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The social evolution of sleep: sex differences, intragenomic conflicts and clinical pathologies

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Sleep appears to be essential for most animals, including humans. Accordingly, individuals who sacrifice sleep are expected to incur costs and so should only be evolutionarily favoured to do this when these costs are offset by other benefits. For instance, a social group might benefit from having some level of wakefulness during the sleeping period if this guards against possible threats. Alternatively, individuals might sacrifice sleep in order to gain an advantage over mate competitors. Here, we perform a theoretical analysis of the social evolutionary pressures that drive investment into sleep versus wakefulness. Specifically, we: investigate how relatedness between social partners may modulate sleeping strategies, depending upon whether sleep sacrifice is selfish or altruistic; determine the conditions under which the sexes are favoured to adopt different sleeping strategies; identify the potential for intragenomic conflict between maternal-origin versus paternal-origin genes regarding an individual's sleeping behaviour; translate this conflict into novel and readily testable predictions concerning patterns of gene expression; and explore the concomitant effects of different kinds of mutations, epimutations, and uniparental disomies in relation to sleep disorders and other clinical pathologies. Our aim is to provide a theoretical framework for future empirical data and stimulate further research on this neglected topic.

1. Introduction

Sleep—defined as a reversible state of behavioural inactivity, elevated arousal threshold, and homeostatic regulation [1,2]—has been found to occur in all animal species that have been adequately studied [3,4]. Several non-exclusive hypotheses have been offered as to the biological function of sleep, including energy allocation into physiological activities that cannot be performed during the day, adaptive inactivity when activity is costly, metabolite clearance from the brain, maturation of the nervous system during ontogeny, memory consolidation, and synaptic homeostasis [5] (electronic supplementary material, table S1).

Given the apparently important benefits of sleep, individuals who sacrifice sleep would be expected to incur significant costs and, indeed, lack of sleep is known to cause or exacerbate a very wide range of health problems, ranging from cardiovascular diseases [6] and type 2 diabetes [7] to psychological distress [8] and cancer [9]. From an evolutionary perspective, sacrifice of sleep will only have been favoured provided that there are substantial compensating benefits. For instance, a social group may benefit from having its members waking up at different times throughout their sleeping period if this helps to protect the group from potential dangers [10,11]. Alternatively, individuals might sacrifice sleep to gain an advantage over their mate competitors [12–14]. In both of these scenarios, individuals who give up opportunities to sleep may have an

important impact on the survival and mating success of their social partners, making sleep an important aspect of an individual's social behaviour.

However, at a fundamental level, the social evolutionary pressures that have shaped investment into sleep versus wakefulness remain entirely obscure. It is not even clear whether sacrificing sleep is a relatively altruistic activity—incurring costs to the individual and yielding benefits to social partners—or relatively selfish—yielding benefits to the individual at a cost to their social partners. Moreover, the distinction between altruistic versus selfish sleep sacrifice could potentially explain sex differences in sleep schedules and modulate conflicts of interest between an individual's maternal- and paternal-origin genes over the individual's investment into sleep versus wakefulness. Such intragenomic conflict would be expected to underpin a range of medical disorders and pathologies in relation to the biology of sleep.

Here, we investigate under which circumstances individuals are favoured to invest more time into sleep versus wakefulness, with a focus on individuals' social environment, and how that may affect gene expression and explain sleep pathologies. Methodologically, we use a personal fitness approach to kin selection [15,16], the results of which analysis may be interpreted in terms of inclusive fitness [17]. First, we analyse how an individual's sleep schedule may be modulated by the degree of genetic relatedness to their social partners, providing a contrast between altruistic versus selfish sacrifice of sleep. Second, we explore the possibility for sex-specific social evolutionary pressures—arising from sex differences in relatedness between social partners and the associated benefits of sleep sacrifice—to drive sex-specific sleeping schedules. Third, we investigate whether there is potential for intragenomic conflict to occur between an individual's maternal-origin versus paternal-origin genes over the investment that the individual makes into sleep versus wakefulness. Fourth, we use the 'loudest voice prevails' principle [18] to translate such conflict into readily testable predictions concerning patterns of gene expression, specifically 'genomic imprinting'. Finally, we show that these patterns of gene expression lead to readily testable predictions concerning the effects of different kinds of mutations, epimutations, and uniparental disomies on sleep disorders and other pathologies. As these predictions relate largely to data that remain to be collected, our overall aim is to provide a theoretical framework for future empirical work and to stimulate research activity on this neglected topic.

2. Is sleep selfish or altruistic?

Natural selection favours those individuals that pass on more copies of their genes to future generations [19,20]. According to the theory of inclusive fitness, an individual may achieve this either by increasing their own reproductive success (direct fitness) or by increasing the reproductive success of other individuals with whom they share genes in common (indirect fitness) [15,21]. Hamilton's rule [15,21–23] provides an encapsulation of this logic: a social behaviour will be favoured by natural selection so long as $-C + Br > 0$, where C is the loss of reproductive success incurred by the actor, B is the gain in reproductive success by the actor's social partners, and r is the genetic relatedness of the actor to their social partners. This is an extremely general result, that holds irrespective of whether the genetical trait varies in a continuous or discontinuous manner, whether selection is weak or strong, whether genes interact additively or nonadditively, and so on (reviewed by [17]). If the social behaviour stabilizes at an intermediate evolutionary optimum then this must occur when the direct and indirect fitness effects exactly cancel each other out ($-C + Br = 0$; note that this is true even if the cost and benefit of the social behaviour change over evolutionary time). This means that the behaviour must be either altruistic ($C > 0$ and $B > 0$) or selfish ($C < 0$ and $B < 0$) at equilibrium [15,24,25].

Consider an individual who is genetically predisposed to having relatively more sleep. If this leads to an increase in their own reproductive success ($C < 0$) and a decrease in their social partners' reproductive success ($B < 0$), then this sleep strategy may be described as selfish and, conversely, individuals who tend to sacrifice sleep may be described as behaving altruistically. An example of such a scenario is when individuals may choose to sacrifice sleep in order to protect their group from threats during the night, such as surprise attacks from other groups or predators. The genetic relatedness of group mates then determines how much sleep an individual should be favoured to sacrifice in order to protect their group from such dangers. To illustrate such a scenario, we incorporate between-group dispersal into Haldane's [26] classic 'tribe splitting' model of human altruism (see electronic supplementary material for details), revealing that a higher rate of dispersal of individuals between groups, which leads to lower relatedness among group mates, incentivises individuals to devote more time to sleep (figure 1*a* and electronic supplementary material, figure S1*a*).

Conversely, if an individual who sleeps relatively more thereby incurs a loss of reproductive success ($C > 0$) and provides a benefit to their social partners ($B > 0$), then they can be said to be behaving altruistically and, conversely, an individual who has a tendency to sacrifice sleep is behaving selfishly. An example of such a scenario is when individuals may choose to sacrifice sleep in order to pursue mating opportunities, and thereby reduce mating opportunities for their social partners. Again, relatedness between group mates is expected to modulate how much time an individual should devote to sleep in such a scenario, but in the opposite direction from before. Turning again to the tribe-splitting model for an illustration, we reveal that as the rate of individual dispersal increases—and, consequently, the degree of relatedness among group members decreases—individuals are favoured to sleep less (figure 1*b* and electronic supplementary material, figure S1*b*).

So is sleeping selfish, or is it altruistic? This question remains to be answered due to a lack of scientific investigation on how genetic relatedness modulates sleep. With respect to the above scenarios, several studies have shown how predation [27–37] and sexual competition [12–14] modulate the sleep schedule of several species. Moreover, Capellini *et al*. [38] report that total duration of sleep across mammals is lower when individuals are more likely to sleep in a group, which they interpreted as either due to individuals being able to sleep more deeply—and hence not requiring such long periods of sleep—or alternatively owing to time invested in social interaction leaving less time for sleep. In humans, selfish personality traits have been shown to correlate with late sleep onset [39] and short-term mating success [40]. To our knowledge, the potential of genetic relatedness to modulate sleep in all of those scenarios remains to be investigated, and a comparative approach—taken across populations or species—may provide a more definitive means of assessing whether sleeping more is a selfish or an altruistic behaviour.

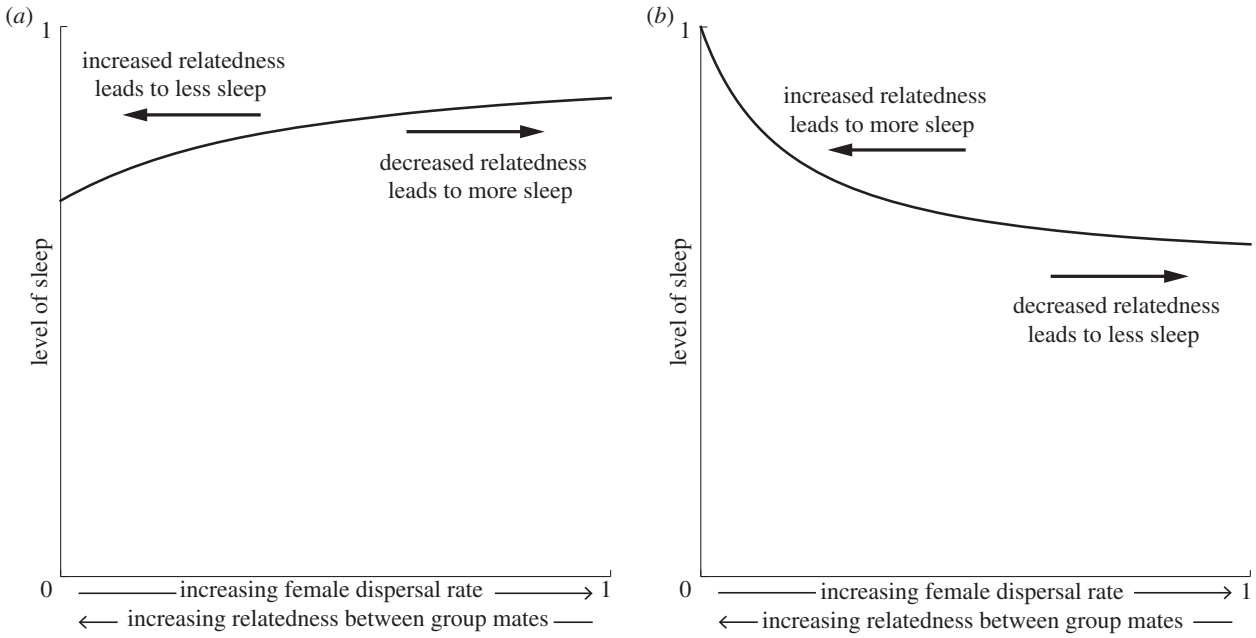

Figure 1. How much an individual should sleep depends on the relatedness between the individuals in a group. When individuals sacrifice sleep in order to remain alert to dangers which may befall the group (*a*), individuals sacrifice more sleep when relatedness is higher, which is the case when female dispersal is lower. When individuals sacrifice sleep in order to gain an advantage over their mate competitors (*b*), individuals sacrifice more sleep when relatedness is lower, which is the case when female dispersal is higher. The following parameter values were used for both panels: male dispersal rate $d_m = 0$; budding dispersal rate $d_B = 1$; number of adult females $n_f = 4$; number of adult males $n_m = 4$; minimum level of sleep $m = 0.05$; and benefits of sleeping throughout the night $b_f = b_m = 1$. Additionally, in (*a*) the level of a threat is $a = 1$ and the mating opportunities that females and males can obtain through sleep sacrifice is $c_f = c_m = 0$, while in (*b*) the level of a threat is $a = 0$ and the mating opportunities that females and males can obtain through sleep sacrifice is $c_f = c_m = 1$. Here, we consider female-biased dispersal—see electronic supplementary material, figure S1 for male-biased dispersal.

3. Sex differences in sleeping behaviour

Above we have shown that sleep is expected to be modulated by its impact on the reproductive success of the individual and the individual's social partners. However, the components of direct ($-C$) and indirect (Br) fitness are liable to be different for females and males, and this suggests that females and males may be favoured to adopt different sleep schedules.

If individuals sacrifice sleep in order to protect their group against night-time dangers, then there is no obvious reason to suspect that this should incur different costs (C) or provides different benefits (B) to their social partners. Nevertheless, females and males might differ with respect to how genetically related they are to their group mates ($r_f \neq r_m$), and this alone could drive sex differences in sleeping habits. Ancestral human populations may have been characterized by female-biased dispersal [41], which would have led to females and males being differently related to their group mates. Returning to the tribe-splitting model for the purpose of illustration (see electronic supplementary material for details), we show that female-biased dispersal—which leads to females being less related to their group mates than are males ($r_f < r_m$)—leads to females being favoured to invest more in sleep than are males, when sleep sacrifice is altruistic (figure 2*a* and electronic supplementary material, figure S2*a*).

But females and males do differ in many aspects of their biology, particularly in relation to reproduction. Females often invest more time and energy into raising their offspring than males who, not having this limitation, are free to pursue additional mating opportunities with females that remain available [42]. In this sense, females may be seen as a resource for which males have to compete [43], such that sexual selection usually acts more strongly in relation to males. Insofar as these differences in fitness components are relevant to the evolution of sleep, we might expect that these, too, could favour sex-specific sleep patterns. For example, if sleep sacrifice is associated with increased mating opportunities [12] which could offset the costs associated with sleep sacrifice, then males are expected to gain more from sleep sacrifice than are females ($C_m < C_f$). In addition, relatedness is expected to modulate how competitive the males should be. Returning to the tribe-splitting model for illustration (see electronic supplementary material for details), we show that an increased rate of individual dispersal—which reduces genetic relatedness between social partners—leads to more selfishness on the part of males and, therefore, less sleep (figure 2*b* and electronic supplementary material, figure S2*b*).

Sex-specific sleeping patterns have been reported in several non-human animal species. Specifically, some studies report that total sleep length is higher for females (in pectoral sandpipers [12], in great tits [13], and in blue tits [44]) while others report that total sleep length is higher for males (in fruit flies [45], and in mice [46]). In humans, women have been suggested to enjoy better-quality sleep [47–49]. Men are also more likely than women to perform normally during the day with less sleep [50,51], suggesting that in our evolutionary past either: (i) women have been favoured to have more sleep and men have evolved adaptations to reduce the harmful effects of less sleep, or (ii) women need more sleep than men due to basic physiological differences. Regardless, the role of relatedness in modulating any of these patterns in humans or any other animal species has not, to our knowledge, been explored empirically.

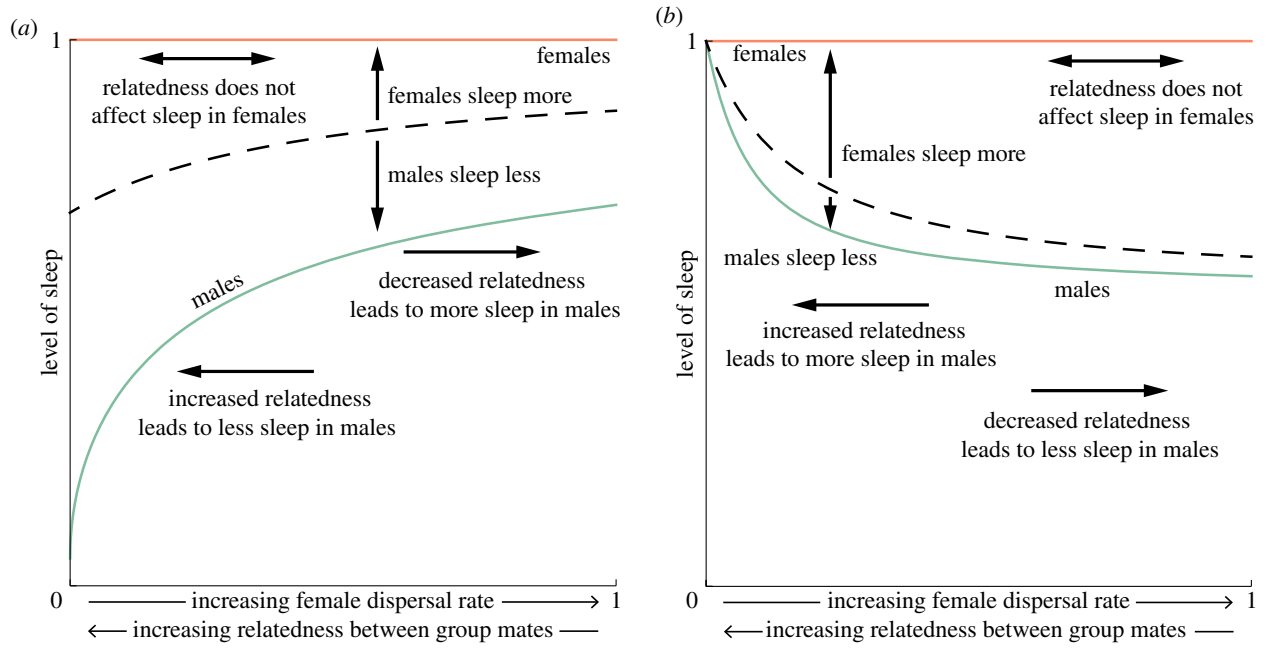

Figure 2. Females and males may be favoured to have different sleeping levels. Given that females are less related to their social partners than males, females favour more sleep when (*a*) the sleep sacrifice is being used to protect the group against threats. When (*b*) sleep sacrifice is being used to increase male reproductive success, females do not favour any sleep sacrifice, with males being the only ones to sacrifice sleep to gain an advantage over their mate competitors. Dashed line represents the favoured level of sleep when this is constrained to be the same for females and males. The following parameter values were used for both panels: male dispersal rate $d_m = 0$; budding dispersal rate $d_B = 1$; number of adult females $n_f = 4$; number of adult males $n_m = 4$; minimum level of sleep $m = 0.05$; and benefits of sleeping throughout the night $b_f = b_m = 1$. Additionally, in (*a*) the level of a threat is $a = 1$ and the mating opportunities that females and males can obtain through sleep sacrifice is $c_f = c_m = 0$, while in (*b*) the level of a threat is $a = 0$ and the mating opportunities that females and males can obtain through sleep sacrifice is $c_f = 0$ and $c_m = 1$, respectively. Here, we consider female-biased dispersal—see electronic supplementary material, figure S2 for male-biased dispersal.

4. Intragenomic conflict, genomic imprinting, and sleep

The genes within an individual do not necessarily agree on how their carrier should interact with social partners. Because an individual can be more related to social partners through one parent than the other, then genes inherited from each of the two parents may differ with regards to the social behaviour that they favour [18,52,53]. Insofar as genetic relatedness is relevant to the evolution of sleeping patterns, maternal-origin genes and paternal-origin genes may then disagree on how much an individual should sleep.

If individuals altruistically sacrifice sleep in order to protect their group mates from danger, then we expect that the genes for which relatedness between social partners is higher will be more strongly favoured to sacrifice their carriers sleep. Using again the tribe-splitting model as an illustration (see electronic supplementary material for details), increasingly female-biased dispersal—which reduces genetic relatedness between social partners with respect to their maternal-origin genes—leads to paternal-origin genes favouring less sleep and maternal-origin genes favouring more sleep (figure 3*a*; electronic supplementary material, figure S3*a* for the opposite pattern when dispersal is male-biased). In contrast, if individuals sacrifice sleep to increase their mating success, then the genes for which relatedness is higher will favour more sleep. Going back to the tribe-splitting model (see electronic supplementary material for details), increasingly female-biased dispersal, leads to paternal-origin genes favouring more sleep and maternal-origin genes favouring less sleep (figure 3*b*; electronic supplementary material, figure S3*b* for the opposite pattern when dispersal is male-biased). These scenarios describe what is known as intragenomic conflicts [18,52,53].

These intragenomic conflicts are predicted to lead to parent-of-origin-specific gene expression—i.e. 'genomic imprinting' [18]. Consider a locus for which increased gene expression leads to more sleep—a 'sleep promoter'. The gene that favours more sleep can get closer to its optimal level of sleep by increasing its own expression. The gene that favours less sleep, in contrast, gets closer to its optimal level of sleep by decreasing its genetic expression. Such changes continue until the gene favouring a lower level of sleep ends up silencing itself, with the gene favouring a higher level of sleep winning the intralocus conflict and, accordingly, setting the level of expression to its own optimum [18] (figure 4; see [55] for a simulation illustration). The logic is reversed for a locus in which increased gene expression leads to less sleep, a 'sleep inhibitor'. In that case, it is the gene that favours lower level of sleep that wins the conflict, and the other gene is silenced (figure 4).

In recent years, there has been a growing interest in the genetic [56–59] and epigenetic [60–66] control of sleep. Epigenetic control comprises any molecular mechanism that changes how genes are expressed without affecting the DNA sequence itself [67] and includes genomic imprinting, which is usually described as involving methylation of the gene's regulatory regions [18]. Several genes involved in the control of sleep have been shown to be imprinted [68–76], but theoretical explanations for such patterns are relatively lacking. The only exception is Haig's [77] study of an intragenomic conflict regarding sleep, where night waking to suckle in newborns is predicted to lead to more maternal care [78,79]. In such

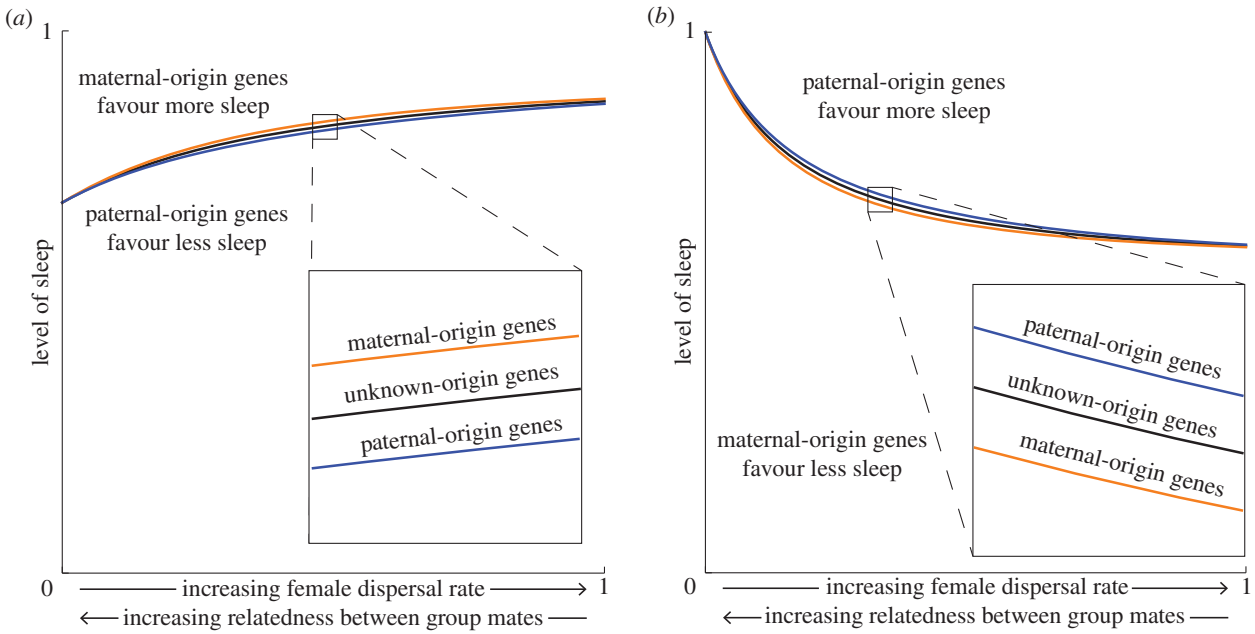

Figure 3. Maternal- and paternal-origin genes disagree regarding how much the individual should sleep. Maternal-origin genes (orange) and paternal-origin genes (blue) will disagree on how much an individual should sleep, which depends upon whether individuals are sacrificing sleep to (*a*) protect the group against threats or (*b*) gain an advantage over their mate competitors (with black being the level favoured by a gene ignorant of its origin). Specifically, given that relatedness is higher for paternal-origin genes, maternal-origin genes favour more sleep and paternal-origin genes less sleep if sleep is selfish (*a*). On the contrary, if sleep is altruistic, then maternal-origin genes favour less sleep and paternal-origin genes more sleep (*b*). The following parameter values were used for both panels: male dispersal rate $d_m = 0$; budding dispersal rate $d_B = 1$; number of adult females $n_f = 4$; number of adult males $n_m = 4$; minimum level of sleep $m = 0.05$; and benefits of sleeping throughout the night $b_f = b_m = 1$. Additionally, in (*a*) the level of a threat is $a = 1$ and the mating opportunities that females and males can obtain through sleep sacrifice is $c_f = c_m = 0$, while in (*b*) the level of a threat is $a = 0$ and the mating opportunities that females and males can obtain through sleep sacrifice is $c_f = c_m = 1$. Here, we consider female-biased dispersal—see electronic supplementary material, figure S3 for male-biased dispersal.

scenarios, paternal-origin genes favour more night waking and maternal-origin genes less night waking because mothers might have future offspring from different fathers [77]. Here, we have shown that social conflicts in adults may also be relevant for the evolution of genomic imprinting in genes controlling sleep.

5. Sleep disorders and genomic imprinting

Genomic imprinting renders individuals functionally haploid at affected loci, and therefore vulnerable to the effects of mutations that would otherwise have been (at least partially) recessive under standard diploid gene expression [80]. These mutations are predicted to result in extreme pathologies [81]. More generally, possible kinds of mutations that can lead to dramatic consequences are: deletions, where a gene is removed from the genome; epimutations, where disruptions occur in the machinery responsible for determining the methylated pattern of a gene; and uniparental disomy, where individuals carry two copies of a maternal- or paternal-origin gene, instead of one of each. In each of these types of perturbations, specific predictions can be made about their consequences at the phenotypical level which are dependent on the selective force that led to individuals sacrificing sleep (figure 4). Conversely, if the phenotypic effect of the mutation is known, then these predictions may be used to infer whether sleep sacrifice is relatively selfish or altruistic (figure 4).

Among the most well-known examples of human pathologies that emerge from a disruption of genomic imprinting patterns are those associated with Prader–Willi and Angelman syndromes, which are hypothesized to have been evolutionarily driven by an intragenomic conflict between maternal-origin and paternal-origin genes in young children with respect to their demand of maternal resources [82]. Because a mother's future offspring might have different fathers, the child's paternal-origin genes favour greater greediness while the maternal-origin genes favour less greediness. Consequently, maternal duplication/paternal deletion of the chromosomic region 15q11-13 results in children with a phenotype that is the result of reduced maternal investment during pregnancy, such as reduced weight, or that result in reduced maternal investment in the newborn, such as poor suckling response, weak cry, and physical inactivity. In contrast, paternal duplications/maternal deletion of the chromosomic region 15q11-13 results in children with a phenotype that is the result of increased maternal care during pregnancy, such as increased weight, or that result in increased maternal care in the new-born, such as prolonged suckling response and physical hyperactivity. Sleep is also affected [68,69,71,75] because night waking in children is predicted to lead to more maternal care [78,79]. Prader–Willi syndrome is then characterized by less night waking to suckle, while Angelman syndrome is characterized by the opposite pattern, with more night waking to suckle [68,69,71,75,77]. While the nature of the conflict is different from what we explore in our analysis, it illustrates how genomic imprinting can affect sleep.

Other disorders have been hypothesized as being associated with genomic imprinting patterns, such as autism and psychopathic disorders [83,84]. Such disorders are considered to be extremes from a phenotypic-continuum, with autism being a 'hyper-altruistic' brain disorder (low cognitive empathy but high emotional empathy; [85]) and psychopathic

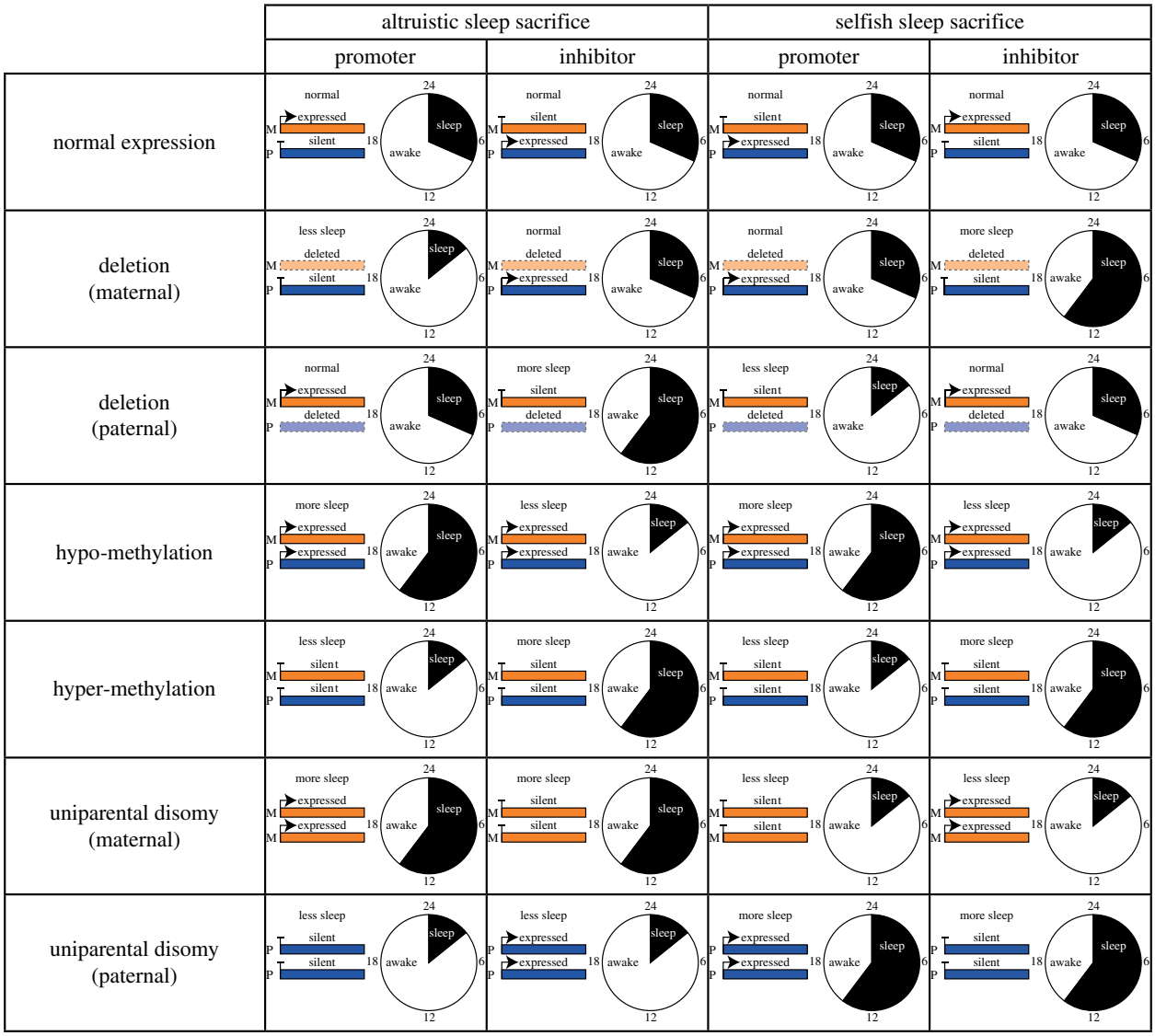

Figure 4. Genomic imprinting of genes responsible for level of sleep and the effects of possible disruptions. Predictions as to which gene is expressed and which gene is silent—maternal-origin gene (M, orange) or paternal-origin genes (P, blue)—when individuals are sacrificing sleep to protect the group against threats (altruism) or to gain an advantage over their mate competitors (selfishness). We consider an example for a gene that promotes sleep (promoter) and an example for a gene that inhibits sleep (inhibitor). In both cases, we assume female-biased dispersal—see electronic supplementary material, figure S4 for male-biased dispersal. Note that for simplicity we assume methylation is associated with gene silencing, as is usually the case in mammals [54]. In cases where methylation is associated with gene activation the outcome for hypo-methylation is expected to be that shown here for hyper-methylation, and vice versa.

disorders a 'hyper-selfish' brain (high cognitive empathy but low emotional empathy; [86]). The intragenomic conflict between maternal-origin genes and paternal-origin genes is over how altruistic an individual should be to their social partners. Therefore, if indeed female-biased dispersal was prevalent throughout human evolution, then relatedness would have been higher for paternal-origin genes and lower for maternal-origin genes. Accordingly, autism would be the result of paternally expressed genes and psychopathy the result of maternally expressed genes. The opposite pattern is expected if male-biased dispersal was present.

Interestingly, in both autistic and psychopathic disorders, sleep is also affected. Accordingly, autism is associated with insomnia and lower levels of sleep [87] while psychopathic disorders tend to be associated with deeper sleep [88–90]. These patterns appear to match the predictions of sleep sacrifice being associated with altruism, but could alternatively be a consequence of anxiety in autism [91] and mental resilience in psychopathic disorders [92]. Others suggest a different continuum, where psychosis—instead of psychopathic disorders—is the opposite extreme of autism [83] and concerning parental-offspring conflict traits, similar to the ones described above for Prader–Willi and Angelman syndromes and with sleep also being affected in an identical way [93].

Some patterns of parent-of-origin gene expression have already been found for genes associated with sleep and not associated with chromosomic regions responsible for Prader–Willi and Angelman syndromes, specifically six genes in an experimental study with mouse strains [72], which suggests that genomic imprinting may be present. More research is necessary to understand if that is indeed evidence of genomic imprinting and if it follows the patterns that we propose. Additionally, genomic-wide association analysis and heritability studies show tentative evidence for a genetic component for several sleeping disorders, such as insomnia [94], obstructive sleep apnoea [95], restless leg syndrome [96], narcolepsy and essential hypersomnia [97], sleepwalking [98], sleep terrors [99], and sleep paralysis [100]. To our knowledge, the

possibility that genomic imprinting is involved in any of those disorders has yet to be investigated.

6. Conclusion

Sleep is not usually considered to be a social behaviour. Here, we have argued that an approach that takes the social impact of sleep into consideration can offer new insights into its evolutionary drivers. We have shown how the social environment may shape an individual's sleeping pattern and also explain sexual differences in sleep requirements. Moreover, our approach also predicts the existence—and patterns—of genomic imprinting in relation to loci that underpin sleep phenotypes. By taking a new approach to the study of sleep, we have integrated our results with what is already known from the literature to present new perspectives. Further empirical work is necessary to determine if relatedness has indeed had a modulating role in the evolution of sleep. If so, then our analysis suggests that it may be crucial for understanding the evolution of sleeping patterns and associated disorders.

Data accessibility. This article has no additional data.

Authors' contributions. G.S.F., S.A.M.V., and A.G. designed the study and wrote the manuscript. G.S.F. led the mathematical analysis.

Competing interests. We declare we have no competing interests.

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Electronic supplementary material for Faria et al. 2018 "The social evolution of sleep: sex differences, intragenomic conflicts and clinical pathologies"

First, we introduce a basic model for the social evolution of sleep with three analyses: 1) absence of sex differences and genes do not know their origin; 2) absence of sex differences and genes know their origin; and 3) sex differences and genes do not know their origin. And second, we introduce an illustrative model for the social evolution of sleep with two analyses: 1) sleep cannot evolve independently in females and males; and 2) sleep can evolve independently in females and males.

1. Basic model of the social evolution of sleep and inclusive fitness predictions

Natural selection favours any gene that is associated with greater individual fitness (Fisher 1930; Price 1970). Assuming vanishingly little genetic variation, this condition may be expressed using the mathematics of differential calculus: $dW/dg > 0$, where g is the genic value of a gene picked at random from the population and W is the relative fitness of the individual carrying this gene (Taylor 1996). We consider three scenarios (defined by the set $A = \{U,M,P\}$), concerning whether the gene's action is independent of its parent of origin (in which case the gene can be considered ignorant of its parent of origin; $A = U$), whether the gene's action is conditional upon it being of maternal origin ($A = M$), or whether the gene's action is conditional upon it being of paternal origin ($A = P$). We assume separate sexes (defined by the set $i = \{m,f\}$), such that a given carrier of the gene may be female ($i = f$) or male ($i = m$). Accordingly, the appropriate measure of relative fitness is a class-reproductive-value-weighted average taken across females and males, i.e. $W = ½W_f + ½W_m$, where W_f is the relative fitness of the female carrying the gene and W_m is the relative fitness of the male carrying the gene (Taylor 1996; Taylor & Frank 1996). The relative fitness of a female may be written as $W_f(x_f, y_f, z_f)$, where x_f is the level of sleep of the focal female, y_f is the average level of sleep of the females in the focal patch, and z_f is the average level of sleep of the females in the population, with values ranging from 0 (no sleep) to 1 (sleep throughout the whole sleeping period). Similarly, the relative fitness of a male may be written as $W_m(x_m, y_m, z_m)$, where x_m is the level of sleep of the focal male, y_m is the average level of sleep of the males in the focal patch, and z_m is the average level of sleep of the males in the population, with values again ranging from 0 (no sleep) to 1 (sleep throughout the whole sleeping period).

Following the approach of Taylor & Frank (1996) for a class-structured population, we may write $dW/dg_{i|A} = ½ (dW_f/dg_{f|A}) + ½ (dW_m/dg_{m|A}) = ½ ((\partial W_f/\partial x_f)(dx_f/dG_f)(dG_f/dg_{f|A}) + (\partial W_f/\partial y_f)(dy_f/dG_f')(dG_f'/dg_{f|A}) + (\partial W_f/\partial y_m)(dy_m/dG_m')(dG_m'/dg_{f|A})) + ½ ((\partial W_m/\partial x_m)(dx_m/dG_m)(dG_m/dg_{m|A}) + (\partial W_m/\partial y_m)(dy_m/dG_m')(dG_m'/dg_{m|A}) + (\partial W_m/\partial y_f)(dy_f/dG_f')(dG_f'/dg_{m|A}))$, where: G_f is the focal female's breeding value, G_f' is the average breeding value of the females in the focal patch; $dx_f/dG_f = dy_f/dG_f' = \gamma_f$ is the mapping between genotype and phenotype in the females; $dG_f/dg_{f|A} = p_{f|A}$ is the consanguinity of the genic actor A in the focal female to the female herself; $dG_f'/dg_{f|A} = p_{ff|A}$ is the consanguinity of the genic actor A in the focal female with a randomly-chosen female on her patch; $dG_m'/dg_f = p_{fm|A}$ is the consanguinity of the genic actor A in the focal female with a randomly-chosen male on her patch; G_m is the focal male's breeding value; G_m' is the average breeding value of the males in the focal patch; $dx_m/dG_m = dy_m/dG_m' = \gamma_m$ is the mapping between genotype and phenotype in the males; $dG_m/dg_m = p_{m|A}$ is the consanguinity of the genic actor A in the focal male to the male himself; $dG_f'/dg_m = p_{mf|A}$ is the consanguinity of the genic actor A in the focal male with a randomly-chose female on his patch; and $dG_m'/dg_m = p_{mm|A}$ is the consanguinity of the genic actor A in the focal male with a randomly-chosen male on his patch. The consanguinity between a genic actor A to its carrier

is the same no matter the class of the genic actor A or the sex that we are considering and, therefore, $p_{f|A} = p_{m|A} = p$. We divide all terms of the right-side of the equation by p to get the kin-selection coefficient of relatedness (see below; Bulmer 1994).

If sleep cannot evolve independently in females and males, then $\gamma_f = 1$ and $\gamma_m = 1$. In this scenario, all derivatives are evaluated at $x_f = x_m = y_f = y_m = z_f = z_m = z$. Accordingly, natural selection favours an increase in the level of sleep in females and males if:

$$(C_f(z) + B_{ff}(z)r_{ff|A} + B_{fm}(z)r_{fm|A}) + (C_m(z) + B_{mm}(z)r_{mm|A} + B_{mf}(z)r_{mf|A}) > 0, \quad \text{(A1)}$$

where: $C_f(z) = \partial W_f/\partial x_f$; $B_{ff}(z) = \partial W_f/\partial y_f$; $B_{fm}(z) = \partial W_f/\partial y_m$; $C_m(z) = \partial W_m/\partial x_m$; $B_{mm}(z) = \partial W_m/\partial y_m$; $B_{mf}(z) = \partial W_m/\partial y_f$; $r_{ff|A} = p_{ff|A}/p$; $r_{fm|A} = p_{fm|A}/p$; $r_{mm|A} = p_{mm|A}/p$; and $r_{mf|A} = p_{mf|A}/p$. If sleep can evolve independently in females and males, then $\gamma_f = 1$ and $\gamma_m = 0$ when analysing sleep in females and $\gamma_f = 0$ and $\gamma_m = 1$ when analysing sleep in males. In this scenario, all derivatives are evaluated at $x_f = y_f = z_f$ and at $x_m = y_m = z_m$. Accordingly, natural selection favours an increase in the level of sleep in females if:

$$C_f(z_f) + B_{ff}(z_f)r_{ff|A} + B_{mf}(z_f)r_{mf|A} > 0 \quad \text{(A2)}$$

and an increase in the level of sleep in males if:

$$B_{fm}(z_m)r_{fm|A} + C_m(z_m) + B_{mm}(z_m)r_{mm|A} > 0. \quad \text{(A3)}$$

1.1 Inclusive fitness predictions when there are no sex differences and genes do not know their origin

We now assume that there are no sex differences and that genes are ignorant to their origin. Therefore, we can simplify the inequality (A1) to $C(z) + B(z)r_U > 0$, where r_U is the average relatedness between a genic actor ignorant to its origin in the focal individual and a random individual in her patch, $C(z)$ is how sleep of the focal individual impacts her own fitness, and $B(z)$ is how the sleep of the focal individual's social partners impacts the fitness of the focal individual. We define a function $J(z^*, r_U) = C(z^*) + B(z^*)r_U$, where z^* represents a sleep optimum (formally, a convergence stable strategy; Christiansen 1991; Taylor 1996). Being a sleep optimum means that the population is at its sleep equilibrium and, therefore, $J(z^*, r_U) = 0$. To be an evolutionary stable equilibrium, it also needs to be convergent stable and the condition $\partial J/\partial z^* < 0$ needs to be met. Making those assumptions, and using the chain rule of derivation, we get $dJ/dr_U = (\partial J/\partial r_U) + (\partial J/\partial z^*)(dz^*/dr_U) = 0$ and, rearranging, $dz^*/dr_U = -(\partial J/\partial r_U)/(\partial J/\partial z^*)$. Defining a function S that returns the sign (positive, negative, or zero), we obtain $S(dz^*/dr_U) = S(\partial J/\partial r_U) = S(B(z^*))$ (Pen 2000; Farrell et al. 2015). Consequently, if social partners' sleep improves the individual's fitness ($B > 0$), then higher relatedness is associated with a higher sleep optimum ($dz^*/dr_U > 0$); if social partners' sleep decreases the individual's fitness ($B < 0$), then higher relatedness is associated with a lower sleep optimum ($dz^*/dr_U < 0$); if social partners' sleep does not affect the individual's fitness ($B = 0$), then higher relatedness is not associated with a higher or lower sleep optimum ($dz^*/dr_U = 0$).

1.2 Inclusive fitness predictions when there are no sex differences and genes know their origin

We now assume that there are no sex differences and that genes do know their origin. Therefore, we can simplify the inequality (A1) to $C(z) + B(z)r_A > 0$, where r_A is the average relatedness between a genic actor A in the focal individual and a random individual in her patch. Following the approach from section 1.1, we get $S(dz^*/dr_A) = S(\partial J/\partial r_A) = S(B(z^*))$

(Pen 2000; Farrell et al. 2015). Therefore, 1) if the sleep of social partners improves an individual's fitness ($B > 0$), then the sleep optimum is higher for maternal-origin genes than it is for paternal-origin genes ($z_M^* > z_P^*$, where z_M^* represents a sleep optimum for the maternal-origin genes and z_P^* represents a sleep optimum for the paternal-origin genes) when relatedness is higher for the former than for the latter ($r_M > r_P$, where r_M represents the average relatedness between a maternal-origin gene in the focal individual and a random individual in her patch and r_P represents the average relatedness between a paternal-origin gene in the focal individual and a random individual in her patch), and the sleep optimum is lower for maternal-origin genes than it is for paternal-origin genes ($z_M^* < z_P^*$) when relatedness is lower for the former than for the latter ($r_M < r_P$); 2) if the sleep of social partners decreases an individual's fitness ($B < 0$), then the sleep optimum is lower for maternal-origin genes than it is for paternal-origin genes ($z_M^* < z_P^*$) when relatedness is higher for the former than for the latter ($r_M > r_P$), and the sleep optimum is higher for maternal-origin genes than it is for paternal-origin genes ($z_M^* > z_P^*$) when relatedness is lower for the former than for the latter ($r_M < r_P$); and 3) if the sleep of social partners does not affect an individual's fitness ($B = 0$), then the sleep optimum for maternal-origin genes is equal to that for paternal-origin genes ($z_M^* \approx z_P^*$) and relatedness does not shape the sleep optimum.

1.3 Inclusive fitness predictions when there are sex differences and genes do not know their origin

We now assume that females and males may have different relatedness values to their social partners, with the costs and benefits associated with a given sleeping schedule being the same. We also assume that genes do not know their origin. Therefore, we can simplify the inequalities (A2) and (A3) to $C_f(z_f) + B_{ff}(z_f)r_{ff|U} + B_{mf}(z_f)r_{ff|U} > 0$ and $C_m(z_m) + B_{mm}(z_m)r_{mm|U} + B_{fm}(z_m)r_{fm|U} > 0$, respectively. For simplicity, we assume that $C_f = C_m = C$ and that $B_{ff} = B_{fm} = B_{mm} = B_{mf} = B$, meaning that the inequalities become $C(z_f) + B(z_f)r_{f|U} > 0$ and $C(z_m) + B(z_m)r_{m|U} > 0$, and $r_{f|U}$ is the average relatedness for the females in their patch and $r_{m|U}$ is the average relatedness for the males in their patch for a genic actor ignorant to its origin. Following the same strategy as in section 1.1, an evolutionary stable equilibrium also needs to be convergent stable and the conditions $\partial J/\partial z_f^* < 0$ and $\partial J/\partial z_m^* < 0$ need to be met (where z_f^* represents a sleep optimum for the females and z_m^* represents a sleep optimum for the males). Using the chain rule of derivation, we get $dJ/dr_{f|U} = (\partial J/\partial r_{f|U}) + (\partial J/\partial z_f^*)(dz_f^*/dr_{f|U}) = 0$, which rearranges to $dz_f^*/dr_{f|U} = -(\partial J/\partial r_{f|U})/(\partial J/\partial z_f^*)$, and $dJ/dr_{m|U} = (\partial J/\partial r_{m|U}) + (\partial J/\partial z_m^*)(dz_m^*/dr_{m|U}) = 0$, which rearranges to $dz_m^*/dr_{m|U} = -(\partial J/\partial r_{m|U})/(\partial J/\partial z_m^*)$. Defining a function S that returns the sign (positive, negative, or zero), we obtain $S(dz_f^*/dr_{f|U}) = S(dz_m^*/dr_{m|U}) = S(\partial J/\partial r_{f|U}) = S(\partial J/\partial r_{m|U}) = S(B(z_f^*)) = S(B(z_m^*))$ (Pen 2000; Farrell et al. 2015) and the same conclusions as in section 1.1 applies.

Therefore, 1) if the sleep of social partners improves an individual's fitness ($B > 0$), then the sleep optimum is higher for females than it is for males ($z_f^* > z_m^*$) when relatedness is higher for the former than for the latter ($r_{f|U} > r_{m|U}$), and the sleep optimum is lower for females than it is for males ($z_f^* < z_m^*$) when relatedness is lower for the former than for the latter ($r_{f|U} < r_{m|U}$); 2) if the sleep of social partners decreases an individual's fitness ($B < 0$), then the sleep optimum is lower for females than it if for males ($z_f^* < z_m^*$) when relatedness is higher for the former than for the latter ($r_{f|U} > r_{m|U}$), and the sleep optimum is higher for females than it is for males ($z_f^* > z_m^*$) when relatedness is lower for the former than for the latter ($r_{f|U} < r_{m|U}$); and 3) if the sleep of social partners does not affect an individual's fitness ($B = 0$), then the sleep optimum for females is equal to that for males ($z_f^* \approx z_m^*$) and relatedness does not shape the sleep optimum.

2. Illustrative model

Life cycle – We consider an infinite diploid population divided into patches (Wright 1931) containing n_f females and n_m males, with every female mating with every male in her patch, and vice versa. During their sleeping period, females and males spent a proportion of this time sleeping – level of sleep – which is necessary for the maintenance of the organism and to cooperate successfully with social partners in their patch. This is counterbalanced by the presence of an external danger, which deleterious effects increase with the level of sleep, and by the probability of gaining mating opportunities, which decreases with the level of sleep. Specifically, a female's fecundity is:

$$f_\mathrm{f} = (x_\mathrm{f} - m)^{b_\mathrm{f}}\left(\frac{y_\mathrm{f}+y_\mathrm{m}}{2} - m\right)\left(1 - \frac{y_\mathrm{f}+y_\mathrm{m}}{2}\right)^{a}\left(\frac{1-c_\mathrm{f}x_\mathrm{f}}{1-c_\mathrm{f}y_\mathrm{f}}\right), \tag{A4}$$

where: m is the minimal amount of sleep that individuals require; b_f defines how the benefits of sleep increase throughout the night for the females (close to 0 the benefits grow exponentially, close to 1 the benefits grow linearly); a is the probability of an external danger being present in the environment; and c_f is the probability of gaining mating opportunities by sacrificing sleep in females. Therefore, $(x_\mathrm{f} - m)^{b_\mathrm{f}}$ defines the maintenance of the focal female's body through sleep, $\left(\frac{y_\mathrm{f}+y_\mathrm{m}}{2} - m\right)$ defines the cooperation within the group, $\left(1 - \frac{y_\mathrm{f}+y_\mathrm{m}}{2}\right)^{a}$ defines the vigilance within the group, and $\left(\frac{1-c_\mathrm{f}x_\mathrm{f}}{1-c_\mathrm{f}y_\mathrm{f}}\right)$ defines the mating competition between the females in the group. Likewise, a male's fecundity is:

$$f_\mathrm{M} = (x_\mathrm{m} - m)^{b_\mathrm{m}}\left(\frac{y_\mathrm{f}+y_\mathrm{m}}{2} - m\right)\left(1 - \frac{y_\mathrm{f}+y_\mathrm{m}}{2}\right)^{a}\left(\frac{1-c_\mathrm{m}x_\mathrm{m}}{1-c_\mathrm{m}y_\mathrm{m}}\right), \tag{A5}$$

where b_m defines how the benefits of sleep increase throughout the night for the males (close to 0 the benefits grow exponentially, close to 1 the benefits grow linearly) and c_m is the probability of gaining mating opportunities by sacrificing sleep in males. Therefore, $(x_\mathrm{m} - m)^{b_\mathrm{m}}$ defines the maintenance of the focal male's body through sleep, and $\left(\frac{1-c_\mathrm{m}x_\mathrm{m}}{1-c_\mathrm{m}y_\mathrm{m}}\right)$ defines the mating competition between the males in the group. Following mating, each female produces a large number of offspring, with an even sex-ratio, in proportion to her fecundity. Adults then die. Juveniles then form groups – or buds – of large size at random within their patch and each group either disperse to a random patch with probability d_B or remain in the focal patch otherwise (Haldane 1932). After budding dispersal, juveniles can still disperse individually, with females dispersing with probability d_f and males dispersing with probability d_m to a random patch or else remaining in their current patch. Following individual dispersal, n_f females and n_m males survive at random within each patch to adulthood, returning the population to the beginning of the life cycle.

Natural selection – Female relative fitness in this model is given by:

$$W_\mathrm{f} = f_\mathrm{f}\left(\frac{1-d_\mathrm{B}}{(1-d_\mathrm{B})F_\mathrm{f}+d_\mathrm{B}\bar{F}_\mathrm{f}} + \frac{d_\mathrm{B}}{\bar{F}_\mathrm{f}}\right), \tag{A6}$$

where: $F_\mathrm{f} = f_\mathrm{f}|_{x_f=y_f}$; and $\bar{F}_\mathrm{f} = f_\mathrm{f}|_{x_f=z_f,\ y_f=z_f,\ y_m=z_m}$. Likewise, male relative fitness in this model is given by:

$$W_\mathrm{m} = \frac{f_\mathrm{m}}{F_\mathrm{m}}F_\mathrm{f}\left(\frac{1-d_\mathrm{B}}{(1-d_\mathrm{B})F_\mathrm{f}+d_\mathrm{B}\bar{F}_\mathrm{f}} + \frac{d_\mathrm{B}}{\bar{F}_\mathrm{f}}\right), \tag{A7}$$

where $F_{\mathrm{m}} = f_{\mathrm{m}}|_{x_m=y_m}$. We can now use the inequalities derived in section 1 to reach the marginal fitness equations for the evolution of sleep and, consequently, to derive the optimal level of sleep for the different scenarios explored in the main text (see below).

Relatedness – The relatedness between a genic actor A in the focal female with a randomly-chosen female in her patch (including the focal female herself) is approximately given by:

$$r_{\mathrm{ff|A}} = \frac{1}{n_{\mathrm{f}}} + \frac{n_{\mathrm{f}}-1}{n_{\mathrm{f}}}(1-d_{\mathrm{f}})^2 r_{\mathrm{A}}, \tag{A8}$$

where: with probability $\frac{1}{n_{\mathrm{f}}}$ the randomly chosen female is the focal female herself, in which case relatedness is 1; and with probability $\frac{n_{\mathrm{f}}-1}{n_{\mathrm{f}}}$ is a different female, in which case they are only related if they are both locals $(1-d_{\mathrm{f}})^2$ and, if so, their relatedness is defined by the relatedness through the genic actor A (r_{A}) in the focal female. The approximation becomes exact in the limit of vanishingly weak selection. For the relatedness between a genic actor A in the focal female with a random male in her patch:

$$r_{\mathrm{fm|A}} = (1-d_{\mathrm{f}})(1-d_{\mathrm{m}})r_{\mathrm{A}}, \tag{A9}$$

and they are only related if they are both locals $(1 - d_{\mathrm{f}})(1 - d_{\mathrm{m}})$ and, if so, their relatedness is defined by the relatedness through the genic actor A (r_{A}) in the focal female. For the relatedness between a genic actor A in the focal male and randomly-chosen male in his patch (including the focal male himself):

$$r_{\mathrm{mm|A}} = \frac{1}{n_{\mathrm{m}}} + \frac{n_{\mathrm{m}}-1}{n_{\mathrm{m}}}(1-d_{\mathrm{m}})^2 r_{\mathrm{A}}, \tag{A10}$$

where: with probability $\frac{1}{n_{\mathrm{m}}}$ the randomly chosen male is the focal male himself, in which case relatedness is 1; and with probability $\frac{n_{\mathrm{m}}-1}{n_{\mathrm{m}}}$ is a different male, in which case they are only related if they are both locals $(1-d_{\mathrm{m}})^2$ and, if so, their relatedness is defined by the relatedness through the genic actor A (r_{A}) in the focal male. For the relatedness between a genic actor A in the focal male with a random female in his patch:

$$r_{\mathrm{mf|A}} = (1-d_{\mathrm{m}})(1-d_{\mathrm{f}})r_{\mathrm{A}}, \tag{A11}$$

and they are only related if they are both locals $(1 - d_{\mathrm{m}})(1 - d_{\mathrm{f}})$ and, if so, their relatedness is defined by the relatedness through the genic actor A (r_{A}) in the focal male. Note that $r_{\mathrm{mf|A}} = r_{\mathrm{fm|A}}$ and, therefore, we use $r_{\mathrm{fm|A}}$ to represent both throughout the rest of the document.

Relatedness through the genic actor A between two different juveniles born in the same patch is then given by $r_{\mathrm{A}} = p_{\mathrm{A}}'/p$, where p_{A}' is the consanguinity through the genic actor A between two individuals born in the same patch and is defined by picking the genic actor A from the focal individual and a random gene from the other individual and calculating the probability that the two are identical by descent (Bulmer 1994). Focusing upon genes ignorant of their origin (A = U) and assuming that consanguinities are at their neutral-equilibrium values, appropriate if selection is weak (Gardner et al. 2011), we write:

$$p_{\mathrm{U}}' = \tfrac{1}{4}\left(\frac{1}{n_{\mathrm{f}}}p + \frac{n_{\mathrm{f}}-1}{n_{\mathrm{f}}}(1-d_{\mathrm{f}})^2 p_{\mathrm{U}}'\right) + \tfrac{1}{4}\left(\frac{1}{n_{\mathrm{m}}}p + \frac{n_{\mathrm{m}}-1}{n_{\mathrm{m}}}(1-d_{\mathrm{m}})^2 p_{\mathrm{U}}'\right) + \tfrac{1}{2}(1-d_{\mathrm{f}})(1-d_{\mathrm{m}})p_{\mathrm{U}}', \tag{A12}$$

where: with probability of ¼ we may have drawn the maternal-origin genes from both individuals, in which case with probability of $\frac{1}{n_\mathrm{f}}$ they share the same mother (and they have consanguinity of p) and with probability of $\frac{n_\mathrm{f}-1}{n_\mathrm{f}}$ they have different mothers (and they will only have consanguinity if both mothers are local, giving a consanguinity of $(1-d_\mathrm{f})^2 p_\mathrm{U}'$); with probability of ¼ we may have drawn the paternal-origin genes from both individuals, in which case with probability of $\frac{1}{n_\mathrm{m}}$ they share the same father (and they have consanguinity of p) and with probability of $\frac{n_\mathrm{m}-1}{n_\mathrm{m}}$ they have different fathers (and they will only have consanguinity if both fathers are local, giving a consanguinity of $(1-d_\mathrm{m})^2 p_\mathrm{U}'$); and with probability of ½ we have drawn the maternal-origin gene from one and the paternal-origin gene from the other and they will only have consanguinity if both these parents are locals (giving a consanguinity of $(1-d_\mathrm{f})(1-d_\mathrm{m})p_\mathrm{U}'$). Rearranging, we get:

$$p_\mathrm{U}' = \frac{n_\mathrm{f}+n_m}{(1-d_\mathrm{f})^2 n_\mathrm{m}+(1-d_\mathrm{m})^2 n_\mathrm{f}+(4-d_\mathrm{f}-d_\mathrm{m})(d_\mathrm{f}+d_\mathrm{m})n_\mathrm{m}n_\mathrm{f}} p, \tag{A13}$$

and the relatedness between two random individuals born in the same patch is then given by $r_\mathrm{U} = p_\mathrm{U}'/p$ (Bulmer 1994). Rearranging, we obtain:

$$r_\mathrm{U} = \frac{n_\mathrm{f}+n_m}{(1-d_\mathrm{f})^2 n_\mathrm{m}+(1-d_\mathrm{m})^2 n_\mathrm{f}+(4-d_\mathrm{f}-d_\mathrm{m})(d_\mathrm{f}+d_\mathrm{m})n_\mathrm{m}n_\mathrm{f}}. \tag{A14}$$

But we can also separate the consanguinity between two juveniles in their maternal- and paternal-origin components. That is:

$$p_\mathrm{U}' = \frac{1}{2}(p_\mathrm{M}' + p_\mathrm{P}'), \tag{A15}$$

and by its turn:

$$p_\mathrm{M}' = \frac{1}{2}\left(\frac{1}{n_\mathrm{f}} p + \frac{n_\mathrm{f}-1}{n_\mathrm{f}}(1-d_\mathrm{f})^2 p_\mathrm{U}'\right) + \frac{1}{2}(1-d_\mathrm{f})(1-d_\mathrm{m})p_\mathrm{U}'; \tag{A16}$$

$$p_\mathrm{P}' = \frac{1}{2}\left(\frac{1}{n_\mathrm{m}} p + \frac{n_\mathrm{m}-1}{n_\mathrm{m}}(1-d_\mathrm{m})^2 p_\mathrm{U}'\right) + \frac{1}{2}(1-d_\mathrm{f})(1-d_\mathrm{m})p_\mathrm{U}'. \tag{A17}$$

Relatedness between two random individuals in the same patch through their maternal-origin genes is then given by $r_\mathrm{M} = p_\mathrm{M}'/p$ (Bulmer 1994) and through their paternal-origin genes by $r_\mathrm{P} = p_\mathrm{P}'/p$ (Bulmer 1994). Rearranging, we obtain:

$$r_\mathrm{M} = \frac{(2-d_\mathrm{f}-d_\mathrm{m})\left(n_\mathrm{f}-d_\mathrm{m}+d_\mathrm{f}(1-n_\mathrm{f})\right)+n_\mathrm{m}(2+d_\mathrm{f}(1-d_\mathrm{m})+d_\mathrm{m}(3-d_\mathrm{m}))}{2n_\mathrm{f}(1-d_\mathrm{m})^2+2n_\mathrm{m}(1-d_\mathrm{f})^2+2n_\mathrm{f}n_\mathrm{m}(4-d_\mathrm{f}-d_\mathrm{m})(d_\mathrm{f}+d_\mathrm{m})}; \tag{A18}$$

$$r_\mathrm{P} = \frac{{d_\mathrm{f}}^2(1-n_\mathrm{f})+d_\mathrm{m}(2+n_\mathrm{f}-d_\mathrm{m}(1-n_\mathrm{m})-3n_\mathrm{m})+2(n_\mathrm{f}+n_\mathrm{m})-d_\mathrm{f}(2-n_\mathrm{f}(3-d_\mathrm{m})+n_\mathrm{m}(1-d_\mathrm{m}))}{2n_\mathrm{f}(1-d_\mathrm{m})^2+2n_\mathrm{m}(1-d_\mathrm{f})^2+2n_\mathrm{f}n_\mathrm{m}(4-d_\mathrm{f}-d_\mathrm{m})(d_\mathrm{f}+d_\mathrm{m})}. \tag{A19}$$

We can replace the equations (A14; A18-19) into the equations (A8-11) to obtain the different coefficients of relatedness for genes ignorant of their origin (A = U), for maternal-origin genes (A = M), and for paternal-origin genes (A = P), respectively.

2.1 Evolution of sleep cannot evolve independently in females and males

Sentinel model – Here we explore the case where individuals can sacrifice sleep to increase the vigilance in their group but not their mating opportunities ($c_f = c_m = 0$). We assume that the benefits that females and males get throughout their sleep is similar ($b_f = b_m = b$), that sleep cannot evolve independently in females and males ($\gamma_f = 1$; $\gamma_m = 1$), and diploidy. We now can obtain the derivatives of the left side of the inequality (A1) and obtain the marginal fitness equation for the present model:

$$\frac{2b\left(2-(1-d_B)^2 r_{ff|A}+(2-d_B)d_B r_{fm|A}-r_{mm|A}\right)(1-z^*)+(2-d_B)d_B(r_{ff|A}+2r_{fm|A}+r_{mm|A})(1+a\,m-(1+a)z^*)}{4(z^*-m)(1-z^*)} = 0. \quad \text{(A20)}$$

Now we can solve the equation (A20) for z^* to get the equation for the optimal level of sleep:

$$z^* = \frac{d_B(2-d_B)(1+am)\left(r_{ff|A}+2r_{fm|A}+r_{mm|A}\right)+2b\left(2\left(1+d_B r_{fm|A}\right)-(1-d_B)^2 r_{ff|A}-r_{fm|A}{d_B}^2-r_{mm|A}\right)}{d_B(1+a)(2-d_B)\left(r_{ff|A}+2r_{fm|A}+r_{mm|A}\right)+2b\left(2\left(1+d_B r_{fm|A}\right)-(1-d_B)^2 r_{ff|A}-r_{fm|A}{d_B}^2-r_{mm|A}\right)}. \quad \text{(A21)}$$

We can now use the equation (A21) with the values of the main text to get the Figure 1*a*, with the assumption that genes are ignorant to their origin (A = U). We can also obtain Figure S1*a* with that same equation, following the same assumption, but now using different values (see below). Similarly, we can obtain the values of sleep favoured by the maternal-origin genes (A = M) and by the paternal-origin genes (A = P) as shown in Figure 3*a* and Figure S3*a*.

Reproductive model – Here we explore the case where individuals can sacrifice sleep to gain additional mating opportunities but not to increase the vigilance in their group ($a = 0$). We assume that females and males benefit from this strategy to the same degree ($c_f = c_m = c$). We also assume that the benefits that females and males get throughout their sleep is similar ($b_f = b_m = b$), that sleep cannot evolve independently in females and males ($\gamma_f = 1$; $\gamma_m = 1$), and diploidy. Using the same approach as above, we get the following marginal fitness equation:

$$\frac{(1-cz^*)\left(d_B(2-d_B)\left(r_{ff|A}+2r_{fm|A}+r_{mm|A}\right)+2b\left(2-(1-d_B)^2 r_{ff|A}+d_B(2-d_B)r_{fm|A}-r_{mm|A}\right)\right)-2c(2-r_{ff|A}-r_{mm|A})(z^*-m)}{4(z-m)(1-cz^*)} = 0. \quad \text{(A22)}$$

Now we can solve the equation (A22) for z^* to get the equation for the optimal level of sleep:

$$z^* = \frac{2cm\left(2-r_{ff|A}-r_{mm|A}\right)+d_B(2-d_B)\left(r_{ff|A}+2r_{fm|A}+r_{mm|A}\right)+2b\left(2-(1-d_B)^2 r_{ff|A}+d_B(2-d_B)r_{fm|A}-r_{mm|A}\right)}{c\left(2\left(2-r_{ff|A}-r_{mm|A}\right)+d_B(2-d_B)\left(r_{ff|A}+2r_{fm|A}+r_{mm|A}\right)+2b\left(2-(1-d_B)^2 r_{ff|A}+d_B(2-d_B)r_{fm|A}-r_{mm|A}\right)\right)}. \quad \text{(A23)}$$

We can now use the equation (A23) with the values of the main text to get the Figure 1*b*, with the assumptions that genes are ignorant to their origin (A = U). We can also obtain Figure S1*b* with that same equation, following the same assumption, but now using different values (see below). As before, we can also obtain the values of sleep favoured by the maternal-origin genes (A = M) and by the paternal-origin genes (A = P) as shown in Figure 3*b* and Figure S3*b*.

2.2 Evolution of sleep can evolve independently in females and males

Sentinel model – We now assume that sleep can evolve independently in females and males. As before, we assume that individuals can sacrifice sleep to increase the vigilance in their group but not their mating opportunities ($c_f = c_m = 0$), that the benefits that females and males get throughout their sleep is similar ($b_f = b_m = b$), that genes are ignorant to their origin (A = U), and diploidy. We now focus on females' sleep ($\gamma_f = 1$; $\gamma_m = 0$). We can obtain the

derivatives of the left side of the inequality (A2) and, with that, the marginal fitness equation for the females:

$$\frac{1}{2}\left(\frac{b(1-r_{\mathrm{ff|U}}(1-d_{\mathrm{B}})^2+r_{\mathrm{fm|U}}d_{\mathrm{B}}(2-d_{\mathrm{B}})}{z_{\mathrm{f}}{}^{*}-m}+d_{\mathrm{B}}(2-d_{\mathrm{B}})(r_{\mathrm{ff|U}}+r_{\mathrm{fm|U}})(\frac{1}{z_{\mathrm{f}}{}^{*}+z_{\mathrm{m}}-2m}-\frac{a}{2-z_{\mathrm{f}}{}^{*}-z_{\mathrm{m}}})\right)=0. \quad \text{(A24)}$$

We now focus on males' sleep ($\gamma_{\mathrm{f}} = 0$; $\gamma_{\mathrm{m}} = 1$). We can obtain the derivatives of the left side of the inequality (A3) and, with that, the marginal fitness equation for the males:

$$\frac{1}{2}\left(\frac{b(1-r_{\mathrm{mm|U}})}{z_{\mathrm{m}}{}^{*}-m}+\ d_{\mathrm{B}}(2-d_{\mathrm{B}})(r_{\mathrm{mm|U}}+r_{\mathrm{fm|U}})(\frac{1}{z_{\mathrm{f}}+z_{\mathrm{m}}{}^{*}-2m}-\frac{a}{2-z_{\mathrm{f}}-z_{\mathrm{m}}{}^{*}})\right)=0. \quad \text{(A25)}$$

Now we can solve the system of equations (A24-25) to get the optimal solutions for both z_{f}* and z_{m}*, similar to what we did above. Those solutions can then be used to get the Figure 2*a*, using the values of the main text. Note, however, that using those values means that the equation (A24) is always positive and, as such, females are selected to sleep as much as possible. This result needs to be incorporated into the equation (A25) when trying to find the males' optimal level of sleep. When doing so, there are two solutions but only one makes sense given the assumptions of the model. A similar pattern is present when using the values of Figure S2*a*. However, after a certain value of male dispersal, the equation is no longer always positive, meaning that we can simply use the solutions of the system of equations (A24-25) to represent the optimal level of sleep for both females and males.

Reproductive model – As in the previous section, here we assume that sleep can evolve independently in females and males, but now we assume that males are the only ones that can sacrifice sleep to increase their mating opportunities ($c_{\mathrm{f}} = 0$) and that individuals do not sacrifice sleep to increase the vigilance in their group ($a = 0$). Once again, we assume that the benefits that females and males get throughout their sleep is similar ($b_{\mathrm{f}} = b_{\mathrm{m}} = b$), that genes are ignorant to their origin (A = U), and diploidy. We now focus on females' sleep ($\gamma_{\mathrm{f}} = 1$; $\gamma_{\mathrm{m}} = 0$). We can obtain the derivatives of the left side of the inequality (A2) and, with that, the marginal fitness equation for the females:

$$\frac{1}{2}\left(\frac{b(1-r_{\mathrm{ff|U}}(1-d_{\mathrm{B}})^2+r_{\mathrm{fm|U}}d_{\mathrm{B}}(2-d_{\mathrm{B}})}{z_{\mathrm{f}}{}^{*}-m}+\frac{d_{\mathrm{B}}(2-d_{\mathrm{B}})(r_{\mathrm{ff|U}}+r_{\mathrm{fm|U}})}{z_{\mathrm{f}}{}^{*}+z_{\mathrm{m}}-2m}\right)=0. \quad \text{(A26)}$$

We now focus on males' sleep ($\gamma_{\mathrm{f}} = 0$; $\gamma_{\mathrm{m}} = 1$). We can obtain the derivatives of the left side of the inequality (A3) and, with that, the marginal fitness equation for the males:

$$\frac{1}{2}\left(\frac{b(1-r_{\mathrm{mm|U}})}{z_{\mathrm{m}}{}^{*}-m}+\frac{d_{\mathrm{B}}(2-d_{\mathrm{B}})(r_{\mathrm{mm|U}}+r_{\mathrm{fm|U}})}{z_{\mathrm{f}}+z_{\mathrm{m}}{}^{*}-2m}-\frac{c_{\mathrm{m}}(1-r_{\mathrm{mm|U}})}{1-c_{\mathrm{m}}z_{\mathrm{m}}{}^{*}}\right)=0. \quad \text{(A27)}$$

Note that equation (A26) is always positive under the assumptions of the model. Therefore, we can simply assume that females are selected to not sacrifice any sleep. Incorporating such assumptions into equation (A27) means that two solutions are possible for z_{m}*. Only one makes sense given the assumptions of the model and, therefore, we can use the values of the main text to get Figure 2*b* and, using different values (see below), Figure S2*b* to represent the males' optimal level of sleep.

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Table S1. The different hypothesized functions for sleep.

Hypothesis	Definition	References
Energy allocation	Organisms are selected to allocate energy to basic functions - growth, maintenance, reproduction - in a manner that maximizes energy use. During the waking period, organisms are selected to downregulate costly biological activities, such as the ones that allow the maintanance of the tissues. Those same activities are then upregulated during sleep, a period where energy is not being used by the individual in other activities, such as foraging or collecting environmental information.	St-Onge 2013; Schmidt 2014.
Adaptive inactivity	Depending on their ecology, organisms will have periods of time where none of their biological requirements can be satisfied due to biotic and abiotic environmental factors. Organisms are selected to maximize the efficiency of their behaviour, therefore being selected to reduce energy use when activity can be costly and not beneficial.	Meddis 1975; Siegel 2009; Field and Bonsall 2018.
Metabolic clearance	During the waking period, individuals accumulate metabolites in the interstitial space of their cerebral cortex as a result of their normal activity. Those metabolites are then cleared away during sleep.	Xie et al 2013; Herculano-Houzel 2015.
Maturation	Sleep allows for the development of the central nervous and sensorimotor systems and their correspondent functions.	Roffwarg et al 1966; Frank et al 2001; Shaffery et al 2002; Blumberg 2015; Bridi et al 2015.
Memory consolidation	Sleep promotes consolidation of memories acquired during the waking state into a network of long-term memories.	Jenkins and Dallenbach 1924; Karni et al 1994; Maquet 2001; Stickgold 2005; Born et al 2006; Diekelmann and Born 2010.
Synaptic homeostasis	Sleep normalizes synaptic activity to normal levels after a waking period where synapses are being triggered by learning and environmental stimulus, therefore restoring neuronal selectivity and the ability to learn new memories.	Tononi and Cirelli 2003; 2006; 2014.

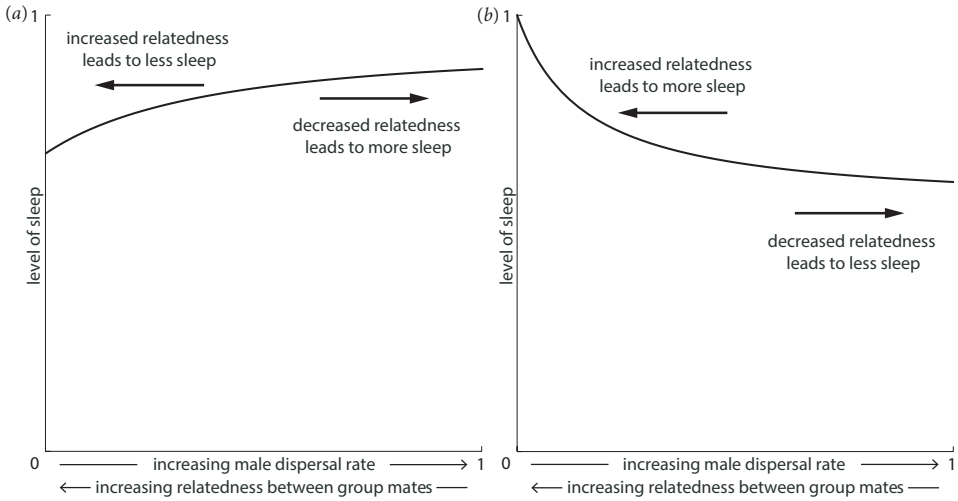

Figure S1. How much an individual should sleep depends on the relatedness between the individuals in a group. When individuals sacrifice sleep in order to remain alert to dangers which may befall the group (*a*), individuals sacrifice more sleep when relatedness is higher, which is the case when male dispersal is lower. When individuals sacrifice sleep in order to gain an advantage over their mate competitors (*b*), individuals sacrifice more sleep when relatedness is lower, which is the case when male dispersal is higher. The following parameter values were used for both panels: female dispersal rate $d_f = 0$; budding dispersal rate $d_B = 1$; number of adult females $n_f = 4$; number of adult males $n_m = 4$; minimum level of sleep $m = 0.05$; and benefits of sleeping throughout the night $b_f = b_m = 1$. Additionally, in (*a*) the level of a threat is $a = 1$ and the mating opportunities that females and males can obtain through sleep sacrifice is $c_f = c_m = 0$, while in (*b*) the level of a threat is $a = 0$ and the mating opportunities that females and males can obtain through sleep sacrifice is $c_f = c_m = 1$. Here, we consider male-biased dispersal.

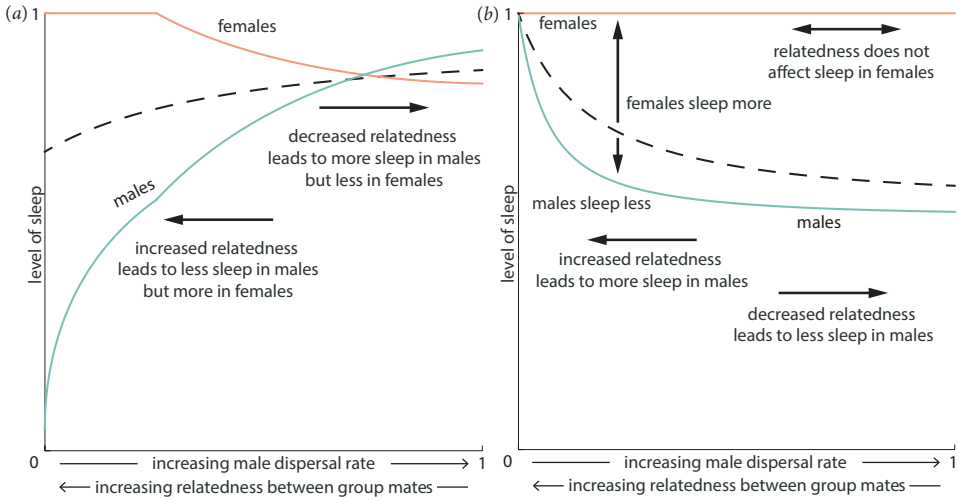

Figure S2. Females and males may be favoured to have different sleeping levels. Given that females are more related to their social partners than males, females favour less sleep when (*a*) the sleep sacrifice is being used to protect the group against threats. When (*b*) sleep sacrifice is being used to increase male reproductive success, females do not favour any sleep sacrifice, with males being the only ones to sacrifice sleep to gain an advantage over their mate competitors. Dashed line represents the favoured level of sleep when this is constrained to be the same for females and males. The following parameter values were used for both panels: female dispersal rate $d_f = 0$; budding dispersal rate $d_B = 1$; number of adult females $n_f = 4$; number of adult males $n_m = 4$; minimum level of sleep $m = 0.05$; and benefits of sleeping throughout the night $b_f = b_m = 1$. Additionally, in (*a*) the level of a threat is $a = 1$ and the mating opportunities that females and males can obtain through sleep sacrifice is $c_f = c_m = 0$, while in (*b*) the level of a threat is $a = 0$ and the mating opportunities that females and males can obtain through sleep sacrifice is $c_f = 0$ and $c_m = 1$, respectively. Here, we consider male-biased dispersal.

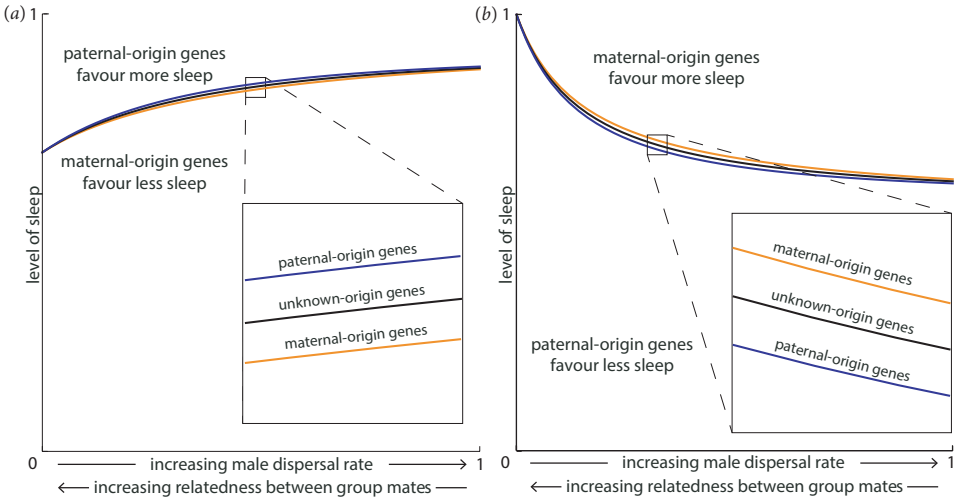

Figure S3. Maternal- and paternal-origin genes disagree regarding how much the individual should sleep. Maternal-origin genes (orange) and paternal-origin genes (blue) will disagree on how much an individual should sleep, which depends upon whether individuals are sacrificing sleep to (*a*) protect the group against threats or (*b*) gain an advantage over their mate competitors (with black being the level favoured by a gene ignorant of its origin). Specifically, given that relatedness is higher for maternal-origin genes, maternal-origin genes favour less sleep and paternal-origin genes more sleep if sleep is selfish (*a*). In constrast, if sleep is altruistic, then maternal-origin genes favour more sleep and paternal-origin genes less sleep (*b*). The following parameter values were used for both panels: female dispersal rate d_f = 0; budding dispersal rate d_b = 1; number of adult females n_f = 4; number of adult males n_m = 4; minimum level of sleep m = 0.05; and benefits of sleeping throughout the night $b_f = b_m = 1$. Additionally, in (*a*) the level of a threat is a = 1 and the mating opportunities that females and males can obtain through sleep sacrifice is $c_f = c_m = 0$, while in (*b*) the level of a threat is a = 0 and the mating opportunities that females and males can obtain through sleep sacrifice is $c_f = c_m = 1$. Here, we consider male-biased dispersal.

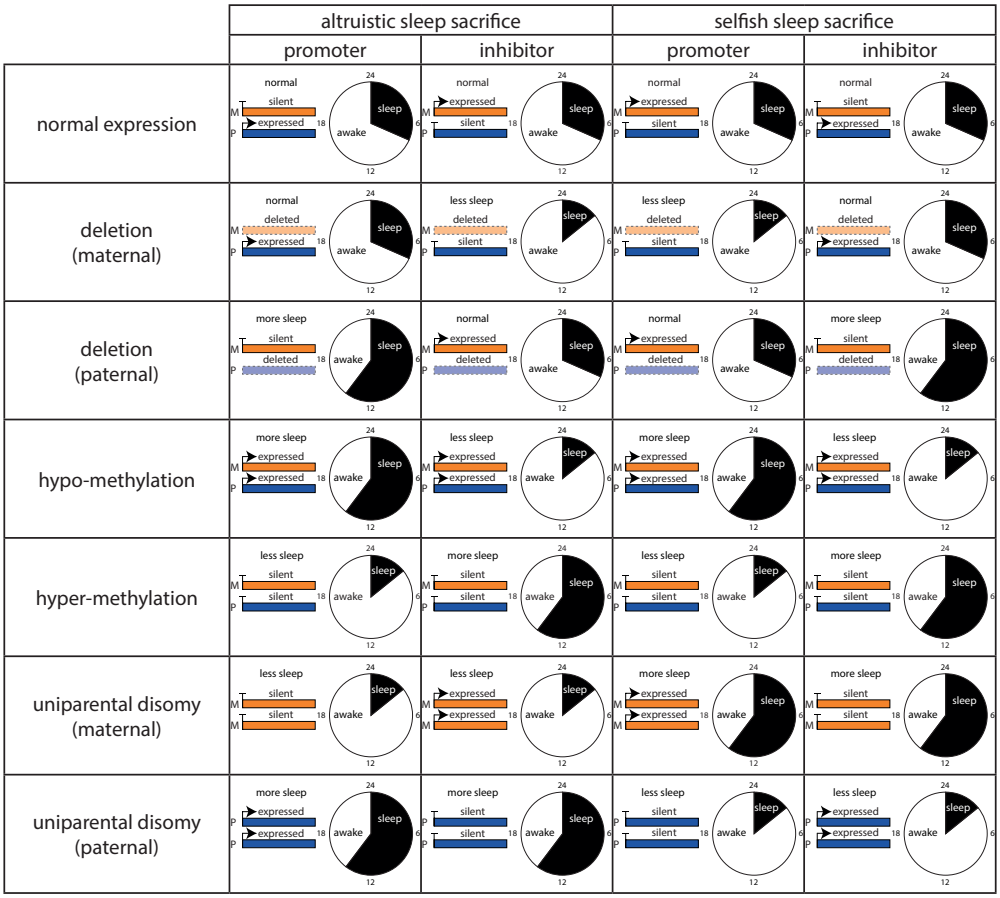

Figure S4. Genomic imprinting of genes responsible for level of sleep and the effects of possible disruptions. Predictions as to which gene is expressed and which gene is silent – maternal-origin gene (M, orange) or paternal-origin genes (P, blue) – when individuals are sacrificing sleep to protect the group against threats (altruism) or to gain an advantage over their mate competitors (selfishness). We consider an example for a gene that promotes sleep (promoter) and an example for a gene that inhibits sleep (inhibitor). In both cases, we assume male-biased dispersal. Note that for simplicity we assume methylation is associated with gene silencing, as is usually the case in mammals (Bird 2002). In cases where methylation is associated with gene activation the outcome for hypo-methylation is expected to be that shown here for hyper-methylation, and vice versa.