Pathogen stress and living organisation: A cross-cultural analysis

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Abstract

Simulations and animal studies provide a model where paratisim risk is associated with less modular social group sizes. Therefore, this study will examine whether the adoption of modular living in humans has been an adaptive strategy to minimise pathogen stress. Subsequently, codes for pathogens stress are analysed for their correlation with variables for household form, as described for 186 socieites in the Standard Cross-Cultural Sample. The resulting analysis found that pathogen stress is significantly higher for societies which adopt more communal living styles, but there is no differenece in mobile or contagious pathogens which would be expected for the mechanism of such a difference. More studies are still needed to rule out possible confoudning factors.

Keywords

behavioural ecology, pathogen, SCCS, household form

Introduction

Pathogenic stress is a mechanism which affects human fitness, as exposure to diseases reduces fitness in human populations. Fitness load is imparted through survival age to time-of-reproduction and fecundity. Living organisation is a specific measure of how families live in permanent dwellings, from single family units to communal living. This paper aims to present a novel analysis on the relationship between pathogen prevalence and domestic-living organisation across societies.

Domestic organisation refers to coresidual groups, rather than other commercial or functional meanings (Yanagisako 1979). Historically, the emergence of single-family living has been associated with economic factors, particularly the transition into horticultural and agricultural societies. Another factor is industrialisation, which influenced the division of labour and the rise of patriarchal property inheritance. Many other complex mechanisms also account for living organisation, including life expectancy and age-of-menarche (Yanagisako 1979). There are several factors through which close-quartered living conditions may increase parasitic risk and exposure. Firstly, living in large group sizes often correlates with high population densities, which increases pathogen spread; living in close quarters can increase close contact with other people, and large group sizes can carry a higher level of individuals infected with a virus (Rifkin et al. 2012). Presumably, these are the factors which increase pathogen stress in communal-living organisations, which drives seletion for modular living. Pathogens have been modelled to have decreased efficacy when communities are more modular, like those that occur around single-family living. Simulations of social networking have predicted that pathogen success is negatively impacted by community modularity—that is, subgroupings (Griffin and Nunn 2012). These simulations confirm pathogen stress in communities as a selective pressure, which negatively affects the fitness of individuals in large groupings. Reducing the size of community organisation may increase the fitness of individuals in the community. The study attributes the increase of close-contact as the mechanism for increased disease stress and the subsequent ease that infections spread through communities where this occurs. Another metaanalysis found a consistent positive correlation between host group size and the prevalence and intensity of contagious pathogens across many natural populations and species (Côté and Poulinb 1995). Pathogenic risk increases in more aggregated, less modular, community structures due to the increased encounter probability of the pathogen (Nunn and Altizer 2006).

There is a caveat to these generalisations. It is important to make the distinction between contagious and mobile diseases; an analysis found that mobile infectious disease risk decreased with group size (Côté and Poulin 1995). This is likely due to the mechanism of 'encounter-dilution effect' where an increase in overall group size decreases the chance of any one individual being affected by a parasite, in a similar mechanism to decreasing predation risk (Rifkin et al. 2012). Similarly, optimal group size can be a balance between higher within-group risk and reduced between-group risk of mobile pathogens, since larger between-group distance correlates with larger group sizes (Wilson et al. 2003). However, it is unclear whether increased distance between living groups is a factor. Regardless, to gain a better understanding of what mechanism pathogen stress is having on domestic organisation, pathogens should be examined as contagious and mobile.

Other animal models have been studied for their relationship between parasitic risk and group size. This paper will examine whether pathogen stress has shaped the various group-living organisations seen across societies, through its direct impact on fitness. Infectious diseases, such as the pathogens studied, can be maintained in the population over time despite their deleterious fitness load (Cochran et al. 2000). Thus, infectious diseases shape the evolution of human societies. Living organisation may be just one method of sanitary engineering through which societies have mitigated infectious disease, which is over and above what medical advancements have achieved (Eaton et al. 2002).

The general hypothesis examined will explore that if group size and living organisation increases transmission of diseases through relatives, then pathogen stress should be a factor in determining living organisation, or optimal living organisation. Furthermore, if the consensus in the literature about pathogen stress increasing in larger group sizes is true, then it could be predicted that more nuclear or less communal-living styles could be an adaptive strategy which reduces pathogen stress in societies.

Methods

Variables

Sample

The samples were taken from the Standard Cross-Cultural Sample (SCCS) codebook as described by Murdock and White (1969). The codebook describes many characteristics from 186 small societies; data from each society's living organisation are used for analysis. This codebook has the advantage of containing stratified samples, which reduces any false causalities between variables due to common ancestries or relations. Additionally, the societies are largely non-industrialised which reduces the industrialisation effect on living organisation as discussed above.

Pathogen

Pathogen stress codes are described by Cashden and Steele (2013), who originally sum the total stress of eight diseases for each society. These stress codes include scores for *dengue*, *typhus*, *plague (bubonic and pneumonic)*, *filariae (guinea worm and lymphatic)*, *schistosomes*, *leishmanias*, *trypanosomes* and *malaria*. Two extra pathogens—*leprosy* and *spirochetes*, originally coded by Low (1994)—are also included. Cashden and Steele's (2013) coding system was preferred as it contained a finer-tuned four-point scale vs Low's three-point scale. These pathogens represent a mixture of contagious and mobile diseases described in Table 5.1. Contagious refers to transmission through human-to-human contact, mobile refers to transmission through an animal vector (such as fleas, mosquitoes, snails etc.).

Table 5.1: Pathogens included and transmission mode

Pathogen	Transmission	Source
dengue	mobile	Cashden and Steele 2013
typhus	mobile	Cashden and Steele 2013
plague	mobile & contagious	Cashden and Steele 2013
filariae	mobile	Cashden and Steele 2013
schistosomes	mobile	Cashden and Steele 2013
leishmanias	mobile	Cashden and Steele 2013
trypanosomes	mobile	Cashden and Steele 2013
malaria	mobile	Cashden and Steele 2013
leprosy	contagious	Low 1994
spirochetes	contagious	Low 1994

Living organisation

Out of all the variables coded by Murdock and White (1969) that describe how families and communities live, one variable was chosen for consistency and simplicity. '*Household form*' (v67) was chosen as it best captures the family living organisation and thus the group sizes in which these societies reside. Of importance, this variable has complete data for all 186 societies. Household form categorises: single family dwellings, family homesteads, large communal structures, multifamily households, and mutidwelling households (mdh); the latter can be divided into husband rotates, individual married man or woman, married pair and husbands separate. To reflect the hypothesis, these variables were combined to roughly characterise larger group-living to smaller, more modular-living organisations (Table 5.2). The grouping of the variables were maintained across analyses for consistency.

Table 5.2: Re-coded household form variables. Multidwelling households = mdh

Re-coded variable	Household form
modular	single family family homestead
communal	large communal structure multifamily household husband rotates (mdh) individuals (mdh) married (mdh) husband separate (mdh)

Analyses

Analysis 1

Cashden and Steele's (2013) eight pathogen stress values are combined from low to high (Table 5.3). These codes were used for analysis 1 as a more recent and independent check to establish general patterns in pathogen stress. The score of one to four for each pathogen intensity is summed, providing a total sum for each society. For this analysis, the stress codes were treated as categorical variables to first establish a relationship between pathogen stress codes and the living organisation. A contingency table is applied to test for a statistically significant relationship. For the chi-square test, the hypotheses were as follows:

- Null: Pathogen stress and household form are independent.
- Alternative: Pathogen stress codes are associated with household form; knowing something about one provides information about the other.

Table 5.3: Recategorised pathogen codes

Pathogen stress	Rating
8–14	low
15–21	medium
22–27	high

Source: Pathogen codes from Cashden and Steele (2013)

Analysis 2

To further investigate the nature of the relationship between the variables, the pathogen stress codes were treated quantitatively. Cashden and Steele (2013) combined their pathogen codes with Low's (1994) and produced z-scores which represent the variance from the mean, for the sum of all ten pathogens (Table 5.1). The z-scores were averaged for the modular and communal variables. The z-scores were then plotted against the household form as a bar graph.

Analysis 3

A third analysis was undertaken to account for the differences between contagious and mobile pathogens, as the ten pathogens are largely biased towards vector-based transmission modes. However, only Low's (1994) codes separated pathogen codes individually, and only *leprosy* and *spirochetes* are contagious. Therefore, the scores from 1–3 for these pathogens were averaged for each society and averaged when combined in the same manner as household form. The average score was plotted on a bar graph. Sample deviations were taken from the average *leprosy* and *spirochetes* score data for every society, before combination.

Results

Analysis 1

The contingency table analysis showed there is a very significant relationship between the household form and the level of pathogen stress ($\chi^2 = 10.1$, $p = 0.00640933$), with a significance level of $p = 0.05$ (Table 5.4). Deviations from the null suggests the variables of pathogen stress and household form are not independent of each other, and knowing something about one increases our understanding of the other. The contingency table only displays evidence for a relationship between the variables, it does not provide meaningful information about whether a pathogen is statistically higher for one or the other variable. However, these results are, so far, in support of our evolutionary hypothesis: living organisation affects how pathogens are transmitted and thus the pathogen stress experienced by a community.

Table 5.4: Contingency analysis table for household and recategorised pathogen codes.

	Low	Medium	High	Total
communal	16 (23.67)	26 (25.67)	20 (12.67)	62
modular	55 (47.33)	51 (51.33)	18 (25.33)	124
total	71	77	38	186

Occurences of each pathogen stress rating for each homestead variable is displayed in the table. Values in parentheses indicate expected frequencies: expected = column sum * row sum / total sum, for example, (71*62)/186 = 23.67.

Analysis 2

Figure 5.1 displays that the modular-living variable has, on average, pathogen-stress scores below the mean of all the samples in the codebook. Conversely, the communal variables have pathogen-stress scores which are above the average. The z-scores used for this analysis combine the two contagious

pathogens not included in Analysis 1. This, combined with the large deviation from the mean seen in the modular-living variable, provides convincing evidence in support of the hypothesis that modular family living reduces pathogen stress experienced in these communities.

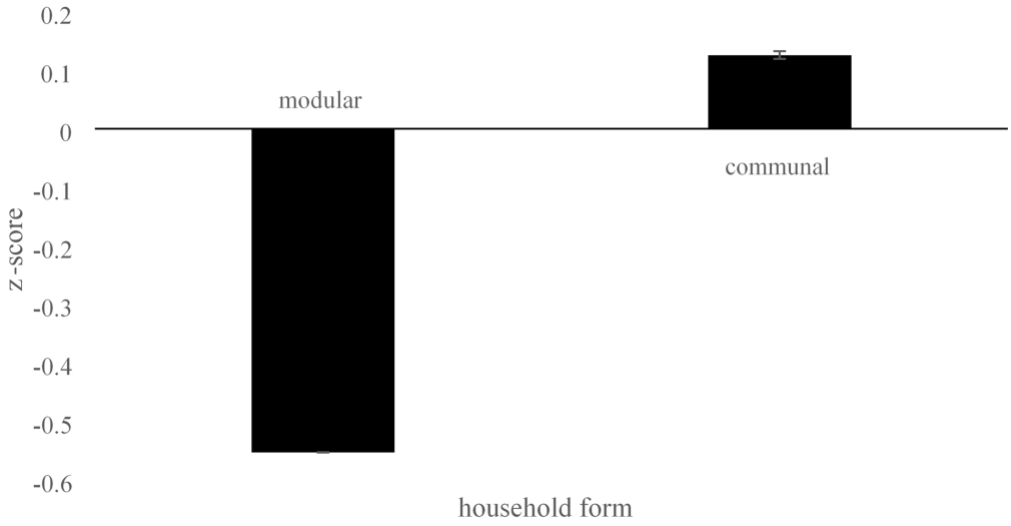

Figure 5.1: Average deviance from the mean pathogen stress in household form.

Z-scores for the total 10 pathogens as scored by Cashden and Steele (2013). Error bars are the standard error, since z-scores represent deviations from the population.

Analysis 3

In Figure 5.2—just the contagious pathogens—*leprosy* and *spirochetes* were combined as these were the only available contagious codes which were coded separately. The results show that there is a minute increase in the communal variable, however the standard deviations from these pathogen averages were too large to establish any real difference between the variables. This does not provide any evidence to support that contagious pathogens have a higher incidence in societies with larger, more communal, living styles.

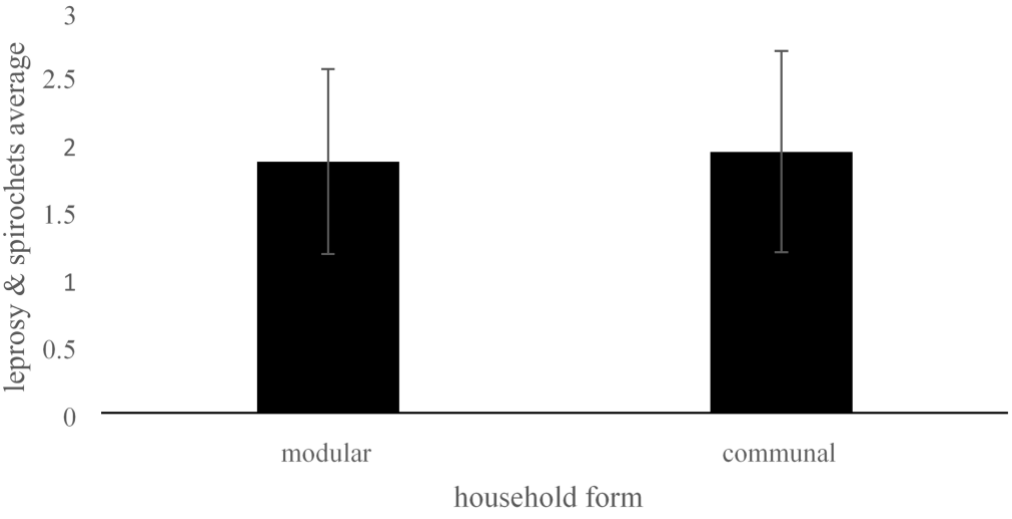

Figure 5.2: Contagious pathogens and household form.

Leprosy and *spirochetes* were averaged, as coded by Low (1994). Error bars are standard deviations.

Discussion

For Analysis 1, it was found that there was a significant relationship between the level of pathogen stress and modular and large group-living household forms. The χ^2_{value} was larger than the χ^2_{crit} value, therefore we can reject the null hypothesis (that the variables are independent), and accept the alternative (that household form has a relationship with the level of pathogen stress experienced by a society). However, the contingency table treats the pathogen-stress scores as counts or categorical variables. This analysis suggests that pathogen stress is a factor which shapes living organisation, but does not address whether or not it is related to more modular or more communal living.

For Analysis 2, the z-scores, as coded by Cashden and Steele (2013), provide a quantitative way to assess the magnitude of pathogen stress among the societies. The analysis found that societies which have adopted more modular living do experience less-than-average pathogen stress, for all 10 pathogens. This analysis provides more robust evidence for the specific evolutionary hypothesis that pathogen stress is a selective pressure which has led to the adaption of more modular living to reduce its negative fitness impact. In other words, pathogen stress is a factor which is shaping the way these societies live.

Analysis 3, however, proved that there was no real difference between the variables when just the contagious variables of *leprosy* and *spirochetes* were considered. However, this could be attributed to the fact that in Low's (1994) coding system, a score of 1 (out of 3) is the equivalent of the pathogen being absent from the society, and the pathogen is not being recorded. This could mean that societies where these pathogens have not yet been recorded are skewing the data towards the appearance of having a low prevalence in the society. Nevertheless, this analysis does not support the hypothesis of more communal-living societies experiencing more pathogen stress due to transmission through others in the household.

The possibility that a confounding factor is producing the correlations which support the hypothesis cannot be ruled out. This adds more complexity which needs to be addressed with further analysis. For example, it is known that populations in tropical environments experience more pathogen stress, and that local populations experience varying degrees of susceptibility, immunity, and resistance (Vasseur and Quintana-Murci 2013). Specifically, the bias towards transmission through animal vectors increases the geographic variations, since climate, especially, affects these animals' ability to survive (Cashden and Steele 2013). Analysis between these factors and living organisation should be undertaken. Other potential analyses which could account for correlations between the variables include stratifying the 186 societies by geographical region, population density, and polygyny prescence.

Another such factor which could be confounding the effects of living organisation and pathogen stress is that of polygyny. One SCCS study (Low 1990) found that polygyny was positively correlated with pathogen stress in the 186 codebook societies—in all geographic locations and population densities. Polygyny is a variable which, understandably, affects household form (Yanagisako 1979). It is possible that the rise of nuclear single-family living correlates with a reduction in polygyny and thus the reduction in pathogen stress. Further study could either stratify the samples for polygyny, or test for correlation between the variables of household form and polygyny to confirm or reduce the validity of the hypothesis.

Other parasitic infection models have found that there is an optimum size for host group clusters, in terms of increasing the survival rate against mobile pathogens (Watve and Jog 1997). Again, the mechanism for this was the reduction of transmission of the parasite, and the extinction rate of the pathogen before infection. This occurred under the caveat that increased host clustering also increased between-group distance (distance can be taken to mean distance between water and other resource-sharing or settling distance). Likewise, more gregarious organisms have been found to invest less in immune responses than solitary organisms (Wilson et al. 2003). However, the mechanism for this also assumes increased between-group distance as a way to reduce overall pathogen infection. The analysis undertaken here did not find any evidence for the decrease in pathogen stress in larger clustering, despite the pathogen codes being heavily biased towards mobile transmission. It could be that societies living

in the communal-living arrangements do not decrease their distance with other clustering living groups, or they are above the optimal group size for humans.

In agreement with the finding in this paper, a recent metaanalysis also found a positive correlation between group size and mobile parasitism (Rifkin et al. 2012), despite the 'encounter dilution' theory. One mechanism suggested for the correlation is a detection increase, where parasites can more easily detect hosts in larger congregations. The size of living arrangements' effect on sociality and interactions is also an especially important factor in human's parasite susceptibility. This could explain why parasitism risk is particularly low in birds vs. mammal models despite overall grouping size (Rifkin et al. 2012). One model examined relatively smaller social networks, and found that overall pathogen prevalence is low when groups are more isolated in their interactions (Griffin and Nunn 2012). Interestingly, there was a lower transmission probability per interaction when one individual dominates social interactions. Specifically, this provides the mechanism of reduced pathogen risk in modular-living arrangements, despite overall population densities (which were not examined in this paper). As pathogen risk is a selection pressure which favours those who interact with fewer individuals to reduce parasitism risk, in the context of the overall hypothesis, this explains a nonrandom adaption of more modular group-living. Or, if these living arrangements arise for other reasons, parasite risk could be the selection pressure which favours the adaption of this living arrangement over others (Griffin and Nunn 2012).

The hypothesis states that modular living is a behavioural adaption to reduce the negative fitness impact of pathogen stress. Despite the results from this analysis—which suggest that pathogen stress is reduced in societies with modular living—the exact mechanism for this cannot be confirmed from this study. It is likely that selection for more isolated group-living reduces the vulnerability for pathogen infection through less-available transmission opportunities. This increases fitness—by directly increasing survival rates/reproduction, or by decreasing resources invested in immune defences (Wilson et al. 2003). Whether it shapes or selects for the living arrangement is not clear.

To conclude, this analysis reports a statistically significant relationship between pathogen stress and living organisation, and that more modular-living includes pathogen stress below average compared to more communal-living structures. An increase in just contagious pathogen risk was not seen in the more modular-living structures; overall pathogen stress for mobile parasites is supported in the literature, despite models of encounter-dilution effect and optimal group sizing. This is likely due to the modular social aspect in human societies, or a lack of decrease in distance between living groups. Overall, the evidence supports the hypothesis that pathogen risk is a selective factor in the adaption of more modular-living styles. More study is needed to exclude the possibility of other cofounding factors like polygyny and geographic variance.

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