Mate Choice and the Persistence of Maternal Mortality

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Abstract

Maternal mortality remains one of the leading causes of death in women of reproductive age in developing countries, and a major concern in some developed countries. It is puzzling why such a condition has not been reduced in frequency, if not eliminated, in the course of evolution. Maternal mortality is a complex phenomenon caused by several physiological and physical factors. Among the physical factors, maternal mortality due to fetopelvic disproportion remains controversial. Several explanations including evolution of bipedal locomotion, rapid brain growth, and nutritional changes and life style changes in settler communities have been proposed. The influences of human reproductive biology and sexual selection have rarely been considered to explain why maternal mortality persisted through human evolution. We entertain the hypothesis that irrespective of the causes, the risks of all factors causing maternal mortality would be aggravated by disassortative mating, specifically male preference for younger females who are generally small statured and at higher risk of obstetric complications. Maternal mortality arising due to sexual selection and mate choice would have the long-term effect of driving widowers toward younger women, often resulting in “child marriage,” which still remains a significant cause of maternal mortality globally. Evolutionarily, such a male driven mating system in polygamous human populations would have prolonged the persistence of maternal mortality despite selection acting against it. The effects may extend beyond maternal mortality because male-mate choice driven maternal mortality would reduce average reproductive life spans of women, thus influencing the evolution of menopause.

Keywords

mate choice, maternal mortality, sexual dimorphism, polygamy, obstetric dilemma, fetopelvic disproportion

Introduction

Maternal mortality continues to be one of the leading causes of death in women of reproductive age, with global estimates of ~200 to 400 deaths per 100 000—the higher incidences occurring in Sub-Saharan Africa, South Asia, and parts of the Middle East.¹⁻⁴ In the 1800s and the beginning of the 1900s, maternal mortalities were close to 1 in 100 births^{2,3,5-8} (Figure 1). These statistics accentuate the historical failure of societies for neglecting women’s health and well-being. In 1990, the World Health Organization (WHO) established 8 Millennium Development Goals; one of which was to improve maternal health and reduce maternal mortality by 3 quarters, by the year 2015. As a result of improved health care, maternal mortality has dropped by 45% since 1990.¹⁰

However, from an evolutionary standpoint, data in Figure 1 may warrant some retrospection; if recent historical rates of maternal mortality seem high, rates in prehistorical times—in hunter-gatherer hominin societies—could have been far greater. Maternal mortality is an ominous predicament because both mother and offspring are often at risk. Considering that ancestral human population sizes were likely quite small,¹¹ high maternal mortality rates may have proved dire for our

ancestors. The impending demise of females and offspring (particularly at disproportionate numbers) will invariably reduce mean population fitness. Hence, it is expected that natural selection should have acted strongly against maternal mortality through human evolutionary history. Yet, the high incidences in historical and modern populations would suggest otherwise and prompts evolutionary investigations of factors that can explain its persistence.

Maternal mortality occurs due to a variety of physiological and physical problems. These range from obstetric hemorrhage, embolism, and cardiovascular disorders that are among the top 5 causal factors to obstructive problems caused by disproportionately large fetal sizes relative to the mother’s pelvis, which account for about 6% to 8% of maternal mortality

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Figure 1. Trends of maternal mortality ratio (per 100 000 births) in different populations over time. Data obtained from studies by WHO,⁴ Loudon,⁸ and Klasen⁹; and Gapminder (<http://www.gapminder.org/data/>). Note that the graph only shows the major trends in each country, more detailed time trends can be obtained/viewed on Gapminder and WHO (http://www.who.int/gho/maternal_health/mortality/maternal/en/). *Data from rural Germany see the study by Klasen⁹, German maternal mortality rates from 1900 onward are from Gapminder.

worldwide.¹²⁻¹⁵ Of these, fetopelvic disproportion (FPD) and the evolution of difficult birth in humans has received much attention, and still remains a contentious issue. Some studies even suggest that FPD may perhaps act as a disposing factor for other causes of maternal mortality such as pregnancy-related sepsis and postpartum hemorrhage.^{16,17}

Fetopelvic Disproportion and Maternal Mortality

Human babies are large relative to the female pelvis, and the birth canal is rather elongated with variable diameters and angles.^{18,19} Due to the tight fit of the fetus in the birth canal, a series of rotational movements are required for the fetus to negotiate the birth canal during parturition, thus making birth-giving a painful and extended process.¹⁹ Whereas some anthropologists refer to the tight fit between the fetus and the pelvic canal as the obstetric dilemma,²⁰ for evolutionary biologists (and in this article), the obstetric dilemma refers to a hypothesis concerning human evolution. The obstetric dilemma is generally accepted to be a byproduct of the antagonistic selective pressures of bipedalism and optimal parturition.^{21,22} However, recent evidence indicates that the hominin pelvis evolved under much more complex drift-selection regimes, affected by a variety of factors²³ including bipedalism, climate, diet, and changing environments. Emerging studies also challenge the traditional idea of the obstetric dilemma suggesting that difficult birth is a recent phenomenon resulting from secular changes in diet and life style associated with agriculture.^{24,25}

Differing views are intensely debated and the scanty fossil record of hominins as well as the variety of methods used to determine hominin trait evolution adds to the confusion.²⁶

Fetopelvic Disproportion Through Human Evolution

Nevertheless, some lines of evidence do raise the possibility that FPD may perhaps have been a problem for at least a part of, if not throughout human evolutionary history. Patterns of pelvic changes in the fossil record strongly suggest that natural selection acted against obstructed labor, favoring anatomical changes for improved parturition.^{18,27,28} DeSilva's²⁹ allometric simulations suggest that compared to great apes and earlier hominins such as *Ardipithecus* (~5.8-5.2 million years ago [ma]), *Australopiths* (~4.2 ma) may have birthed large infants relative to female pelvic proportions. The fossil record also shows extensive variability in pelvic size estimates, ranging from pelvic proportions that may result in higher incidences of FPD in some samples of *Australopiths*, and none in other samples, see the study by Wells et al²⁵ for a review. Indeed, interpretations from scanty fossil evidence must be done with caution, but if this large variation is real, it would imply that there was substantial genetic variability for selection to act upon. This indirectly supports the possibility that obstructed labor was a problem, to varying degrees, in the past. As such, the persistence of obstructed labor suggests that new factors may have impeded the strength of natural selection against FPD. For instance, declines in overall human health and stature in postagricultural societies³⁰ may have exacerbated the risks of maternal mortality.^{25,31} More recently, Mitteroecker et al's mathematical model³² suggests that increased use of Caesarian delivery may have also exacerbated the risks of FPD across generations. Here, we entertain a hypothesis of how sexual selection, sexual antagonism, and human mating strategies may have prolonged the persistence of maternal mortality due to FPD and other causes, despite selection against it.

Sexual Selection, Mate Choice, and Maternal Mortality

Superficially, FPD may appear to be a problem for the mothers and of the mothers, but it is also influenced by the fathers. Increasing fetal size would exert pressure on female pelvic size to evolve, and thus the evolution of both traits can be expected to correlate.³³ Moreover, these traits would be affected by sexual dimorphism and sexual selection through mate choice, which together can influence the prevalence of FPD and maternal mortality in populations.

Sexual selection can produce life history changes in either sex³⁴ and sexual dimorphism can result from natural selection acting differently on male and female life history "niches" (eg, food acquisition and consumption). In addition, sexual selection/conflict can influence the evolution of sex specific traits in one sex that are not necessarily optimal for survival.³⁵ Even at the genetic level, the genetic load can be different for males and females,³⁶ and deleterious mutations can often strongly affect one sex while in the other sex, the effects may be modest or

absent altogether.³⁷ There is also evidence in some animals that changes in sexual dimorphism can occur as a response to selection on size-related fertility.³⁸ Yet, the problem of FPD and maternal mortality in general has rarely been discussed in the light of sexual selection. Geugan et al³⁹ have shown that size related mortality factors have the potential to influence the nature of sexual size dimorphism (SSD) in human populations, but whether or not changes in SSD driven by sexual selection can influence the proportion of FPD in a population has not been fully explored. A few studies have shown that SSD and parental stature can impact the risks of FPD and that these risks vary across populations.^{31,40} Mitteroecker et al's⁴¹ model of obstetric selection in humans clearly indicates the considerable influence of males on the problem of FPD. Their model suggests that a proportion of the population may always face the risk of FPD, and males exacerbate the risk-fraction of the population, that is, they make the fraction larger. Although they discuss the possible influence of sexual dimorphism and fitness differences between the sexes, the link between the two and the relevance of human reproductive strategies remain unexplored. Since FPD has received so much attention, we use FPD as a model to illustrate our hypothesis, which should also apply to other causes of maternal mortality.

The Hypothesis—Mate Choice, Maternal Mortality and Menopause: An Evolutionary 'Axis'

We present an argument, that regardless of the factors involved in causing maternal mortality, 3 aspects of the human mating system: (1) "incompatible" mating due to SSD, particularly mating between disproportionately large males and small females, and (2) age biased male mate choice, that is, male preference for younger women, along with (3) polygamy, serial monogamy or repeated mating by males; would have reduced the efficacy of selection against maternal mortality in the past and prolonged its persistence in human populations.

Sexual Size Dimorphism can Increase the Risk of Maternal Mortality

Parental Statures and the Risks of FPD

Assuming genetic variation for SSD, and given that a proportion of maternal mortality occurs due to FPD, a proportion of FPD could arise from size-incompatible mating (disproportionate size/height differences between partners). While superficially, women's size could be taken as the factor affecting maternal mortality, it is not unreasonable to ask if sexual dimorphism can influence the risk and magnitude of maternal mortality due to obstetric complications. This is likely since males also contribute to the production of neonates, but what are paternal influences on fetal proportions?

Several studies show that paternal stature affects fetal phenotype.^{42–46} For instance, in Klebanoff et al's⁴² Danish study, the association between paternal stature and offspring birth

weight was explained primarily by differences in paternal influence on fetal growth rate rather than gestation length. In a British cohort study, Knight et al⁴³ found that paternal height correlated with birth weight and birth length independently of maternal influence. However, neonatal size is a quantitative trait and both parents must exert influence over it. There is, however, much variation in this pattern across populations. Leary et al⁴⁴ showed that the influence of maternal stature on birth weight was consistent across populations, while that of the father's was less consistent. The association between parental statures on fetal phenotype does raise the possibility that sexual dimorphisms in populations might have the potential to influence the risk of obstetric problems.

Mascie-Taylor and Boldsen⁴⁷ examined the relationship between spousal height differences and pregnancy outcomes (reproductive success and abnormal outcomes) in a large British sample. Their analyses showed that parental height differences correlated with the risk of pregnancy-related complications including FPD. Spouses with greater differences in height had a higher probability of pregnancy-related problems, including FPD. Another study showed that maternal size and parental height differences influenced birth weight, and parental height differences as much as 20 to 30 cm affected the likelihood of requiring emergency cesarean sections (CSs).⁴⁰ The key finding relevant to our hypotheses is that women with relatively tall partners compared to women with relatively short partners had a higher risk of emergency CS, particularly in high birth weight cases. Since women with short stature are already at highest risk of adverse pregnancy outcomes, parental height differences appeared to have little additional effect on the risk of emergency CS. However, even average and tall women were at higher risk of emergency CS if their partners were relatively taller.⁴⁰ Indeed, these data may not be "evidence" for the influence of sexual dimorphism on FPD risk, but they do indirectly reinforce the possibility. More recently, Mitteroecker and Fischer⁴⁸ showed that the mother's head size and stature, parental height differences, and fetal head proportions can interact variably to determine adverse pregnancy outcomes.

Sexual Dimorphism, FPD, and Maternal Mortality Through Human Evolution

Through the changing face of sexual dimorphism during human evolution, there would have been some degree of non-overlapping distribution of the sizes of males and females. Under a random mating system, a proportion of mating would have involved small statured females and tall males, and a fraction of these, as Stulp et al⁴⁰ have suggested, would have relatively higher risks of FPD. Height is heritable and is influenced by environmental conditions, which will affect the magnitude of risk arising from size incompatible matings across populations with variable height distributions. Nevertheless, in all of these populations, one can expect that the greater the height-difference between partners, the higher the relative risk of adverse pregnancy outcomes.

Moreover, in general, women in most populations appear to prefer men taller than themselves as mates,⁴⁹⁻⁵¹ although there are exceptions in certain cultures.⁵² Thus, the extent of sexual dimorphisms and the nature of mate choice in populations can determine the frequencies of size-incompatible matings that will occur, and among these matings, a fraction would suffer adverse pregnancy outcomes. As such, it would not be unreasonable to expect that the frequency of size incompatible mating in *Australopithecines* (avg: males ~4'11, females ~3'5) would have been higher than in later *Homo* (avg: males ~5'8, females ~5'5). In such a case, one may expect that there would have been strong selection against at least a fraction of matings, particularly if FPD caused maternal (and fetal) mortality. Indeed, there may have been significant variation, not only in the fraction of such matings, but also in the outcomes, across populations. In some modern populations Geugan et al³⁹ found women to be taller in populations with high maternal mortality.

If this theory is verified, then selection against small statured women would, among other factors, explain why the decline in sexual dimorphism in hominins appeared to be largely due to an increase in female size.^{26,53} Even if the advent of agriculture resulted in the reduction of human stature³⁰ one would expect stature changes in both sexes, unless females were for some reason more adversely affected. On the other hand, it is possible that agriculture associated changes in diet and life style may have affected the nature of developmental plasticity of the female birth canal and fetal proportions. Huseynov et al,⁵⁴ have shown that female pelvic development is rather pliable and responds to environmental and biological changes. Mitteroecker and Fischer⁴⁸ found evidence of correlated selection on head proportions (both mother and fetal) and pelvic dimensions. Secular declines in stature and nutritional changes may have dissociated the equilibrium between maternal and fetal developmental plasticity, which could have aggravated the fit between the fetus and the birth canal.

One problem with a model dependent on the degree of sexual dimorphism and rates of size-incompatible unions is the extent of the height difference that actually poses a risk. It is also important to take into account the significant variability of stature and pelvic dimensions across geographical populations and ethnicities.^{31,55-58} Accordingly, one can expect significant variability in incidences of size incompatibility derived FPD across populations. Another problem is the frequency of size incompatible matings that do occur, which as one may argue, be too small to have major consequences over short evolutionary timescales. These issues will have to be researched, but both these factors become quite relevant if sexual selection (mate choice and mating system) is considered, as we outline in the next section. Quite importantly, invoking sexual selection expands our arguments beyond just maternal mortality due to FPD arising from size-incompatibilities. Outcomes of the following hypotheses would apply across the gamut of causal factors (physical and physiological).

Mate Choice, and Preference for Younger Women Would Exacerbate Maternal Mortality

Young Mothers Are at Higher Risk of Maternal Mortality

There is substantial evidence to indicate that historically and in contemporary populations, younger women, particularly adolescents, who are likely to be small statured, are at higher risk of adverse pregnancy related outcomes^{2,59,60} (Figure 2A). In a large US cohort analyses between 1995 and 2004, Malabarey et al⁶⁰ found that adolescent mothers (aged 12-17 years) were at a higher risk of CS compared to women of older age groups. This risk was particularly high if adolescent mothers had macrosomic babies, compared to older mothers who had macrosomic babies. Other studies show that the risk of dystocia in adolescent women is likely due to their "underdeveloped" smaller pelvis.^{61,62} Interestingly, Lenhard et al⁶³ found smaller sagittal midpelvic diameters in women who underwent caesarian deliveries due to FPD compared to women with no risk of FPD. Tague⁶⁴ found anatomical evidence that the female pelvis continued to grow well past adolescence in 3 prehistoric populations of Amerindians. Particularly, the obstetrically relevant pelvic dimensions were much smaller in remains of 18 to 24 year old women compared to remains of women past the age of 25. Volgyi et al⁶⁵ found that although teens may generally reach their mothers' height by the age of 18, their pelvic widths had yet to reach proportions comparable to their mothers'. Recent studies also show that the female pelvis continues to grow in adulthood and reaches its most obstetrically optimal morphology between the ages of 25 and 30 years, and then declines past 40.^{48,54,66} These data provide compelling evidence for age-related pregnancy outcomes. In addition to obstructed labor, there is substantial evidence that adolescent mothers are generally at a higher risk of maternal mortality due to eclampsia, hemorrhage, and sepsis, as well as indirect causes such as malaria and rickets in certain populations^{15,59,67} (Figure 2B and C). These data become relevant if FPD acts as a disposing factor for risk of eclampsia and hemorrhage, for example see study by Fantu et al.¹⁶

Age-Biased Male-Mate Choice and Risks of Maternal Mortality

One mechanism that can significantly affect the incidence of maternal mortality, apart from height/size-dependent mate choice, is an age-based mate choice system, that is, *either women preferring older men as mates, or males preferring younger women as mates*. In either case, there is likely to be a height difference between partners, depending on the extent of sexual dimorphism in the population.

Although there has traditionally been much emphasis on female choice, since males are relatively physically stronger, male mate choice may have been more relevant for humans in a resource limited and competitive system. Stronger males would have the ability to monopolize resources and mates. Puts⁶⁸ has

Figure 2. A, Maternal mortality rates in mothers of different ages data obtained from the study by Say et al.¹⁵ B, Proportion of adverse pregnancy-related outcomes in mothers of different age groups, data obtained from study by Ganchimeg et al.⁵⁹ C, Severe pregnancy outcomes in mothers of different age groups, data obtained from study by Ganchimeg et al.⁵⁹

argued that ancestral male mating success may have been more dependent on male–male competition than other forms of sexual selection. The physical prowess of males would allow them to acquire multiple long- or short-term mates by force.⁶⁸ Despite these speculations of ancestral human mating systems, what is important is that a multigenerational system creates the possibility of older, powerful men having more access to younger women either by force or because they have and control more resources. In many contemporary populations, there is substantial evidence that younger and older men tend to prefer women younger than themselves as mates, and women tend to be attracted to older men.^{69–74}

An age-based mate choice system, particularly one where male preference is skewed toward younger females, would aggravate the risk of obstructive complications. As Ganchimeg et al.’s⁵⁹ data indicate, even in contemporary populations, pregnancy in teen mothers occurs at an alarming rate in many countries. Child and adolescent marriage is a deep rooted cultural and social practice in many developing countries.⁷⁵ Low-income countries are more likely to have malnourished

children and therefore adolescent girls may not be anatomically and physiologically “prepared” for pregnancy.^{61,63,65,66,76} Ancestral populations, where human life expectancy in a resource limited environment was about 30 to 40 years maybe slightly higher in other conditions, see the study by Gurven and Kaplan,⁷⁷ would likely tend to reproduce much earlier than contemporary populations. In this case, size incompatible matings due to age-biased male mate choice would have been a bigger problem in the past than it is today. Moreover, when traits such as promiscuity and/or polygamy are included in such a system, there will be constant selection on female phenotypes of particular age groups, which over time can lead to sex specific differences. However, the deleterious trait under selection may persist longer despite selection against it. For instance, Tuljapurkar et al.⁷⁸ showed that older males’ preference for younger women, polygyny, and gerontocracy have played an important role in shaping the evolution of senescence in humans. Similarly, Morton et al.⁷⁹ have shown that menopause could arise as an indirect evolutionary consequence of accumulating deleterious fertility-affecting mutations due to male

preference to mate with younger women. Such studies, rooted in population genetics theory, shed light on how maladaptive traits can evolve *and persist* despite being counter-intuitive to selectionists' expectations.

Mate Choice and Polygamy Would Make Maternal Mortality Persist Longer in Populations

Using the example of FPD, if size incompatible matings result in maternal (and infant) mortality, one may expect that under such selective pressure, the genetic variation underlying the size-incompatibility would decrease, resulting in reduced sexual dimorphism. This reduction would occur faster in a monogamous population because the genetic variations of both parents will be affected (assuming death of mother and infant in every instance). Of course, apart from the extent of SSD and incidence of maternal mortality due to FPD, ecological and environmental factors can affect the rate and intensity of such a process. This deduction (among other relevant factors) would support the reduction of sexual dimorphism observed from *Australopithecines* to *Homo*. However it would not support Plavcan's⁵³ findings that the decrease in sexual dimorphism leading up to *Homo erectus* appears primarily due to increases in female body size, and not so much of male size. This trend is curious even if ecological factors are more likely determinants of changes in sexual dimorphism, because both sexes are likely to benefit in favorable conditions.

Males Are Less Affected by Maternal Mortality in a Polygamous System

Size is a quantitative trait and selection on one sex would affect both sexes. Furthermore, a promiscuous or polygamous system can influence the sex specific change of a size-related fertility trait. This is because males will not be affected as much as females are, particularly if males have more opportunities to mate than females. In such a system, selective pressure will be stronger on the female phenotype, particularly if maternal mortality was high.

Elucidating mating behaviors in early hominins is an elusive task but evidence suggests that most early hominins were promiscuous in some form or another.⁸⁰ Even "pair-bonding," which eventually became widespread, probably began as transient, sex-for-food exchanges.^{28,81} Under a promiscuous and competitive system, "dominant" or "more successful" human males would have controlled resources and had unimpeded access to females.⁸⁰⁻⁸³ In early agricultural societies, polygamy would have intensified, particularly for resource-rich men who can gain several wives.^{78,84-87} Even contemporary populations are not strictly monogamous, due to the practice of remarriage (serial monogamy). Both polygamy and serial monogamy increase reproductive success of men compared to women.⁸⁸ In such a system, the loss of fitness from maternal mortality is offset with male-gains of fitness (from access to multiple partners and/or remarriage). However small the frequency of such

occurrences (size-incompatible matings), a polygamous mating system ensures male fitness gains in consecutive generations regardless of female demise. Thus, the rate of selection against the risks of maternal mortality will be reduced, contributing to its prolonged persistence. The qualitative nature of this conclusion would not change even if men suffered some loss of fitness due to maternal mortality. Even if men lose some fitness due to death of the mother and child, they can engage in additional matings. Such males may indeed have relatively lower fitness than other males who do not face similar fitness loss. Nevertheless, in the end, male fitness remains relatively higher than that of females in such a mating system.

In an age-biased mating system, male mate choice and maternal mortality also have the potential to influence the evolution of menopause. Morton et al⁷⁹ developed a population genetics model of the human mating system and showed that nonrandom mating involving male preference for younger females would lead to accumulation of female infertility causing mutations in older females, resulting in the evolution of menopause. When this is considered from the perspective of our theory, the reproductive "axis" of male mate choice, male preference to mate with younger women, and maternal mortality, can reinforce one another and potentially reduce average female reproductive life span through generations. This would in turn drive male mate choice toward younger women over time, and based on Morton et al's⁷⁹ finding, the reinforcing cycle of male preference for younger women, maternal mortality, and the reduction of female reproductive lifespan can be expected to accelerate the evolution of menopause by accelerating the rate of female-infertility causing mutations.

Concluding Remarks

The role of sexual selection/antagonism has not been adequately explored to address an inherently human reproductive problem. Indeed, the underlying causes of obstructive labor are complex and varied.^{24,25} The short- and long-term changes in the risks of birthing through time, and the extent of its phenotypic plasticity in modern times may well be due to the interactions of a variety of factors that have changed through time. In this manuscript, our intention was to illuminate the relevance of sexual selection and human reproductive biology, particularly of sexual dimorphism and mate choice to the *persistence* of maternal mortality, not its causes. As we have reviewed, interactions between sexual dimorphism and mate preference for younger women have the potential to affect the risk of obstructed labor and maternal mortality over time. Indeed, dietary shifts due to a change from a hunter gatherer to an agricultural society may have additionally exacerbated the degree of the risk. But patterns of mating, particularly the prevalence polygamy and serial monogamy, may also have changed for the same reason, additionally affecting the magnitude of delivery-related risks.

Even though our theory is based on mating systems and focuses on size-incompatible and age-biased matings, from a broader perspective, it shows that maternal mortality is not a

problem of female biology alone. It is a problem of human reproductive biology affected by both males and females. As such, measures to “cure” maternal mortality as a female affliction will be insufficient, although certain genetic variations may be acting as disposing factors. If our theory holds, it would show that maternal mortality is an evolving consequence of human reproductive biology, affected by interactions between polygamy, mate choice, and fitness differences between the sexes. As Mitteroecker et al’s⁴¹ modeling work showed, males by virtue of being unaffected by all aspects of maternal mortality have the ability to exacerbate the problem. Our hypothesis provides an explanation of *how* males exacerbate the problem—through male driven, age-biased mate choice, and polygamy. Our hypothesis can be implemented in a population genetics model and tested via simulations, as Morton et al⁷⁹ did.

Authors’ Note

R.S.S. conceived the theoretical basis of the hypothesis, interpreted data, designed, and wrote the manuscript. S.J. contributed to hypothesis development, surveyed literature, collected information, interpreted data, designed, and wrote the manuscript. A.K.G. surveyed literature, collected information, and contributed to writing the manuscript.

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