SOCIOSEXUAL NORMS, THE BEHAVIOURAL IMMUNE SYSTEM HYPOTHESIS AND THE RED QUEEN

ABSTRACT

Pathogen pressure has played an extremely influential role in human adaptive evolution. It is well established that this role includes extensive selective sweeps upon the genetic substrates of antibody repertoires. A growing volume of research suggests that this role may extend to the evolution of sophisticated cognitive mechanisms via which individuals detect and avoid initial infection. In humans, such mechanisms may play an influential role in the emergence, establishment and reinforcement of cultural norms of behaviour. This is termed the behavioural immune system hypothesis. Sexual behaviour, which represents a significant potential route to infection, is the subject of widely differing attitudes and norms throughout the world. Herein, it is hypothesised that norms of restrictiveness may tend to vary along gradients of pathogen prevalence. It is further hypothesised that differential fitness costs between the sexes, arising from pathogen stress, may contribute towards explaining the existence of differing levels of sexual permissiveness. To account for the contingency that the results yielded track in the opposite to that which is hypothesised, the Red Queen hypothesis (wherein the faster adaptive rate of pathogens predicts a preference for genetic diversity – and thus less restrictive social norms) is put forth as a counterpoint. Improved characterisation of the biocultural basis of differing sexual norms promises to provide insights useful in anthropological research, as well as public health planning and risk mitigation.

INTRODUCTION

Although antecedent ideas concerning the influence of pathogen prevalence on human behaviour exist, the concept of a behavioural immune system (BEH) is formally of a relatively recent vintage (Schaller 2006; Thornhill & Fincher 2014). It refers to a suite of cognitive mechanisms that enable organisms to detect and avoid sources of parasitic infection within their environments. This ostensibly stands in contrast to the biological immune system (BIO), consisting of the genetic and cellular mechanisms of defence against such threats. The broad assertion that organisms (including humans) detect and avoid pathogens has been relatively uncontroversial and has not met with repudiation in any systematic way. In this regard the BEH is so far a paradigm alone and apart, witnessing little challenge to its primary tenets. Nonetheless, much remains to be elucidated in terms of the extent of its influence, and disputes exists concerning certain minutiae of its mechanisms.

This research will examine norms of sexual permissiveness and hygiene in the Standard Cross-Cultural Sample (SCCS), focusing on those which – being linked to sexual reproduction – may have strong connections to selective processes and pressures. It is further notable that differences in infection risk and levels of innate reproductive investment between the sexes have been invoked to explain the establishment of observably different sociosexual behavioural norms. If sexually differentiated norms are strongly influenced by pathogen prevalence, this may constitute a level of support and a novel application for the behavioural immune system hypothesis. This support would exist only in the form of correlation and would therefore be indirect and circumstantial in itself. However, it would highlight productive directions for future research. In order to better characterise the background of this proposal, it is necessary to consider in greater detail what the BEH entails, the bases upon which it is predicated, as well as the purposes to which it has been put. It is held likely the behavioural hygiene hypothesis will elicit greater support.

LITERATURE REVIEW

Pathogen pressure represents the most significant driver of adaptive evolution in many lineages, including humans, exerting great influence upon both natural and sexual selection dynamics (Ejsmond et al. 2014; Fumagalli et al. 2011; Thornhill & Fincher 2014). The effects of infection, morbidity and differential survival on the genetic and cellular substrates of organisms' immune repertoires are increasingly well characterised. Yet, profound as this impact is, it may provide an incomplete portrait of human immune adaptation. It has been argued that selective processes have also acted upon cognitive mechanisms and behavioural strategies that serve to mitigate infection risk (Schaller 2006). Before proceeding, it is necessary to examine certain fundamental predicates. In particular, there is a need to consider the distinctive benefits that might attend evolved behavioural mechanisms of aversion.

Selection would be less likely to favour adaptations that imposed costs while merely duplicating existing benefits, ergo there is a need to consider the deficiencies and attendant costs of immune function. Several arguments have been set forth in this regard. Antibodies of the BIO can only be reactive by nature, responding to an infection already in progress. Quite apart from the harm that can be sustained before an infection is quelled, immune surveillance is a highly metabolically costly trait. Activation of the inflammatory mechanisms associated with the BIO involves substantial physiological resources. For instance, an increase of ~13% in basal metabolic rate is required to raise the core body temperature of an adult human by just 1°C (Schaller 2015). This immune response is, in itself, debilitating to the individual. Firstly, there is a direct physiological toll and even a mortality risk attending system-wide inflammation. Secondly, this debilitating process presents an opportunity cost, as the resources used in combating an infection might otherwise be invested in fitness-increasing activities – such as resource gathering, reproduction or care for kin (Schaller 2015). It is

therefore apparent that some advantages would not merely be duplicated in successfully detecting and avoiding infection risk.

The plausibility of the behavioural immune system hypothesis gains additional support by virtue of some important properties that are intrinsic to most human pathogens. Because pathogens are typically so small as to be undetectable in the absence of complex instruments, the evolution of aversion mechanisms may include the overgeneralisation of risk perception (Miller & Maner 2012). Pressure towards this end lies not only in the minuscule size of the selective agent, but also the substantially differing costs imposed by false-positive and false-negative risk assessment – sometimes termed the 'smoke detector' principle (Nesse 2005). False-positive assessment may produce behaviours that are not in themselves adaptive and which may even be moderately maladaptive. This is rendered plausible by the potential for false negative risk assessment to result in a dramatic loss of fitness, potentially including prolonged morbidity, infertility or premature death. Consequently, phenomena (particularly visible deviations from a template of normality) that have no actual relationship to infectious agents (and which may not be in any sense pathological) could fall under the aegis of reflexive aversion. These topics are addressed herein, but it is first prudent to consider reasons for circumspection.

We may doubt that selection could act upon human populations in a sufficiently strong manner to elicit identifiable adaptive behaviours. In order for selection to act on organisms with long generation times and low reproductive rates, exposure would likely need to have occurred over a long duration, or else be extremely severe. Although the endemic phase of many pathogens appears to have accelerated with the onset of the Neolithic (particularly with regard to the advent of animal agriculture, with demographic transitions resulting in greater population sizes and with the reduction of group mobility), it is increasingly apparent that many pathogens have an ancient relationship with human populations (Wolfe et al. 2007). Disease, considered broadly, was not a topic of trivial concern to premodern populations. We may further note that the evolution of cognitive mechanisms to actively prevent infection may be heavily suppressed, due to countervailing fitness benefits arising from social interaction. Such benefits may include predator detection, sex, or collective foraging (Curtis 2014). Yet there is a case to be made that, in some lineages, this renders the existence of an evolved BEH

more probable, not less.

Behavioural mitigation of disease risk is phylogenetically widespread, which ostensibly lends some support to the idea of an evolved adaptation. Due caution should be exercised, as many such observations are anecdotal rather than systematic (Curtis 2014). Even so, some trends are notable. As parasites are often adapted to a limited range of hosts, the highest infection risk is likely to stem from contact with members of the same species, or the refuse of the same, particularly in high densities. Preponderantly solitary living therefore features prominently among infection avoidance mechanisms in many taxa (Curtis 2014). Humans, however, are anything but solitary-living. To the contrary, humans are extremely social and distinguished from most other taxa by the extent of our predilection to shape our environmental niches.

It is notable that highly social species are more likely to specialise in forms of environmental niche modification to prevent parasite infection (Curtis 2014). As a species that could almost be considered social by obligation, we might therefore expect humans to be more in need of sensitive mechanisms of detection and aversion than would more solitary taxa (Neuberg et al. 2011; Tybur & Lieberman 2016). Further to which, as cultural organisms, this could include not only individual-level mechanisms of revulsion, but the establishment and collective reinforcement of complex behavioural norms. There is more than a *prima facie* case for the idea that selection has acted upon human cognitive and cultural mechanisms of infection avoidance. Although norms that serve to reduce pathogen transmission may partially be explained by cultural evolution, drift and inertia, these phenomena interact with (and arguably emerge from) a biological substrate, consisting of aversive emotions and cognitive processes (Schaller & Murray 2010). Central to this argument is the phenomenon of disgust.

Disgust arising from from observation of images of contaminants, disgust arising from the direct experience of gustatory distaste and disgust arising from the observation of transgressions of moral norms all involve comparable activation of the levator labii muscle region of the face, characteristic of

an oral-nasal rejection response (Chapman 2009). This may imply the exaptation of the latter form of disgust sensitivity from the former in human evolution (Curtis et al. 2011; Lieberman & Patrick 2014; Schaller 2014). The expression of disgust, amplified when expressed collectively, functions to manipulate individuals towards adherence to cultural norms (Curtis et al. 2011; Wu & Chang 2012). Between-population differences in disgust sensitivity may arise from variation in relevant allele frequencies – or, by virtue of gene-environment feedback mechanisms driving developmental plasticity, the differential expression of a shared genotype between populations in differing ecological contexts (Schaller & Park 2011; Schaller 2015). Candidate alleles that might explain variation between populations are hitherto relatively little investigated. However, one possible link between the experience of disgust as an evolved mechanism for infection risk reduction and innate immunity is the serotonergic system. Serotonin receptors, enriched in the gut, are associated with emetic and peristaltic responses, as well as aversive dietary conditioning (Rubio-Godoy et al. 2007). Improved resolution of these mechanisms is possible. However, the purpose in raising them here is only to support the assertion that moral and other mechanisms of disgust share substantial similarities and, quite likely, common origins.

We may be curious as to the mechanisms by which aversion to infection risk develops. In most mammals, the vomeronasal organ mediates both positive and negative olfactory perception (Adams et al. 1984, Boillat 2015, Cooper & Batnagar 1976, D'Aniello et al. 2017, Dinka et al. 2016, Keverne 1989, Vaccarezza et al. 1981). Importantly, this includes at least some primates (Hohenbrink et al. 2013, Laska & Salazar 2015). In great apes, for whom visual acuity (with the evolution of trichromatic vision, this may be less important or of negligible impact (Laska & Salazar 2015; Smith et al. 2014). However, there is evidence that major histocompatibility complex (MHC)– more specifically MHC 1 peptides – are secreted via the skin microbiota and reflect immunological differences. However, there is evidence for selection of both self-alike and self-divergent peptides (Leinders-Zufall et al. 2004). Disease states often have distinct olfactory as well as visual profiles (Schute 2005), therefore this is also a topic of considerable interest in considering any outcomes yielded).

In light of the intimately intertwined nature of biology and culture in disgust sensitivity, some further consideration should be given to the conceptual separation of BEH and BIO. These should be understood as misnomers to some degree. It has been demonstrated that mere viewing of disease symptoms promotes heightened production of the pro-inflammatory cytokine interleukin-6 upon subsequent pathogen challenge, ergo the behavioural immune system may influence its biological counterpart (Schaller et al. 2010). Perhaps less intuitively, the relationship also proceeds in the opposite direction, in that recent illness can predict heightened attention and aversion to others who are ill (Miller & Maner 2011). Biological immune function and behavioural immune function are therefore not completely separable. It may also be erroneous to speak of 'the' behavioural immune system, in light of differential expression of traits (Gangestad & Grebe 2014; Schaller & Park 2011). Yet, although the terminology is imprecise, it will suffice for the present purpose. At this juncture, it is prudent to consider the purposes to which the BEH hypothesis has been put. The broad concept of an evolved disgust mechanism associated with perceived infection risk has received no direct opposition of note, although certain applications have produced inconsistent results.

The BEH hypothesis has been used to test predictions about certain forms of prejudice and cultural preference for modes of social organisation. Here we must return to the concept that infection risk perception and disgust may be overgeneralised, in light of the widely differing severity of false-positive and false-negative errors. Notably, such findings include the stigmatisation of visible attributes that do not have a basis in pathogen infection, such as obesity (Park et al. 2007), physical disabilities unrelated to infection (Park et al. 2003; Ryan et al. 2012) and older age (Duncan & Schaller 2009). These tendencies are more pronounced when subjective perception of infection risk is high. Evidence of disgust concerning norm violations in a more general sense, modulated by perceived infection risk, also exists. This includes the existence of strong associations between pathogen prevalence and more rigidly conservative, authoritarian and xenophobic cultural norms (Murray et al. 2013; Terrizzi et al. 2013). Similarly, pathogen prevalence is positively correlated with collectivist (Cashdan & Steele 2013) and conformist (Wu & Chang 2012; Wu et al. 2015) attitudes. Nonetheless such predictions have not been consistently strong; measures of xenophobia, for instance, variably show a moderately strong (Murray et al. 2013) and

statistically insignificant (Cashdan & Steele 2013) relationship with pathogen prevalence. Scope exists to further expand the range and specificity of variables tested. Saliently, differences observed between populations may also exist between the sexes within populations, where potential fitness costs differ.

Countervailing tendencies in pathogen pressure exist between the sexes, which will be examined in this analysis. Females, on average, have stronger immune responses than males as a product of different endocrine endowments (Foo et al. 2016), which implies a higher risk in the latter. Conversely, differing levels of reproductive investment between the sexes render female reproductive capacity a scarce 'resource', which social norms may serve to guard (Low 1990). Notably, differences in levels of innate reproductive investment between the sexes have been invoked to explain the establishment of observably different sociosexual behavioural norms (eg. Low 1989). It is plausible that other sexual norms are similarly affected by pathogenic threats and differential fitness costs. For instance, Fincher and Thornhill (2008a, 2008b) identify pathogen stress as a significant correlate of endogamy. If other sexual norms vary along a gradient of pathogen prevalence, this may constitute a level of support for the behavioural immune system hypothesis. If notable disparities exist between the sexes, this may represent a productive avenue for further inquiry.

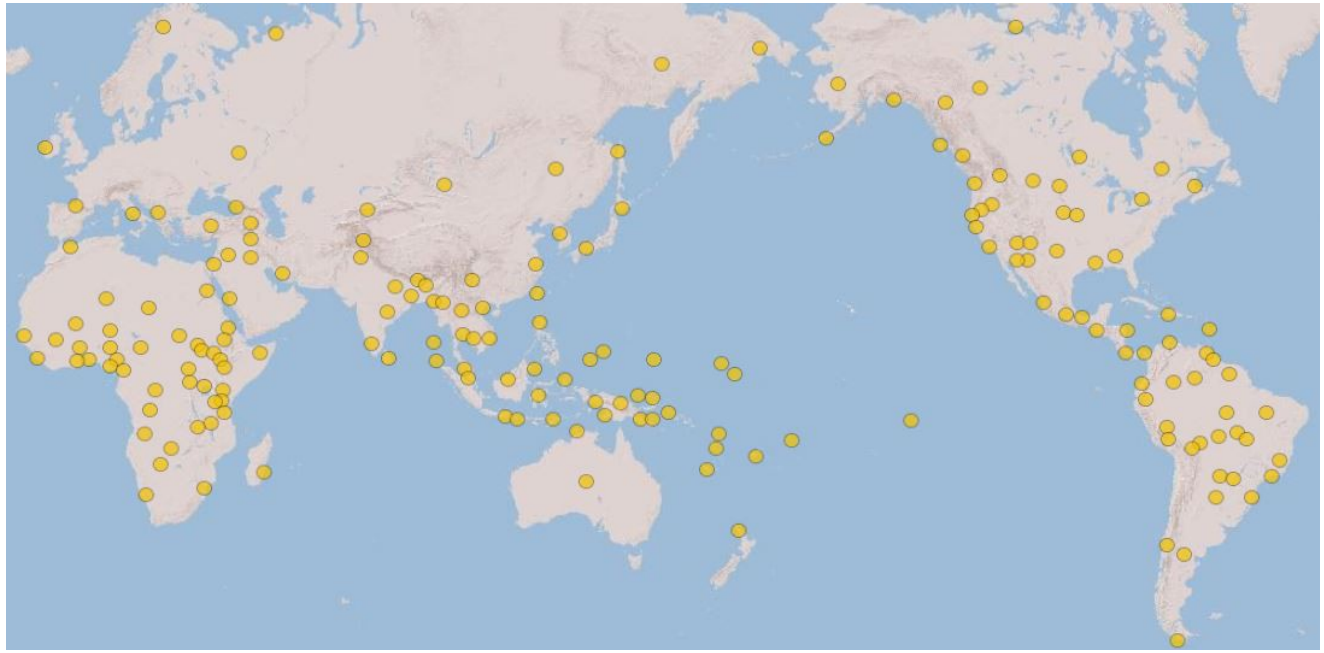
The recent revision and expansion of pathogen codes for the SCCS (Cashdan & Steele 2013) permits testing of hypotheses pertaining to variation in sociosexual norms as a function of pathogen prevalence. Examining the inculcation of differences in hygiene-related socialisation between the sexes, using other datasets, is not novel (see for example Low 1989). Testing of the relationship between pathogen prevalence and various cultural behaviours, using the SCCS specifically, would not be novel either (Cashdan & Steele 2013; Murray et al. 2013; Nettle 2009). However, analysis of sexually differentiated behavioural norms, as a product of pathogen prevalence, has not been conducted using the SCCS dataset. The significance of the proposed analysis therefore lies in an extension of prior work, insofar as the study will examine norms related to the differential inculcation of sociosexual hygiene, between the sexes and between disparate cultural groups.

METHODS

OVERVIEW

This analysis will use an existing data set, namely the SCCS, ergo the research to be conducted will not involve any field activity. The proposed analysis constitutes a case study, insofar as the SCCS represents a sample of global cultural diversity, to which the results may be generalised. The SCCS is coded in terms of ordinal variables, the ranks of each of which may have either numerical or qualitative descriptions. In this analysis, normalisation of the independent variables also results in a continuous scale. All analysis was performed in Stata 15. The spatial distribution of coded societies is visible in F**igure 1**.

Figure 1: Spatial distribution of SCCS-coded societies.



DATA COLLECTION

1. The data collection methods underpinning the SCCS, as well as its notable advantages for cross-cultural comparisons, are summarised here. The 186 societies represented are premodern (non-industrial) and preponderantly small-scale. This is useful, as the hypothesis to be tested concerns the effects of pathogen prevalence on the norms of comparable groups. The societies represented are globally dispersed, providing a worldwide portrait, which is of use in testing the universality (or otherwise) of the proposed hypothesis. The societies are also stratified by both language and region, which helps to ensure that similarities arising from shared history are minimised (Hruschka & Hackman 2014; Murdock & White 1969). Cultures exist in a constant state of flux, ergo each is assigned a focal year; a point at which the generalisations made for each variable are most appropriately representative (Murdock & White 1969). The fact that these focal years usually fall in the mid-20th century or earlier assists in mitigating the effect of increasingly globalised cultural norms.

   The enumerated cultures are also chosen to reflect the epicentre of larger cultural groupings, which likely limits the level of relatedness and cultural similarity between cultures, which may occur at territorial margins (Murdock & White 1969). Collection and use of epidemiological and demographic data in the present day could be confounded by these factors, regarding the purpose at hand. The fact that the SCCS represents a standard in cross-cultural research means that several variables of interest to the research at hand have been coded. This analysis will use the 2003 SCCS dataset and recently expanded pathogen codes. This is due to the fact that novel data, useful to this investigation, has been added to it in prior work (Cashdan & Steele 2013). This analysis will use this work as a template. One criticism of the Low (1998) variables is that the data collected is not time-adjusted and the Cashdan and Steele variables (2013) are better matched in this regard. Nonetheless both are examined. Finally, it is notable that the SCCS is incomplete. That is to say, not all societies have values for all studied variables. **Table 1** describes the dependent variables used, the abbreviations assigned, the type of variable (ie. Binary or ordinal), codes used in

the analysis, as well as the original variable codes. **Table 2** uses the same format for the independent variables in the analysis.

Binary dependent variables were modelled in Stata using the logit command, while ordinal dependent variables were modelled using the ologit command for each independent variable in Table 2. To obtain odds ratios the additional command "or" was added to the model.

Table 1: Dependent variables.

Variable	Abbrev.	Type	Codes		Original Variable	Reference
Polygamy	v79_1r	Binary	0 = Monogamous 1 = Polygamous		Polygamy (v79) 1 = Polyandry - primarily monogamous with some plural husbands 2 = Monogamy 3 = Polygyny < 20% plural wives (if more frequent than polyandry) 4 = Polygyny > 20% plural wives (if more frequent than polyandry)	Murdock & Wilson 1972
Elite Multi-Wives	v868_1	Binary	0 = No 1 = Yes		Multiple wives for Leaders, Headmen, Chiefs (v868) 0 = No, or unimportant 1 = Yes, or Leaders have more wives than commoners	White 1988
Permit Extramarital (M) Permit Extramarital (F)	xmarm xmarf	Binary	xmarm 0 = No (3,4) 1 = Yes (1,2)	xmarf 0 = N (2,3,4) 1 = Y (1)	Extramarital Sex (v169) 1 = Single standard (both allowed) 2 = Double standard (husbands only) 3 = Double standard (both forbidden, women punished more) 4 = Single standard (both condemned equally)	Broude & Greene 1976
Increasing Polygyny	v860_1	Ordinal	1 = Monogamy prescribed or preferred 2 = Polygyny preferred by leaders and men if higher social class 3 = Polygyny preferred by most men		Cultural Basis of Polygyny (v860) 1 = Monogamy prescribed 2 = Monogamy preferred, but exceptional cases of polygyny 3 = Polygyny preferred by individual men with leadership attributes (chiefs, medicine men, outstanding hunters) 4 = Polygyny preferred by men of a higher social class: men of wealth, rank, nobility, etc. 5 = Polygyny preferred by most men and attained by most men of sufficient years or wealth to obtain wives.	White 1988
Premarital Sex Freq (M) Premarital Sex Freq (F)	v166_2 v167_2	Ordinal Ordinal	1 = Occasional or uncommon 2 = Moderate 3 = Universal		Frequency of Premarital Sex – Male (v166) / - Female (v167) 1 = Universal 2 = Moderate	Broude & Greene 1976

				3 = Occasional 4 = Uncommon	
Sexual Expression (AB)	v827	Ordinal	Reduced to 3 categories	Sexual Expression in Adolescent Boys (v827) and Girls (v828) [1]	Barry & Schlegel 1984
Sexual Expression (AG)	v828	Ordinal	###How?	Scale from 0-10, where: 2 is not approved, 5 is normally and generally approved, and 8 is strongly approved and values	
Sexual Nonrestraint (AB)	v829	Ordinal	Reduced to 3 categories	Sexual Nonrestraint in Adolescent Boys (v829) and Girls (v830) [2]	Barry & Schlegel 1984
Sexual Nonrestraint (AG)	v830	Ordinal	###How?	Scale from 0-10, where: 2 is strictly and effectively prohibited, 5 is disapproved, and 8 is condoned and not generally punished.	

[1] Encouragement of sexual behavior, taking into account its frequency, emotional intensity, importance, and variety (including range of partners) in adolescence. Heterosexual intercourse is the principal criterion, but heterosexual foreplay, masturbation, homosexuality, sexual jokes, and exposing the genitals were also considered. The absence of sexual restraints such as taboos or restrictions on heterosexual intercourse and other erotic behavior, including heterosexual play, masturbation, and homosexuality. A high degree of modesty, such as the requirement to keep the genitals constantly covered in public, indicates moderate restraint. Incest taboos, if highly emphasized or widely extended, are considered as indicators of restraint.

Table 2. Independent variables.

Variable	Abbrev.	Codes	Variable Definition	Ref.
Low (1988) Pathogen Pressure	v1260	Same as original	Total Pathogen Stress (v1260) Scale from 7-21; sum of pathogen codes, where 1 is absent, 2 is present, and 3 is serious, for the following pathogens: leishmanias (v1253), trypanosomes (v1254), malaria (v1255), schistosomes (v1256), filariae (v1257), spirochetes (v1258), and leprosy (v1259)	Low 1988
Low Tertiles – G	v1260_G	1 = Low pathogen pressure (lowest tertile) 2 = Moderate pathogen pressure (middle tertile) 3 = High pathogen pressure (highest tertile)	Tertiles based on data percentiles (0-38.71, 38.72-67.20, 67.21-100) Stata command: xtile [variable name] = v1260, nquantiles(3). 7-11 = Low pathogen pressure (lowest tertile) 12-14 = Moderate pathogen pressure (middle tertile) 15-21 = High pathogen pressure (highest tertile)	Low 1988
Low Tertiles – C	v1260_C	1 = Low pathogen pressure (lowest tertile) 2 = Moderate pathogen pressure (middle tertile) 3 = High pathogen pressure (highest tertile)	Tertiles based on combinations of 7 pathogen scores (eg. a score of 1 for each pathogen gives the combination 1111111). With 36 possible combinations, 12 combinations were allocated to each level). 7-12 = Low pathogen pressure (lowest tertile)	Low 1988

			13-15 = Moderate pathogen pressure (middle tertile) 16-21 = High pathogen pressure (highest tertile)	
Cashdan & Steele (2013) Pathogen Pressure	pathogenscore	Same as original	Sum Score of Individual Pathogen Codes (v2177) Scale from 8-27 Scale used 1-4 8 pathogens, 5 in Common with Low and 3 additional	Cashdan & Steele 2013
Cashdan Tertiles – G	pathogenscore_g	1 = Low pathogen pressure (lowest tertile) 2 = Moderate pathogen pressure (middle tertile) 3 = High pathogen pressure (highest tertile)	Tertiles based on data percentiles (0-33.33, 33.34-67.20, 67.21-100) Stata command: xtile [variable name] = v2177, nquantiles(3). 8-13 = Low pathogen pressure (lowest tertile) 14-19 = Moderate pathogen pressure (middle tertile) 20-27 = High pathogen pressure (highest tertile)	Cashdan & Steele 2013
Cashdan Tertiles – C	pathogenscore_c	1 = Low pathogen pressure (lowest tertile) 2 = Moderate pathogen pressure (middle tertile) 3 = High pathogen pressure (highest tertile)	Tertiles based on combinations of 8 pathogen scores of 1-4 (eg. a score of 1 for each pathogen gives the combination 1111111). With 165 possible combinations, 52 combinations were allocated to levels low and high, but 61 combinations to moderate). 8-17 = Low pathogen pressure (lowest tertile) 18-22 = Moderate pathogen pressure (middle tertile)	Cashdan & Steele 2013

			23-27 = High pathogen pressure (highest tertile)	
Region	v200	Same as original	1 = Africa, exclusive of Madagascar and the Sahara 2 = Circum-Mediterranean (N Africa, Europe, Turkey, Caucusus, Semitic Near East) 3 = East Eurasia, including Madagascar and islands in the Indian Ocean 4 = Insular Pacific, including Australia, Indonesia, Formosa, and Philippines 5 = North America (indigenous societies to the Isthmus of Tehuantepec) 6 = South America, including Antilles, Yucatan, and Central America	Gray 1998

Tables 3 and 4 give descriptive statistics for dependent and independent variables respectively. These summary statistics include sample size, category frequencies and related proportions.

Table 3: Descriptive statistics for dependent variables.

Variable	N	Missing	Categories	Freq.	Prop.

Polygamy	186	0	Monogamous		16.7%
			Polygamous	155	83.3%
Elite Multi-Wives	155	31	No	94	60.1%
			Yes	61	39.9%
Permit Extramarital (M)	109	77	No	48	44.0%
			lTHESISYes	61	64.0%
Permit Extramarital (F)	109	77	No	96	88.1%
			Yes	13	11.9%
Increasing Polygyny	180	6	Monogamy prescribed or preferred	60	33.3%
			Polygyny preferred by leaders…	75	41.7%
			Polygyny preferred by most men	45	25.0%
Premarital Sex Freq (M)	102	84	Occasional or uncommon	24	23.5%
			Moderate	18	17.6%
			Universal	60	58.8%
Premarital Sex Freq (F)	109	77	Occasional or uncommon	39	35.8%
			Moderate	19	17.4%
			Universal	51	46.8%
Sexual Expression (AB)	104	82	Not approved	27	26.0%
			Normally and generally approved	33	31.7%
			Strongly approved and valued	44	42.3%
Sexual Expression	102	84	Not approved	37	36.2%

(AG)					
			Normally and generally approved	31	30.4%
			Strongly approved and valued	34	33.6%
Sexual Nonrestraint (AB)	96	90	Strictly and effectively prohibited	21	21.9%
			Disapproved	30	31.3%
			Condoned and not punished	45	46.9%
Sexual Nonrestraint (AG)	105	81	Strictly and effectively prohibited	36	34.3%
			Disapproved	32	30.5%
			Condoned and not punished	37	35.2%

Table 4: Descriptive statistics for independent variables.

Variable	N	Missing	Categories	Freq.	Prop.	Tertiles		Freq.	Prop.
Low (1988)	186	0	7	30	28.3%	G	Low	72	38.7%
			8	9	4.8%		Moderate	53	28.5%

Pathogen Pressure [1]			9	9	4.8%		High	61	32.8%
			10	4	2.2%				
			11	20	10.8%	C	Low	92	49.5%
			12	20	10.8%		Moderate	49	26.3%
			13	13	7.0%		High	45	24.2%
			14	20	10.8%				
			15	16	8.6%				
			16	16	8.6%				
			17	9	4.8%				
			18	9	4.8%				
			19	6	3.2%				
			20	3	1.6%				
			21	2	1.1%				
Cashdan & Steele (2013) Pathogen Pressure [2]	186	0	8	24	12.9%	G	Low	62	33.3%
			9	4	2.2%		Moderate	63	33.9%
			10	7	3.8%		High	61	32.8%
			11	4	2.2%				
			12	11	5.9%	C	Low	100	53.8%
			13	12	6.5%		Moderate	58	31.2%
			14	9	4.8%		High	28	15.1%
			15	13	7.0%				
			16	6	3.2%				
			17	10	5.4%				
			18	14	7.5%				
			19	11	5.9%				
			20	8	4.4%				
			21	15	8.1%				
			22	10	5.4%				

			23	7	3.8%
			24	12	6.5%
			25	6	3.2%
			26	2	1.1%
			27	1	0.6%
Region	186	0	Africa	28	15.1%
			Circum-Med.	28	15.1%
			East Eurasia	34	18.3%
			Insular Pacific	31	16.7%
			N. America	33	17.7%
			S. America	32	17.2%

[1] Low (1988) Pathogen Pressure as continuous variable: M=12.6, SD=3.78

[2] Cashdan & Steele (2014) Pathogen Pressure as continuous variable: M=16.4, SD=5.37

ANALYSIS

This section describes the rationale for the variables and analytical methods chosen.

Pathogen prevalence:

Total pathogen stress (hereafter TPS; SCCS variable 1260) is an ordinal scale consisting of the aggregate scores of SCCS variables 1253-1259. Cashdan and Steele (2013) created a normalised pathogen z-score from this data and their own original research, the product of which is a numerical scale that will be utilised in this analysis. This data is available in the electronic supplementary material associated with the aforementioned paper. Pathogens considered herein include leishmanias, trypanosomes, malaria, schistosomes, filariae, dengue, typhus, spirochetes and plague. Each entry is localised within 200 km of the focal points of the societies described. TPS represents the independent variable in this analysis.

V1253 to v1260 were coded by Low (1988). Cashdan and Steele (2013) derived their z-scores directly from five of the Low codes and 3 additional cdes,The notes suggest that the Cashdan & Steele ones are better time-matched to the focal dates for each society's data.

Sociosexual norms:

In order to test the hypothesis that sociosexual norms should tend toward increasing restrictiveness along gradients of increasing pathogen prevalence, it was necessary to choose suitable proxies for which adequate data was available. It is also important that data concerning the phenomena in question be presented for men and women separately or as poles of a spectrum relating to gendered norms, as one of the extended aims of the study is to detect sex biases in levels of restrictiveness. Additional variables may be considered. Where necessary, some categories will be collapsed in order to ensure adequate sample sizes, if this can be done without losing essential nuance.

· V68: 'Form of Family' (concerning polygamy vs. polygyny).

· V166: 'Frequency of premarital sex – female' and V167: 'Frequency of premarital sex – male'.
· V170: 'Frequency of extramarital sex – male' and V171: 'Frequency of extramarital sex – female'.
· V596: 'No double standard in regard to premarital sex'.
· V597: 'No double standard in regard to extramarital sex'.
· V827: 'Sexual expression in adolescents – boys' and V828: 'Sexual expression in adolescents – girls'.
· V829: 'Sexual nonrestraint in adolescents – boys' and V830: 'Sexual nonrestraint in adolescents – girls'.

HYPOTHESIS

For each test:
$\mathbf{H_0}$: There will be no correlation between pathogen prevalence and greater restrictiveness of sociosexual norms.
$\mathbf{H_A}$: There will be a correlation between pathogen prevalence and greater restrictiveness of sociosexual norms.

Comparisons will be made between sex-specific variables to elucidate sex biases in the norms described. These include variables pertaining to polygyny, polygamy, degrees of sexual nonrestraint in adolescents, cultural permission for extramarital sex, as well as the actual frequency of extramarital sex. A dimension of social class is included to ascertain whether multiple wives are only or preponderantly afforded to elite classes.

Analytical methods:

Spatial autocorrelation can be a fatal methodological issue in such analyses; that is, phenomena that are closer together are more likely to be highly positive correlated. For this reason, political complexity and region) are included as 'dummy' variables to enhance spatial segregation (ie. reduce the risk of spatial autocorrelation) and thus the independence of data points (Cashdan & Steele 2013). Because this analysis seeks to test whether or not pathogen prevalence is associated with greater restrictiveness in sexual mores, dependent variables will be reverse-coded as the need arises. This will facilitate ease of analysis and will ultimately also improve the structural clarity of figures presented.

In line with the theoretical background and methodology outlined, this analysis aims to determine whether pathogen prevalence is associated with restrictive and sex-biased sexual norms in the SCCS data set. Logistic regression was chosen as an analytical method as it allows for a stronger test of a directional hypothesis. To this end, dependent variables have been recoded as binary values, without appreciably losing the desired information. The conflation of categories is listed in table 1 (overleaf).

Table 5 Dependent variables compacted to binary or tertile values.

Variable	Abbrev.	Type	Codes		Original Variable	Reference
Polygamy	v79_1r	Binary	0 = Monogamous 1 = Polygamous		Polygamy (v79) 1 = Polyandry - primarily monogamous with some plural husbands 2 = Monogamy 3 = Polygyny < 20% plural wives (if more frequent than polyandry) 4 = Polygyny > 20% plural wives (if more frequent than polyandry)	Murdock & Wilson 1972
Elite Multi-Wives	v868_1	Binary	0 = No 1 = Yes		Multiple wives for Leaders, Headmen, Chiefs (v868) 0 = No, or unimportant 1 = Yes, or Leaders have more wives than commoners	White 1988
Permit Extramarital (M) Permit Extramarital (F)	xmarm xmarf	Binary	xmarm 0 = No (3,4) 1 = Yes (1,2)	xmarf 0 = N (2,3,4) 1 = Y (1)	Extramarital Sex (v169) 1 = Single standard (both allowed) 2 = Double standard (husbands only) 3 = Double standard (both forbidden, women punished more) 4 = Single standard (both condemned equally)	Broude & Greene 1976
Increasing Polygyny	v860_1	Ordinal	1 = Monogamy prescribed or preferred 2 = Polygyny preferred by leaders and men if higher social class 3 = Polygyny preferred by most men		Cultural Basis of Polygyny (v860) 1 = Monogamy prescribed 2 = Monogamy preferred, but exceptional cases of polygyny 3 = Polygyny preferred by individual men with leadership attributes (chiefs, medicine men, outstanding hunters) 4 = Polygyny preferred by men of a higher social class: men of wealth, rank, nobility, etc. 5 = Polygyny preferred by most men and attained by most men of sufficient years or wealth to obtain wives.	White 1988
Premarital Sex Freq (M) Premarital Sex Freq (F)	v166_2 v167_2	Ordinal Ordinal	1 = Occasional or uncommon 2 = Moderate 3 = Universal		Frequency of Premarital Sex – Male (v166) / - Female (v167) 1 = Universal 2 = Moderate 3 = Occasional 4 = Uncommon	Broude & Greene 1976
Sexual Expression (AB) Sexual Expression (AG)	v827 v828	Ordinal Ordinal	Reduced to 3 categories in the version of the SCCS used.		Sexual Expression in Adolescent Boys (v827) and Girls (v828) [1] Scale from 0-10, where: 2 is not approved, 5 is normally and generally approved, and 8 is strongly approved and values	Barry & Schlegel 1984

Increasing Polygyny	v860_1	Ordinal	1 = Monogamy prescribed or preferred 2 = Polygyny preferred by leaders and men if higher social class 3 = Polygyny preferred by most men	Cultural Basis of Polygyny (v860) 1 = Monogamy prescribed 2 = Monogamy preferred, but exceptional cases of polygyny 3 = Polygyny preferred by individual men with leadership attributes (chiefs, medicine men, outstanding hunters) 4 = Polygyny preferred by men of a higher social class: men of wealth, rank, nobility, etc. 5 = Polygyny preferred by most men and attained by most men of sufficient years or wealth to obtain wives.	White 1988
Sexual Nonrestraint (AB)	v829	Ordinal	Reduced to 3 categories in the version of the SCCS used.	Sexual Nonrestraint in Adolescent Boys (v829) and Girls (v830) [2] Scale from 0-10, where: 2 is strictly and effectively prohibited, 5 is disapproved, and 8 is condoned and not generally punished.	Barry & Schlegel 1984
Sexual Nonrestraint (AG)	v830	Ordinal			

[1] These variables are considered to constitute a reasonable sample of attitudes to sexual permissiveness. Encouragement of sexual behaviour, taking into account its frequency, emotional intensity, importance, and variety (including range of partners) in adolescence Heterosexual intercourse is the principal criterion, but heterosexual foreplay, masturbation, homosexuality, sexual jokes, and exposing the genitals were also considered.

RESULTS

Table 6 presents the odds ratios for the logit and ologit models. We present below descriptions of the results for models with significant effects including the logit model for xmarm (C) and the ologit models for polygyny (L, LQ, C, LG, LC, CG and CC)

1. xmarm (C)

For this model the Cashdan & Steele pathogen score is treated as a continuous variable. In this simple linear regression the odds ratio of 1.077 ($p<.0.05$) indicates that for a one-unit increase in the Cashdan & Steele pathogen score the odds of extramarital sex for males increases by a factor of 1.077.

Polygyny (L, LQ, C, LG, LC, CG, CC)

For models L, LQ, C, LG, LC, GC and CC odds ratios are significant for some levels of pathogen stress. When Low's pathogen scores are considered to be continuous, 1.207 is the proportional odds ratio for a 1-unit increase in Low pathogen score on polygyny level. Thus, for a one-unit increase in Low pathogen score the odds of polygyny level 3 (polygyny preferred by most men) versus the combined levels 1 and 2 (monogamy preferred by leaders and men of high status) are 1.207 times greater. Likewise for a one unit increase in the Low pathogen score the odds ratio of the combined high and middle polygyny level (levels 2 and 3) versus level 1 (monogamy preferred) is also 1.207 times greater. Again, the introduction of the quadratic term is significant ($p<0.001$) indicating that the relationship may be curvilinear. The pattern for C and CQ is similar but not significant.

Considering the tertile form of the pathogen score data it can be seen that for the Low tertiles ordinal logistic regressions (LG and LC) moderate pathogen pressure compared to low pathogen pressure leads to odds ratios of <1, while for high pathogen pressure compared to low pathogen

pressure the ORs are 4.12 ($p<0.001$) and 6.574 ($p<001$) respectively. These odds ratios are for both polygyny level 3 versus the combined level 1 and 2 and for polygyny levels 2 and 3 versus level 1. The Cashdan & Steel ordinal logistic regressions (CG and CC) have ORs >1 for moderate pathogen pressure compared to low pathogen pressure. This difference is possibly due to the inclusion of more pathologies for the Cashdan & Steele tertiles. The OR for high pathogen pressure versus low pathogen pressure is also highly significant ($p<0.01$) for CG and CC.

Other patterns can be observed in some models but they are non-significant. Table 7 gives the signs for odds ratios relative to 1, that is, +indicates >1 and – indicates <1. Listed below are some of these consistent patterns.

1. Polygamy. For LG, LC, GC and CC the odds ratio for moderate pathogen stress is <1 while the odds ratio for high pathogen stress is >1.
2. Elite multi-wives. For LC, CG and CC the odds ratios for both moderate and high pathogen stress are <1.
3. Permit extramarital (male). For LG, CG and the odds ratios for both moderate and high pathogen stress are >1.
4. Permit extramarital (female). Only CG and CC are consistent with the odds ratio for moderate pathogen stress <1 and for high pathogen stress >1.
5. PM sex frequency (male). The two Low tertile models are consistent in odds ratio signs, but they differ in the odds ratio sign for high pathogen pressure from models CG and GG.
6. PM sex frequency (female). The regression models L, LQ, C and CQ are consistent with each other. However, for the tertile models, only CG and CC are consistent with the odds ratios for moderate and high pathogen stress <1.
7. Sexual expression (adolescent boys). Models L, LQ, C and CQ are consistent in direction. For the tertile models LG, CG and CC odds ratios for moderate and high pathogen stress are <1.
8. Sexual expression (adolescent girls). The models L, LQ, C and CQ are consistent in direction, The Low tertile models differ from the

Cashdan & Steele models.

9. Sexual non-restraint (adolescent boys). Only the two Cashdan & Steele models (CG and CC) are consistent in signage.

10. Sexual non-restraint (adolescent girls). Again, only the two Cashdan & Steele models (CG and CC) are consistent in signage.

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Table 6: Odds ratios for logit and ordered logit models.

	L	LQ	C	CQ	LG	LC	CG	CC
Polygamy								
Low Pathogens	1.080	0.566						
v1260sq		1.027						
Cashdan Pathogens			1.021	0.833				
pathogenscoresq				1.006				
Low G Moderate					0.841			
Low G High					1.700			
Low C Moderate						0.821		
Low C High						1.684		
Cashdan G Moderate							0.917	
Cashdan G High							1.429	
Cashdan C Moderate								0.785
Cashdan C High								2.663
N	186	186	186	186	186	186	186	186
AIC	169.5	168.7	171.3	172.6	171.6	172.0	172.7	170.7
Elite Multi-Wives								
Low Pathogens	1.018	1.412						
v1260sq		0.987						
Cashdan Pathogens			0.978	1.315				
pathogenscoresq				0.991				
Low G Moderate					1.685			
Low G High					0.933			
Low C Moderate						0.775		
Low C High						0.753		
Cashdan G Moderate							0.802	

Cashdan G High							0.670	
Cashdan C Moderate								0.883
Cashdan C High								0.441
N	155	155	155	155	155	155	155	155
AIC	211.6	212.4	211.3	211.2	211.4	213.1	212.8	211.1
xmarm								
Low Pathogens	1.075	1.262						
v1260sq		0.994						
Cashdan Pathogens			1.077*	1.212				
pathogenscoresq				0.996				
Low G Moderate					1.624			
Low G High					1.778			
Low C Moderate						0.923		
Low C High						1.481		
Cashdan G Moderate							1.618	
Cashdan G High							2.614	
Cashdan C Moderate								1.545
Cashdan C High								2.167
N	109	109	109	109	109	109	109	109
AIC	151.5	153.3	149.3	151.0	153.8	154.6	151.7	153.2
xmarf								
Low Pathogens	0.963	1.444						
v1260sq		0.984						
Cashdan Pathogens			1.024	1.003				
pathogenscoresq				1.001				
Low G Moderate					0.683			
Low G High					0.971			
Low C Moderate						1.333		
Low C High						0.852		
Cashdan G Moderate							0.667	
Cashdan G High							1.714	
Cashdan C Moderate								0.604
Cashdan C High								2.095
N	109	109	109	109	109	109	109	109
AIC	83.4	84.9	83.5	85.5	85.4	85.4	84.0	83.7

p<0.05, ** p<0.01, *** p<0.001

Table 6 Odds ratios for logit and ordered logit models (cont.).

	L	LQ	C	CQ	LG	LC	CG	CC
Increasing Polygyny								
Low Pathogens	1.207***	0.390***						
v1260sq		1.047***						
Cashdan Pathogens			1.081**	0.844				
pathogenscoresq				1.008				
Low G Low					1.000			
Low G Moderate					0.873			
Low G High					4.123***			
Low C Low						1.000		
Low C Moderate						0.732		
Low C High						6.574***		
Cashdan G Low							1.000	
Cashdan G Moderate							1.394	
Cashdan G High							2.692**	
Cashdan C Low								1.000
Cashdan C Moderate								1.582
Cashdan C High								3.846**
N	180	180	180	180	180	180	180	180
AIC	370.3	354.3	385.4	385.3	373.8	364.9	387.7	386.5
PM Sex Freq M								
Low Pathogens	1.019	0.962						
v1260sq		1.002						
Cashdan Pathogens			0.975	1.077				
pathogenscoresq				0.997				
Low G Low					1.000			
Low G Moderate					0.612			
Low G High					1.540			
Low C Low						1.000		
Low C Moderate						0.685		
Low C High						1.978		
Cashdan G Low							1.000	
Cashdan G Moderate							0.768	
Cashdan G High							0.870	
Cashdan C Low								1.000

Cashdan C Moderate								0.630
Cashdan C High								0.582
N	102	102	102	102	102	102	102	102
AIC	201.4	203.4	201.1	202.9	200.3	200.0	203.3	202.0
PM Sex Freq F								
Low Pathogens	1.027	1.266						
v1260sq		0.992						
Cashdan Pathogens			1.003	1.292				
pathogenscoresq				0.992				
Low G Low					1.000			
Low G Moderate					1.015			
Low G High					1.399			
Low C Low						1.000		
Low C Moderate						0.746		
Low C High						1.473		
Cashdan G Low							1.000	
Cashdan G Moderate							0.922	
Cashdan G High							0.985	
Cashdan C Low								1.000
Cashdan C Moderate								0.787
Cashdan C High								0.916
N	109	109	109	109	109	109	109	109
AIC	229.7	231.3	230.0	230.5	231.3	230.4	232.0	231.7

- p<0.05, ** p<0.01, *** p<0.001

Table 6 (cont.): Odds ratios for logit and ordered logit models.

	L	LQ	C	CQ	LG	LC	CG	CC
Sex Expression (AB)								
Low Pathogens	0.974	0.816						
v1260sq		1.007						
Cashdan Pathogens			0.967	0.961				
pathogenscoresq				1.000				
Low G Moderate					0.981			
Low G High					0.756			
Low C Moderate						1.046		
Low C High						0.832		
Cashdan G Moderate							0.526	
Cashdan G High							0.556	
Cashdan C Moderate								0.510
Cashdan C High								0.842
N	104	104	104	104	104	104	104	104
AIC	230.0	231.7	229.3	231.3	231.8	232.0	229.9	229.6
Sex Expression (AG)								
Low Pathogens	1.030	1.099						
v1260sq		0.997						
Cashdan Pathogens			1.004	1.005				
pathogenscoresq				1.000				
Low G Moderate					1.675			
Low G High					1.205			
Low C Moderate						1.133		
Low C High						1.133		
Cashdan G Moderate							0.698	
Cashdan G High							0.810	
Cashdan C Moderate								0.690
Cashdan C High								1.379
N	102	102	102	102	102	102	102	102
AIC	229.2	231.2	229.6	231.6	230.3	231.5	230.9	230.1
Sex Non-R (AB)								
Low Pathogens	1.011	0.881						
v1260sq		1.005						
Cashdan Pathogens			0.954	1.020				

pathogenscoresq				0.998				
Low G Moderate					0.610			
Low G High					1.248			
Low C Moderate						1.158		
Low C High						1.377		
Cashdan G Moderate							0.596	
Cashdan G High							0.492	
Cashdan C Moderate								0.729
Cashdan C High								0.678
N	96	96	96	96	96	96	96	96
AIC	207.8	209.6	206.1	208.0	207.9	209.3	207.5	209.0
Sex Non-R (AG)								
Low Pathogens	1.014	1.147						
v1260sq		0.995						
Cashdan Pathogens			0.951	1.315				
pathogenscoresq				0.990				
Low G Moderate					0.865			
Low G High					1.348			
Low C Moderate						1.126		
Low C High						1.552		
Cashdan G Moderate							0.831	
Cashdan G High							0.458	
Cashdan C Moderate								0.697
Cashdan C High								0.480
N	105	105	105	105	105	105	105	105
AIC	236.2	238.1	234.1	233.7	237.4	237.2	235.3	236.3

* p<0.05, ** p<0.01, *** p<0.001

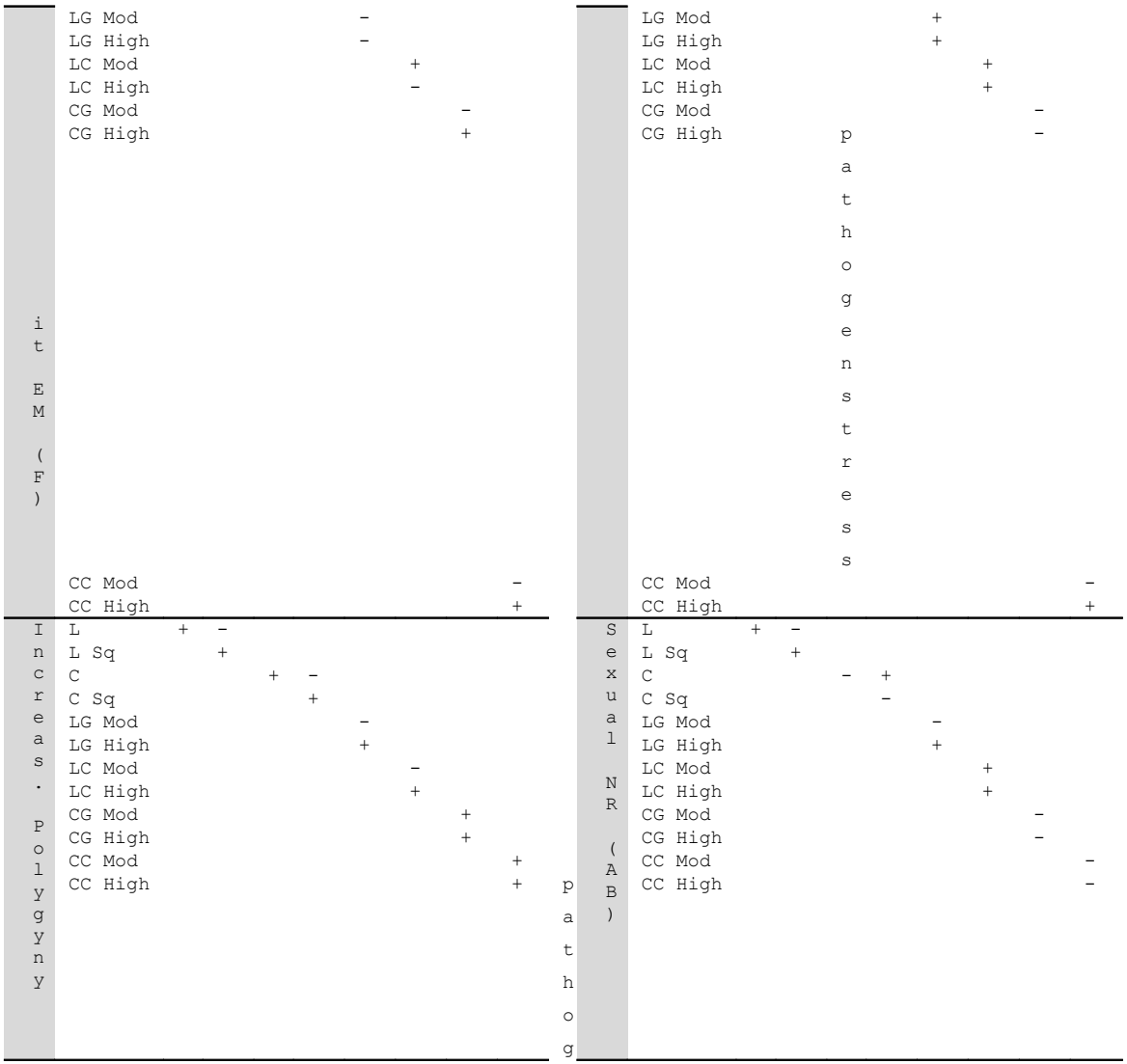
Table 7 Signs for model results: Normal DVs.

		L	LQ	C	CQ	LG	LC	CG	CC
Poly	L	+	−						
	L Sq		+						
	C			+	−				
	C Sq				+				

		L	LQ	C	CQ	LG	LC	CG	CC
PM	L	+	−						
	L Sq		+						
	C			−	+				
S	C Sq				−				

gamy	LG Mod					–			
	LG High					+			
	LC Mod						–		
	LC High						+		
	CG Mod							–	
	CG High							+	
	CC Mod								–
	CC High								+
Elite Multi-Wives	L	+	+						
	L Sq		–						
	C			–	+				
	C Sq				–				
	LG Mod					+			
	LG High					–			
	LC Mod						–		
	LC High						–		
	CG Mod							–	
	CG High							–	
	CC Mod								–
	CC High								–
● Permit EM (M)	L	+	+						
	L Sq		–						
	C			+	+				
	C Sq				–				
	LG Mod					+			
	LG High					+			
	LC Mod						–		
	LC High						+		
	CG Mod							+	
	CG High							+	
	CC Mod								+
	CC High								+
Perm	L	–	+						
	L Sq		–						
	C			+	+				
	C Sq				+				

ex Fre	LG Mod					–			
	LG High					+			
	LC Mod						–		
	LC High						+		
	CG Mod							–	
	CG High							–	
	CC Mod								–
	CC High								–
e PM Sex Freq (F)	L	+	+						
	L Sq		–						
	C			+	+				
	C Sq				–				
	LG Mod					+			
	LG High					+			
	LC Mod						–		
	LC High						+		
	CG Mod							–	
	CG High							–	
	CC Mod								–
	CC High								–
Sexual Exp (AB)	L	–	–						
	L Sq		+						
	C			–	–				
	C Sq				+				
	LG Mod					–			
	LG High					–			
	LC Mod						+		
	LC High						–		
	CG Mod							–	
	CG High							–	
	CC Mod								–
	CC High								–
	L	+	+						
	L Sq		–						
	C			+	+				
	C Sq				–				


Increas. Polygyny
LG Mod
LG High
LC Mod
LC High
CG Mod
CG High
CC Mod
CC High
L
L Sq
C
C Sq
LG Mod
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LC Mod
LC High
CG Mod
CG High
CC Mod
CC High
pathogen
Sexual NR (AB)
LG Mod
LG High
LC Mod
LC High
CG Mod
CG High
CC Mod
CC High
L
L Sq
C
C Sq
LG Mod
LG High
LC Mod
LC High
CG Mod
CG High
CC Mod
CC High

en stress

Sexual NR (AG)	L	+	+						
	L Sq		-						
	C			-	+				
	C Sq				-				
	LG Mod					-			
	LG High					+			
	LC Mod						+		
	LC High						+		
	CG Mod							-	
	CG High							-	
	CC Mod								-
	CC High								-

ETHICS STATEMENT

The SCCS (Murdock 1969) is a widely accepted tool for the purpose of cross-cultural analysis. Although some coded variables used herein refer to minor individuals, it is the understanding of the author that use of this information does not breach contemporary standards of ethical practice as these individuals are not identifiable.

DISCUSSION AND CONCLUSION

It can hardly escape notice that polygyny is the variable of greatest statistical significance, at multiple levels, in both the Cashdan & Steele 2013 and Low 1994 data sets. Ostensibly this might appear to support either, rendering the discussion moot. Yet this lays fertile ground for a different interpretation which has no such complications. While females remain comparably promiscuous under high pathogen pressure. The male bias towards multiple spouses might be better by the sexy-son hypothesis, first proposed by Ronald Fisher and which has received considerable support (Weatherhead T.J. & Robertson RJ, 1979). This may seem unintuitive insofar as it divides the male's parental provisioning ability. Under this hypothesis, female choice is of great importance, as the hypothesis contains the argument that females should choose to mate with the most desirable (typically high-status or physically healthy) males as men do to some degree, thus to have the most desirable offspring in survival terms under conditions of high mortality. However, each reproductive event is often more costly, terms of investment of time, risk and metabolic resources, than it is for males and this is reflected in the decision to seek the provisioning of a high status male. By contrast, male reproduction is less costly. In any event, in neither of the hypotheses tested is it apparent that there a compelling argument to be made that sexual promiscuity aversion should be sex-biased.

LIMITATIONS

· For many societies no data is coded in regard to the topics of interest. This is recognised as a limitation on the strength of the analysis. This could have been resolved to a degree by chained multiple imputation (whereby reasonable values are assigned to cells missing data).

· Sexual norms are unlikely to be modulated by pathogen stress alone; modes of social organisation are subject to heterogeneous pressures (such as foraging strategies, or norms of inheritance). Due to the low counts in many variables, as well as the fact that data is missing for many societies (which differ between variables), employment of a more exhaustive set of variables would likely result in an analysis with no useful data to analyse.

· The premise of this analysis is partially (though only partially) contingent on awareness of the routes of disease transmission in studied populations; this is not universally the case and tends to reflect a WEIRD view of the phenomenon. While it is held that the biomedical theory of disease mission is valid, a substantial proportion of cultures in the SCCS are more likely to attribute illness to aggression by spirits, to sorcery, or to other supernatural causes that do not discernibly resemble a germ theory of disease or analogue thereof (Murdock et al. 1980). This does not mean they could not plausibly act in the manner describe from the outset, but they may report the incidence of disease very differently.

· The SCCS does not document diseases whose primary route of transmission is sexual interaction. Having such data may be useful in further analyses. The historical sources from which pathogen prevalence data was derived have a low spatial resolution and are not of the same quality and thoroughness as contemporary cultural or epidemiological surveys. However, given the need to use historical sources appropriate to the focal years of the societies represented, this was judged to be is an insoluble issue.

· The TPS given in the SCCS and in the newer data generated by Cashdan and Steele (2013) is composed of the total score of each individual pathogen score combined. Whether this provides a valid representation of the true strength of selective pressure likely to have been exerted by an overall parasite

burden is uncertain. Multiple low scores may produce a rank equivalent to a single high score, and it is not clear that these represent equivalent pressures. In the absence of the ability to derive more detailed and quantitative pathogen prevalence data, however, this issue is also likely insoluble. Because the societies described in the SCCS have focal years significantly removed from the present, little can be done toward the end of increasing the precision of pathogen prevalence estimates. Searches for additional historical sources to better refine pathogen prevalence data revealed no candidate literature. This is, therefore, yet again beyond the author's capacity to solve.

· Pathogen codes in the SCCS are heavily biased towards categories of disease transmitted via arthropod vectors, which are significantly more prevalent in tropical regions. Thus the correlation between disease prevalence and latitude is likely to be exaggerated. Diseases that follow human-to-human respiratory droplet transmission routes to infection are thus underrepresented, as are those for which other (non-arthropod) zoonotic reservoirs exist (Cashdan 7 Steele 2013). Some extremely important pathogens with cosmopolitan distributions, such as tuberculosis, are not represented. Most critically, diseases for which the primary vector is sexual contact are not represented. Each of these issues is, unfortunately, insoluble in the absence of more complete historical data.

· Extant or historically recent societies occupying a premodern niche are not necessarily directly comparable to ancestral groups, despite likely sharing certain important similarities (such as modes of social organisation, foraging strategies, mobility patterns and political).
· This study only tests a correlation, not a direct causal relationship. It could be that pathogen prevalence is high because sexual mores are looser. Inferences and deductions derived are therefore treated as topics of interest, but not 'proof' as such.
Other statistical techniques may be required to fully investigate these data, for example, polychoric principal component analysis.

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