 39, 155-160.
- Fincher, C. L. & Thornhill, R. (in press). Parasite-stress promotes in-group assortative sociality: the case of strong family ties and heightened religiosity. *Behavioral and Brain Sciences*
- Gottfredson, L. S. (1997). Why *g* matters: the complexity of everyday life. *Intelligence*, 24, 79-132.
- Guernier, V., Hochbert, M. E. & Guégan, J. (2004). Ecology drives the worldwide distribution of human diseases. *PLoS Biology*, 2, 740-746.

- Holliday, M. A. (1986). Body composition and energy needs during growth. In F.Falkner, & J. M. Tanner (Eds.), *Human growth: a comprehensive treatise*,*Volume 2.* (pp. 101-117) New York: Plenum.
- Kanazawa, S. (2008). Temperature and evolutionary novelty as forces behind the evolution of general intelligence. *Intelligence*, 36, 99-108.
- Lubinski, D., Benbow, C. P., Webb, R. M., & Bleske-Rechek, A. (2006). Tracking exceptional human capital over two decades. *Psychological Science*, 17, 194-199.
- Lynn, R. & Vanhanen, T. (2002). IQ and the wealth of nations. Westport: Praeger.
- Lynn, R. & Vanhanen, T (2006). IQ and global inequality. Augusta: Washington Summit
- McDaniel, M. A. (2006). Estimating state IQ: measurement challenges and preliminary correlates. *Intelligence*, 34, 607-619.
- Møller, A. P. (1998). Evidence of larger impact of parasites on hosts in the tropics: investment in immune function within and outside the tropics. *Oikos*, 82, 265-270.
- Murdock, G. P. (1949). Social Structure. New York: Macmillan.
- Nunn, C. L., Altizer, S. M., Sechrest, W., & Cunninghamj, A. A. (2005). Latitudinal gradients of parasite species richness in primates. *Diversity and Distributions*, 11, 249-256.
- Oesterdiekhoff, G. W. & Rindermann, H. (2007). The spread of AIDS in developing countries: a psycho-cultural approach. *Journal of Social, Political and Economic Studies*, 32, 201-222.

- Reeve, C. L. (2009). Expanding the *g*-nexus: further evidence regarding the relations among national IQ, religiosity and national health outcomes. *Intelligence*, 37, 495-505.
- Reeve, C. L. & Basalik, D. (2010). Average state IQ, state wealth and racial composition as predictors of state health statistics: partial support for 'g' as a fundamental cause of health disparities. *Intelligence*, 38, 282-289.
- Rindermann, H. & Meisenberg, G. (2009). Relevance of education and intelligence at the national levels for health: the case of HIV and AIDS. *Intelligence*, 37, 383-395.
- Templer, D. I. & Arikawa, H. (2006). Temperature, skin color, per capita income, and IQ: an international perspective. *Intelligence*, 34, 121-139.
- Saadat, M. (2008). Consanguinity and national IQ scores. *Journal of Epidemiology and Community Health*, 62, 566-567.
- Wicherts, J. M., Dolan, C. V. & van der Maas, H. L. J. (2010). A systematic literature review of the average IQ of sub-Saharan Africans. *Intelligence*, 38, 1-20.
- Woodley, M. A. (2009). Inbreeding depression and IQ in a study of 72 countries. *Intelligence*, 37, 268-276.

Chapter 5: Infectious disease and the relationship of the hygiene hypothesis to IQ.

Christopher G. Eppig, Corey L. Fincher and Randy Thornhill

This chapter is formatted for submission to the journal Intelligence.

Infectious disease and the relationship of the hygiene hypothesis to IQ

Christopher Eppig<sup>a</sup>, Corey L. Fincher<sup>a</sup>, Randy Thornhill<sup>a</sup>

<sup>a</sup>Biology Department, MSC03 2020, 1 University of New Mexico, Albuquerque, NM 87131, USA

Contact Information for Corresponding Author:

UNM Biology Department MSC03 2020

1 University of New Mexico

Albuquerque, NM 87131

(505)277-4999

e-mail: <a href="mailto:ceppig@unm.edu">ceppig@unm.edu</a>

#### Abstract

In this study, we investigate the previously documented positive relationship between asthma and IQ. Given that infectious disease has been implicated as a cause of variation in both IQ and asthma rates, we propose that the link between asthma and IQ is not directly causal, but the result of both being affected by the common cause of infectious disease. We attempt to reconcile the effects of both infectious disease and vitamin D deficiency on asthma. Across the 50 USA states, we found that both ambient light intensity (which contributes to vitamin D production) and infectious disease contribute to regional variation in rates of asthma (r = -0.28 and r = -0.35, respectively). Average IQ across the states and rates of asthma correlated positively (r = 0.25), but this relationship dropped close to zero when the effects of infectious disease were removed (r = 0.016).

Keywords: parasite-stress hypothesis, hygiene hypothesis, vitamin D, development, biodiversity.

#### Introduction

Previous studies have found a positive relationship between cognitive ability and rates of asthma on the individual level (e.g. Sacher et al., 1994; Benbow, 1986; reviewed in Jensen and Sinha, 1993). In our 2010 study (Eppig et al., 2010), we proposed the hypothesis that infectious disease may be the reason that IQ and asthma are related,

because infectious disease appears to influence both IQ and risk of asthma. It is therefore possible for IQ and asthma rates to correlate without any direct causal relationship.

Eppig et al. (2010, 2011) found evidence that infectious disease is the primary cause of the global diversity of human intelligence, across world nations (2010) and USA states (2011). Infectious disease correlated with average IQ at r = -0.79 to r = -0.82 across nations (Eppig et al., 2010) and r = -0.67 across states (Eppig et al., 2011). These relationships remained robust when wealth, education, and other variables thought to influence the distribution of human intelligence were controlled. Our hypothesis — the parasite-stress hypothesis — states that parasitic infections result in the loss of large amounts of energy that would otherwise have gone to building a metabolically expensive brain. (We use a broad, ecological definition of "parasite" to mean any infectious organism) Hassall and Sherratt (2011) repeated our cross-national analysis of IQ variation, controlling for spatial autocorrelation, and found further support for infectious disease as the primary variable of importance.

Three hypotheses exist to explain variation in the prevalence of asthma. The Geschwind-Behan hypothesis (1982) argues that fetal testosterone simultaneously affects many factors including intelligence and risk of developing asthma. Benbow (1986) invoked this hypothesis to explain the positive link between IQ and asthma. This hypothesis has led to many testable predictions, but it has been scientifically controversial, however, as many subsequent tests have failed to support its various predictions (e.g. McKeever and Rich, 1990; Segalowitz et al., 1994).

A second hypothesis for the prevalence of asthma, as well as certain other autoimmune disorders, is the hygiene hypothesis. First proposed by Stachan (1989), the

hygiene hypothesis states that low exposure to infectious organisms during early development produces a hyperactive immune system that manifests as asthma, allergies, or other, chronic autoimmune disorders. Although the mechanisms involved are unclear, there is mounting evidence that this hypothesis is correct (e.g. Ege et al., 2011; Peters et al., 2006; Vogel et al., 2008; Gangal and Chowgule, 2009).

A third hypothesis for the variation in prevalence of asthma rates involves vitamin D deficiency. Although there is good evidence of the relationship between vitamin D deficiency and increased risk of asthma (e.g. Camargo et al., 2007; Devereux et al., 2007; Litonjua and Weiss, 2007), the reason for this relationship is not understood. Krstic (2011) tested the vitamin D hypothesis on the regional level. It was found that rates of asthma in metropolitan areas of Australia and the United States were predicted positively by latitude (USA:  $r^2 = 0.22$ , p < 0.001, n = 97; Australia:  $r^2 = 0.73$ , p < 0.01, n = 8), and negatively by temperature (USA:  $r^2 = 0.17$ , p < 0.001, n = 97) and insolation (solar radiation) (USA:  $r^2 = 0.15$ , p < 0.001, n = 97), but not predicted by pollution (USA:  $r^2 =$ 0.002, p = 0.66, n = 97). Latitude was found to be the best predictor of asthma rates in this study, but this is not necessarily consistent with the broader predictions of the vitamin D-deficiency hypothesis. Latitude is highly predictive (negative) of insolation, of course, but it is not a direct measure. Unless some other correlate of latitude besides sun intensity contributes to vitamin D production, this hypothesis should tacitly predict that light intensity should predict rates of asthma better than latitude, assuming accurate measurements of all three variables. A variable that latitude does predict, and strongly so, is infectious disease (e.g. Guernier et al, 2004), which is known to contribute to variation in rates of asthma in accord with the hygiene hypothesis (e.g. Strachan, 1989; Ege et al.,

2011; Gangal and Chowgule, 2009). It is likely, then, that Krstic (2011) found latitude to be such a good predictor of asthma rates because latitude encompassed variation in both light intensity and infectious disease.

Our primary objective in this study is to determine whether the relationship between IQ and asthma is driven by infectious disease. This necessitates first testing the hygiene hypothesis across states, as our prediction is contingent on infectious disease being an important cause of asthma. Our secondary objective is to determine the relative impact of sun intensity and infectious disease on rates of asthma across states of the USA. Third, we will determine whether infectious disease is a mediator of the relationship between latitude and rates of asthma.

We have four predictions: (1) parasite stress and rates of asthma will correlate negatively across USA states, (2) IQ and asthma will correlate positively across USA states, and (3) the correlation between asthma and IQ will be reduced when the effects of infectious disease are removed. Finally, we will use the methods described by Baron and Kenny (1986) to determine the mediating effect of infectious disease on the relationship of asthma and latitude. To this end, we predict the following will be true: (1) latitude will correlate negatively and significantly with infectious disease; (2) infectious disease will correlate negatively and significantly with rates of asthma; (3) infectious disease will be a better predictor of asthma rates than will latitude; and (4) when the effects of infectious disease are removed, the ability of latitude to predict rates of asthma will be reduced. Since the vitamin D hypothesis is theoretically compatible with the hygiene hypothesis, we do not necessarily predict that the correlation between latitude and rates of asthma to

disappear entirely when infectious disease is controlled. These hypotheses are compatible because neither precludes the other from influencing rates of asthma independently.

### Methods

Average USA state IQ was taken from McDaniel (2006a). These data were based on average scores on the national assessment of educational progress (NAEP) test. We have previously explained the reason for the use of this IQ data set as opposed to others (Eppig et al., 2011). In short, this measure is the most representative of the general population, and is not subject to the same systematic biases as other IQ measures (see also McDaniel, 2006b).

Parasite-stress data were taken from Fincher and Thornhill (in press). These data are based on mortality and morbidity statistics for notifiable infectious diseases from the USA National Centers for Disease Control and Prevention (CDC). The list of notifiable diseases changes from year to year, but may include tuberculosis, tetanus, pertussis, cholera, meningitis, measles, and many others. It does not include diseases that are not primarily caused by infectious organisms, such as diabetes, heart disease and cancer.

Rates of asthma were taken from the USA National Centers for Disease Control and Prevention (CDC, 2009), and are measured as the percent of adults in a state who currently have asthma. This is the same measure used by Krstic (2011).

Latitude was taken from Rand McNally & Company (1995), and represents the geographic center of each state.

Data on average yearly insolation was collected by the National Renewable Energy Laboratory (NREL, 1994) at 239 stations across the United States from 1961-1990. Data given were annual insolation averaged across these years at each site. To estimate average yearly insolation within a USA state, values were averaged from all of the data collected at stations within a state, ranging from one to 17 stations. Light intensity was measured as kWh/m²/day on flat, horizontal (0° tilt) collectors.

We did not test any variables predicted by the Geschwind-Behan hypothesis because the necessary data, such as testosterone levels or related traits, was not available on the regional level. However, this hypothesis is theoretically compatible with both the vitamin D hypothesis and the hygiene hypothesis for predicting the prevalence of asthma, and compatible with the parasite-stress hypothesis for predicting the geographical variation of average IQ. All of these hypotheses necessarily require complicated physiological pathways that testosterone may play a role in.

#### Results

One-tailed p-values were used for all analyses (unless otherwise noted) for two reasons: (1) our hypothesis that IQ and asthma are related via infectious disease has clear directional predictions, and (2) previous studies have found these same directions of association.

Across USA states, parasite stress and asthma rate correlated at r = -0.35 (p = 0.006, n = 50). Average state IQ and asthma rate correlated at r = 0.25 (p = 0.042, n = 50). Parasite stress correlated with IQ at r = -0.67 (p < 0.0001, n = 50), as we have

previously shown (Eppig et al., 2011). The partial correlation between average state IQ and asthma rate with the effects of parasite stress removed was r = 0.016 (p = 0.41, n = 50).

Average yearly insolation correlated significantly with asthma rate at r = -0.28 (p = 0.026, n = 50) but not significantly with infectious disease at r = 0.22 (two-tailed p = 0.12, p = 50). In a multiple regression predicting rates of asthma with both parasite stress and insolation simultaneously, infectious disease was significant (p = 0.014, std beta = -0.31) and insolation was marginally significant (p = 0.065, std beta = -0.21). The whole-model  $r^2$  was 0.17 (two-tailed p = 0.014, n = 50). Variance inflation factor (VIF) was 1.05, indicating that multicolinearity was very low and not a problem.

In determining the role of infectious disease as the mediator between latitude and asthma, we have already reported the correlation between asthma and parasite stress. Latitude correlated with asthma rates at r=0.26 (p=0.03, n=50) and with parasite stress at r=-0.45 (p=0.0005, n=50). The partial correlation between latitude and asthma rates with the effects of infectious disease removed was r=0.13 (p=0.17, n=50). In a multiple regression using latitude and parasite stress to predict asthma rates, parasite stress was a significant predictor (p=0.03, std beta = -0.29, VIF = 1.25) but latitude was not (p=0.17, std beta = 0.14, VIF = 1.26). The whole-model  $r^2$  was 0.14 (two-tailed p=0.028, n=50).

Additional correlations are shown in Table 1.

Due to the strong correlation between insolation and IQ (Table 1), we used average insolation and parasite stress simultaneously to predict average IQ in a multiple regression. The whole model  $r^2$  was 0.604 (two-tailed p < 0.0001, n = 50). Both parasite

stress (std beta = -0.58, two-tailed p < 0.0001, VIF = 1.05) and insolation (std beta = -0.41, two-tailed p < 0.0001, VIF = 1.05) were significant in the model.

#### **Discussion**

We found evidence that infectious disease is the lurking variable that explains the relationship between IQ and rates of asthma. In doing so, we also found support for the hygiene hypothesis. Rates of infectious disease and rates of asthma correlated negatively across 50 states (r = -0.35, p = 0.006, n = 50). Consistent with previous findings, average state IQ correlated positively with rates of asthma (r = 0.25, p = 0.042). When controlling for the effects of infectious disease, the correlation between IQ and asthma dropped close to zero (r = 0.016, p = 0.41, n = 50). We found a negative relationship between light intensity and asthma (r = -0.28, p = 0.026, n = 50), which is consistent with previous studies testing the vitamin D hypothesis (e.g. Krstic, 2011). When removing the effects of infectious disease, this correlation was only marginally significant (r = -0.22, p = 0.065, n = 50). In a multiple regression predicting rates of asthma, infectious disease (std beta = -0.31) accounted for a higher portion of the variance than insolation (std beta = -0.21 did.

Although the effects of insolation on asthma rates were only marginally significant when infectious disease was taken into account, the predictive power and significance of insolation only dropped slightly. Therefore, while we cannot conclude that insolation does not affect rates of asthma, our data does suggest that infectious disease may have a greater impact. Krstic's (2011) study did not investigate the impact of

variables related to other hypotheses, but it did have two advantages over our own: (1) the geographic resolution was better because the level of analysis was metropolitan areas instead of states, and (2) it covered two large nations (USA and Australia). Future research can attempt to resolve these issues while simultaneously investigating the impact of more variables.

We also found evidence that infectious disease is a powerful mediator of the relationship between latitude and asthma reported by Krstic (2011). We found that latitude and asthma rates correlate significantly and positively at r=0.26 (p=0.03, n=50). When the effects of infectious disease was removed from this relationship, the predictive power of latitude was reduced considerably (r=0.13, p=0.17, n=50). Third, infectious disease was a better predictor of asthma rates than latitude was. This indicates a strong mediating effect according to Baron and Kenny (1986). The correlation between latitude and asthma rates did not drop to zero when infectious disease was removed, which is consistent with our findings that both infectious disease and insolation contribute independently to the distribution of asthma rates.

Our findings begin to develop a larger picture of the relationships among latitude, light intensity, infectious disease, asthma, and IQ (Figure 1). Latitude contributes to variation in both light intensity and infectious disease. Both light intensity and infectious disease appear to contribute to variation in rates of asthma, with our findings showing better predictive power for parasites than for insolation. Finally, infectious disease predicts variation in average IQ. Asthma rates and IQ, which correlated positively in our current study, appear to have no direct link, but seem to be connected through infectious disease. We also found a very strong relationship between insolation and average state IQ

(r = -0.53, p < 0.0001, n = 50) that we did not predict. This relationship remained robust when infectious disease was taken into account.

Further study of the relationship between infectious disease and asthma may shed light on the relationship between infectious disease and IQ. In our 2010 study (Eppig et al., 2010), we proposed two mechanisms through which infectious disease may reduce IQ: (1) that infections temporarily draw energy away from brain development, and (2) that early exposure to infectious disease may result in a developmental pathway that permanently diverts energy away from the brain and into immune function. This latter mechanism resembles, if only superficially, predictions made by the hygiene hypothesis: that early interactions with infectious disease can produce lasting effects on the immune system. Future research could investigate this further.

# Acknowledgements

The authors thank Rachael Falcon and Kenneth Letendre for their helpful suggestions.

# References

- Baron, R. M. & Kenny, D. A. (1986). The moderator-mediator variable distinction in social psychological research: conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology*, *51*, 1173-1182.
- Benbow, C. P. (1986). Physiological correlates of extreme intellectual precocity. *Neuropsychologia*, 24, 719-725.
- Center for Disease Control (CDC). (2009). CDC Asthma BRFSS 2009 Prevalence tables and maps. Retrieved July 16, 2011 from <a href="http://cdc.gov/asthma/brfss/09/brfssdata.htm">http://cdc.gov/asthma/brfss/09/brfssdata.htm</a>.
- Devereux, D., Litonjua, A. A., Turner, S. W., Craig, L. C. A., McNeill, G., Martindale, S., Helms, P. J., Seaton, A., & Weiss, S. T. (2007). Maternal vitamin D intake during pregnancy and early childhood wheezing. *American Journal of Clinical Nutrition*, 85, 853-859.
- Carmango, C. A. Jr, Rifas-Shiman, S. L., Litonjua, A. A, Rich-Edwards, J. W., Weiss, S.T., Gold, D. R., Kleinman, K., & Gillman, M. W. American Journal of Clinical Nutrition, 85, 788-795.
- Ege, M. J., Mayer, M. Normand, A.-C., Genuneit, J., Cookson, W. O. C. M., Braun-Fahrlënder, C., Heederik, D., Piarroux, R., & von Mutis, E. (2011). Exposure to environmental microorganisms and childhood asthma. *New England Journal of Medicine*, *364*, 701-709.

- Eppig, C., Fincher, C. L. & Thornhill, R. (2010). Parasite prevalence and the worldwide distribution of cognitive ability. *Proceedings of the Royal Society B*, 277, 3801-3808.
- Eppig, C., Fincher, C. L and Thornhill, R. (2011). Parasite prevalence and the distribution of intelligence among the states of the USA. *Intelligence*, *39*, 155-160.
- Fincher, C. L. & Thornhill, R. (in press). Parasite-stress promotes in-group assortative sociality: the case of strong family ties and heightened religiosity. *Behavioral and Brain Sciences*.
- Gangal, C. & Chowgule, R. (2009). Infections in early life and susceptibility to allergic diseases: relevance of hygiene hypothesis. *Current Science*, *96*, 784-793.
- Geschwind, N. & Behan, P. (1982). Left-handedness: association with immune disease, migrane, and developmental learning disorder. *Proceedings of the National Academy of Sciences*, 79, 5097-5100.
- Jensen, A. R. & Sinha, S. N. (1993). Physical correlates of human intelligence. In P. A. Vernon (Ed.), *Biological approaches to the study of human intelligence* (pp. 139-242). Norwood: Ablex
- Hassall, C. & Sherratt, T. N. (2011). Statistical inference and special patterns in correlates of IQ. *Intelligence*, *39*, 303-310.
- Krstic, G. (2011). Asthma prevalence associated with geographical latitude and regional insolation in the United States of America and Australia. *PLOS One*, 6(4), 1-9
- Litonjua, A. A. & Weiss, S. S. (2007). Is vitamin D deficiency to blame for the asthma epidemic? *Journal of Allergy and Clinical Immunology*, *120*, 1031-1035.

- McDaniel, M. A. (2006a). Estimating state IQ: Measurement challenges and preliminary correlates. *Intelligence*, *34*, 607-619.
- McDaniel, M. A. (2006b). State preferences for the ACT versus SAT complicates inferences about SAT-derived state IQ estimates: a comment on Kanazawa (2006). *Intelligence*, *34*, 601-606.
- McKeever, W. F. & Rich, D. A. (1990). Left handedness and immune disorders. *Cortex*, 26, 33-40.
- National Renewable Energy Laboratory (NREL). (1994). Solar radiation data manual for flat-plate and concentrating collectors. Golden, Colorado: National Renewable Energy Laboratory.
- Peters, M., Kauth, M., Schwarze, J., Körner-Rettberg, C., Riedler, J., Nowak, D., Braun-Fahrländer, C., von Mutius, E., Bufe, A., & Holst, O. (2006). Inhalation of stable dust extract prevents allergen induced airway inflammation and hyperresponsiveness. *Thorax*, *61*, 134-139.
- Rand McNally & Company. (1995). *Goode's World Atlas, 19<sup>th</sup> edition*. Rand McNally & Company, USA.
- Sacher, Y., Laor, A. & Danon, Y. L. (1994). Longitudinal study on the prevalence of asthma among Israeli young adults. *Israeli Journal of Medical Sciences*, *30*, 564-572.
- Segalowitz, S. J., Berge, B. E., Lawson, S., & Brown, D. (1994). If you can replicate the handedness-immune disorder effect, the more power to you. *Brain and Cognition*, 26, 217-227.

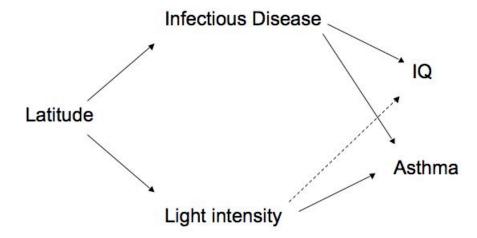
- Strachan, D. P. (1989). Hay-fever, hygiene, and household size. *British Medical Journal*, 299, 1259-1260.
- Vogel, K., Blümer, N., Korthals, M., Mittelstädt, J., Garn, H., Ege, M., von Mutius, E., Gatermann, S., Bufe, A., Goldman, T., Schwaiger, K, Renz, H., Brandau, S., Bauer, J., Heine, H., & Holst, O. (2008). Animal shed *Bacillus licheniformis* spores possess allergy-protective as well as inflammatory properties. *Journal of Allergy and Clinical Immunology, 122*, 307-312.

Table 1: Zero order correlations among all variables.

	1.	2.	3.	4.	5.
1. IQ		-0.67***	-0.53***	0.25†	0.59***
2. Parasite Stress			0.22	-0.35*	-0.45**
3. Insolation				-0.28†	-0.78***
4. % Asthma					0.26†

5. Latitude

 $N=50 \ states \ for \ all \ correlations. \ All \ p-values \ are \ two-tailed. \ \dagger p<0.1, \ *p<0.05, \ **p<0.01, \ ***p<0.001.$ 



(Figure 1: Directions of influence tested in this study. Solid lines are those predicted collectively by the hygiene hypothesis, the vitamin D-deficiency hypothesis and the parasite-stress hypothesis. Dashed line represents a statistically robust relationship not predicted by any of these hypotheses. All influences are negative.)