THE DEMOGRAPHIC TRANSITION IS ROOTED IN SOCIAL STRESSING OF MALES TRIGGERING EPIGENETICALLY INDUCED INFERTILITY OF EXOGAMOUS (FEMALE) OFFSPRING, TO COMPROMISE OUT-GROUP REPRODUCTION, FORESTALLING NATAL GROUP EXTINCTION

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ABSTRACT

A hitherto unidentified underlying basis of the globally ubiquitous demographic transition is suspected from causality-testing economic models, problems with anthropological hypotheses, and the longstanding opacity of aetiology unrelieved by integrating theory from different fields. Given a newly found main factor of population density, there is an apparent biological basis that rather than out-breeding depression is likely the now-replicated finding of chronic crowding stress of male mammals epigenetically transmitted to female offspring only, triggering multi-generational fertility decline and aberrant reproduction-related behaviours. The mechanistic core is found in humans. It's adaptive in aiding natal-group survival of neighbouring-group hostility, through exogamous natal-group females severely compromising out-group male reproduction, without impeding that of natal-group males; this complementing apparent female clique-based facultative co-operative breeding.

Keywords: demographic transition, social stress, male, epigenetic, out-group

IMPASSE

There continues, as ever, to be little or no advance in understanding the profound decline in fertility known as the demographic transition (DT), first outlined almost a century ago by Thompson (1929). Considered globally ubiquitous (e.g. Lee & Reher, 2011), DT has always been theorised as essentially an economic phenomenon, currently most commonly in the form of *unified growth theory* (e.g. Cervellati & Sunde, 2013), which combines economic development with another putative root factor, emphasised by demographers, of falling child mortality. This last some now claim is the key driver (Mühlhoff, 2021; Ranganathan, Swain & Sumpter, 2015), though it's itself symptom more than cause. Aetiology in economics terms is strained by the heterogeneity of conditions in which DT takes place—any permutation of rich or poor, urban or rural—indicating the likelihood of a more profound underlying basis.

The lack of progress is despite a vast literature that is regularly complained of as being as messily contradictory as it is large (e.g. Shenk et al, 2013). There is "no agreement among researchers regarding the main cause" (Orekhov, Prichina & Shchennikova, 2019). It is "impossible to be precise about the various causal factors", is Frejka's (2016) conclusion, noting this confirms that of 70 years ago by Notestein (1945 and 1953). It cannot even be determined as to what *level* of explanation is primarily causative, according to Colleran (2016), nor the direction of causation (e.g. O'Sullivan, 2013), nor if putative causes are not instead *products* of DT or in a reciprocal relationship with it (e.g. Canning, 2011). Not only is there "no accepted theory of human fertility" (Leridon, 2015), and no single model, with instead "a great diversity" (Zavala, 2022), but no means of deciding between them, given "studies describe and quantify but don't explain", Elspeth (2021) complains. Vishnevsky (2017) goes even further, concluding that existing models "do not allow us to go beyond the descriptive level, and, in fact do not give reason to speak of a theory"; echoing Teitelbaum (1987), who criticised DT modelling as "notably lacking in such components of theories as a specifiable and measurable mechanism of 'causation'" (p421). The upshot, and confirming that modelling thus far has been woeful, is that none has proved to have any predictive value at all (Snopkowski & Kaplan, 2018).

With the quest to find a general aetiology all but stalled, it has given way to studies of particular instances—specific countries within certain periods (the great majority of papers now being published)—or appeals for more complex aetiology and/or to integrate approaches.

Complexity—*economic complexity*—is itself posited as a new factor by Innocenti, Vignoli & Lazzeretti (2021), and chaos & complexity theory is invoked by Erçetin, Ray & Sen (2019). Asking *what can we learn from evolutionary demography?*, Sear, Lawson, Kaplan & Shenk (2016) are unable to point to progress in unravelling complexities; only its prospect in demography taking a more evolutionary biological direction, that Morita (2018) hopes will incorporate behavioral ecology, evolutionary psychology and cultural evolution, in the hope of a "deeper understanding". There appears no less of an impasse in studying from a biological (evolutionary, or a cultural evolution) perspective as from the economic. It may be that indeed it is complexity that has precluded elucidation, but an apparent requirement for ever more theoretical elaboration is a telling sign in science that new, fundamentally different, simpler hypotheses are required.

THE IMPLAUSIBILITY OF CURRENT THEORY

A theory of fertility according to economic principles is inherently implausible. As Pritchett & Viarengo (2012) outline and decry, in economics not only are children regarded as mere *goods*, but *inferior goods* (goods for which demand declines with income); this deemed necessary to explain why socio-economic improvement is linked with declining fertility, given that the overall premise in economics is a motivation to maximise the acquisition of goods, in a cost-benefit analysis by a rational actor. Evolutionary psychology (not to mention common sense) shows decisively that no part of this premise is a basis of human behaviour or cognition. Resources (goods) are instrumental to having offspring, which last are (and are profoundly felt to be) the embodiment of their parents' futures. Children are in no sense goods. Neither is deciding whether or not to have progeny predicated on a child being an indirect means of acquiring resources; that is, envisaged as a potential acquirer of resources in their own right, on behalf of the parents.

In economic modelling it is taken that in precluding child labour the onset of schooling renders children a burden instead of an asset. It is even claimed that as "the generalized child price relative to the child quality price rose, the rising opportunity cost of education was as decisive for the transition as the parental shift to child quality" (Foreman-Peck & Zhou, 2019). Yet a claim that the association of schooling with declining fertility is causal is non-cogent in the very terms of economic modelling. In former times (and as currently in some parts of the world),

even when offspring were maximally exploited as labour they had no economic value for several years, until after middle childhood, and then could be of only marginal value as extra helping hands, either in the home/farm-based economy or in very-low-paid employment. It's by reason of the extra value it conferred to labour—even to child labour—that schooling became necessary, so as to prepare for the different, higher skill-sets required by a more technologically developed economy. Notwithstanding in that this meant children entering the labour market at an older age, in that they did so as more formal, better-paid employees, they thereby contributed significantly more to the family household. In this way, economic development likely actually facilitated fertility rather than precipitated the decline that is the foundational claim in DT hypothesising.

Hardly more plausible is a particular biological perspective that DT may be (straightforwardly) adaptive in there being overall a reproductive pay-off through a smaller number of offspring receiving much higher parental investment. It turns out this produces no more or actually fewer grandchildren and great-grandchildren (Goodman, Koupil & Lawson, 2012; confirming previous findings), and even with very high levels of economic development an anticipated bounce-back of fertility has not materialised (Gaddy, 2021). This is hardly surprising, as economically enhanced upbringing may bring to fruition but cannot actually improve offspring in terms of genetic quality or fertility, the basis respectively of female and male mate choice, which anyway is a zero-sum phenomenon, so that attempting to raise all in effect raises none. So, the conditions that give rise to lower numbers of offspring are likely to result in the maintenance of a lower rate of reproduction, rather than a longer-term uptick.

There has been a failure to establish even the simple cultural evolution hypothesis of kin influencing fertility decision-making—transmitting pro-natal ideation—that in its absence purportedly precipitates DT. Only "mixed evidence" (Stulp & Barrett, 2021). A review by Sear & Coall (2011) reveals merely a "tentative" link to fertility, and even then, this is of mixed direction – fertility may be either decreased or increased in any given set of circumstances, with different kin relationships often being opposed in their impact. In a further review of all studies, Mathews & Sear (2013) find at most only partial support, a very weak effect only, and (of particular surprise) no specific kin relationship as a predominant conduit—not even the female's mother-in-law, who has a very strong evolved interest in her son's pair-bond partner increasing her

reproduction. The data anyway is merely correlational, and they concede "an alternative explanation is that there is some *unmeasured common factor* which predisposes women both to remain close to their kin, and to have relatively high fertility". Also could be posited not presence or absence of some hitherto undiscovered factor that rather indirectly impacts fertility, but one that more directly undermines it.

A CLOSER LOOK AT CAUSATION

Attempting to discover elusive causation, economist-demographers have used sophisticated comparative tests across competing hypotheses. Shenk et al (2013, 2011) used *likelihood theory* as a basis of weighing and deciding even between models whose predictions are not mutually exclusive. In considering several putative principal drivers of DT—child mortality decline, the economic costs of raising children, the benefits of investing in them, and the transmission of low-fertility social norms—they uncovered multiple causal pathways, yet failed to identify a root aetiology. *Panel co-integration estimators* and *Granger causality testing* likewise reveal only a circular causality: Herzer, Strulik & Vollmer (2012) show that the fertility changes in DT are both cause and consequence. In their first footnote, they suggest an as yet hidden underpinning to this circular causality: "Nevertheless it could be that Granger causality (testing) fails to identify true causality. It could be that the co-integrated variables are driven by another neglected process" (Herzer et al., p367). In general, what demographers and economists have discovered may well be merely inter-related second-order processes consequent to an underlying primary cause hitherto entirely missed.

POPULATION DENSITY IS THE NEWLY FOUND (PROXIMAL) FACTOR

A recently emerging likely basis—that is, a proxy (indirect measure) of a basis—of DT is population density. De la Croix & Gobbi (2017) studied both developed and developing countries, and "find a causal relationship from population density to fertility such that a rise in density from 10 to 1,000 inhabitants per square kilometer corresponds to a decrease in fertility of about 0.7 children. The corresponding half-life for population dynamics is of the order of four-five generations". When De la Croix & Gobbi's data was subjected to a new, better statistical analysis focused on effect size, population density was shown to be a still stronger determinant by a factor of three (Sterck, 2019). A still more recent investigation of 174 nations, controlling for many variables, by Rotella, Varnum, Sng & Grossmann (2021), reveals the same "robust"

association both within- and between-countries. The initial finding of the phenomenon, seemingly ignored at the time, was by Lutz et al (2006) from a study across 145 countries, in a follow up of work in 2002 (Lutz & Quiang). Controlling for key social and economic variables, they found "a consistent and significant negative relationship between human fertility and population density" and "that individual fertility preferences also decline with population density".

As to what mechanism explains why population density seemingly is key, Rotella's team set store by an application of life history theory. LHT (cogently outlined by Ellis et al, 2009) posits a distinction between *fast* and *slow* life history traits; *slow* traits being later onset of puberty, older age of first sexual intercourse & first birth, and increased parental investment. *Fast* traits are held to be prompted by a *harsh* and *unpredictable* environment. LHT is seen to apply to DT in emerging benign conditions precipitating a *slow* life history and consequent falls in fertility, but there are major problems here. The environment attending early stages of DT might be considered to be the very sort of *harsh* and *unpredictable* conditions that would lead to not a *slow* but a *fast* life history: a giving way of profound socio-economic stability of extended-family based small-scale community to internal migration and insecure wage labour in relatively anonymous urban concentrations or new industrialised or agribusiness settlements. It might be argued that a benign and predictable environment emerges only later in economic development, after DT has already ensued, at least in its early stages; and even that a benign, predictable environment is itself in part the consequence of a wide adoption of *slow* life history traits. Such contradiction in conceptualisation is a general issue with LHT, according to Stearns & Rodrigues (2020), notably in determining what is and what is not (and instead the opposite of) a *harsh* environment, and what constitutes a *fast* as opposed to a *slow* life history.

In any case, outline in terms of LHT appears more descriptive than explanatory: a now time-honoured criticism of what is put forward as DT theory. Are the traits less cause than manifestation of a changed attitude to fertility, begging the question as to what is the basis of the changed attitude? And might a changed attitude to fertility itself be a by-product of—a second-order response to—reduced fertility? With fertility reduction being so profound, and fertility matters so central in biology, an underpinning biological aetiology has to be strongly suspected.

COULD ROOT CAUSATION BE OUT-BREEDING DEPRESSION?

Conceivably, as Helgason et al (2008) suggest, this could be out-breeding depression (OD): the well-known phenomenon across species that as genetic distance between sexual partners becomes progressively greater, genetic incompatibilities arise that lower fertility (reproductive output), eventually to the point of sterility, even non-viability. It is the general case, irrespective of species, that fertility measured against relatedness (consanguinity) is an n-shaped curve. Very close relatedness produces the opposite *in*-breeding depression (from the pairing up of deleterious recessive alleles), a degree of non-relatedness being optimal in producing hybrid vigour, but still greater non-relatedness may start to lead to OD.

Helgason et al show that fertility (children and grandchildren produced) indeed follows an n-shaped curve, peaking at an average level of consanguinity of that between third and fourth cousins, in marked contrast to closer relatedness—at the level of second cousins or closer—in which case fertility is decreased, as would be expected through in-breeding. Fertility falls off significantly and monotonically (steadily) with decreasing relatedness from the level of third to fourth cousins (a coefficient of relatedness between roughly 0.75% and 0.2%), and to an extent sufficient to explain the fertility decline seen in DT. The dataset is Iceland's meticulous genealogical records from 1800 to 1965, the period during which there had been a change from a rural to an urban society, when the mean kinship between partners fell by a factor of ten, to that of fifth cousins. Weller & Santos (2013), using data from Brazil, likewise find third cousins to be the level of consanguinity at which fertility is greatest, with a difference on average of almost one child between the progeny of related and non-related couples. Endorsing Helgason, Fox (2015) stresses the importance of cross-cousin marriage in a balance between in- and out-breeding he dubs *mediogamy*, that he finds to be the strategy common to humans, mammals, birds, fish and insects to optimise fertility. It may well be that the third-to-fourth cousins sweet spot of maximum fertility is a primary determinant of the ancestral maximum or optimal size of human groups and their fractal fission-fusion nature, as appears to be represented in *Dunbar's number* (e.g. and including a discussion of relatedness, Dunbar & Sosis, 2018)—especially given evidence against the hypothesis that this is due to a ceiling to the number of other individuals with whom an individual has the cognitive capacity to deal (Lindenfors, Wartel & Lind, 2021).

The problem is that albeit the data fits the theory in being n-shaped, it's a correlation, not a causal relationship. There is an absence of research clearly demonstrating OD in humans, or to show at what degree of unrelatedness it would or might become apparent. Owing to inappropriate ideological concerns (the fear of the possibility of providing support for racism) there is a complete dearth of research extending out of animal studies to humans, bar what is purported to be the first evidence of it, in 2007 (Meyer et al), and this may be criticised for relying on a particular interpretation of the data. According to Reich (2018), studies of inter-breeding between modern human ethnic groups have found no evidence of OD. In general (across species), Frankham et al (2011) point out that "empirical data indicated that populations in similar environments had not developed OD even after thousands of generations of isolation", and that whether or not sub-populations have been separated in the same or different environments is key. They predict low OD for sub-populations separated for less than 20 generations in similar environments. Although there are human sub-populations that prior to very recent mutual contact had been separated for vastly more than 20 generations in different, often very different environments, and with not insignificantly contrasting genotypes, these are along ethnic fault-lines, which have little relevance to most historical DT, as largely it took place within ethnically homogeneous populations. Yet major internal migration often over considerable distances attended DT, so OD has to be considered likely a significant or highly significant contribution to DT, even if for now it cannot be regarded as the primary cause.

SOCIAL STRESS AS ROOT CAUSATION

The merely correlational nature of the data could indicate some other biological mechanism. Most obviously the stress caused by what may co-occur with decreasing mutual relatedness of spouses: potential *crowding stress* simply from the close proximity and high number of others, as a consequence of greater social mobility through dislocation in employment patterns leading to urbanisation and increasing population density. Alternatively, or additionally, the general relative absence of kin—not merely lesser kinship between pair-bond partners—may be stressful either in itself or through its being proxy for something that is. Non-kin in comparison to kin do not clearly share interests, essentially are strangers, and in some respects at least are likely to provide competition, which is an additional dimension to proximity and number of others in considering the impact of population density. Rather than *crowding*

stress per se, therefore, it is more appropriate to consider more generally *social stress*. And with increased population density in mutual relative anonymity a permanent new global reality, any stress caused would be chronic, which is known to have far more adverse, often opposite impacts to stress that is merely acute, having different neuro-hormonal physiology (e.g. Musazzi, Treccani & Popoli, 2015).

Social stress or more specifically *crowding stress* is very well recognised in mammals and in fish, not least in its negative impact on fertility. There is a considerable literature on all this (that is beyond the scope of this review), including on the endocrinology of the impact on the mammalian female menstrual system, with human females being no different in stress causing reproductive suppression (Wasser & Isenberg, 1986). Interestingly, females are more susceptible to experiencing *crowding stress* from same-sex others, as, unlike for males, this is not a function of proximity but of total numbers (Brown & Grunberg, 1995).

For obvious ethical reasons there is far less work on humans than on animal models, though one particular scenario of ostensible *social* or *crowding stress* experienced by human non-kin in close proximity that *has* attracted research is that of co-wives in polygynous households (and their evolutionary homologues, polygynous gorilla females), who sustain significant, even profound negative impact on fertility. Stress-induced infertility is a principal theme of the findings by Tabi, Doster & Cheney (2010) in their investigation of the lives of co-wives. The stress they feel would be more severe still but for male presence: it is now thought that pair-bonding (originally in the form of polygyny) in apes evolved from the need for individual males to be placed between small groups of females to prevent intra-female conflict. [This also is beyond the scope of discussion here: for a mini-review and discussion, placing in context, see Moxon (2021).] Note that again, here we would seem to be dealing with what would best be dubbed *social* rather than specifically *crowding stress*, given the mutual competition among co-wives for the attentions of their shared high-mate-value pair-bond partner.

It is difficult to disentangle the different types or elements of *social stress*, and using mammal models as the basis of understanding human response to population density may be unwarranted with humans instead appearing to cope (de Waal, Aureli & Judge, 2000). Yet this is in terms of not reacting with aggression, which is a superficial appreciation. Social withdrawal is the chief response (Evans et al, 2000), as it often is in mammals. O'Brien (2009) concludes that

people become "psychologically aloof, while also being socially removed from many of their neighbours", leading to a sharp fall in pro-social behaviours, with ensuing anti-social behaviours. Gomez (2017), citing Gray (2001), claims that urban communities, notwithstanding attempts at behavioral and societal modifications, succumb "to the abnormalities occasioned by the experience of cognitive overload or the perception of crowding that follows as a psychological effect of absolute increases in population density" (p9). Watve (2012) claims that "today's unprecedented density of human population is a super-normal stimulus that could change human behavior as well as physiology substantially." Fletcher (2007) theorises that for individuals struggling with the stress of social interaction there is actually a ceiling beyond which it drives reduction in residential density. If this is the case, then for the very many with nowhere else to go*, social stress* levels may rise to seriously compromise functionality.

That *crowding* or more generally *social stress* likely is key is apparent globally in concentration of population in nodes rather than in a uniform distribution. There has been an overwhelming shift to urban living, but the ubiquity of population concentration is still more apparent in considering rural-to-rural migration, that in lower-income countries is far more common than rural-to-urban (Lucas, 2007); with male labourers relocating from subsistence to plantation agriculture, and moving village between dry farming and irrigated areas. This entails proximity to non-kin & strangers and increased population density. In Ghana, for example, rural-rural permanent and seasonal migration of men so as to send remittances home has been the predominant pattern (Primavera, 2005). This was evident several decades ago in Kenya, where 40% of all migration was rural-rural as against 33% that was rural-urban (Oucho, 1984). That split was 68%/25% more recently in Nepal, and similarly in India (Deshingkar & Grimm, 2004). Oucho argued that: "students of migration have shown unwarranted obsession with rural-urban at the expense of rural-rural migration in the developing countries" (Oucho, 1984, p123). Deshingkar & Grimm (2004, pp11, 20 & 21) point out that rural-rural migration is: "the least visible because such migration is usually missed by official surveys (through) the inability to capture seasonal and part-time occupations; covering only registered migrants; and … owing to scattered locations of sending and receiving areas".

THE LIKELY BIOLOGICAL ROOT CAUSATION IN EPIGENETIC TRANSMISSION OF SOCIAL STRESS

A particularly interesting feature of *social stress*, as researched in mammals, is that there are sex-specific effects: a distinct response in males but not females—and this is held specifically to be in respect of *crowding stress* (Laviola et al, 2002). There is paternal transmission to offspring over several generations of chronic variable stress (e.g. Rodgers et al, 2015) or chronic paternal social defeat stress (Dietz et al, 2011); and epigenetic changes within the brains of stressed male individuals (Hunter, 2012). Most profoundly and intriguingly—and again specifically, supposedly, regarding *crowding stress*—is the triggering of an epigenetic transmission to female offspring to produce physiological fertility decline and degradation of reproductive-related behaviours of great effect sizes; this being in turn further transmitted down the female line for at least another two generations (the male's f1, f2 and f3 female offspring) (Saavedra-Rodríguez & Feig, 2012). This has been replicated by Kong et al (2021), who created social instability so as to induce stress, and pinpointed the epigenetic mechanism as the methylation of CAT genes in sperm. Previous investigation of the mechanism shows it entails reduced levels in sperm of certain microRNAs (nucleotide chains transcribed from DNA that regulate gene expression) as the response to stress exposure, and that the same sperm microRNAs were likewise altered *in men* who had been raised in families that were abusive, dysfunctional, or both (Dickson, Paulus & Mensah, 2018), suggesting the mechanism is homologous. That is, it has been evolutionarily conserved, leaving it present in humans as well as in lower mammals: as affirmed in a review by Wilson & Wallingford (2021). Note that in order for the stress to produce a transmissable effect, it has to be chronic, and not merely acute (Fennell *et al,* 2020), which is what applies in the global great increases in population density and concentration in urban and rural nodes.

Further refining research of the phenomenon, (Manners et al, 2019) find that prolonged chronic unpredictable stress of adolescent males not only transmits specifically along the female lineage (to offspring and grand-offspring), but that there is a *protective* impact on male offspring, which is also trans-generational; with this being distinguished in such males from normal (unstressed) males by differentially expressed genes within the amygdala of the brain, altering certain signalling pathways. Further confirming that the depressive impact is on female offspring only, Mashoodh, Habrylo, Gudsnuk & Champagne (2022) likewise discover that there is an

opposite impact on male offspring. Whereas female offspring show increased anxiety and depression-like behaviors, male offspring conversely demonstrate *reductions* in anxiety- and depression-behaviors, and, moreover, *promotion of exploratory behaviour*. It would appear that in contrast to reproductive suppression of female offspring, for male offspring the same mechanism *positively promotes reproduction*.

AN ADAPTATION TO REDUCE OUT-GROUP THREAT

This clearly defined, remarkable and surprising mechanism, is initially a puzzle. If it is some sort of adaptation serving temporarily to strategically reduce reproduction (if such could be theoretically conceived), then why, if female reproduction is suppressed, is male reproduction *actually promoted*? Evidently it is not a population-reduction adaptation, at least not primarily, per se. It does not appear to be a maladaptive by-product of an entirely different mechanism; one that although itself adaptive has a deleterious parallel impact. Such an adaptation would have to confer enormous value if it were ever to have been selected to fixation despite all the other selection against it through its deleterious baggage. For this mechanism to be adaptive it would be assumed that it has to *increase* reproduction overall over some time-scale, but a more nuanced understanding is that selection pressure would be greater if, rather, or more specifically, it functioned to *forestall reproductive collapse, indeed local group extinction.* Note that there is no invocation here of *group selection*; either naive or as recently reformulated. The stale debate on that topic having resolved into several empirically equivalent theoretical positions, and not just multi-level selection, satisfactorily explaining evident mutualism. [See the referenced discussion in several of the present author's papers, e.g. Moxon, 2017.]

It might be envisaged that in times of severe environmental, ecological stress and degradation, temporary reduction of reproduction serves to avoid the complete exhaustion of local resources and thereby to avoid local extinction, but unless this were not universal—so as to allow *some* females to continue to reproduce—the mechanism would itself tend to cause the very outcome it serves to forestall. If anyway a temporary *complete* cessation of reproduction could be adaptive, this would not account for why male reproduction is not only unsuppressed but boosted, nor why females rather than males specifically are targeted.

The mystery is resolved by taking into account human female exogamy. Given that human females, along with those of many other species, mostly *marry out* (thereby preventing in-

breeding depression), then male to female epigenetically transmitted fertility reduction would impact mainly not on natal group reproduction but on that of those out-group(s) to which females are exported. Indeed, there is likely no impact on natal group reproduction, in that of the females who remain, most or all would be either of low fertility and don't reproduce (or do so minimally), or are those few retained by—and likely themselves the offspring of—higher status (higher mate-value) natal males. These males, in being high status, are themselves physiologically protected from the impact of stress, so do not produce fertility-compromised offspring: neither female targets nor male vectors. This is through glucocorticoid stress hormones in high-rankers (in comparison to low-ranking males) quickly reverting to a low baseline (e.g. Summers & Winberg, 2006), and the higher testosterone levels in high-ranking males leading to a decrease in glucocorticoid levels owing to mutual antagonism (Glenn, 2009)—presumably through testosterone blocking Type II glucocorticoid receptors. Furthermore, the afore-mentioned actual *promotion* of male reproduction by the epigenetic mechanism would actually *boost* specifically natal-group reproduction, given that males, unlike females, *remain* in the natal group.

The adaptive value of the mechanism now becomes apparent. As it impacts out-group but not natal group reproduction, it would be of great assistance in forestalling any threat of natal group extinction in the face of hostility from an out-group. Such hostility would be chronically stressful for males, and would exist and persist in any scenario in which there is a threat of natal group extinction, not least that from severe local environmental (ecological) stress and degradation. In this scenario, even if there is no threat initially from such an out-group, such a threat would ensue, given that environmental stress almost certainly would be a simultaneous major problem for both the natal group and any groups adjacent, obliging inter-group contest over dwindling resources (De Dreu, Gross & Reddmann, 2022).

Such inter-group conflict, as with other inter-group conflict scenarios, might tend to produce group-reciprocal reproductive suppression of females. Albeit to some extent asymmetrical according to relative group strength, the mechanism then would function also to bring about a more general population reduction, across groups, that is potentially adaptive if, again, it forestalls extinction of each group. And note that adjacent groups are anyway usually related, in likely being sub-divisions of a former single group, given human fission-fusion

sociality, so an *inclusive fitness* perspective is applicable. As it's a mechanism that in extremis would precipitate what it otherwise precludes, overshoot is always a danger, yet if even merely statistically it avoids natal group extinction more than it would occur anyway, then there would be selection pressure in its favour.

COMPLEMENTARY FACULTATIVE CO-OPERATIVE BREEDING IN FEMALE CLIQUES

The mechanism would complement what appears to be human female facultative co-operative breeding in the functioning of human female cliques (Moxon, 2021). As outlined by Powell-Lunder (2013), Wiseman (2016) and Harley & MacNeil (2017), these typically consist of four or five females, the most physically, that is sexually attractive (fertile) of whom is dubbed the *queen bee*. Rather than an alpha in a dominance hierarchy, she's a *first among equals,* exerting firm control over the others, who take pains to serve her on pain of expulsion, though they (especially the *deputy*, the second most fertile female) are biding their time and ultimately aspire to usurp her. The facultative co-operative breeding hypothesis is that in order to head off the distinct possibility of the extinction of the local group at times of local severe environmental, ecological stress and degradation (or when for other reasons there are impossibly limited resources), the *queen bee* either behaviourally and/or physiologically reproductively suppresses the others at least to some degree if not completely. They thereby become available to serve as alloparents, usefully expressing their otherwise redundant reproductive motivation. The *queen bee* then faces no competition (or less so) for resources, as she would if there were parallel breeding females. This helps to ensure that at least one couple per clique produces offspring and that they survive, instead of risking no offspring surviving from any female clique member. The natal group apparently features one clique (often referred to in US high schools as the *populars*) that according to fertility criteria is predominant, and it may be that in extremis only this clique—and, therefore, just one couple—is allocated a reproductive role, with the rest of the local-group females remaining barren.

Clique-based facultative reproductive repression by *queen bees* further reveals that there is no problem posed to natal group reproduction by the epigenetic mechanism herein outlined. To reiterate, any natal females who do not *marry out* tend to be polarised between those anyway who would not reproduce (low-fertility females) and those particularly earmarked for reproduction (highly fertile females, pair-bonded with the most high-mate-value natal-group

males). Given such males do not confer epigenetically-induced reduction in fertility to their offspring (because high-status males are protected from the impact of stress), then natal group reproduction is not compromised. Apparently, the reduced female fertility resulting from clique-based facultative co-operative breeding would work in tandem with the mechanism to epigenetically transmit fertility reduction to females who become out-group members. Both aid natal-group survival by restricting reproduction; one as a temporary strategic narrowing of reproductive output of the natal group, the other as a longer-term blanket imposition on out-group competitors.

CONCLUSION

The root cause of DT is most likely the herein outlined epigenetic transmission to females of *social stress* experienced by males as a result of either disproportionate presence of non-related others, or sheer population density, or both, causing physiological fertility decline and aberrant reproduction-related behaviour that together very significantly lower reproductive output. There are surely additional contributions from direct impact of *social stress* (on both males and females) and—albeit an uncertainty of impact ranging from barely significant to large or very large indeed—from out-breeding depression. But even if DT were this simple at root, its manifestation would be at different levels, in various second-, and third-order processes, physiological, psychological, behavioural and socialital, giving the appearance of an intractable complex chain and mesh of causation. Those who hold DT to be a multi-factorially complex cultural phenomenon, elusive as to locus or level of causation, are obliged to consider that the logic of evolution dictates that all necessarily arises out of biology. Any and every evolutionary elaboration, not least a facility to engage in culture, functions to feed back to fine-tune and reinforce the very biology that gave rise to it. Otherwise, there would be no basis for such facility to have evolved in the first place. [For a fleshing out of this key point, see Moxon, 2010.] There has to be root biological cause, and the truly remarkable mechanism here outlined is the clear favourite as primary.

IMPLICATIONS

Taking *social stress* and the epigenetic mechanism here outlined as key (and possibly also out-breeding depression) the upshot is that with the root cause of DT being biological rather than cultural, it's not clear what or if changes could be made to ameliorate it. That is, there is a

question as to what changes if any could be made to living space so as to reduce the sense of *social stress* for any given population structure or density. [It's an unwarranted assumption in any case that global population reduction from its current and projected extremely high, likely unsustainable level is undesirable.] It is easy to see what would *not* work: to try to counteract the problem DT poses of adverse support ratios of workers to non-workers by population replacement through immigration is counter-productive, as this actually still further intensifies DT, so will exacerbate demographically driven economic implosion. It may be that the social milieu that does not precipitate *social stress* in that it more resembles ancestral communities in terms of structure and size—likely importantly that they feature a sufficient presence of relatively consanguinous individuals and/or a sufficient absence of non-consanguineous individuals—is impossible to re-achieve in highly developed societies. However, if a much greater proportion of the population is either obliged to or comes to realise again profoundly the value of remaining close to family and community roots, perhaps a significant difference could ensue. There may already be data available to be able to consider these questions, and now that the very basis of DT can be appreciated (rather than just its complexity), attempts to answer them might now be made.

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