Do wealth and inequality associate with health in a small-scale subsistence society?

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Abstract

In high-income countries, one's relative socio-economic position and economic inequality may affect health and well-being, arguably via psychosocial stress. We tested this in a small-scale subsistence society, the Tsimane, by associating relative household wealth (n=871) and community-level wealth inequality (n=40, Gini = 0.15 – 0.53) with a range of psychological variables, stressors, and health outcomes (depressive symptoms [n=670], social conflicts [n=401], non-social problems [n=398], social support [n=399], cortisol [n=811], BMI [n=9926], blood pressure [n=3195], self-rated health [n=2523], morbidities [n=1542]) controlling for community-average wealth, age, sex, household size, community size, and distance to markets. Wealthier people largely had better outcomes while inequality associated with more respiratory disease, a leading cause of mortality. Greater inequality and lower wealth were associated with higher blood pressure. Psychosocial factors didn't mediate wealth-health associations. Thus, relative socio-economic position and inequality may affect health across diverse societies, though this is likely exacerbated in high-income countries.

Introduction

It is relatively uncontroversial that people with greater access to resources – usually operationalized as income, wealth, or broader indicators of socio-economic position, rank or status[1] – are likely to be in better health, as resources can be converted into better nutritional status, access to health care, or insulation against health risks. Such benefits of *absolute* rank are also commonly found in non-human primates (Cowlishaw and Dunbar, 1991; Pusey et al., 1997; Snyder-Mackler et al., 2020; van Noordwijk and van Schaik, 1999). However, there is increasing evidence that relative access to resources, i.e. one's relative position in a socio-economic hierarchy, may also affect health. Across developed societies, there is causal evidence for a health gradient along socio-economic hierarchies, independent of absolute wealth or use of health care services (Ecob and Davey Smith, 1999; Marmot et al., 1991; Oakes et al., 1973; Sorlie et al., 1995; Wolfson et al., 1993). In other words, these studies find that *relative* rank - how one compares to others - is a critical variable in determining health outcomes (Anderson et al., 2012; Luttmer, 2005; Snyder-Mackler et al., 2020; Wood et al., 2012).

The steepness of socio-economic hierarchies (i.e. income or wealth *inequality*) is also associated with both physical and mental health outcomes - including self-rated health, all-cause mortality, heart disease, respiratory disease, obesity, or homicide rates - independent of absolute wealth (Nowatzki, 2012; Pickett and Wilkinson, 2015; Wilkinson and Pickett, 2006). While these findings are hotly debated and tests of this inequality hypothesis have been critiqued on methodological grounds (Kondo et al., 2009; Lynch et al., 2004; Macinko et al., 2003; Subramanian and Kawachi, 2004; Wagstaff and van Doorslaer, 2000), a formal meta-analysis on studies controlling for individual wealth found significant associations between inequality and mortality or self-rated health in high-income countries (Kondo et

[1] We use status or rank interchangeably to refer to one's position in a hierarchy. In the present study, we focus on household wealth because it was most widely available, but show that it correlates with other measures of status (see Methods)

al., 2009). Thus, relative position in a socio-economic hierarchy and the steepness of such hierarchies seem to matter for health.

The most cited mechanism for such hierarchy-health associations is that hierarchies cause psychosocial stress, which in turn leads to poorer health outcomes (Chen and Miller, 2013; Pickett and Wilkinson, 2015). Chronic stress leads to altered hypothalamic-pituitary-adrenal (HPA) axis function, including chronically elevated cortisol levels. Increased cortisol can cause neural atrophy, cardiovascular damage, obesity, or immunosuppression, all resulting in increased susceptibility to chronic and infectious disease (Aiello et al., 2018; Garcia et al., 2017; Kunz-Ebrecht et al., 2004; Quon and McGrath, 2014; Sapolsky, 2004). In addition, submission in status competition and learned helplessness are associated with depression in humans and other primates (Hagen, 2011; Nesse, 2000; Stieglitz et al., 2014). Experimental studies in nonhuman primates show that dominance rank also affects gene expression and immune function (Snyder-Mackler et al., 2016; Tung et al., 2012). Related results in humans show that early life experiences and other forms of social stress are also associated with increases in inflammation and blunted immunological responses to cortisol (Aiello et al., 2018; Miller et al., 2014, 2011, 2009).

But why are hierarchies stressful or otherwise detrimental to health? An evolutionary-medicine perspective suggests that many detrimental health outcomes may result from adaptive *tradeoffs*, as fitness gains are prioritized over detrimental health outcomes, *developmental constraints*, as long term negative health effects result from short term accommodations to conditions during development, or from evolutionary *mismatch*, as our bodies struggle to deal with conditions atypical for our species (Eaton et al., 2002; Gluckman et al., 2016; Lea et al., 2017; Nesse and Williams, 1994; Wells et al., 2017). Given the consistent fitness benefits of high status (Stulp et al., 2016; Von Rueden and Jaeggi, 2016), and given that fitness is always relative, humans arguably have evolved motivations for status-striving that are independent of one's absolute access to resources (Johnson et al., 2012; Shenk et al., 2016). Status-

82 striving activates the stress response, and not just for low-rankers: depending on how rank is achieved
83 and maintained, high- or low-ranking individuals may be more stressed (Abbott et al., 2003; Sapolsky,
84 2005). Crucial to who is stressed is the availability of social support, which can be as or even more
85 important for health and fitness as rank per se (Sapolsky, 2005; Snyder-Mackler et al., 2020). Other
86 factors primarily impact low-ranking individuals: in many primate (and some human) societies,
87 subordinates are regularly subjected to aggression and intimidation by higher-ranking individuals (Silk,
88 2003), resulting in the lack of control and learned helplessness that often cause depression (Sapolsky,
89 2005, 2004).

90 Greater inequality, i.e. steeper hierarchies, entail more skewed payoff distributions and thus
91 also favor more intense competition and risk-taking as behavioral strategies, especially among low-
92 ranking individuals; this is argued to explain the persistent association between income inequality and
93 homicide rates, as most homicides result from escalated contests over status (Daly, 2016; Daly and
94 Wilson, 1997). If skewed pay-off distributions and oppression of low-rankers favor life-history strategies
95 focused on short-term payoffs (i.e. "faster" life-history strategies *sensu* Wells et al., 2017), this could
96 also explain hierarchy-health associations via present-oriented decision-making at the expense of long-
97 term health (Daly and Wilson, 1997; Griskevicius et al., 2011; Pepper and Nettle, 2014). These
98 relationships are expected even when hierarchies are based on prestige, rather than dominance, since
99 prestige-based hierarchies still correlate with social support, insulation against shocks, influence, sense
100 of control, and access to mates (Gurven et al., 2000; Sugiyama and Sugiyama, 2003; von Rueden et al.,
101 2014).

102 Thus, stress and negative health consequences due to socio-economic hierarchies can result
103 from perpetual status-striving, unequal distribution of social support, lack of control and learned
104 helplessness, intensified competition especially among low-ranking individuals, and from physiological
105 accommodations to generally "faster" life histories. In short, hierarchies may cause stress and affect

health largely because individuals engage in competitive strategies that function to maximize fitness at the expense of health, while failure to succeed in such competition negatively affects mental health. In addition, if hierarchies constrain access to resources individuals may face developmental constraints causing long-term tradeoffs that negatively impact health.

While such adjustments of physiology and behavior to the local competitive environment may in principle generalize to all human societies, the effects of hierarchy on health may be exacerbated in industrialized, high-income countries due to mismatch. Specifically, such societies could represent a mismatch with the ancestral environments in which our competitive strategies have evolved because (i) socio-economic hierarchies may be steeper and more rigid than was typical of our hunter-gatherer ancestors (Borgerhoff Mulder et al., 2009; Kaplan et al., 2009), and include features such as lack of kin support, limited upward mobility, structural violence, and systemic racism, all of which are well-known to negatively affect health (Gravlee, 2009; Sapolsky, 2004), and (ii) novel lifestyle factors such as obesogenic diets, lack of physical activity and chronic inflammation turn previously relatively harmless responses, such as temporarily elevated blood pressure or depressed mood, into "mismatch" diseases, such as hypertension, atherosclerosis, and major depression (Gurven et al., 2012; Kaplan et al., 2017; Miller and Raison, 2016; Stieglitz et al., 2015). In sum, mismatch diseases often arise when risk factors that used to elicit an adequate acute response become chronic problems, which could well be the case with modern socio-economic hierarchies, and interact with novel lifestyle factors that push our physiology into novel and unhealthy ranges.

In summary, humans, much like other primates, are sensitive to their relative rank and the distribution of fitness outcomes, and adjust behavior and physiological responses accordingly, resulting in negative influences of hierarchy on health. Several open questions remain, however. First, the inequality hypothesis remains hotly debated, since parsing inequality from other correlated variables is difficult and requires careful statistical methods. Second, it remains unclear to what extent the observed

health consequences of relative status and inequality in high-income countries (i) represent tradeoffs of potentially adaptive responses to lower relative rank and/or to inequality, or (ii) are caused by evolutionary mismatch, i.e. novel conditions that cause maladaptive outcomes. If health consequences stem from tradeoffs from adaptive responses then hierarchy should be associated with health in any population, independent of absolute access to resources. However, if the impacts of status and inequality are caused by evolutionary mismatch, then we would not expect detrimental effects on health in all societies, though we might observe related physiological responses in a subclinical range.

Small-scale societies practicing traditional subsistence lifestyles (henceforth: "subsistence societies") are an important test case for the universality of hierarchy-health associations as they generally have more informal, egalitarian hierarchies with relatively high individual autonomy and mobility (Borgerhoff Mulder et al., 2009; Kaplan et al., 2009; Mattison et al., 2016), and suffer from infectious rather than chronic disease as major sources of morbidity and mortality (Eaton et al., 1988; Gurven et al., 2007, 2016; Gurven and Kaplan, 2007; Kaplan et al., 2017; Pontzer et al., 2018). Further, individuals in many subsistence societies have immune systems that are well calibrated by frequent exposure to pathogens and microbiota, and predominantly experience acute responses to infections (Blackwell et al., 2016a; McDade, 2005), unlike the chronic low-grade inflammation that links stress to hypertension, cardiovascular disease, and depression in high-income countries (Gurven et al., 2008a). Lastly, competition for mates and resources in such societies is usually fairly local, meaning that the scale at which relative rank and inequality should be measured is more recognizable than in large-scale modern societies with mass media, where people are simultaneously part of many hierarchies. Thus, subsistence societies may help us discern whether associations between hierarchy and health are caused by tradeoffs expected in any society, by evolutionary mismatch in modern, industrialized populations, or a combination of both.

Few studies have examined associations between rank or inequality and health in subsistence societies. Among Dominican farmers, socio-economic indicators were unrelated to cortisol levels whereas local influence was associated with lower cortisol (Decker, 2000). Among egalitarian Garisakang horticulturalists in Papua New Guinea, higher income coming from greater market exposure was associated with higher cortisol, whereas other locally relevant measures of wealth and status were not (Konečná and Urlacher, 2017). While results are mixed, there is some converging evidence that suggests market integration generates psychosocial stress in subsistence societies, arguably due to the threat of cultural loss and discrimination often experienced through contact with majority groups.

Among Tsimane forager-horticulturalists in Bolivia, it has been reported that traditional forms of status generally support a status-health gradient, but studies on income or wealth show mixed results. In a sample of four communities, politically influential men had lower cortisol and a lower incidence of respiratory infection, though there were also many null results, and higher income was associated with higher cortisol (von Rueden et al., 2014). In one village, women's political influence was associated with improved growth and health outcomes for their children (Alami et al., 2020). Across 13 Tsimane villages, relative wealth was associated with better self-reported health (Undurraga et al., 2010); however, average self-reported health was lower in wealthier villages. In a larger sample of villages, relative income associated with lower BMI among individuals with smaller support networks (Brabec et al., 2007).

In terms of the relationship between inequality and health within communities, studies among Tsimane have also shown mixed results. One study found no association between income inequality and body fat (Godoy et al., 2005), but income inequality was associated with more negative emotions (Godoy et al., 2006). Greater wealth inequality did not associate with self-reported health in one study (Undurraga et al., 2010) but did associate with better self-reported health and lower self-reported stress

in another, controlling for individual and village wealth level (Undurraga et al., 2016). Overall, these results provide mixed evidence for associations between inequality and health.

Here we test for links between hierarchy and health among the Tsimane, expanding upon previous studies in several ways. First, we simultaneously assess the effects of within-community relative wealth, mean community wealth, and community-level wealth inequality. Second, while previous studies have mostly relied on just one or two indirect health outcomes such as BMI, we include thirteen different dependent variables (Table 1) capturing various health outcomes, including infectious disease morbidity, psychological well-being, social conflicts and connections, and other stressors. Third, we explicitly test whether these psychological and social variables and other stressors (henceforth "psychosocial variables") mediate links between wealth and health, as predicted if the adverse health effects of hierarchy occur through psychosocial stress. Note though that some of these "psychosocial" variables may also be associated with health through more direct mechanisms, e.g. non-social problems (food insecurity, debt, etc.) may cause stress but also represent poorer access to resources, which could affect health through energetic constraints. Fourth, we greatly increase the sample size relative to previous studies with inequality measured in 40 communities and wealth in 871 households, representing approximately one quarter of the Tsimane population (see Table 1, Figure 1-figure supplement 1). Thus, our study represents the most comprehensive test of hierarchy-health associations in a subsistence society.

We specifically test the following predictions stemming from the hypotheses that relative socio-economic position as well as the steepness of socio-economic hierarchies affect health and well-being, and that these effects are mediated by psychosocial stress.

P1: Higher relative wealth is associated with better psychosocial and health outcomes

P2a: Greater wealth inequality is associated with worse psychosocial and health outcomes, and

P2b: this should hold especially for low-rankers

P3: Psychosocial variables mediate wealth and inequality-health links found under P1 and P2

Table 1 gives an overview of all variables used to test these predictions.

* Insert Table 1 about here *

Study Population

The Tsimane are a population of >16,000 Indigenous Amerindians living in >90 communities at the edge of the Amazon basin in lowland Bolivia. Tsimane communities consist of dispersed household clusters tied together by networks of kinship, cooperative production and consumption (Hooper et al., 2015; Jaeggi et al., 2016) as well as usually a school and soccer field. Community meetings convene to discuss and resolve important matters, including conflicts within the community. As such, we treat the community as the salient scale of status competition (Alami et al., 2020; von Rueden et al., 2018, 2008, 2019, 2014), and calculated relative wealth and inequality at this level.

The Tsimane remained relatively isolated from the larger Bolivian economy until the 1970's and still widely practice traditional subsistence (swidden horticulture, hunting, and fishing), which contributes >90% of their calories (Gurven et al., 2017; Kraft et al., 2018). Cattle, introduced by missionaries and ranchers, are owned by a small minority of Tsimane. Over the past few decades, wage labor opportunities with loggers or ranchers and produce sales in the local market towns of San Borja and Yucumo have been increasing, as have formal schooling, Spanish fluency, and access to modern amenities such as electricity and health care. The population thus exhibits quantifiable gradients of modernization (see Fig. 1).

In terms of morbidity and mortality, the Tsimane are characterized by high infectious disease burden, with respiratory infections as the leading cause of death at all ages (Gurven et al., 2007). Additionally, parasites such as helminths and giardia are highly prevalent (Blackwell et al., 2013; see also

Table 2). These conditions result in frequent, acute immune responses (Blackwell et al., 2016a) but still a low incidence of chronic conditions such as hypertension or atherosclerosis, due to high levels of physical activity and other protective factors (Gurven et al., 2012, 2016, 2009; Kaplan et al., 2017).

Results

Tsimane wealth and inequality

Because wealth varied considerably by age (Fig. 1A), we used an age-corrected measure of relative wealth that reflects one's wealth relative to this age trajectory (see Methods). This corrects for random variation in the age structure of sampled communities, and arguably better captures the essence of relative socio-economic rank: what matters is how one compares to others, relative to general trends such as wealth (status, influence, etc.) accumulating with age. At the high end of the wealth distribution (Fig. 1B), much of the variation was driven by livestock, especially cattle. Fig. 1C-D illustrate variation in mean wealth and wealth inequality among the study communities. Mean wealth was generally lowest in communities located in the interior forest (Fig 1C, bottom right), which are remote and inaccessible by road for much of the year (due to washed-out bridges); and in those communities downriver from San Borja (Fig 1C, top), which experience frequent flooding and are within or adjacent to a protected bioreserve that limits resource extraction. Somewhat unexpectedly, mean wealth was higher further from the market town of San Borja (correlation between mean wealth and distance to market $r=0.36$, $df=38$, $p=0.02$). We operationalized inequality by calculating community-level Gini coefficients for age-corrected wealth (see Methods). Wealth inequality was generally higher in communities closer to the market towns of San Borja and Yucumo, where Tsimane can sell produce and purchase market goods, though some villages near towns also show low inequality (Fig 1D) (correlation between Gini and distance to market $r= -0.38$, $df=38$, $p=0.01$). Inequality was marginally lower in richer communities ($r= -$

0.22, df=38, p=0.17). Community size was not significantly related to distance (r= -0.18, p=0.26), mean wealth (r=0.11, p=0.50), or inequality (r=0.00, p=0.99). In sum, villages near towns had both higher inequality and lower mean wealth due to both more wealthy individuals and more very poor individuals in these communities.

Insert Figure 1 about here

Modeling strategy

To examine the effects of household wealth and community wealth inequality on psychosocial or health outcomes, we used Bayesian multilevel models with appropriate controls and random effects at the individual, household, and community level (see Methods). Wealth was divided into relative wealth, centered on the community mean, and mean community wealth. Operationalizing wealth this way means we are in principle able to tease apart within-community wealth differentials, i.e. one's position in the local socio-economic hierarchy, from community-level differences in access to resources, i.e. mean community wealth (Kreft et al., 1995). However, in practice, models with wealth centered on the village produced virtually identical estimates to models with wealth centered on the sample as a whole (see Supplementary file 1a-1m), largely because villages did not differ strongly in mean wealth (median -0.03, range: -1.0 to 0.66 z-scores, 80% between -0.43 and 0.37). Thus, community relative and population relative wealth were highly correlated (r=0.92).

Bayesian models produce a posterior distribution of parameter estimates that can be summarized in various ways (McElreath, 2020). Here we provide coefficient plots (Figures 2-5) showing posterior medians, as well as 75% and 95% highest posterior density intervals; we also provide prediction plots as supplements to these figures. In the text we report results as standardized coefficients (β) for Gaussian models or as log odds (β) and odds ratios (OR) for logistic models, both

represented by the posterior mean, as well as the proportion of the posterior above zero ($P_{>0}$), i.e. the likelihood of a positive association. Higher or lower values of this number represent stronger certainty for a non-zero effect, while values near 0.5 indicate complete uncertainty about the direction of an association, if any. In addition, we report Cohen's *d* as a standardized measure of effect size to allow comparison between continuous and binary variables; *d* is reported as the posterior median and the median absolute deviation [MAD] (a more robust measure of dispersion than the standard deviation). For simplicity, we refer to effect sizes of d>0.2 as "strong", those >0.1 as "moderate", and consider the rest to be "weak" though potentially still suggestive of a general pattern. Similarly, we refer to posterior support of >0.975 (or <0.025, if negative) as "high certainty" and those with support >0.875 /<0.125 as "moderate certainty", corresponding to the entire 95% or 75% highest posterior density intervals respectively not overlapping with 0, and we consider the rest to be "uncertain". However, we encourage readers to use the full information on the posteriors to inform their own inference. Means and 95% credible intervals for all parameters are reported in Supplementary file 1a-1o. These tables also provide Bayesian R^2 (Gelman et al., 2019) as a goodness of fit measure, indicating that in most models, the predictors and random effects jointly explained about 20-40% of the variance in the data (R^2 range: 0.16-0.91).

Is wealth related to health outcomes?

Overall, for adults, household wealth was associated, with various effect sizes and degrees of confidence, with beneficial health outcomes except gastrointestinal illness, which showed no association (Fig. 2; Supplementary file 1f-1m). Community mean wealth had more mixed associations with health outcomes. Specifically, household wealth was associated with lower systolic blood pressure (β=-0.01, $P_{>0}$=0.37, Cohen's d=-0.01 [0.02]) and lower diastolic blood pressure (β=-0.04, $P_{>0}$=0.02, d=-0.05 [0.02]), though both effect sizes were small and only the latter association had high certainty.

Community mean wealth was strongly and with high certainty associated with lower systolic (β=-0.29, $P_{>0}$=0.00, d=-0.34 [0.09]) and diastolic (β=-0.21, $P_{>0}$=0.01, d=-0.24 [0.11]) blood pressure. Household wealth also associated with better self-rated health (reverse coded β=-0.02, $P_{>0}$=0.14, d=-0.03 [0.02]), lower odds of infectious (β=-0.06 $P_{>0}$=0.26, d=-0.02 [0.05], OR=0.94) and respiratory (β=-0.04 $P_{>0}$=0.69, d=-0.03 [0.05], OR=0.96) illness, and lower total morbidity (β=-0.02, $P_{>0}$=0.23, d=-0.02 [0.04]), though again most effect sizes were small and there was high uncertainty. There was no evidence for an association with gastrointestinal infection. However, there was a moderate though uncertain association between community mean wealth and lower gastrointestinal illnesses (β=-0.32, $P_{>0}$=0.21, d=-0.16 [0.21], OR=0.72). Household wealth was weakly and uncertainly associated with lower BMI (β=-0.01, $P_{>0}$=0.24, d=-0.02 [0.04]), but community mean wealth was weakly associated with higher BMI (β=0.06, $P_{>0}$=0.74, d=0.12 [0.20]). Using population-relative wealth, rather than community relative wealth had little effect on these associations (Supplementary file 1a-1m). In sum, despite mostly small effect sizes and high uncertainty, the general pattern was for wealthier adults to have better outcomes.

For juveniles ≤15 years of age (Fig. 3, Supplementary file 1n & 1o), household wealth was weakly associated with lower total morbidity (β=-0.04, $P_{>0}$=0.06, d=-0.06 [0.04]), and in particular, moderately lower odds of respiratory illness (β=-0.24, $P_{>0}$<0.01, d=-0.13 [0.05], OR=0.79). However, both household and community mean wealth were associated with higher odds of gastrointestinal illness (β=0.13, $P_{>0}$=0.95, d=0.07 [0.04], OR=1.14; β=0.49, $P_{>0}$=0.81, d=0.27 [0.30], OR=1.63) and community mean wealth was associated with other infections (β=0.81, $P_{>0}$=0.87, d=0.44 [0.37], OR=2.25) and higher total morbidity (β=0.05, $P_{>0}$=0.82, d=0.31 [0.33]) with mostly strong effect sizes but high uncertainty. In sum, for juveniles, wealth was moderately associated with reduced risk of respiratory illness, while community wealth was strongly associated with several negative health outcomes.

insert Figure 2 about here

Is inequality related to health outcomes?

For adults, inequality was associated with higher levels of three morbidity-related outcomes and lower levels of four outcomes (Fig. 2, Supplementary file 1f-1m). Consistent with predictions of worse health with inequality (P2a), greater inequality was weakly associated with higher blood pressure (systolic: β=0.05, $P_{>0}$=0.98, d=0.06 [0.03]; diastolic: β=0.02, $P_{>0}$=0.75, d=0.03 [0.04]), and strongly with a greater likelihood of respiratory illness (β=0.35, $P_{>0}$=0.93, d=0.20 [0.13], OR=1.36). Despite these harmful associations with inequality, people in more unequal communities had a strongly lower likelihood of other infections (β=-0.62, $P_{>0}$=0.02, d=-0.33 [0.16], OR=0.54) and to a more uncertain degree, total morbidity (β=-0.07, $P_{>0}$=0.25, d=-0.07 [0.13]), and gastrointestinal infections (β=-0.12, $P_{>0}$=0.22, d=-0.06 [0.09], OR=0.89). Associations with BMI were negligible (β=0.01, $P_{>0}$=0.62, d=0.03 [0.08]).

In contrast, for juveniles (Fig. 3, Supplementary file 1m & 1o), BMI was lower in more unequal communities (β=-0.06, $P_{>0}$=0.05, d=-0.08 [0.05]). Inequality had little effect on total morbidity and was moderately associated with less infectious illness (β=-0.23, $P_{>0}$=0.24, d=-0.13 [0.19], OR=0.79), but greater respiratory illness (β=0.21, $P_{>0}$=0.81, d=0.11 [0.13], OR=1.23) and gastrointestinal illness (β=0.17, $P_{>0}$=0.74, d=0.10 [0.13], OR=1.19), both of which are highly prevalent among juveniles.

Insert Figure 3 about here

Is wealth related to psychosocial outcomes?

For adults, greater household wealth was associated with better outcomes in four of five psychological and social measures, with no association for the fifth (Fig. 2; Supplementary file 1a-1e). Household wealth was strongly and with high certainty associated with having more labor partners (reverse coded β= -0.13, $P_{>0}$=0.01, d=-0.49 [0.20]), and weakly and uncertainly, with fewer depressive symptoms (β=-

0.04, $P_{>0}$=0.14, d=-0.05 [0.05]), fewer non-social problems (i.e. self-reported concerns over food insecurity, debt, and illness; β=-0.06, $P_{>0}$=0.12, d=-0.08 [0.07]), and lower urinary cortisol (β=-0.02, $P_{>0}$=0.27, d=-0.02 [0.04]). There was no support for an association with social conflicts. Unlike household wealth, community mean wealth was not clearly associated with any psychosocial outcome, though there were strong but uncertain associations with more labor partners (reverse coded β=-0.16, $P_{>0}$=0.29, d=-0.77 [0.94]) but also more non-social problems (β=0.28, $P_{>0}$=0.77, d=0.33 [0.45]).

Is inequality related to psychosocial outcomes?

Contrary to predictions, inequality was largely associated with fewer stressors and psychological or social problems (Fig. 2; Supplementary file 1a-1e). The strongest evidence was for fewer non-social problems in more unequal communities (β=-0.15, $P_{>0}$=0.07, d=-0.17 [0.13]), with weak evidence for fewer conflicts (β=-0.04, $P_{>0}$=0.33, d=-0.01 [0.09]), and more labor partners (β=-0.05, $P_{>0}$=0.32, d=-0.29 [0.38]) with more inequality.

Do psychosocial variables mediate relationships between wealth or wealth inequality and health?

We tested the prediction (P3) that the effects of wealth or inequality on health were mediated via psychosocial pathways using formal mediation analysis (Baron and Kenny, 1986; MacKinnon et al., 2007). Specifically, this involves estimating the association between wealth/inequality and psychosocial variables ("path *a*"), as well as between psychosocial variables and health outcomes ("path *b*"); if both are statistically significant and the association between wealth/inequality and health outcomes ("path *c*", or *direct effect*) is weaker, then there is evidence that there is an *indirect effect* of wealth/inequality on health via psychosocial variables (i.e. the psychosocial variable is a mediator). As reported above, paths *a* were mostly supported for household wealth, i.e. household wealth was associated with four of the five psychosocial variables, but not for community wealth or inequality. Supplementary file 1q-1s

present mediation analyses with each health outcome variable and each psychosocial variable as a potential mediator, including estimates of the direct (path *c*, as reported above) and indirect effects, the mediator effects (Path *b*), and the proportion mediated (indirect effect / total effect). See Appendix 1 for a discussion and graphical depiction of the causal relationships assumed by this mediation approach.

The only convincing evidence for mediation was found for depression and non-social problems mediating the effect of household wealth on diastolic blood pressure; specifically, household wealth was negatively associated with diastolic blood pressure (path *c*) as well as with depression and non-social problems (paths *a*; see above, Figure 2), and both depression (β=-0.03, $P_{>0}$=0.20) and non-social problems (β=-0.08, $P_{>0}$=0.05) were themselves negatively associated with diastolic blood pressure (paths *b*). However, there were no other cases where both path *a* and path *b* were well supported, the indirect effects of household wealth, community wealth, or inequality were virtually always 0 for any mediator (including depression and non-social problems), and the proportion mediated was generally small or highly uncertain (Supplementary file 1q-1s). Overall, there was little evidence of mediation.

Effect of covariates on outcomes

Of the included covariates, many were associated with outcomes. For adults (Fig. 4; Supplementary file 1a-1m), age was positively associated with all negative health outcomes except respiratory illness as well as depression and social conflict. Male sex was associated with increased blood pressure but lower depression, conflicts, non-social problems, urinary cortisol, infection illness, and total morbidity, and with better self-rated health. Increasing distance from the market town was associated with increased blood pressure, more conflicts, respiratory illness, and gastrointestinal illness, as well as lower BMI. However, it was also associated with lower depression and urinary cortisol. Community size was generally associated with more positive psychological and social variables, but also higher blood pressure and infection. Household size was associated with worse psychological and social condition,

with the exception of labor partners, which were higher for large households. Results for juveniles largely reflect similar associations (Fig. 5; Supplementary file 1n & 1o).

In some cases the inclusion of covariates improved model R^2 statistics, though in many models changes in fit were negligible (Supplementary file 1a-1o). In general, the inclusion of covariates reduced the variance attributable to random effects for individual, household, and community. Posterior distributions for wealth and inequality associations were all similar whether covariates were included or excluded (i.e. the posteriors overlap substantially), though there were some minor differences between the posterior means that were largely inconsequential for inference.

Insert Figure 4 about here

Insert Figure 5 about here

Is there evidence for more complex wealth-health associations?

Finally, we conducted several post-hoc tests to examine whether wealth-health associations were contingent on sex, or whether relative wealth effects were contingent on levels of inequality and vice versa. For example, inequality could trigger increased stress and competitiveness only in men given a history of higher reproductive skew in males (Daly, 2016) and inequality might affect the wealthier and poorer differently (P2b), i.e. poorer individuals may fare even worse in more unequal contexts. For this reason we included wealth-by-inequality, wealth-by-sex, or inequality-by-sex interactions in models. A number of models favored interactions though there was little consistency across outcomes (Fig. 6; Supplementary file 1p). For depression, systolic and diastolic blood pressure, and self-rated health, poorer men showed worse outcomes than wealthier men, though there was little effect of wealth for women. In contrast, poorer women reported more non-social problems. Poor individuals showed both increased conflicts and reduced labor partners in unequal places, while wealthier individuals reported

more conflicts and fewer labor partners in equal communities. In unequal communities, wealth had little effect on respiratory illness, while in more equal places, wealthier individuals were less likely to be diagnosed with respiratory illness. Contra P2b, there was no consistent indication that inequality was worse for poorer individuals, while males were somewhat more affected by being poor.

insert Figure 6 about here

Discussion

We tested whether within-community relative wealth, community wealth, and community-level wealth inequality were associated with a broad range of psychological, social, and health outcomes in a large sample of households and communities in a relatively egalitarian small-scale subsistence society. Overall, our results showed substantial heterogeneity in terms of the direction and magnitude of associations between wealth, wealth-inequality, and health, which contrasts with the more consistent SES-health gradients in high-income countries. Nevertheless, some findings supported an association between wealth or inequality and health outcomes, though these associations were not mediated by psychosocial factors.

Consistent with the prediction that higher relative position in a socio-economic hierarchy improves outcomes (P1), we found that household wealth relative to others in the community, capturing one's rank within the local socio-economic hierarchy, was associated with lower blood pressure, and for juveniles, lower total morbidity and fewer respiratory infections. Relative household wealth was also generally associated with better health and psychosocial outcomes, but with more uncertainty in the posterior estimates, and for juveniles, relative household wealth was associated with

438 increased gastrointestinal illness. Community mean wealth, capturing the absolute access to resources
439 of households within that community, was also strongly associated with lower blood pressure for adults,
440 but there was high uncertainty in estimates for other outcomes. Conversely, in support of P2a inequality
441 was associated with higher blood pressure in adults and more respiratory disease in both adults and
442 juveniles. It was also associated with lower BMI in juveniles, which in this energetically-limited
443 population likely represents a negative outcome. However, contra P2a inequality was also associated
444 with lower levels of other infections (mostly fungal and yeast infections and lice) as well as fewer non-
445 social problems, and there were several null results (Figures 2 & 3). Although most effect sizes were
446 weak to moderate (most Cohen's d <0.2), these statistically weak results could still have significant
447 biological and clinical impacts, as elaborated below.

448 The finding that higher inequality associated with greater likelihood of respiratory disease is
449 perhaps the most significant in terms of well-being and biological fitness. Respiratory illness is the
450 leading cause of mortality at all ages in this population (Gurven et al., 2007), and continues to be a
451 major source of morbidity. The likelihood of being diagnosed with respiratory illness was predicted to
452 differ >3-fold, 8% to 28%, between the least and most unequal communities indicating substantial
453 fitness costs to inequality. However, this effect of inequality appears to primarily affect wealthy
454 individuals, bringing their prevalence up to the level of poorer individuals (Figure 6). In one Tsimane
455 community (with relatively high average income compared to other communities), von Rueden et al
456 (2014) found lower risk of respiratory infection among influential men but no effect on respiratory
457 infection (though trending in direction of higher risk) for men with higher income. With current data, we
458 cannot determine the mechanism responsible for this association between inequality and respiratory
459 disease. The association could reflect differences in immune function as suggested by other research on
460 psychosocial influences on infectious disease (Aiello et al., 2018; Chen and Miller, 2013; McDade et al.,
461 2016), despite a lack of evidence for psychosocial mediation here. The association between inequality

and respiratory disease could also be spurious, despite our best efforts to control for relevant covariates, or it could reflect differences in exposure not captured by distance to town or community size (such as population density, or frequency of contact with outsiders); in this context, it should be noted that effects of inequality on health are arguably only expected for outcomes for which there is a socio-economic gradient in the first place (Pickett and Wilkinson, 2015), which was not the case for respiratory disease here.

One of the strongest, most certain and most consistent associations of wealth (both household- and community-level) and inequality was with blood pressure, a major contributor to chronic disease in high-income countries. There was a clear socio-economic gradient in blood pressure within and between communities, and blood pressure was higher in more unequal communities. These effects were observed primarily in men. While most Tsimane are not hypertensive and do not have heart disease (Gurven et al., 2012; Kaplan et al., 2017), the predicted effects of wealth and inequality on blood pressure were substantial: systolic blood pressure was predicted to increase by 0.32 SD (i.e. 4.0 mmHg) and diastolic blood pressure by 0.40 SD (3.7 mmHg) in the most unequal compared to the most equal communities; conversely, wealth was protective such that the lowest blood pressures were predicted for people in the richest communities (7.4 mmHg systolic and 4.2 mmHg diastolic lower) and the richest households within communities (1.0 mmHg systolic and 3.1 mmHg diastolic lower). In high-income countries such changes in blood pressure correspond to as much as a 10% change in the risk of major cardiovascular disease events (see Figure 2 in Ettehad et al., 2016). Among the Tsimane, it corresponds to as much as 40 years of age-related increases in blood pressure (Gurven et al., 2012). As novel, obesogenic foods enter the Tsimane diet (Kraft et al., 2018), market integration increases stress (Konečná and Urlacher, 2017; von Rueden et al., 2014), sanitation improves (Dinkel et al., 2020), and protective lifestyle factors like physical activity and helminth infections are changing (Gurven et al., 2013, 2016), people in unequal communities, especially the poor (see Figure 6), may be at increasingly

greater risk of chronic disease. Increases in blood pressure with modernization have also been reported in many other subsistence populations, and may partly stem from stress caused by integrating into a dominant culture (Dressler, 1999; Konečná and Urlacher, 2017). In this context, it is also worth noting that while the range of our village-level Gini values (0.15-0.43) was similar to that of *income* inequality among high-income countries (e.g. Denmark: 0.24, USA: 0.45), it was considerably lower than the range of *wealth* inequality in these countries (e.g. Japan: 0.55, USA: 0.81 (Nowatzki, 2012)). Thus, the reported associations between wealth/inequality and blood pressure may still be relatively harmless for the Tsimane, but lay the foundation for chronic disease under more mismatched conditions.

An alternative interpretation for some of these associations may be that causality is reversed, with poor health leading to less wealth or exacerbated inequality. On the face of it, this seems plausible for respiratory illness, which reduces work productivity. However, the fact that we see no direct association with wealth for adults, and only an association with inequality, seems to argue against such a mechanism. We did find an association between wealth and respiratory disease for juveniles—perhaps having sicker children puts some strain on wealth accumulation. For blood pressure it is harder to imagine how reverse causality might occur, since the blood pressure changes we observed are unlikely to affect wealth. Regardless, a limitation of our data is that we cannot determine the direction of causation given our cross-sectional design. Other confounds might also be possible, for example if people preferentially assort by health or wealth by moving between villages.

Beyond respiratory disease and blood pressure, many associations were inconclusive. This heterogeneous picture may seem surprising given robust directional findings from studies in high-income countries, especially for SES-health gradients. One possibility for this difference is that hierarchy-stress associations produce more consistent health effects in an epidemiological context characterized by chronic, rather than infectious disease. As argued above, our finding that one of the most consistent wealth-health associations was with blood pressure would support this argument, since hypertension is

a risk factor for most chronic disease and consistently associated with SES and inequality in high-income countries (Kim et al., 2008; Shahu et al., 2019), but unlikely to be harmful for most Tsimane (Gurven et al., 2012; Kaplan et al., 2017). However, there are also consistent associations between SES and infectious disease in high-income countries (Aiello et al., 2018; Snyder-Mackler et al., 2020), suggesting that epidemiological context alone does not account for inconsistent results.

Another possible source of heterogeneity is the scale at which relative wealth and inequality are measured. Literature reviews suggest that at an international scale as many as 83% of studies find associations, while in studies of areas the size of neighborhoods, only 45% find associations (Kondo et al., 2012; Pickett and Wilkinson, 2015; Wilkinson and Pickett, 2006). Pickett and Wilkinson (2015) suggest that this heterogeneity reflects the scale at which inequality is perceived as most salient. Here we assessed relative wealth and inequality at the scale of the residential community, a salient arena of daily cooperation and competition (Alami et al., 2020; Gurven et al., 2015, 2008b; Jaeggi et al., 2016; von Rueden et al., 2008, 2014). This local level is also similar in scale to group-level hierarchies in other social species that show hierarchy-associated stress responses (Sapolsky, 2005; Snyder-Mackler et al., 2016; Tung et al., 2012). Furthermore, substituting community-relative wealth with wealth relative to the whole Tsimane population made little difference for results (Supplementary file 1a-1m), suggesting that the choice of scale within this relatively small-scale society did not matter. Modern technologies, such as television, may upset these comparisons and the functioning of hierarchy-related adaptations, by making the global seem local; however few Tsimane have regular access to television and other media. Nevertheless, it is possible that at least some Tsimane perceive inequality in reference to the local non-Tsimane population, or other regions of Bolivia, which was not captured by our study. Interacting with members of the dominant culture can be a source of stress (Dressler, 1999; Konečná and Urlacher, 2017; von Rueden et al., 2014), even if the Tsimane are arguably doing fairly well financially compared to other rural Bolivians (Godoy et al., 2007). Thus, we might not have been able to

capture a relevant scale of comparison for some people, which could explain why associations at other scales were less consistent. However, this argument also applies to studies in high-income countries – where the relevant scales could be anything from neighborhoods to countries – and doesn't necessarily explain why results were inconsistent (as opposed to simply weaker) when measured at a less salient scale.

Finally, another explanation for heterogeneous associations is that our measure of household wealth may capture several distinct dimensions of socio-economic status, with partly orthogonal effects on health. On the one hand, greater wealth affords more respect and influence within communities, which is associated with lower cortisol and better health among the Tsimane (von Rueden et al., 2014) and elsewhere (Decker, 2000); this is likely the dimension captured by our subjective status data. On the other hand, household wealth is accumulated through participation in the market economy, which is associated with greater stress – higher cortisol, blood pressure – among the Tsimane (von Rueden et al., 2014) and elsewhere (Dressler, 1999; Konečná and Urlacher, 2017). The risks of different infectious diseases may also vary along these dimensions, with people who more frequently visit town and interact with outsiders possibly being more exposed to respiratory pathogens (Kaplan et al., 2020). Thus, household wealth may in part be inconsistently associated with health because of these opposing processes.

Several psychosocial variables were directly associated with health. Conflicts and depression were associated with lower BMI and blood pressure, perhaps indicating the effects of stress or lack of access to resources (depression is associated with low productivity among the Tsimane; Stieglitz et al., 2014). Depression and non-social problems were associated with worse self-rated health, again possibly via stress or direct effects of resource availability. However, associations between wealth or inequality and health outcomes were not mediated when including psychosocial variables in models (P3), and there was almost no evidence for indirect effects proceeding through these pathways. An obvious

558 limitation is that our sample sizes for the mediation analysis were smaller than for other analyses
559 (Supplementary file 1q-1s), though most were still large enough to capture any meaningful effect. It is
560 also possible that our measures of psychosocial stress were inadequate, e.g. a single urinary cortisol
561 measure likely captures overall differences in cortisol excretion (Yehuda et al., 2003), but does not
562 capture changes in diurnal cortisol patterns that are typically associated with chronic stress (Garcia et
563 al., 2017; Miller et al., 2007). But the lack of mediation found here may also point to more nuanced
564 mechanisms such as changes in physical activity related to different subsistence strategies, or other
565 lifestyle factors not accounted for here. For subsistence societies experiencing socioeconomic change,
566 whether relative status increases, decreases, or has no effect on stress and health may depend on the
567 status measure and its association with social support. A study of four Tsimane communities found that
568 influential men with greater social support had lower cortisol (von Rueden et al., 2014), but higher cash
569 income associated with higher cortisol (von Rueden et al., 2014; see also Konečná and Urlacher, 2017).
570 In another study of the Tsimane, higher incomes predicted lower BMIs, unless individuals had relatively
571 more social support (Brabec et al., 2007). It therefore remains unclear what mechanisms were
572 responsible for the wealth-health associations found here, though hierarchy is known to affect immune
573 function, and thereby infectious disease morbidity independently of stress and associated HPA activity
574 (Aiello et al., 2018; Miller et al., 2011; Snyder-Mackler et al., 2020, 2016).

575 In sum, we present the most comprehensive test of hierarchy-health associations in a
576 subsistence society to date. In support of an evolutionary argument that conceptualizes hierarchy-
577 health effects as stemming from evolved reaction norms adjusting people's behavior and physiology to
578 the rank and local competitive regime they find themselves in (Daly and Wilson, 1997; Griskevicius et al.,
579 2011; Pepper and Nettle, 2014), we found that wealth and inequality were associated with several
580 health outcomes, though other associations were negligible or in the opposite direction to that
581 predicted. In support of the argument that most hierarchy-health effects in high-income countries are

caused by evolutionary mismatch (Sapolsky, 2004), we found that inequality was associated with blood pressure but in a range unlikely to affect health; however, this association could lead to hypertension, cardiovascular and metabolic disease as inequality further increases due to increased market integration and/or as novel foods and lifestyle factors enter the population (Gurven et al., 2012, 2016; Kaplan et al., 2017; Kraft et al., 2018). Our study thus contributes to an evolutionary approach to public health that considers tradeoffs and mismatch as important links between socio-ecology, lifestyle and health (Eaton et al., 2002; Wells et al., 2017).

Materials and Methods

Data collection and preparation

All data were collected under the auspices of the Tsimane Health and Life History Project (THLHP) (Gurven et al., 2017) by a team of Bolivian medical professionals and Tsimane researchers.

Wealth and wealth inequality.

Wealth data were collected in 2006-2007 and 2013. Here we only included wealth data collected prior to a rare catastrophic flood in February 2014 that destroyed crops and household goods in the vast majority of Tsimane communities (Trumble et al., 2018). Figure 1-figure supplement 1 summarizes how many individuals were included in the sample, out of all individuals ever sampled by the THLHP. Household wealth was assessed through an inventory of commonly owned items including traditional goods, i.e. items manufactured from local organic materials (e.g. canoes, bows and arrows), market goods, i.e. industrially produced items obtained through trade or purchase (e.g. bicycles, motorbikes), and livestock (e.g. pigs, cows), which were subsequently converted into their local market value in Bolivianos and summed (Fig. 1).

Objective household wealth arguably provides only an indirect measure of people's subjective wealth and status (Norton, 2013) but these data were most widely available for this study. Furthermore, household wealth correlated significantly, albeit weakly, with subjective status (Amir et al., 2019; Woolard et al., 2019; $r=0.17$, $df=147$, $P<0.05$) and subjective wealth rank ($r=0.29$, $df=150$, $P<0.001$). Previous work among the Tsimane (Undurraga et al., 2016) has also shown that more visible forms of wealth, such as the household items counted here, influenced subjective health more than less visible forms of wealth, such as the size of cultivated fields. To prevent differences in age sampling between villages from affecting wealth and inequality estimates, we followed Borgerhoff Mulder et al (2009) and adjusted wealth values for the age of the head of household by fitting generalized additive models for location scale and shape (GAMLSS) to the distribution of wealth-by-age to obtain wealth-by-age z-scores. Wealth z-scores derived from GAMLSS, representing centile values, were used in all analyses in part because wealth was skewed in distribution, and also expected to have diminishing returns at higher values (i.e. 100 Bolivianos are worth more to a poor individual than a wealthy one). However, to determine whether z-scoring with GAMLSS altered results by normalizing the shape of the wealth distribution, we also repeated analyses with standardized wealth (i.e. [household wealth – population-average wealth] / standard deviation of population-average wealth), which preserves the skew. There were no qualitative differences in inference between the two methods, largely because z-scoring with GAMLSS primarily affects outliers on the far high end of the distribution. Note that "z-score" can have two slightly different meanings; for wealth and BMI (see below), we generally mean centile values from GAMLSS unless otherwise noted, for all other variables Z scores refer simply to standardized values (i.e. $[x - mean(x)] / \mathrm{sd}(x)$).

Mean wealth and wealth inequality at the community level (for communities with >=9 households) were calculated after converting wealth Z-scores back into equivalent values in Bolivianos at age 50 (see Fig. 1). We used the Gini coefficient to measure inequality; other inequality measures (e.g.

median share, 90/10 ratio) generally correlate highly (r>0.94) with Gini (Kondo et al., 2009) and were therefore not considered. In other studies, local scales of measuring inequality, such as at the community level used here, tend to produce smaller effects on health than those at larger scales, such as states or countries (Kondo et al., 2009; Wilkinson and Pickett, 2006). In the Tsimane context, it is unclear whether that will be the case given low residential mobility and concentration of work and socializing within communities. However, Tsimane visit other communities and sporadically engage in market-based interaction with non-Tsimane, and comparisons with wealthier neighbors can contribute to Tsimane status aspirations (Schultz, 2019). Nevertheless, as mentioned above (Study population), we consider the community to be the most relevant arena for status competition among Tsimane (though substituting community-relative wealth with population-relative wealth made little difference; see Supplementary file 1a-1m). Note that most studies on health effects of inequality use *income* inequality (but see Nowatzki, 2012), which is less unequally distributed than wealth. Cash income among the Tsimane during this study period was sporadic and many households may have no income in a given sampling period, which leads to overestimated Ginis. We therefore preferred wealth and wealth inequality as a more reliable measure of households' long-term access to resources and its distribution.

Psychological, social, and health variables.

The THLHP has been recording biomedical and anthropological data during roughly annual medical examinations and interviews by THLHP physicians and research assistants, on an increasing number of communities since 2002. Here we included any data collected within two years of an individual's wealth data, i.e. the potential range of data was 2004-2009 and 2011-2015. Table 1 summarizes how many individuals out of all the ones with wealth data (see also Figure 1-figure supplement 1) were included for each outcome variable.

Depressive symptoms were measured using an adapted 18-item questionnaire (Stieglitz et al., 2014), the responses to which were summed to yield an overall depression score. The same interview also asked whether participants experienced conflicts with several kinds of social partners as well as non-social problems, such as food insecurity, illness, or debt; affirmative answers were summed to yield a composite measure of social conflicts and non-social problems, respectively. A household's cooperation network was measured as the number of people from different households who helped in that household's fields in a given year. Cortisol was measured in first-morning urine using enzyme-linked immunosorbent assays and corrected for specific gravity (see von Rueden et al., 2014 for details). Body mass index (BMI) Z-scores were calculated by GAMLSS using Tsimane-specific growth curves (Blackwell et al., 2016b) (R package at: www.github.com/adblackwell/localgrowth) as well as the total distribution of Tsimane adult BMIs, representing deviations from the local population average for a given age and sex. Diastolic and systolic blood pressure were measured by THLHP physicians using an aneroid sphygmomanometer. Self-rated general health was measured using a five-point scale from ("very bad" [1] to "excellent" [5]). Morbidity at the time of the medical check-up was assessed by physicians using the ICD-10 classification (International Classification of Disease, 10th edition) and then grouped into 18 clinically meaningful categories following the Clinical Classifications System (https://www.hcup-us.ahrq.gov/toolssoftware/ccs/ccsfactsheet.jsp); morbidities in any of these categories were summed to give a total morbidity score potentially ranging from 0 (no morbidities) to 18 (at least one morbidity in each category). In addition, we also examined the presence/absence of infectious and parasitic diseases (CCS 1, hereafter "infections"), diseases of the respiratory system (CCS 8, "respiratory illness") and diseases of the digestive system (CCS 9, "gastrointestinal illness"), which represent the most common causes of morbidity and mortality in this population (Gurven et al., 2020, 2007). See Table 2 for examples of the six most common diagnoses in these three categories. Distance to the town of San Borja was measured as nearest route (whether by river or road) from the center of the community and

provides a proxy for access to modern amenities. Community size and household size were summarized from complete population censuses conducted regularly by the THLHP. Thus, they include all individuals, not just those sampled for wealth or other covariates.

Data analysis

Prior to analysis, all variables except binary variables were standardized into Z-scores. Urinary cortisol was log transformed prior to standardization to reduce skew, as is common practice. All outcomes were modeled as Gaussian, except the presence/absence of specific morbidities (Bernoulli). Each analysis modeled an individual-level outcome as a function of individual-, household-, and community-level characteristics (Table 1). Thus, we fit the following base model for each outcome:

$$\text{Outcome}_{ijkl} \sim \beta_0 + (\beta_1 * \text{Sex}_j) + (\beta_2 * \text{Age}_j) + (\beta_3 * \text{relative household wealth}_k) + (\beta_4 * \text{Community-level Gini}_l) + (\beta_5 * \text{Community-level mean wealth}_l) + (\beta_6 * \text{Community Size}_l) + (\beta_7 * \text{Distance of community to market town}_l) + (\beta_8 * \text{Household Size}_l)\ u_j + u_k + u_l + e_{ijkl}$$

wherein the subscripts denote measurement *i*, individual *j*, household *k*, and community *l*, respectively. β_0 is the intercept, all other β's are slopes, *u*'s are random intercepts, and *e* is the residual error (not available for Bernoulli responses). Variance inflation factors indicated virtually no collinearity among predictors (all VIFs <3).

In order to test whether potential wealth-health associations were mediated by psychosocial stress we re-ran all health models (blood pressure, self-rated health, total morbidity, infections, respiratory and gastrointestinal illness) with pertinent psychosocial variables as covariates and used the *mediation* function in the *sjstats* package (Lüdecke, 2021) to estimate direct and indirect effect. In addition, we also ran a series of exploratory analyses in which we added interaction terms.

We used Bayesian multilevel models fit with the *brms* package v. 2.13.5. (Bürkner, 2017) in R 4.0.2. for all analyses. All models used regularizing priors (fixed effects: normal, mean=0, SD=1; random effects: half-cauchy, location=0, scale=2) which imposes conservatism on parameter estimates and reduces the risk of inferential errors (Gelman et al., 2013; McElreath, 2020). All models converged well as assessed by inspecting trace plots and standard diagnostics (all Rhat =<1.01). All data and R code are available at https://doi.org/10.5281/zenodo.4567498 with any updates at https://github.com/adblackwell/wealthinequality.

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References

Abbott DH, Keverne EB, Bercovitch FB, Shively C a., Mendoza SP, Saltzman W, Snowdon CT, Ziegler TE, Banjevic M, Garland T, Sapolsky RM. 2003. Are subordinates always stressed? A comparative analysis of rank differences in cortisol levels among primates. *Horm Behav* **43**:67–82. doi:10.1016/S0018-506X(02)00037-5

Aiello AE, Simanek AM, Stebbins RC, Dowd JB. 2018. Psychosocial influences on infectious disease. *Routledge Int Handb Psychosoc Epidemiol*.

Alami S, Rueden C Von, Seabright E, Kraft TS, Blackwell AD, Stieglitz J, Kaplan HS, Gurven MD, von Rueden CR, Blackwell AD, Seabright E, Kraft TS, Kaplan HS, Gurven MD. 2020. Mother's social status is associated with child health in a horticulturalist population. *Proc R Soc B - Biol Sci* **287**:20192783.

Amir D, Valeggia C, Srinivasan M, Sugiyama LS, Dunham Y. 2019. Measuring subjective social status in children of diverse societies. *PLoS One* **14**:e0226550. doi:10.1371/journal.pone.0226550

Anderson C, Kraus MW, Galinsky AD, Keltner D. 2012. The Local-Ladder Effect: Social Status and Subjective Well-Being. *Psychol Sci* **23**:764–771. doi:10.1177/0956797611434537

Baron RM, Kenny DA. 1986. The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *J Pers Soc Psychol* **51**. doi:10.1037//0022-3514.51.6.1173

Blackwell AD, Martin M, Kaplan H, Gurven M. 2013. Antagonism between two intestinal parasites in humans: the importance of co-infection for infection risk and recovery dynamics. *Proc R Soc B* **280**:20131671. doi:10.1098/rspb.2013.1671

Blackwell AD, Trumble BC, Maldonado Suarez I, Stieglitz J, Beheim B, Snodgrass JJ, Kaplan H, Gurven M. 2016a. Immune function in Amazonian horticulturalists. *Ann Hum Biol* **43**:382–396.

744 doi:10.1080/03014460.2016.1189963

745 Blackwell AD, Urlacher SS, Beheim BA, von Rueden C, Jaeggi A V., Stieglitz J, Trumble BC, Gurven MD,
746 Kaplan HS. 2016b. Growth references for Tsimane forager-horticulturalists of the Bolivian Amazon.
747 *Am J Phys Anthropol* **162**:441–461. doi:10.1002/ajpa.23128

748 Borgerhoff Mulder M, Bowles S, Hertz T, Bell A, Beise J, Clark G, Fazzio I, Gurven M, Hill K, Hooper PL,
749 Irons W, Kaplan H, Leonetti D, Low B, Marlowe F, Mcelreath R, Naidu S, Nolin D, Piraino P, Quinlan
750 R, Schniter E, Sear R, Shenk M, Smith EA, von Rueden C, Wiessner P. 2009. Intergenerational
751 wealth transmission and the dynamics of inequality in small-scale societies. *Science (80-)* **326**:682–
752 688. doi:10.1126/science.1178336

753 Brabec M, Godoy RA, Reyes-García V, Leonard WR. 2007. BMI, income, and social capital in a native
754 Amazonian society: Interaction between relative and community variables. *Am J Hum Biol* **19**:459–
755 474.

756 Bürkner P-C. 2017. brms: An R package for Bayesian multilevel models using Stan. *J Stat Softw* **80**.

757 Chen E, Miller GE. 2013. Socioeconomic Status and Health: Mediating and Moderating Factors. *Annu Rev*
758 *Clin Psychol*. doi:10.1146/annurev-clinpsy-050212-185634

759 Cowlishaw G, Dunbar R. 1991. Dominance rank and mating success in male primates. *Anim Behav*
760 **41**:1045–1056.

761 Daly M. 2016. Killing the competition: Economic inequality and homicide. New York: Routledge.
762 doi:10.4324/9780203787748

763 Daly M, Wilson M. 1997. Life expectancy, economic inequality, homicide, and reproductive timing in
764 Chicago neighborhoods. *Br Med J* **314**:1271–2174.

765 Decker SA. 2000. Salivary cortisol and social status among Dominican men. *Horm Behav* **38**:29–38.
766 doi:10.1006/HBEH.2000.1597

767 Dinkel KA, Costa ME, Kraft TS, Stieglitz J, Cummings DK, Gurven M, Kaplan H, Trumble BC. 2020.
768 Relationship of sanitation, water boiling, and mosquito nets to health biomarkers in a rural
769 subsistence population. *Am J Hum Biol* **32**:1–13. doi:10.1002/ajhb.23356

770 Dressler WW. 1999. Modernization, stress, and blood pressure: New directions in research. *Hum Biol* **71**.

771 Eaton S.Boyd, Strassman BI, Nesse RM, Neel J V, Ewald PW, Williams GC, Weder AB, Eaton Stanley B,
772 Lindeberg S, Konner MJ, Mysterud I, Cordain L. 2002. Evolutionary health promotion. *Prev Med*
773 *(Baltim)* **34**:109–118. doi:10.1006/pmed.2001.0876

774 Eaton SB, Konner M, Shostak M. 1988. Stone agers in the fast lane: chronic degenerative diseases in
775 evolutionary perspective. *Am J Med* **84**:739–749. doi:10.1016/0002-9343(88)90113-1

776 Ecob R, Davey Smith G. 1999. Income and health: what is the nature of the relationship? *Soc Sci Med*
777 **48**:693–705. doi:10.1016/S0277-9536(98)00385-2

778 Ettehad D, Emdin CA, Kiran A, Anderson SG, Callender T, Emberson J, Chalmers J, Rodgers A, Rahimi K.
779 2016. Blood pressure lowering for prevention of cardiovascular disease and death: A systematic
780 review and meta-analysis. *Lancet*. doi:10.1016/S0140-6736(15)01225-8

781 Garcia AR, Gurven MD, Blackwell AD. 2017. A matter of perception: perceived socio-economic status
782 and cortisol on the island of Utila, Honduras. *Am J Hum Biol*. doi:10.1002/ajhb.23031

783 Gelman A, Carlin JB, Stern HS, Dunson DB, Vehtari A, Rubin DB. 2013. Bayesian data analysis, 3rd editio.
784 ed. New York: Chapman and Hall.

785 Gelman A, Goodrich B, Gabry J, Vehtari A. 2019. R-squared for Bayesian regression models. *Am Stat*

786 **73**:307–309.

787 Gluckman PD, Beedle AS, Buklijas T, Low F, Hanson M. 2016. Principles of evolutionary medicine. Oxford:
788 Oxford Unniversity Press.

789 Godoy R a., Reyes-García V, McDade T, Huanca T, Leonard WR, Tanner S, Vadez V. 2006. Does village
790 inequality in modern income harm the psyche? Anger, fear, sadness, and alcohol consumption in a
791 pre-industrial society. *Soc Sci Med* **63**:359–372. doi:10.1016/j.socscimed.2006.01.021

792 Godoy R, Byron E, Reyes-García V, Vadez V, Leonard WR, Apaza L, Huanca T, Pérez E, Wilkie D. 2005.
793 Income inequality and adult nutritional status: Anthropometric evidence from a pre-industrial
794 society in the Bolivian Amazon. *Soc Sci Med* **61**:907–919. doi:10.1016/j.socscimed.2005.01.007

795 Godoy R, Reyes-Garcia V, Huanca T, Leonard WR, McDade T, Tanner S, Seyfried C. 2007. On the measure
796 of income and the economic unimportance of social capital: Evidence from a native Amazonian
797 society of farmers and foragers. *J Anthropol Res* **63**:239–260.

798 Gravlee CC. 2009. How race becomes biology: Embodiment of social inequality. *Am J Phys Anthropol*
799 **139**:47–57. doi:10.1002/ajpa.20983

800 Griskevicius V, Tybur JM, Delton AW, Robertson TE. 2011. The influence of mortality and socioeconomic
801 status on risk and delayed rewards: A life history theory approach. *J Pers Soc Psychol* **100**:1015–
802 1026. doi:10.1037/a0022403

803 Gurven M, Allen-Arave W, Hill K, Hurtado M. 2000. “It’s a Wonderful Life”: signaling generosity among
804 the Ache of Paraguay. *Evol Hum Behav* **21**:263–282.

805 Gurven M, Blackwell AD, Rodríguez DE, Stieglitz J, Kaplan H. 2012. Does blood pressure inevitably rise
806 with age? Longitudinal evidence among forager-horticulturalists. *Hypertension* **60**:25–33.
807 doi:10.1161/HYPERTENSIONAHA.111.189100

808 Gurven M, Jaeggi A V., Kaplan H, Cummings D. 2013. Physical activity and modernization among Bolivian
809 Amerindians. *PLoS One* **8**:e55679. doi:10.1371/journal.pone.0055679

810 Gurven M, Jaeggi A V, Von Rueden CR, Hooper PL, Kaplan H. 2015. Does market integration buffer risk,
811 erode traditional sharing practices, and increase inequality? A test among Bolivian forager-farmers.
812 *Hum Ecol* **43**:515–530. doi:10.1007/s10745-015-9764-y

813 Gurven M, Kaplan H, Stieglitz J, Trumble B, Blackwell AD, Beheim B, Hooper P. 2017. The Tsimane Health
814 and Life History Project: Integrating anthropology and biomedicine. *Evol Anthropol* **26**:54–73.
815 doi:10.1002/evan.21515

816 Gurven M, Kaplan H, Winking J, Finch C, Crimmins EM. 2008a. Aging and inflammation in two
817 epidemiological worlds. *J Gerontol A Biol Sci Med Sci* **63**:196–9.

818 Gurven M, Kaplan H, Zelada Supa A. 2007. Mortality experience of Tsimane Amerindians of Bolivia:
819 Regional variation and temporal trends. *Am J Hum Biol* **19**:376–398. doi:10.1002/ajhb

820 Gurven M, Kaplan HS. 2007. Longevity among hunter-gatherers: a cross-cultural examination. *Popul Dev*
821 *Rev* **33**:321–365.

822 Gurven M, Kraft TS, Alami S, Adrian JC, Linares EC, Cummings D, Rodriguez DE, Hooper PL, Jaeggi AV,
823 Gutierrez RQ, Suarez IM, Seabright E, Kaplan H, Stieglitz J, Trumble B. 2020. Rapidly declining body
824 temperature in a tropical human population. *Sci Adv* **6**:eabc6599. doi:10.1126/sciadv.abc6599

825 Gurven M, Zanolini A, Schniter E. 2008b. Culture sometimes matters: Intra-cultural variation in pro-
826 social behavior among Tsimane Amerindians. *J Econ Behav Organ* **67**:587–607.

827 Gurven MD, Kaplan H, Winking J, Eid Rodriguez D, Vasunilashorn S, Kim JK, Finch C, Crimmins E. 2009.
828 Inflammation and infection do not promote arterial aging and cardiovascular disease risk factors
829 among lean horticulturalists. *PLoS One* **4**:e6590. doi:10.1371/journal.pone.0006590

Gurven MD, Trumble BC, Stieglitz J, Blackwell AD, Michalik DE, Finch CE, Kaplan HS. 2016. Cardiovascular disease and type 2 diabetes in evolutionary perspective: A critical role for helminths? *Evol Med Public Heal* **2016**:338–357. doi:10.1093/emph/eow028

Hagen EH. 2011. Evolutionary theories of depression: A critical review. *Can J Psychiatry* **56**:716–726. doi:10.1177/070674371105601203

Hooper PL, Gurven M, Winking J, Kaplan HS. 2015. Inclusive fitness and differential productivity across the life course determine intergenerational transfers in a small-scale human society. *Proc R Soc B - Biol Sci* **282**:20142808.

Jaeggi A V, Hooper PL, Beheim BA, Kaplan HS, Gurven MD. 2016. Reciprocal exchange patterned by market forces helps explain cooperation in a small-scale society. *Curr Biol* **26**:2180–2187. doi:10.1016/j.cub.2016.06.019

Johnson SL, Leedom LJ, Muhtadie L. 2012. The dominance behavioral system and psychopathology: Evidence from self-report, observational, and biological studies. *Psychol Bull* **138**:692–743.

Kaplan H, Thompson RC, Trumble BC, Wann LS, Allam AH, Beheim B, Frohlich B, Sutherland ML, Sutherland JD, Stieglitz J, Rodriguez DE, Michalik DE, Rowan CJ, Lombardi GP, Bedi R, Garcia AR, Min JK, Narula J, Finch CE, Gurven M, Thomas GS. 2017. Coronary atherosclerosis in indigenous South American Tsimane: a cross-sectional cohort study. *Lancet* **15**:1–9. doi:10.1016/S0140-6736(17)30752-3

Kaplan HS, Hooper P, Gurven M. 2009. The evolutionary and ecological roots of human social organization. *Philos Trans R Soc B - Biol Sci* **364**:3289–3299. doi:10.1098/rstb.2009.0115

Kaplan HS, Trumble BC, Stieglitz J, Mendez Mamany R, Gutierrez Cayuba M, Maito Moye L, Alami S, Kraft T, Quispe Gutierrez R, Copajira Adrian J, Thompson RC, Thomas GS, Michalik DE, Eid Rodriguez D,

Gurven MD. 2020. Voluntary collective isolation as a best response to COVID-19 for indigenous populations? A case study and protocol from the Bolivian Amazon. *Lancet* **6736**:1–8. doi:10.1016/S0140-6736(20)31104-1

Kim D, Kawachi I, Hoorn S Vander, Ezzati M. 2008. Is inequality at the heart of it? Cross-country associations of income inequality with cardiovascular diseases and risk factors. *Soc Sci Med* **66**. doi:10.1016/j.socscimed.2007.12.030

Kondo N, Sembajwe G, Kawachi I, van Dam RM, Subramanian S V, Yamagata Z. 2009. Income inequality, mortality, and self rated health: meta-analysis of multilevel studies. *BMJ* **339**:b4471. doi:10.1136/bmj.b4471

Kondo N, van Dam RM, Sembajwe G, Subramanian S V., Kawachi I, Yamagata Z. 2012. Income inequality and health: The role of population size, inequality threshold, period effects and lag effects. *J Epidemiol Community Health* **66**. doi:10.1136/jech-2011-200321

Konečná M, Urlacher SS. 2017. Male social status and its predictors among Garisakang forager-horticulturalists of lowland Papua New Guinea. *Evol Hum Behav* **38**:789–797. doi:10.1016/J.EVOLHUMBEHAV.2017.05.005

Kraft TS, Stieglitz J, Trumble BC, Martin M, Kaplan H, Gurven M. 2018. Nutrition transition in 2 lowland Bolivian subsistence populations. *Am J Clin Nutr* 1–13. doi:10.1093/ajcn/nqy250

Kreft IGG, de Leeuw J, Aiken LS. 1995. The Effect of Different Forms of Centering in Hierarchical Linear Models. *Multivariate Behav Res* **30**:1–21. doi:10.1207/s15327906mbr3001_1

Kunz-Ebrecht SR, Kirschbaum C, Marmot M, Steptoe A. 2004. Differences in cortisol awakening response on work days and weekends in women and men from the Whitehall II cohort. *Psychoneuroendocrinology* **29**:516–528. doi:10.1016/S0306-4530(03)00072-6

874 Lea AJ, Tung J, Archie EA, Alberts SC. 2017. Developmental plasticity. *Evol Med Public Heal* **2017**:162–
875 175. doi:10.1093/emph/eox019

876 Lüdecke D. 2021. sjstats: Statistical Functions for Regression Models (Version 0.18.1).
877 doi:10.5281/zenodo.1284472

878 Luttmer EFP. 2005. Neighbors as negatives: Relative earnings and well-being. *Q J Econ* **120**:963–1002.
879 doi:10.1162/003355305774268255

880 Lynch J, Smith GD, Harper S, Hillemeier M, Ross N, Kaplan G a, Wolfson M. 2004. Is income inequality a
881 determinant of population health? Part 1. A systematic review. *Milbank Q* **82**:5–99.

882 Macinko JA, Shi L, Starfield B, Wulu JT. 2003. Income inequality and health: A critical review of the
883 literature. *Med Care Res Rev* **60**:407–452. doi:10.1177/1077558703257169

884 MacKinnon DP, Fairchild AJ, Fritz MS. 2007. Mediation analysis. *Annu Rev Psychol* **58**:593–614.
885 doi:10.1146/annurev.psych.58.110405.085542

886 Marmot MG, Stansfeld S, Patel C, North F, Head J, White I, Brunner E, Feeney A, Smith GD. 1991. Health
887 inequalities among British civil servants: the Whitehall II study. *Lancet* **337**:1387–1393.
888 doi:10.1016/0140-6736(91)93068-K

889 Mattison SM, Smith EA, Shenk MK, Cochrane EE. 2016. The evolution of inequality. *Evol Anthropol*
890 *Issues, News, Rev* **25**:184–199. doi:10.1002/evan.21491

891 McDade TW. 2005. THE ECOLOGIES OF HUMAN IMMUNE FUNCTION. *Annu Rev Anthropol* **34**:495–521.
892 doi:10.1146/annurev.anthro.34.081804.120348

893 McDade TW, Georgiev A V, Kuzawa CW. 2016. Trade-offs between acquired and innate immune
894 defenses in humans. *Evol Med public Heal* **2016**:1–16. doi:10.1093/emph/eov033

McElreath R. 2020. Statistical rethinking: A Bayesian course with examples in R and Stan, 2nd editio. ed. Boca Raton, FL: CRC Press.

Miller AH, Raison CL. 2016. The role of inflammation in depression: From evolutionary imperative to modern treatment target. *Nat Rev Immunol* **16**:22–34. doi:10.1038/nri.2015.5

Miller GE, Chen E, Fok AK, Walker H, Lim A, Nicholls EF, Cole S, Kobor MS. 2009. Low early-life social class leaves a biological residue manifested by decreased glucocorticoid and increased proinflammatory signaling. *Proc Natl Acad Sci U S A* **106**:14716–21. doi:10.1073/pnas.0902971106

Miller GE, Chen E, Parker KJ. 2011. Psychological stress in childhood and susceptibility to the chronic diseases of aging: moving toward a model of behavioral and biological mechanisms. *Psychol Bull* **137**:959–97. doi:10.1037/a0024768

Miller GE, Chen E, Zhou ES. 2007. If it goes up, must it come down? Chronic stress and the hypothalamic-pituitary-adrenocortical axis in humans. *Psychol Bull*. doi:10.1037/0033-2909.133.1.25

Miller GE, Murphy MLM, Cashman R, Ma R, Ma J, Arevalo JMG, Kobor MS, Cole SW. 2014. Greater inflammatory activity and blunted glucocorticoid signaling in monocytes of chronically stressed caregivers. *Brain Behav Immun* **41**:191–199. doi:10.1016/j.bbi.2014.05.016

Nesse RM. 2000. Is depression an adaptation? *Arch Gen Psychiatry* **57**:14–20.

Nesse RM, Williams GC. 1994. Why we get sick: The new science of Darwinian medicine. New York: Vintage.

Norton MI. 2013. All ranks are local: Why humans are both (painfully) aware and (surprisingly) unaware of their lot in life. *Psychol Inq* **24**:124–125. doi:10.1080/1047840X.2013.794689

Nowatzki NR. 2012. Wealth inequality and health: A political economy perspective. *Int J Heal Serv*

916 **42**:403–424. doi:10.2190/HS.42.3.c

917 Oakes TW, Syme SL, Feldman R, Friedman GD, Siegelaub AB, Collen MF. 1973. Social factors in newly
918 discovered elevated blood pressure. *J Health Soc Behav* **14**:198. doi:10.2307/2137111

919 Pepper G V, Nettle D. 2014. Socioeconomic disparities in health behaviour: An evolutionary perspective
920 In: Gibson MA, Lawson DW, editors. Applied Evolutionary Anthropology2. New York: Springer. pp.
921 225–243.

922 Pickett KE, Wilkinson RG. 2015. Income inequality and health: A causal review. *Soc Sci Med* **128**:316–
923 326. doi:10.1016/j.socscimed.2014.12.031

924 Pontzer H, Wood BM, Raichlen DA. 2018. Hunter-gatherers as models in public health. *Obes Rev* **19**:24–
925 35. doi:10.1111/obr.12785

926 Pusey AE, Williams J, Goodall J. 1997. The influence of dominance rank on the reproductive success of
927 female chimpanzees. *Science (80-)* **277**:828–831. doi:10.1126/science.277.5327.828

928 Quon EC, McGrath JJ. 2014. Subjective socioeconomic status and adolescent health: A meta-analysis.
929 *Heal Psychol* **33**:433–447. doi:10.1037/a0033716

930 Sapolsky RM. 2005. The influence of social hierarchy on primate health. *Science (80-)* **308**:648–52.
931 doi:10.1126/science.1106477

932 Sapolsky RM. 2004. Why Zebras don't get ulcers, 3rd ed. New York: Holt.

933 Schultz AF. 2019. Status determinants, social incongruity and economic transition: Gender, relative
934 material wealth and heterogeneity in the cultural lifestyle of forager-horticulturalists. *PLoS One*
935 **14**:e0220432. doi:10.1371/journal.pone.0220432

936 Shahu A, Herrin J, Dhruva SS, Desai NR, Davis BR, Krumholz HM, Spatz ES. 2019. Disparities in

Socioeconomic Context and Association With Blood Pressure Control and Cardiovascular Outcomes in ALLHAT. *J Am Heart Assoc* **8**. doi:10.1161/JAHA.119.012277

Shenk MK, Kaplan HS, Hooper PL. 2016. Status competition, inequality, and fertility: implications for the demographic transition. *Phil Trans R Soc B* **371**:20150150. doi:10.1098/rstb.2015.0150

Silk JB. 2003. Practice random acts of aggression and senseless acts of intimidation: The logic of status contests in social groups. *Evol Anthropol Issues, News, Rev* **11**:221–225. doi:10.1002/evan.10038

Snyder-Mackler N, Burger JR, Gaydosh L, Belsky DW, Noppert GA, Campos FA, Bartolomucci A, Yang YC, Aiello AE, Rand AO, Harris KM, Shively CA, Alberts SC, Tung J. 2020. Social determinants of health and survival in humans and other animals. *Science (80-)* **368**:eaax9553. doi:10.1126/science.aax9553

Snyder-Mackler N, Sanz J, Kohn JN, Brinkworth JF, Morrow S, Shaver AO, Grenier J-C, Pique-Regi R, Johnson ZP, Wilson ME, Barreiro LB, Tung J. 2016. Social status alters immune regulation and response to infection in macaques. *Science (80-)* **354**.

Sorlie PD, Backlund E, Keller JB. 1995. US mortality by economic, demographic, and social characteristics: the National Longitudinal Mortality Study. *Am J Public Health* **85**:949–56. doi:10.2105/AJPH.85.7.949

Stieglitz J, Jaeggi A V, Blackwell AD, Trumble BC, Gurven M, Kaplan HS. 2014. Work to live and live to work: Productivity, transfers, and psychological well-being in adulthood and old age In: Weinstein M, Lane MA, editors. Sociality, Hierarchy, Health: Comparative Biodemography. Washington, DC: National Academies Press. pp. 195–220. doi:10.17226/18822

Stieglitz J, Trumble BC, Thompson ME, Blackwell AD, Kaplan H, Gurven M. 2015. Depression as sickness behavior? A test of the host defense hypothesis in a high pathogen population. *Brain Behav Immun*

959 **49**:130–139. doi:10.1016/J.BBI.2015.05.008

960 Stulp G, Sear R, Schaffnit SB, Mills MC, Barrett L. 2016. The Reproductive Ecology of Industrial Societies,
961 Part II: The Association between Wealth and Fertility. *Hum Nat* **27**:445–470. doi:10.1007/s12110-
962 016-9272-9

963 Subramanian S V., Kawachi I. 2004. Income inequality and health: What have we learned so far?
964 *Epidemiol Rev* **26**:78–91. doi:10.1093/epirev/mxh003

965 Sugiyama LS, Sugiyama MS. 2003. Social roles, prestige, and health risk: Social niche specialization as a
966 risk-buffering strategy. *Hum Nat* **14**:165–190. doi:10.1007/s12110-003-1002-4

967 Trumble BC, Stieglitz J, Jaeggi A V, Beheim BA, Schwartz M, Seabright E, Cummings D, Kaplan HS, Gurven
968 M. 2018. Parental hormones are associated with crop loss and family sickness following
969 catastrophic flooding in lowland Bolivia. *Physiol Behav* **193**:101–107.
970 doi:10.1016/j.physbeh.2018.02.028

971 Tung J, Barreiro LB, Johnson ZP, Hansen KD, Michopoulos V, Toufexis D, Michelini K, Wilson ME, Gilad Y.
972 2012. Social environment is associated with gene regulatory variation in the rhesus macaque
973 immune system. *Proc Natl Acad Sci* **109**:6490–6495. doi:10.1073/pnas.1202734109

974 Undurraga EA, Nica V, Zhang R, Mensah IC, Godoy RA. 2016. Individual health and the visibility of village
975 economic inequality: Longitudinal evidence from native Amazonians in Bolivia. *Econ Hum Biol*.
976 doi:10.1016/j.ehb.2016.06.004

977 Undurraga EA, Nyberg C, Eisenberg DTA, Magvanjav O, Reyes-García V, Huanca T, Leonard WR, McDade
978 TW, Tanner S, Vadez V, Godoy R. 2010. Individual wealth rank, community wealth inequality, and
979 self-reported adult poor health: A test of hypotheses with panel data (2002-2006) from native
980 amazonians, bolivia. *Med Anthropol Q* **24**:522–548. doi:10.1111/j.1548-1387.2010.01121.x

van Noordwijk MA, van Schaik CP. 1999. The effects of dominance rank and group size on female lifetime reproductive success in wild long-tailed macaques, Macaca fascicularis. *Primates* **40**:105–30. doi:10.1007/BF02557705

von Rueden C, Alami S, Kaplan H, Gurven M. 2018. Sex differences in political leadership in an egalitarian society. *Evol Hum Behav* **39**:402–411. doi:10.1016/J.EVOLHUMBEHAV.2018.03.005

von Rueden C, Gurven M, Kaplan H. 2008. The multiple dimensions of male social status in an Amazonian society. *Evol Hum Behav* **29**:402–415. doi:10.1016/j.evolhumbehav.2008.05.001

Von Rueden CR, Jaeggi A V. 2016. Men's status and reproductive success in 33 nonindustrial societies: Effects of subsistence, marriage system, and reproductive strategy. *Proc Natl Acad Sci* **113**:10824–10829. doi:10.1073/pnas.1606800113

von Rueden CR, Redhead D, O'Gorman R, Kaplan H, Gurven M. 2019. The dynamics of men's cooperation and social status in a small-scale society. *Proc R Soc B Biol Sci* **286**:20191367. doi:10.1098/rspb.2019.1367

von Rueden CR, Trumble BC, Emery Thompson M, Stieglitz J, Hooper PL, Blackwell AD, Kaplan HS, Gurven M. 2014. Political influence associates with cortisol and health among egalitarian forager-farmers. *Evol Med public Heal* **2014**:122–33. doi:10.1093/emph/eou021

Wagstaff A, van Doorslaer E. 2000. Income inequality and health: What does the literature tell us? *Annu Rev Public Health* **21**:543–67. doi:10.1111/j.1467-8276.2007.01012.x

Wells J, Sear R, Stearns S, Nesse R, Johnstone R. 2017. Evolutionary public health: introducing the concept. *Lancet* **390**:500–509. doi:10.1016/S0140-6736(17)30572-X

Wilkinson RG, Pickett KE. 2006. Income inequality and population health: A review and explanation of the evidence. *Soc Sci Med* **62**:1768–1784. doi:10.1016/j.socscimed.2005.08.036

1003 Wolfson M, Rowe G, Gentleman JF, Tomiak M. 1993. Career earnings and death: A longitudinal analysis
1004 of older Canadian men. *J Gerontol* **48**:S167–S179. doi:10.1093/geronj/48.4.S167

1005 Wood AM, Boyce CJ, Moore SC, Brown GDA. 2012. An evolutionary based social rank explanation of why
1006 low income predicts mental distress: A 17 year cohort study of 30,000 people. *J Affect Disord*
1007 **136**:882–888. doi:10.1016/j.jad.2011.09.014

1008 Woolard K, Garcia AR, Stieglitz J, Kaplan HS, Gurven M, Trumble BC. 2019. Do perceptions of social
1009 status and material wealth associate with biomarkers of health in a relatively egalitarian
1010 population? *Am J Hum Biol* **31**.

1011 Yehuda R, Halligan SL, Yang RK, Guo LS, Makotkine I, Singh B, Pickholtz D. 2003. Relationship between
1012 24-hour urinary-free cortisol excretion and salivary cortisol levels sampled from awakening to
1013 bedtime in healthy subjects. *Life Sci* **73**. doi:10.1016/S0024-3205(03)00286-8

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Tables

Table 1: Overview of study variables and descriptive statistics. For an overview of the sample relative to all people known to the THLHP and at risk of having wealth data see Figure 1-figure supplement 1

Variable	N	Obs	Median	SD	Min	Max
Adult Outcomes: Psychosocial						
Depression [possible range 16-64]	528	670	40.0	7.1	23.0	62.0
Conflicts [possible range 0-4]	342	401	2.0	0.7	0.0	4.0
Labor partners [count] a	304	399	2.0	2.0	1.0	13.0
Non-social problems [possible range 0-7]	339	398	3.0	1.2	0.0	7.0
Urinary cortisol [pg/ml]	588	811	155,191	149,602	93	851,308
Adult Outcomes: Health						
BMI [kg/m^2] b	1,901	5,179	23.3	2.8	16.0	36.6
Systolic blood pressure [mmHg]	1,622	3,195	110.0	12.8	60.0	190.0
Diastolic blood pressure [mmHg]	1,622	3,195	70.0	10.0	24.0	136.0
Self-rated health [1 Excellent- 5 Very Bad]	1,307	2,523	4.0	0.5	1.0	5.0
Total morbidity [possible range 0-18] c	1,306	1,542	2.0	1.1	0.0	5.0
Infections/parasites [yes/no] c	1,306	1,542	25.2%			
Respiratory disease [yes/no] c	1,306	1,542	21.9%			
Gastrointestinal [yes/no] c	1,306	1,542	36.3%			
Adult Predictors						
Age [years]	1,931	5,383	35.0	15.1	16.0	91.0
Sex [0=female, 1=male]	1,931	5,383	46.2			
Juvenile Outcomes: Health						
BMI [kg/m2] b	1,765	4,747	16.6	2.1	10.2	27.6
Total morbidity [count] c	1,423	1,569	1.0	0.8	0.0	4.0
Infections/parasites [yes/no] c	1,423	1,569	13.6%			
Respiratory disease [yes/no] c	1,423	1,569	42.4%			
Gastrointestinal [yes/no] c	1,423	1,569	41.2%			
Juvenile Predictors						
Age [years]	1,772	4,783	7.0	4.1	0.0	15.0
Sex [0=female, 1=male]	1,772	4,783	49.6			
Household Predictors						
Household size	871	1,045	4.0	2.7	1.0	14.0
Household wealth [Bs]	871	1,045	7,675	5,675	386	56,664
Community Predictors						
Community size [Adults >15]	40	55	72.0	81.2	27.0	346.0
Distance to market town [km]	40	55	43.0	44.2	5.0	140.0
Mean community wealth [Bs]	40	55	8,373	2,331	3,930	16,250
Community wealth inequality [Gini]	40	55	0.27	0.07	0.15	0.53

a Reverse coded in analyses to make higher values worse outcomes.

b Whether higher or lower BMI is better is a bit ambiguous: in high-income countries higher BMI is associated with worse health, lower status and greater inequality, whereas in low-income countries the reverse may be true.

c see Table 2 for an overview of the most common morbidities by category.

Table 2: Overview of the most common morbidities. Three of the most common CCS categories (number in parentheses) and the 6 most prevalent diagnoses within each category (in decreasing order down rows, ICD-10 codes in parentheses). Musculoskeletal conditions (CCS 13) were also common but not analyzed independently

Infectious and parasitic diseases (CCS 1)	**Diseases of the respiratory system (CCS 8)**	**Diseases of the digestive system (CCS 9)**
Pediculosis due to Pediculus humanus capitis (B85.0)	Acute nasopharyngitis [common cold] (J00)	Intestinal helminthiasis (B82.0)
Tinea unguium (B35.1)	Acute streptococcal tonsillitis; unspecified (J03.00)	Infectious gastroenteritis and colitis (A09)
Candidiasis of vulva and vagina (B37.3)	Streptococcal pharyngitis (J02.0)	Dyspepsia (K30)
Pediculosis; unspecified (B85.2)	Acute upper respiratory infection; unspecified (J06.9)	Gastro-esophageal reflux disease with esophagitis (K21.0)
Superficial mycosis; unspecified (B36.9)	Acute bronchitis due to Mycoplasma pneumonia (J20.0)	Giardiasis [lambliasis] (A07.1)
Necatoriasis (B76.1)	Bronchopneumonia; unspecified organism (J18.0)	Gastritis; unspecified, without bleeding (K29.70)

Figure legends

Figure 1: Overview of wealth and inequality distributions. A) Mean wealth by age of household head. B) Mean wealth by population-level wealth Z-score. C) Map of study communities (n=40) and mean wealth at the community level. D) Map of community-level wealth inequality. Note: A and B use raw wealth, while C and D are based on age-corrected values. Heat maps in C and D give a rough sense of the distribution; circle size indicates the number of sampled households (range = 9-81). Data for individual villages are not directly shown to protect confidentiality. Yucumo and San Borja are local market towns inhabited by non-Tsimane, Mission is the site of a Catholic mission and the largest Tsimane settlement.

Figure 2: Wealth and inequality posterior parameter values for models with adults (> 15 years). Points are posterior medians and lines are 75% (thick) and 95% (thin) highest posterior density intervals. Numbers in each panel represent the proportion of the posterior distribution that is greater than zero ($P_{>0}$). All models control for age, sex, distance to market town, and community size. Rough categories of dependent variables (psychosocial, continuous health outcomes, and binary health outcomes) are distinguished by rows and colors. For the first two rows, the outcomes are expressed as Z scores, the bottom row as log odds. See supplements 1-3 for predicted associations of household wealth, community wealth, and wealth inequality, respectively.

Figure 1-figure supplement 1: Overview of the sample. *Ever sampled by THLHP* refers to the period potentially included in this study, i.e. up to December 2015; note that this sample includes 92 communities. Target communities (N=40) were those with any wealth data collected during the periods included here (2006/2007, 2013). The main reason why 2075 people who lived in a target community did not have wealth data is likely that no one in their household was available to be interviewed about their assets, most likely because they were temporarily absent from the community (e.g. people sometimes stay near their far-away fields, go on extended hunting trips, or visit town or other communities). The majority of the 681 individuals who lived in a household with wealth data but lacked

age, sex, and data on at least one of the outcome variables were most likely small children and infants who had not yet been sampled in detail. For a further missingness breakdown of the sample by specific outcome variable see Table 1.

Figure 2-figure supplement 1: Predicted conditional effects of relative household wealth on all psychosocial and health outcomes for adults. Lines are posterior means and shaded areas are 95% credible intervals on mean values. Numbers in each panel represent the proportion of the posterior distribution that supports the predicted negative association between wealth and the outcome ($P_{<0}$). All predictions control for age, sex, inequality, distance to market town, community size, and mean community wealth, holding all other variables at the mean, with sex=female. Rough categories of dependent variables (psychosocial, continuous health outcomes, and binary health outcomes) are distinguished by rows and colors. For the first two rows, the outcomes are measured as Z scores, the bottom row as probabilities.

Figure 2-figure supplement 2: Predicted conditional effects of mean community wealth on all psychosocial and health outcomes for adults. Lines are posterior means and shaded areas are 95% credible intervals on mean values. Numbers in each panel represent the proportion of the posterior distribution that supports the predicted negative association between wealth and the outcome ($P_{<0}$). All predictions control for age, sex, inequality, distance to market town, community size, and mean community wealth, holding all other variables at the mean, with sex=female. Rough categories of dependent variables (psychosocial, continuous health outcomes, and binary health outcomes) are distinguished by rows and colors. For the first two rows, the outcomes are measured as Z scores, the bottom row as probabilities.

Figure 2-figure supplement 3: Predicted conditional effects of wealth inequality (Gini coefficients) on all psychosocial and health outcomes for adults. Lines are posterior means and shaded areas are 95%

credible intervals on mean values. Numbers in each panel represent the proportion of the posterior distribution that supports the predicted negative association between wealth and the outcome ($P_{<0}$). All predictions control for age, sex, inequality, distance to market town, community size, and mean community wealth, holding all other variables at the mean, with sex=female. Rough categories of dependent variables (psychosocial, continuous health outcomes, and binary health outcomes) are distinguished by rows and colors. For the first two rows, the outcomes are measured as Z scores, the bottom row as probabilities.

Figure 3: Wealth and inequality posterior parameter values for models with juveniles (≤ 15 years). Points are posterior medians and lines are 75% (thick) and 95% (thin) highest posterior density intervals. Numbers in each panel represent the proportion of the posterior distribution that is greater than zero ($P_{>0}$). All models control for age, sex, distance to market town, and community size. Rough categories of dependent variables (continuous health outcomes and binary health outcomes) are distinguished by rows and colors. For the first row, the outcomes are measured as Z scores, the bottom row as log odds. See supplement 1 for predicted associations of household wealth, community wealth, and wealth inequality.

Figure 3-figure supplement 1: Predicted conditional effects of household wealth, community wealth, and inequality (Gini coefficients) on all health outcomes for juveniles (<15 years). Lines are posterior means and shaded areas are 95% credible intervals on mean values. Numbers in each panel represent the posterior probability, i.e. the proportion of the posterior that supports an association between inequality and the outcome. All predictions control for age, sex, distance to market town, and community size, holding all other variables at the mean, with sex=female. For the first two columns, the outcomes are measured as Z scores, the remainder as probabilities.

Figure 4: Covariate posterior parameter values for models with adults (> 15 years). Points are posterior medians and lines are 75% (thick) and 95% (thin) highest posterior density intervals. Numbers in each

panel represent the proportion of the posterior distribution that is greater than zero ($P_{>0}$). Full models are given in Supplementary file 1a-1m. Rough categories of dependent variables (psychosocial, continuous health outcomes, and binary health outcomes) are distinguished by rows and colors. For the first two rows, the outcomes are measured as Z scores, the bottom row as log odds.

Figure 5: Covariate posterior parameter values for models with juveniles (≤ 15 years). Points are posterior medians and lines are 75% (thick) and 95% (thin) highest posterior density intervals. Numbers in each panel represent the proportion of the posterior distribution that is greater than zero ($P_{>0}$). Full models are given in Supplementary file 1n & 1o. Rough categories of dependent variables (continuous health outcomes, and binary health outcomes) are distinguished by rows and colors. For the first row, the outcomes are measured as Z scores, the bottom row as log odds.

Figure 6. Interactions between sex, wealth, and inequality. Plots show the predicted values for each outcome and Gini z-score. Red shading indicates poorer individuals (wealth z = -2), blue indicates wealthier individuals (z = 2). For each model the proportion of the posterior >0 is shown in the numbers above: GxW = Gini x Wealth; SxG = Sex x Gini; SxW = Sex x Wealth.

Supplementary files:

Supplementary file 1 contains the following tables with additional information on the statistical models.

Supplementary file 1a: Model summary – Depression

Supplementary file 1b: Model summary – Social conflicts

Supplementary file 1c: Model summary – Fewer Labor partners

Supplementary file 1d: Model summary – Non-social problems

Supplementary file 1e: Model summary – Cortisol

Supplementary file 1f: Model summary – BMI

Supplementary file 1g: Model summary – Systolic blood pressure

Supplementary file 1h: Model summary – Diastolic blood pressure

Supplementary file 1i: Model summary – Worse Self-rated health

Supplementary file 1j: Model summary – Total morbidity

Supplementary file 1k: Model summary – Infections

Supplementary file 1l: Model summary – Respiratory illness

Supplementary file 1m: Model summary – Gastrointestinal illness

Supplementary file 1n: Gaussian model summaries for juveniles

Supplementary file 1o: Logistic model summaries for juveniles

Supplementary file 1p: Overview of exploratory interaction effects

Supplementary file 1q: Mediation of wealth effects

Supplementary file 1r: Mediation of inequality effects

Supplementary file 1s: Mediation of mean community wealth effects

Appendix 1: Causal relationships assumed by mediation analysis

The causal relationships between independent variable, mediator and dependent variable assumed by standard mediation analysis are depicted in the DAG below.

insert Appendix 1-figure 1 here

In our case, the independent variables are absolute wealth, relative wealth and inequality, the mediators are the psychosocial variables, and the dependent variables are the health outcomes (see Supplementary file 1q-1s).

The next causal diagram highlights a potential problem of this approach, which treats each mediator separately even though several mediators are present.

insert Appendix 1-figure 2 here

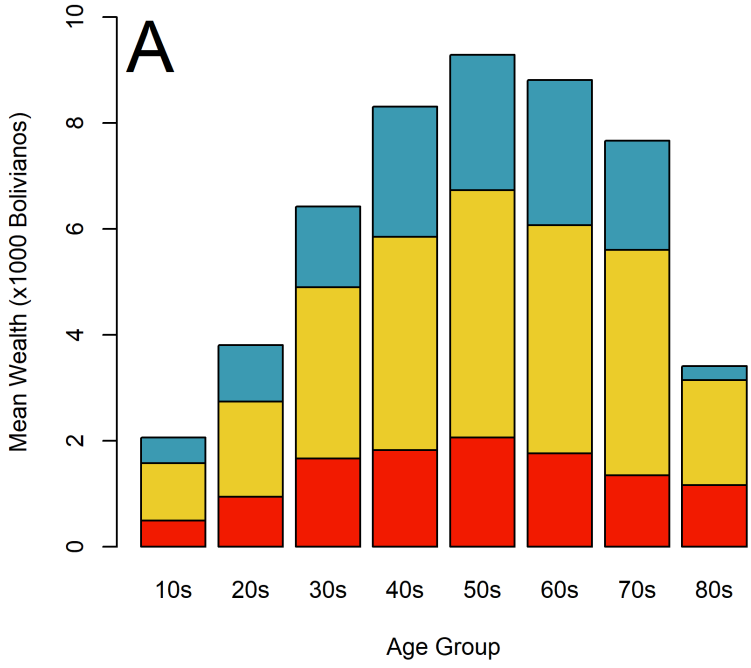
For example it could be that wealth/inequality cause both higher levels of depression and higher levels of cortisol. In this scenario, leaving one mediator out prevents us from accurately estimating the "direct effect" of our independent variable on health outcomes, limiting us only to the total effect absent the mediating pathway.

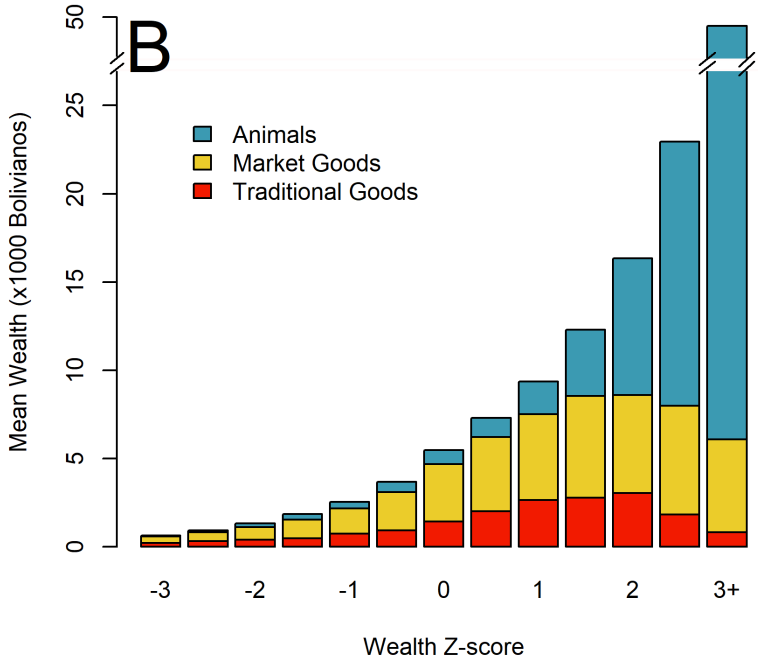
Furthermore, our results could also be influenced by collider bias in the case that different mediating variables are themselves causally linked, as in the following DAG:

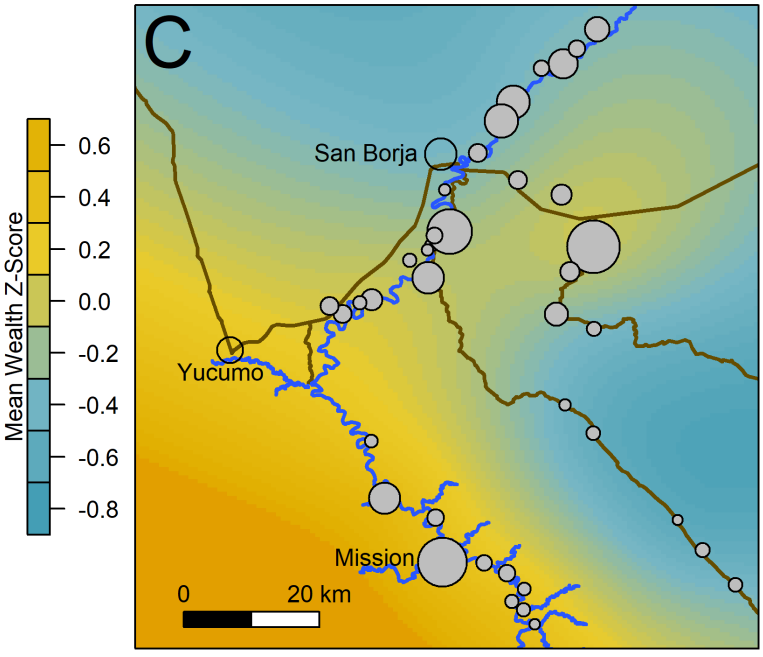
insert Appendix 1-figure 3 here

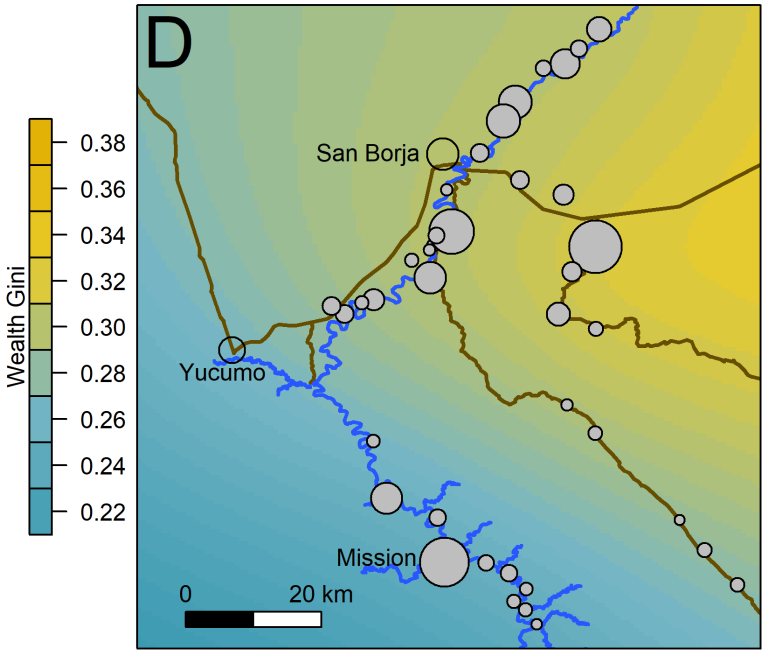
Following the example above, it could be that cortisol also independently causes higher levels of depression (or vice versa). In this situation, the mediating psychosocial variable 1 functions as a collider between wealth / inequality and psychosocial variable 2, and we cannot properly assess the impact of our independent variables on the outcome without conditioning on all mediation variables simultaneously.

Despite these potential problems, we preferred the present approach of treating each mediator independently because including all mediators in the same analysis would have required imputing most of the values, because the sample overlap was small. Furthermore, we have good reasons to believe that this approach would not change inference. Namely, in a previous version of the mediation analysis (https://www.medrxiv.org/content/10.1101/2020.06.11.20121889v1) we performed a PCA on all psychosocial variables and found that they were relatively uncorrelated, as the main PC only contained depression loadings. Furthermore, we had included depression, non-social problems, and cortisol (with imputation) in the same models with the same inference as in the present version of the analysis: there was no convincing evidence for mediation.


A
Mean Wealth (x1000 Bolivianos)
0
2
4
6
8
10
10s
20s
30s
40s
50s
60s
70s
80s
Age Group


B
Mean Wealth (x1000 Bolivianos)
0
5
10
15
20
25
50
Animals
Market Goods
Traditional Goods
-3
-2
-1
0
1
2
3+
Wealth Z-score


C
Mean Wealth Z-Score
0.6
0.4
0.2
0.0
-0.2
-0.4
-0.6
-0.8
San Borja
Yucumo
Mission
0
20 km


D
Wealth Gini
0.38
0.36
0.34
0.32
0.30
0.28
0.26
0.24
0.22
San Borja
Yucumo
Mission
0
20 km

Ever sampled by THLHP: 12197 individuals

↓

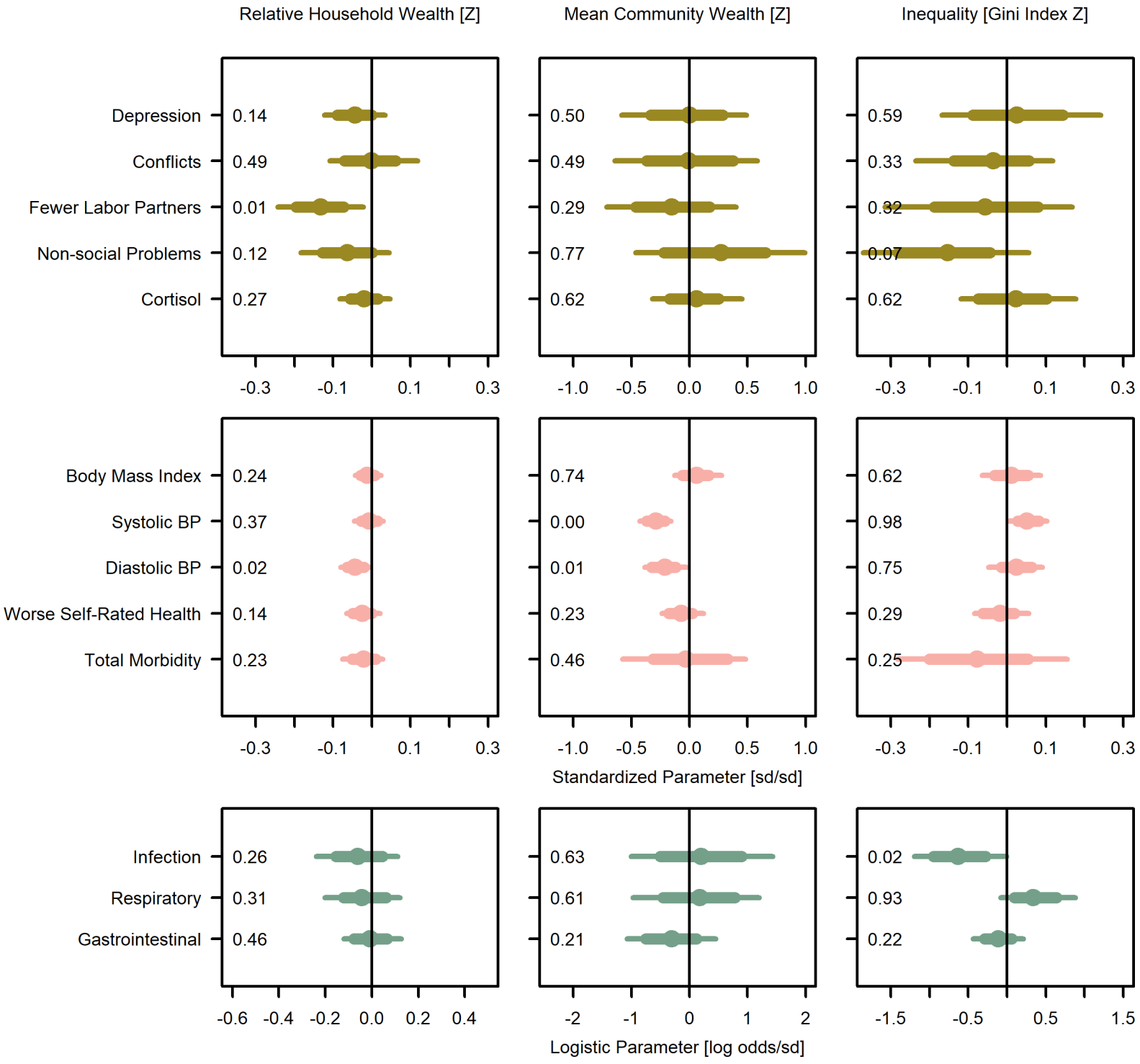
Lives in a community targeted for wealth data: 6310 individuals

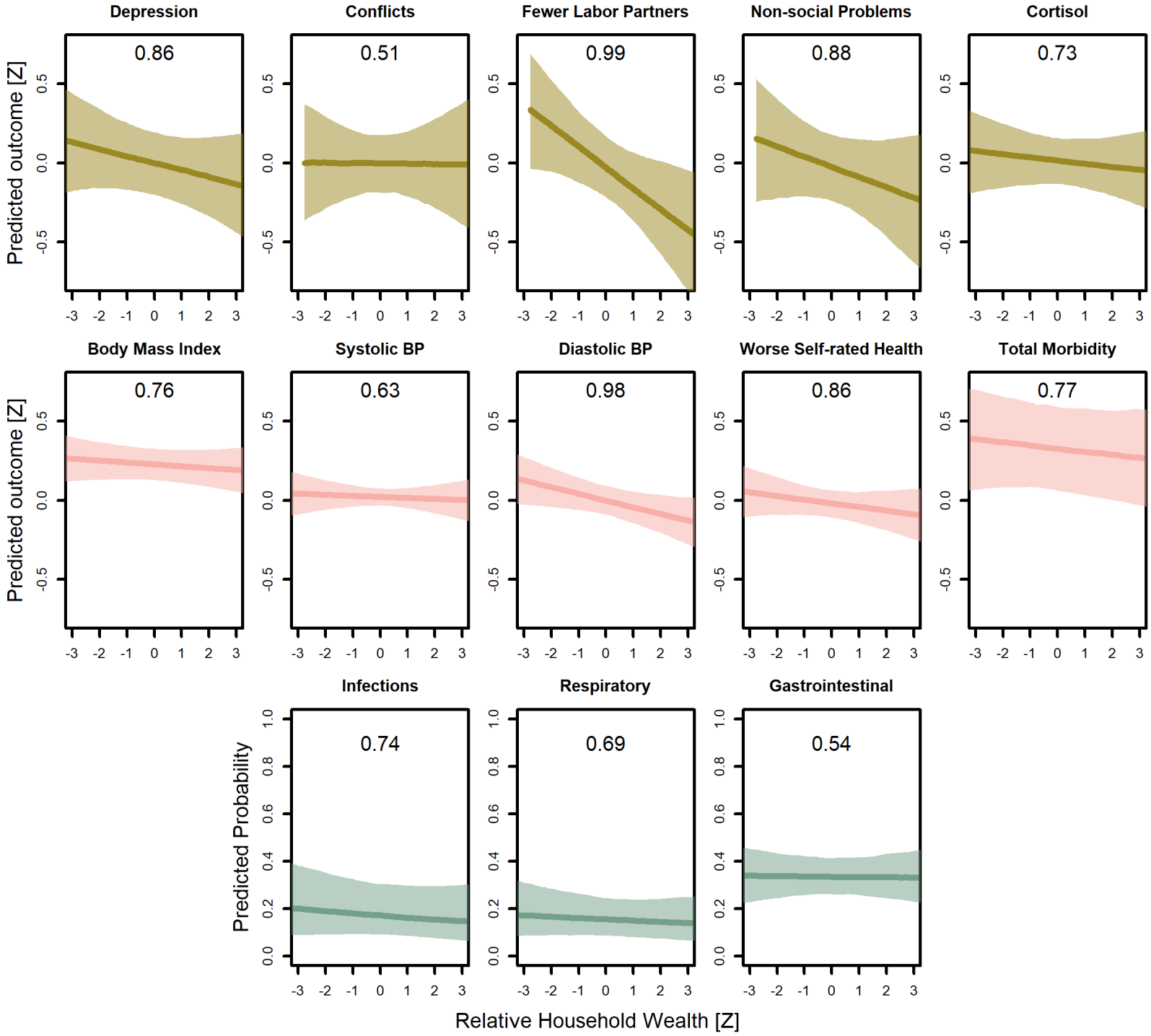
↓

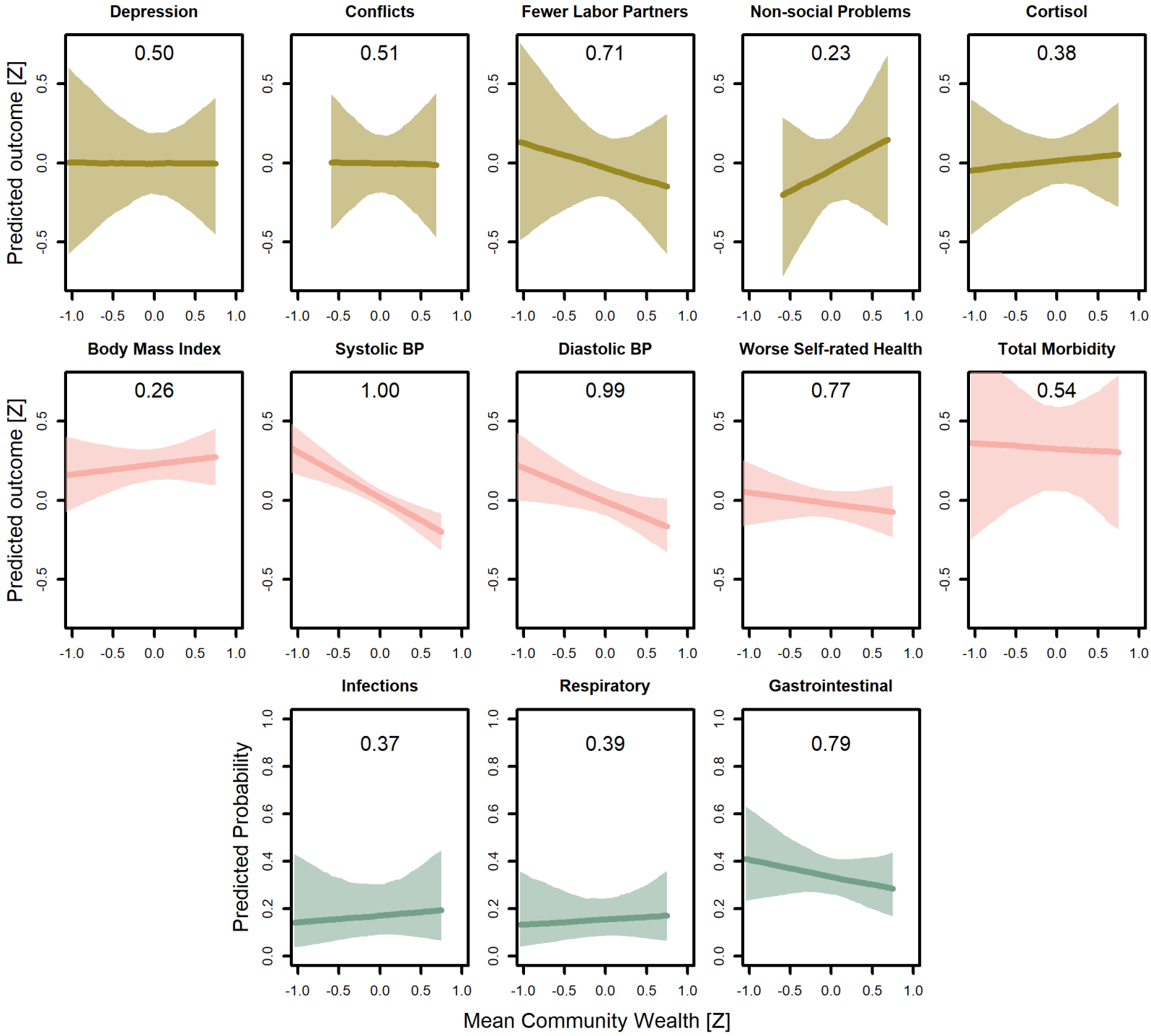
Has wealth data: 4235 individuals

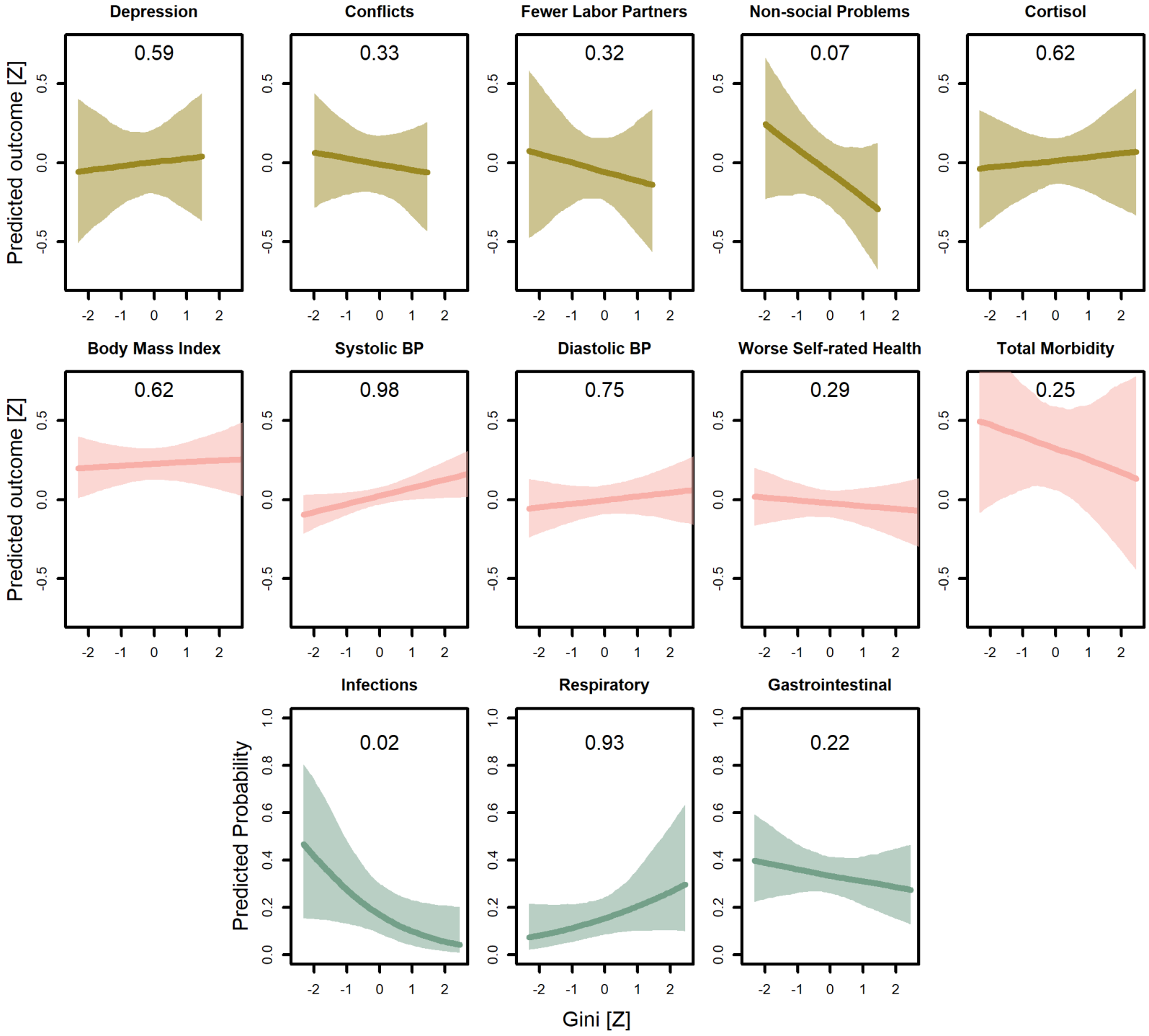
↓

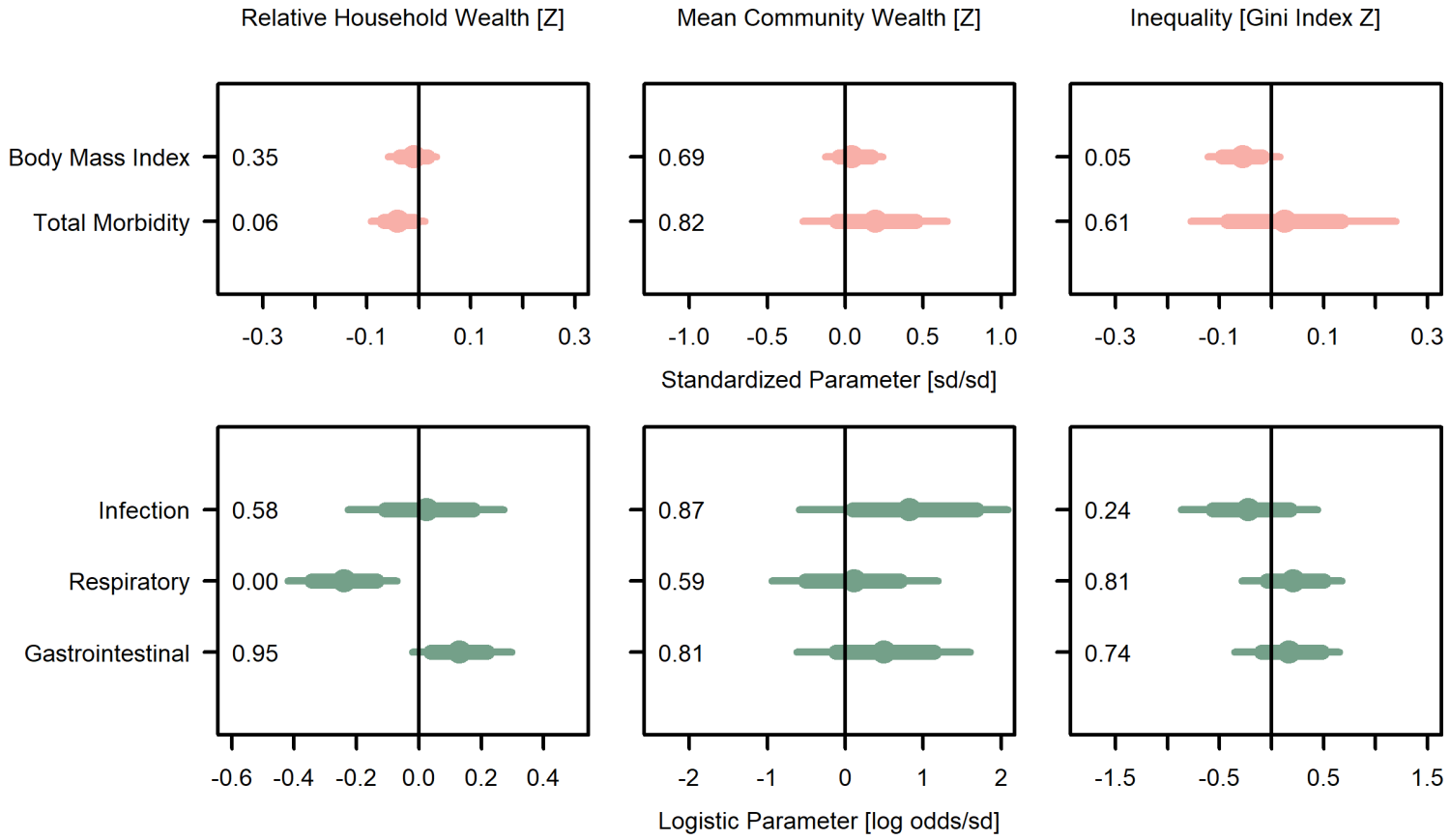
Has age, sex, + any outcome: 3554 individuals

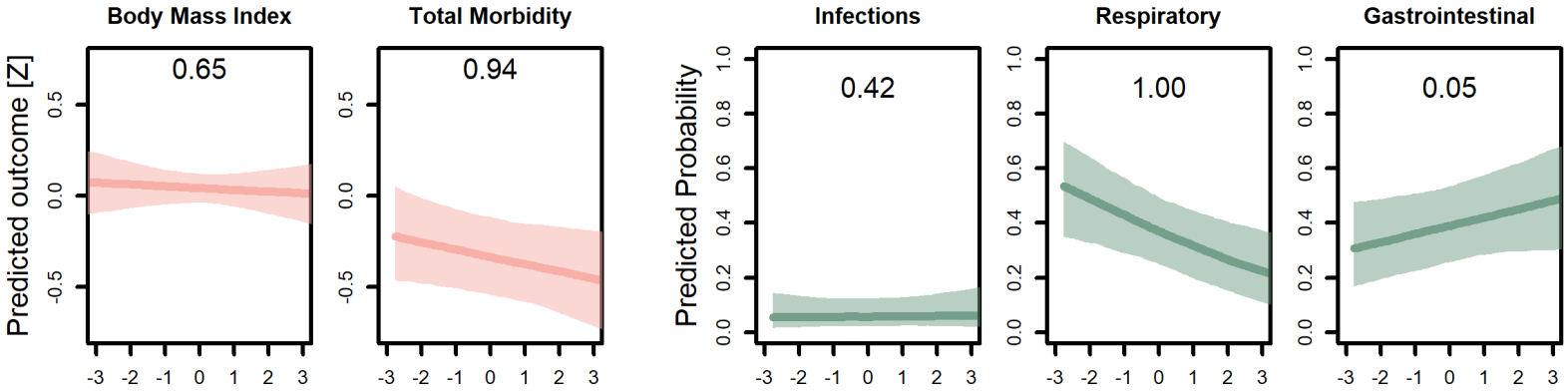

Relative Household Wealth [Z]
Mean Community Wealth [Z]
Inequality [Gini Index Z]
Depression
Conflicts
Fewer Labor Partners
Non-social Problems
Cortisol
0.14
0.49
0.01
0.12
0.27
0.50
0.49
0.29
0.77
0.62
0.59
0.33
0.32
0.07
0.62
Body Mass Index
Systolic BP
Diastolic BP
Worse Self-Rated Health
Total Morbidity
0.24
0.37
0.02
0.14
0.23
0.74
0.00
0.01
0.23
0.46
0.62
0.98
0.75
0.29
0.25
-0.3
-0.1
0.1
0.3
-1.0
-0.5
0.0
0.5
1.0
Standardized Parameter [sd/sd]
Infection
Respiratory
Gastrointestinal
0.26
0.31
0.46
0.63
0.61
0.21
0.02
0.93
0.22
-0.6
-0.4
-0.2
0.0
0.2
0.4
-2
-1
0
1
2
-1.5
-0.5
0.5
1.5
Logistic Parameter [log odds/sd]

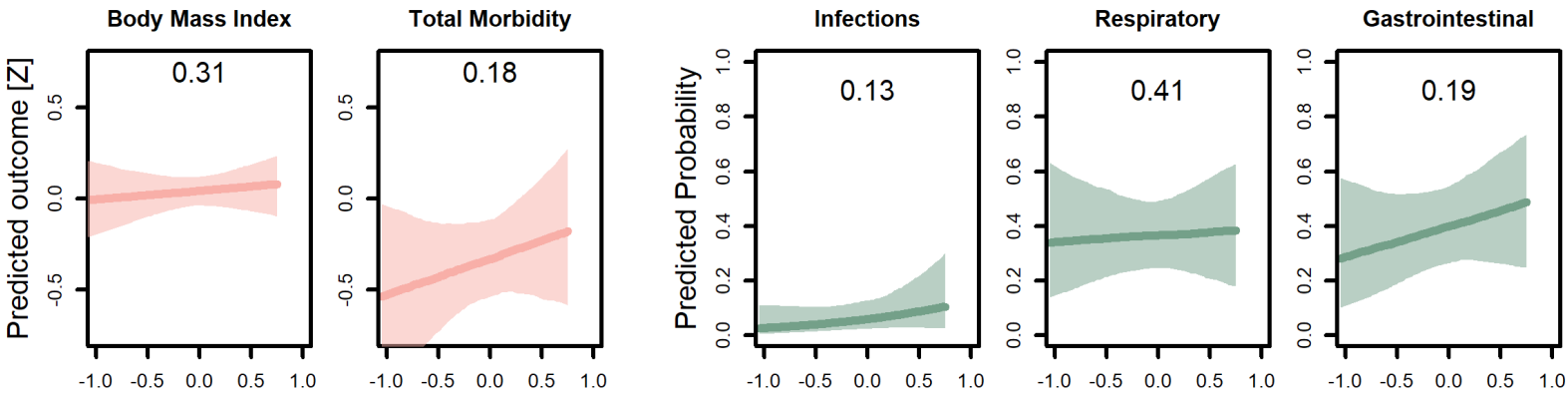

Depression
0.86
Conflicts
0.51
Fewer Labor Partners
0.99
Non-social Problems
0.88
Cortisol
0.73
Body Mass Index
0.76
Systolic BP
0.63
Diastolic BP
0.98
Worse Self-rated Health
0.86
Total Morbidity
0.77
Infections
0.74
Respiratory
0.69
Gastrointestinal
0.54
Predicted outcome [Z]
Predicted Probability
Relative Household Wealth [Z]

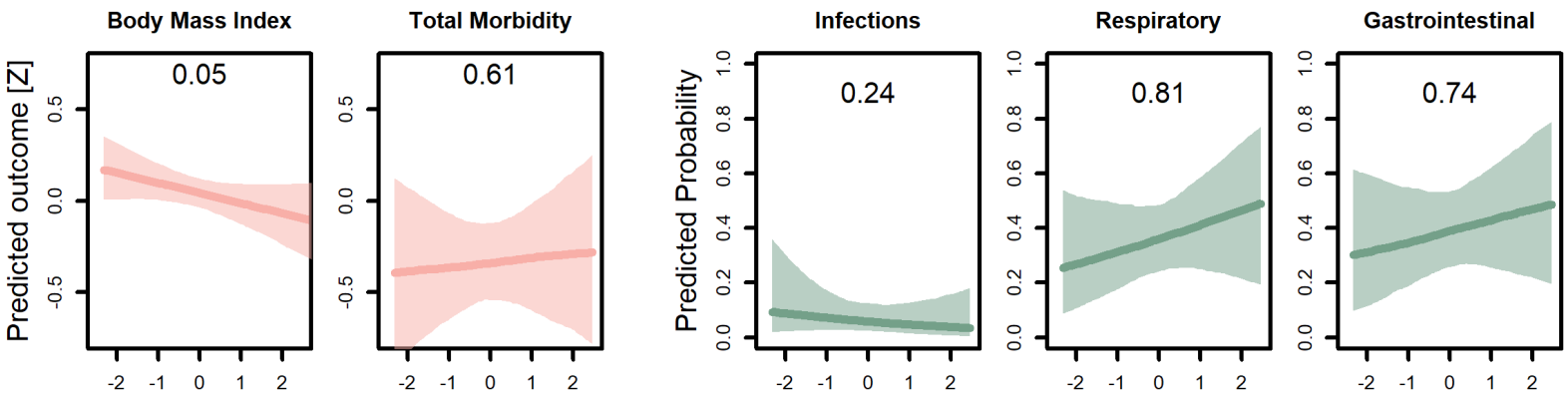

Depression
0.50
Conflicts
0.51
Fewer Labor Partners
0.71
Non-social Problems
0.23
Cortisol
0.38
Body Mass Index
0.26
Systolic BP
1.00
Diastolic BP
0.99
Worse Self-rated Health
0.77
Total Morbidity
0.54
Infections
0.37
Respiratory
0.39
Gastrointestinal
0.79
Predicted outcome [Z]
Predicted Probability
Mean Community Wealth [Z]

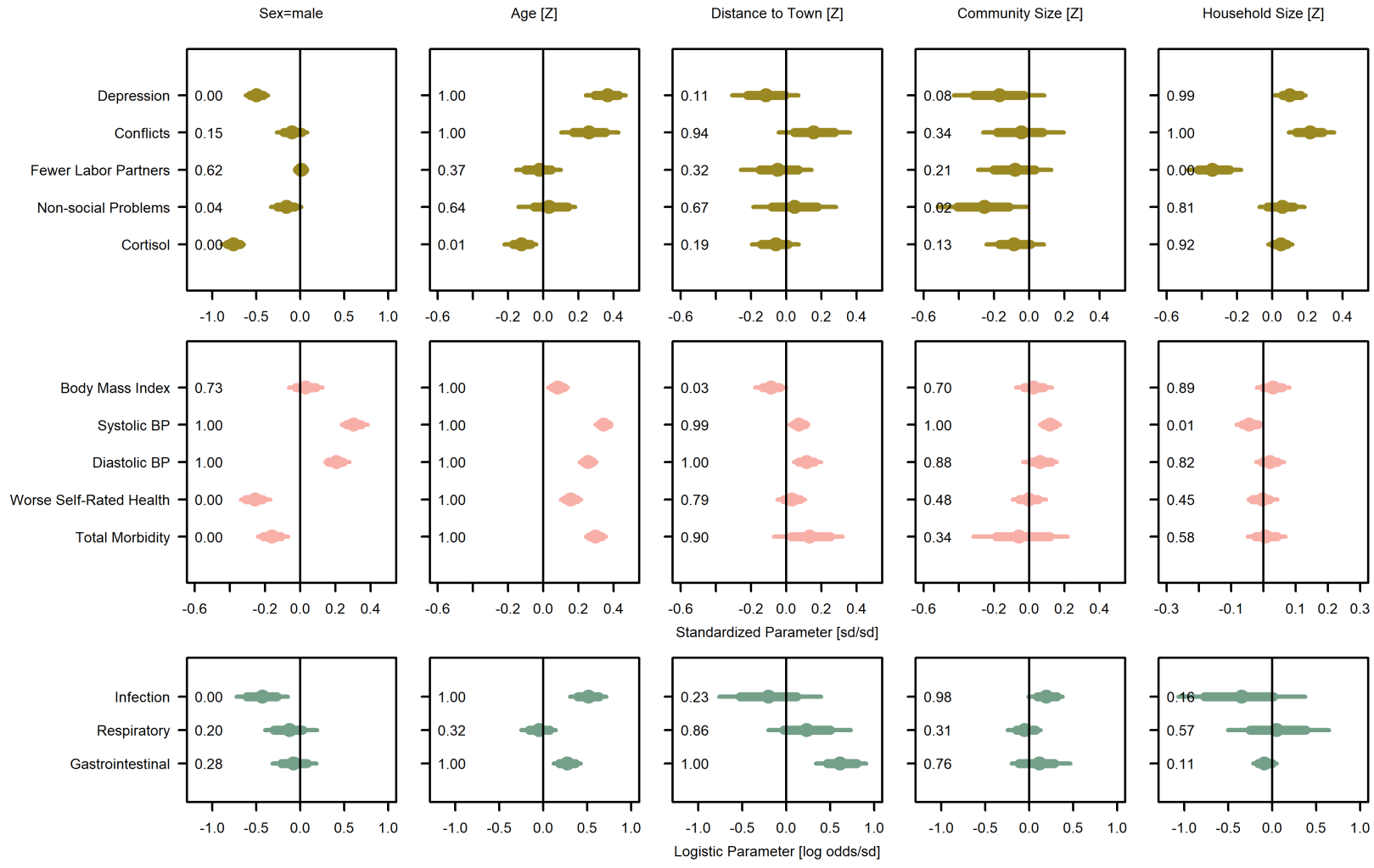

Depression
0.59
Conflicts
0.33
Fewer Labor Partners
0.32
Non-social Problems
0.07
Cortisol
0.62
Body Mass Index
0.62
Systolic BP
0.98
Diastolic BP
0.75
Worse Self-rated Health
0.29
Total Morbidity
0.25
Infections
0.02
Respiratory
0.93
Gastrointestinal
0.22
Predicted outcome [Z]
Predicted Probability
Gini [Z]

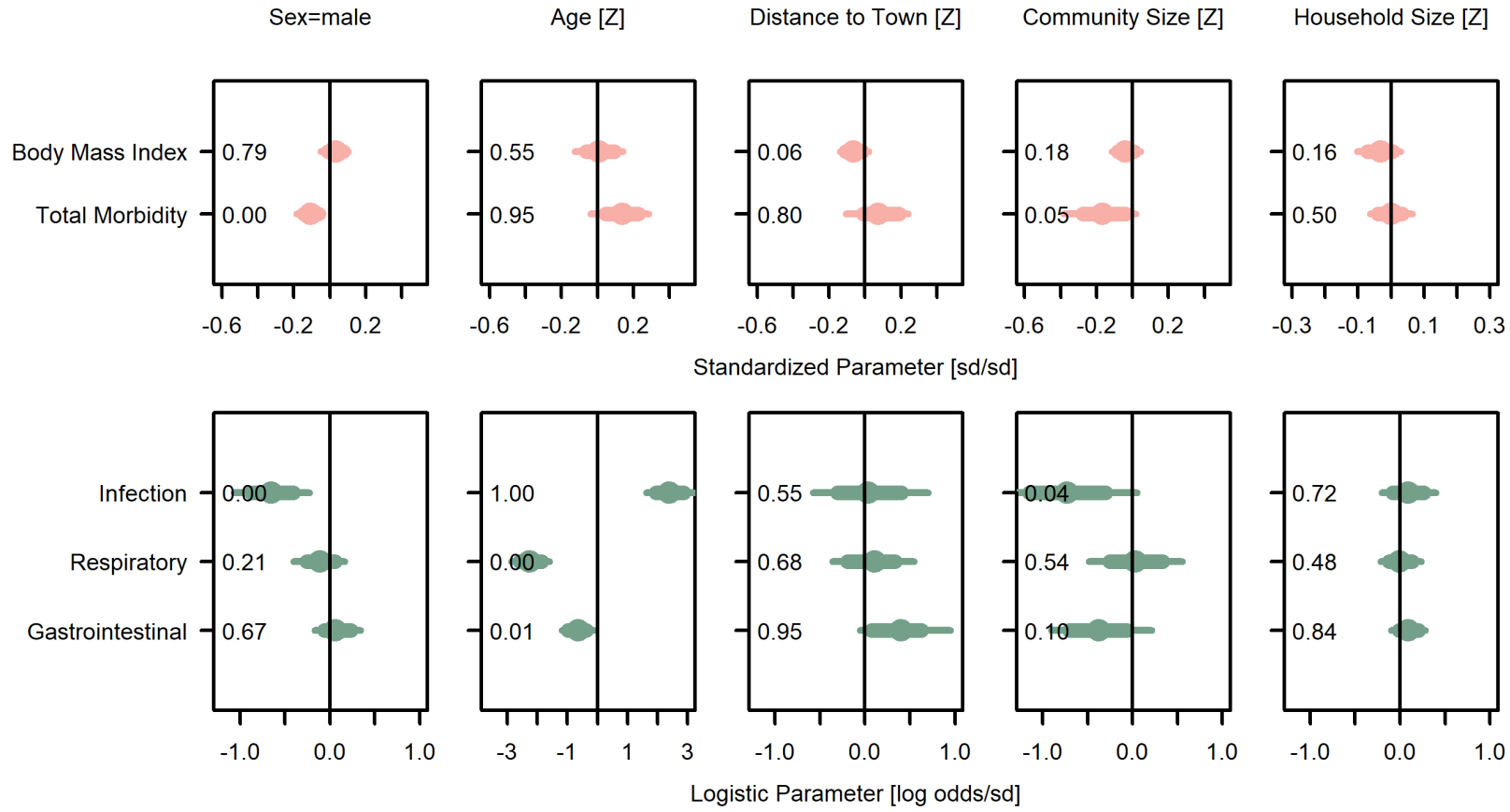

Relative Household Wealth [Z]
Mean Community Wealth [Z]
Inequality [Gini Index Z]
Body Mass Index
Total Morbidity
0.35
0.06
0.69
0.82
0.05
0.61
-0.3
-0.1
0.1
0.3
-1.0
-0.5
0.0
0.5
1.0
Standardized Parameter [sd/sd]
Infection
Respiratory
Gastrointestinal
0.58
0.00
0.95
0.87
0.59
0.81
0.24
0.81
0.74
-0.6
-0.4
-0.2
0.0
0.2
0.4
-2
-1
0
1
2
-1.5
-0.5
0.5
1.5
Logistic Parameter [log odds/sd]

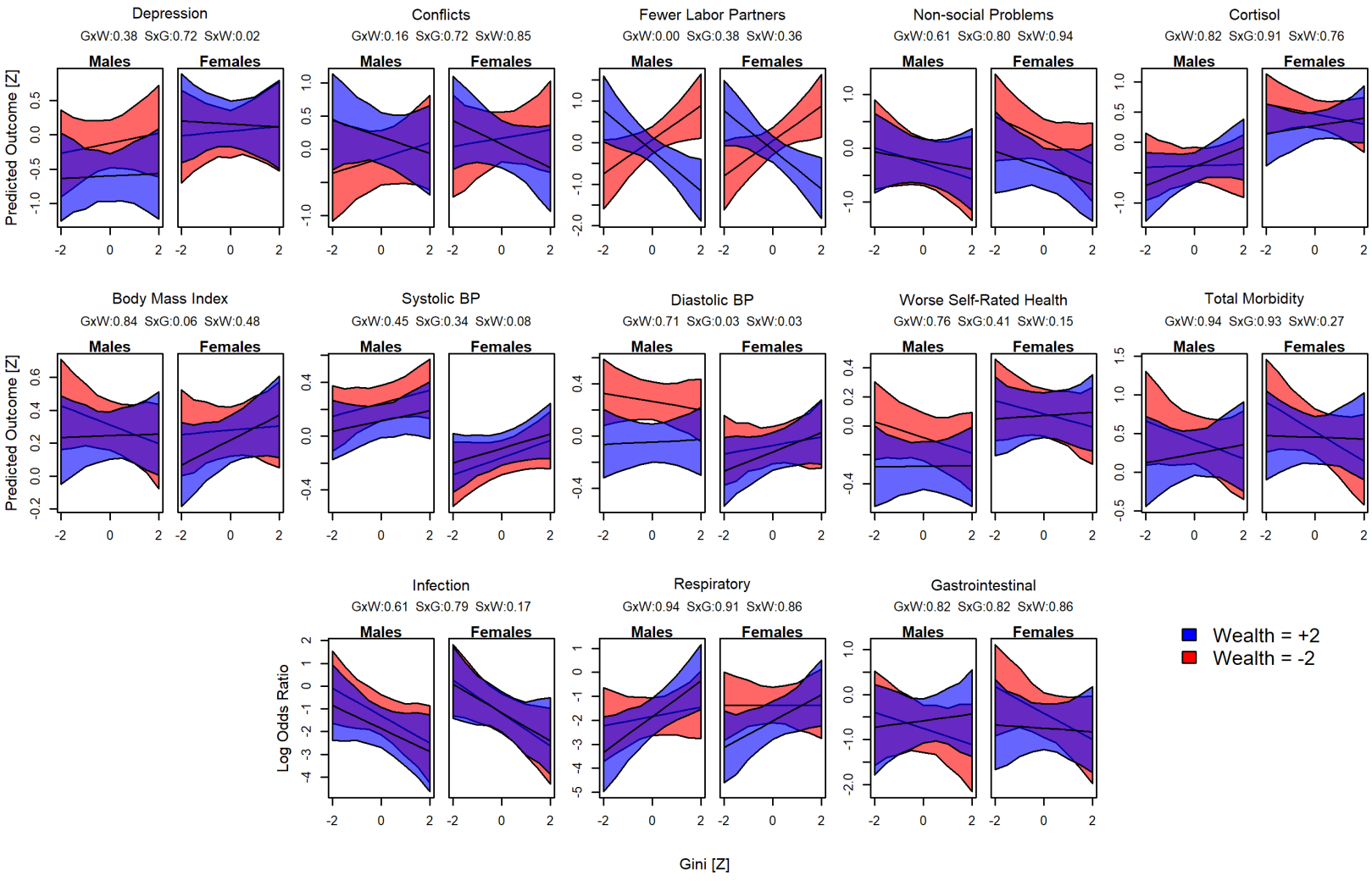

Body Mass Index
Total Morbidity
Infections
Respiratory
Gastrointestinal
Predicted outcome [Z]
Predicted Probability
0.65
0.94
0.42
1.00
0.05
Relative Household Wealth [Z]

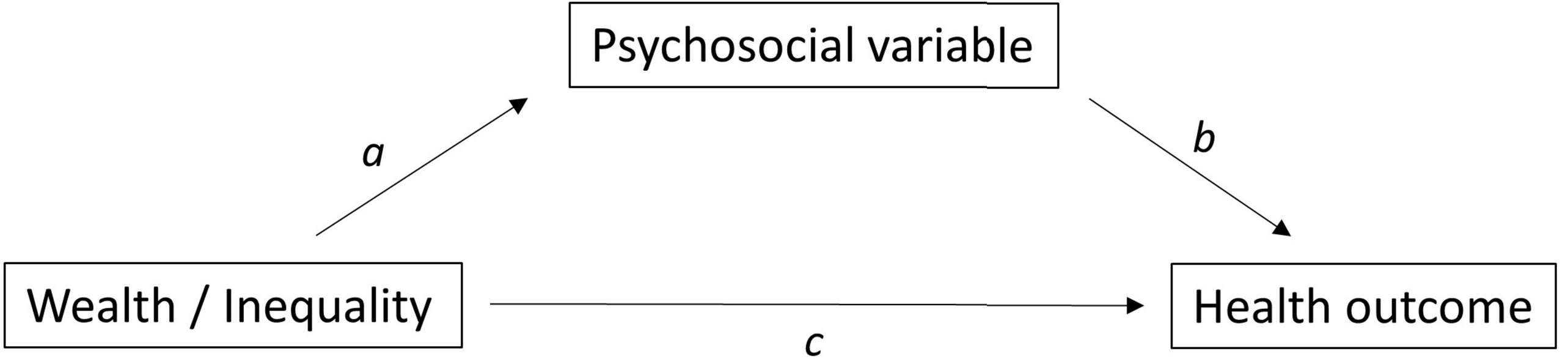

Body Mass Index
Total Morbidity
Infections
Respiratory
Gastrointestinal
Predicted outcome [Z]
Predicted Probability
0.31
0.18
0.13
0.41
0.19
Mean Community Wealth [Z]

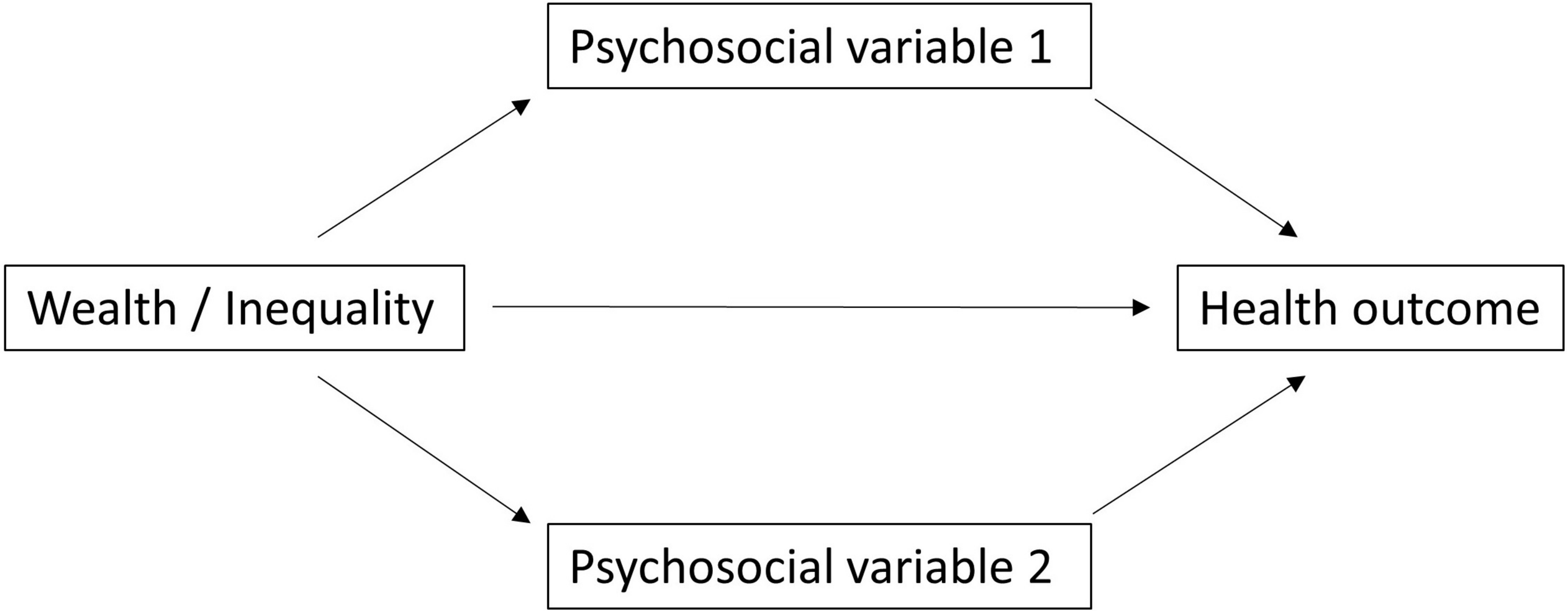

Body Mass Index
Total Morbidity
Infections
Respiratory
Gastrointestinal
Predicted outcome [Z]
Predicted Probability
0.05
0.61
0.24
0.81
0.74
Inequality [Gini Index Z]

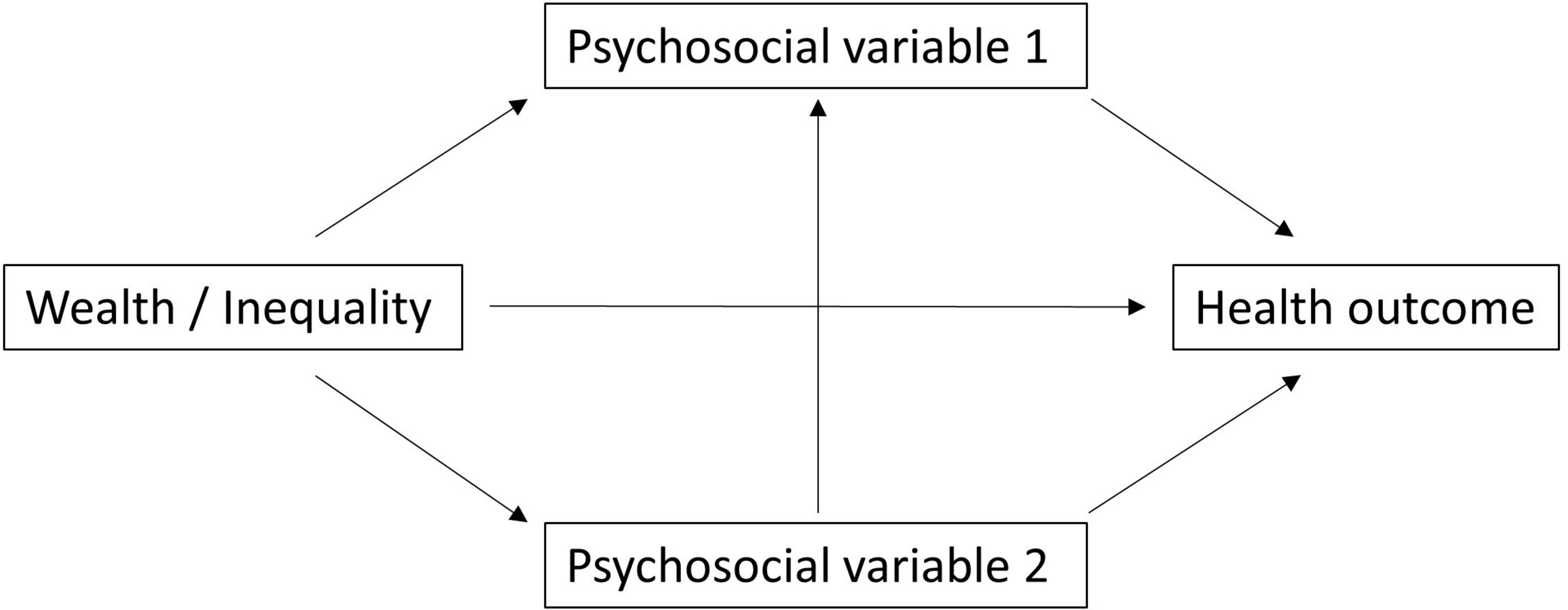

Sex=male
Age [Z]
Distance to Town [Z]
Community Size [Z]
Household Size [Z]
Depression
Conflicts
Fewer Labor Partners
Non-social Problems
Cortisol
0.00
0.15
0.62
0.04
0.00
1.00
1.00
0.37
0.64
0.01
0.11
0.94
0.32
0.67
0.19
0.08
0.34
0.21
0.02
0.13
0.99
1.00
0.00
0.81
0.92
Body Mass Index
Systolic BP
Diastolic BP
Worse Self-Rated Health
Total Morbidity
0.73
1.00
1.00
0.00
0.00
1.00
1.00
1.00
1.00
1.00
0.03
0.99
1.00
0.79
0.90
0.70
1.00
0.88
0.48
0.34
0.89
0.01
0.82
0.45
0.58
Standardized Parameter [sd/sd]
Infection
Respiratory
Gastrointestinal
0.00
0.20
0.28
1.00
0.32
1.00
0.23
0.86
1.00
0.98
0.31
0.76
0.16
0.57
0.11
Logistic Parameter [log odds/sd]


Sex=male
Age [Z]
Distance to Town [Z]
Community Size [Z]
Household Size [Z]
Body Mass Index
Total Morbidity
0.79
0.00
0.55
0.95
0.06
0.80
0.18
0.05
0.16
0.50
-0.6 -0.2 0.2
-0.6 -0.2 0.2
-0.6 -0.2 0.2
-0.6 -0.2 0.2
-0.3 -0.1 0.1 0.3
Standardized Parameter [sd/sd]
Infection
Respiratory
Gastrointestinal
0.00
0.21
0.67
1.00
0.00
0.01
0.55
0.68
0.95
0.04
0.54
0.10
0.72
0.48
0.84
-1.0 0.0 1.0
-3 -1 1 3
-1.0 0.0 1.0
-1.0 0.0 1.0
-1.0 0.0 1.0
Logistic Parameter [log odds/sd]


Depression
GxW:0.38 SxG:0.72 SxW:0.02
Conflicts
GxW:0.16 SxG:0.72 SxW:0.85
Fewer Labor Partners
GxW:0.00 SxG:0.38 SxW:0.36
Non-social Problems
GxW:0.61 SxG:0.80 SxW:0.94
Cortisol
GxW:0.82 SxG:0.91 SxW:0.76
Body Mass Index
GxW:0.84 SxG:0.06 SxW:0.48
Systolic BP
GxW:0.45 SxG:0.34 SxW:0.08
Diastolic BP
GxW:0.71 SxG:0.03 SxW:0.03
Worse Self-Rated Health
GxW:0.76 SxG:0.41 SxW:0.15
Total Morbidity
GxW:0.94 SxG:0.93 SxW:0.27
Infection
GxW:0.61 SxG:0.79 SxW:0.17
Respiratory
GxW:0.94 SxG:0.91 SxW:0.86
Gastrointestinal
GxW:0.82 SxG:0.82 SxW:0.86
Males
Females
Predicted Outcome [Z]
Log Odds Ratio
Gini [Z]
Wealth = +2
Wealth = -2


Psychosocial variable
a
b
Wealth / Inequality
Health outcome
c


Psychosocial variable 1
Wealth / Inequality
Health outcome
Psychosocial variable 2


Psychosocial variable 1
Wealth / Inequality
Health outcome
Psychosocial variable 2