Romantic Love and Sleep

Subjects: Biology
Contributor: Adam Bode

“Romantic love is a motivational state typically associated with a desire for long-term mating with a particular individual. It occurs across the lifespan and is associated with distinctive cognitive, emotional, behavioural, social, genetic, neural, and endocrine activity in both sexes. Throughout much of the life course, it serves mate choice, courtship, sex, and pair-bonding functions. It is a suite of adaptations and by-products that arose sometime during the recent evolutionary history of humans”.

Sleep is common in the animal kingdom, although it takes various forms. Sleep in humans is defined “on the basis of both behaviour of the person while asleep and the related physiologic changes that occur to the waking brain’s electrical rhythm in sleep”. Behavioural characteristics of sleep include lack of mobility or slight mobility, closed eyes, a characteristic species-specific sleeping posture, reduced response to external stimulation, quiescence, increased reaction time, elevated arousal threshold, impaired cognitive function, and a reversible unconscious state. It includes non-rapid eye movement (NREM) sleep and rapid eye movement (REM) sleep. Non-rapid eye movement sleep is characterised by synchronised electroencephalographic activity, mildly reduced muscle tone, and slow rolling eye movements. Rapid eye movement sleep is characterised by theta or saw tooth waves and desynchronised electroencephalographic activity, moderately to severely reduced or absent muscle tone, and rapid eye movements. There are three stages of NREM sleep (i.e., N1, N2, N3) and one stage of REM sleep.

Keywords: romantic love ; sleep ; Tinbergen ; mechanisms ; functions ; evolution

1. Romantic Love

“Romantic love is a motivational state typically associated with a desire for long-term mating with a particular individual. It occurs across the lifespan and is associated with distinctive cognitive, emotional, behavioural, social, genetic, neural, and endocrine activity in both sexes. Throughout much of the life course, it serves mate choice, courtship, sex, and pair-bonding functions. It is a suite of adaptations and by-products that arose sometime during the recent evolutionary history of humans” [1] (p. 21).

As above-mentioned, Bode and Kushnick [1] describe the mechanisms, development across the lifespan, functions, and evolutionary history of romantic love in detail. In summary, romantic love is caused by social and interactive characteristics: reciprocal liking, propinquity, social influence, and the filling of needs [2][3][4]. It is generated by psychological mate choice mechanisms: mate preferences [5], attraction [6], and sexual desire [7]. Specific genetic polymorphisms that regulate dopamine 2 receptors, vasopressin receptors, oxytocin receptors, dopamine 4 receptors, and dopamine transmission are associated with romantic love [8][9]. Romantic love is driven by activity in various neurobiological systems: mesolimbic reward pathway (e.g., ventral tegmental area, nucleus accumbens, amygdala, and medial prefrontal cortex [10]), emotion regulation (e.g., amygdala, anterior cingulate cortex, and the insula) (see [1]), sexual desire and arousal (e.g., caudate, insula, putamen, and anterior cingulate cortex) [11][12], social cognition (e.g., amygdala, insula, and medial prefrontal cortex) (see [1]), and others [13]. It is also caused by endocrinological activity in multiple systems: those that regulate sex hormones (i.e., testosterone, follicle-stimulating hormone, luteinising hormone), serotonin, dopamine, oxytocin, cortisol, and nerve growth factor [14][15][16][17][18][19][20][21]. Romantic love can first emerge in childhood [22], becomes more frequent and expresses with most of its characteristics in adolescence, but manifests throughout the lifespan [23].

Romantic love serves the evolutionary functions of mate choice [24], courtship [24], sex [25], and pair-bonding [26]. Romantic love probably evolved by co-opting mother–infant bonding sometime prior to, or following, the human line split from our common ancestor with chimpanzees and bonobos [1].

2. Sleep

2.1. Definition, Characteristics, and Measurement

Sleep is common in the animal kingdom, although it takes various forms [27][28]. Sleep in humans is defined "on the basis of both behaviour of the person while asleep and the related physiologic changes that occur to the waking brain's electrical rhythm in sleep" [29] (p. 7). Behavioural characteristics of sleep include lack of mobility or slight mobility, closed eyes, a characteristic species-specific sleeping posture, reduced response to external stimulation, quiescence, increased reaction time, elevated arousal threshold, impaired cognitive function, and a reversible unconscious state. It includes non-rapid eye movement (NREM) sleep and rapid eye movement (REM) sleep. Non-rapid eye movement sleep is characterised by synchronised electroencephalographic activity, mildly reduced muscle tone, and slow rolling eye movements. Rapid eye movement sleep is characterised by theta or saw tooth waves and desynchronised electroencephalographic activity, moderately to severely reduced or absent muscle tone, and rapid eye movements [29]. There are three stages of NREM sleep (i.e., N1, N2, N3) and one stage of REM sleep [30].

Sleep can be measured in multiple ways. These can include self-report [31], observational [32], and objective methods (see [33]). Self-report measures can include the collection of data about sleep onset, sleep timing, sleep duration, wake after sleep onset (WASO), sleep quality, restoration after sleep, sleep regularity, and causes of sleep disturbance (see, for example, [31][34][35][36][37][38]). Observational methods can identify the behavioural characteristics of sleep. The two most common objective measures of sleep are the accelerometry or actigraphy, which measures movement of the individual, and polysomnography (PSG) [39]. Polysomnography is a systematic process used to collect physiologic parameters during sleep [40]. It involves a combination of electroencephalogram (EEG), electro-oculogram (EOG), electromyogram (EMG), electrocardiogram (ECG), pulse oximetry, and measures of airflow and respiratory effort. It is the only means of assessing stages of sleep and is the gold standard for sleep research and diagnosing sleep disorders. Despite PSG being the gold standard, the full range of methods may be useful in detecting different aspects of sleep. **Figure 1** presents a list of the features of sleep according to Tinbergen's [41] four questions.

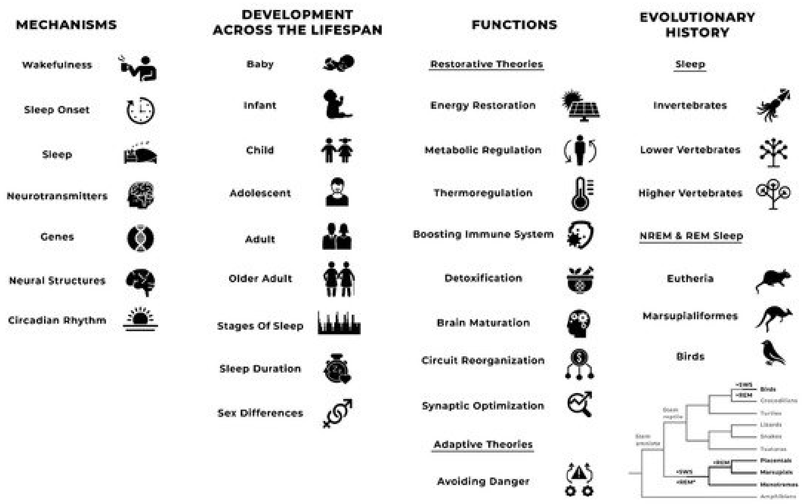

Figure 1. List of features of sleep according to Tinbergen's four questions. Features of sleep are presented according to the order in which they are presented in this article. Cladogram taken from Lesku and colleagues [42].

2.2. Mechanisms

Mechanisms relating to different aspects of sleep have been studied in both animals and humans (see [43][44]). Wakefulness is regulated by the basal forebrain, lateral hypothalamus, tuberomammillary nucleus, and brainstem, with involvement of norepinephrine, dopamine, serotonin, acetylcholine, histamine, hypocretin, and neuropeptide S systems (see [45] for review). Sleep onset is induced by cytokines and hormones, adenosine, prostaglandins, anandamide, and urotensin II (see [45] for review). A number of neural structures regulate sleep: the suprachiasmatic nucleus (SCN), basal forebrain, medial, lateral, and ventrolateral preoptic nuclei, and brainstem (see [45] for review). The primary neurochemical mechanisms that regulate sleep include gamma-aminobutyric acid and acetylcholine (see [45] for review). Some immediate-early genes are up- or downregulated during sleep compared to the waking state (see [45] for review). Specifically, the preoptic area, basal forebrain, and cortical sleep-active neuronal nitric oxide synthase neurons may play particularly important roles in NREM sleep (see [45] for review). The pedunculopontine/laterodorsal tegmental nuclei, sublaterodorsal nucleus, medullary reticular formation, and parts of the hypothalamus probably play specific roles in REM sleep (see [45] for review).

One of the key mechanisms that regulates the sleep–wake cycle as well as metabolism, heart rate, blood pressure, body temperature, renal activity, and hormone secretion is the circadian rhythm. The circadian rhythm results from environmental cues (i.e., light exposure) as well as an endogenous circadian timing system. This timing system throughout the body is largely regulated by two clusters of neurons in the SCN, located in the anterior hypothalamus, which coordinate overt rhythms through neuronal and hormonal outputs [46]. Another key mechanism might be the basic rest–activity cycle, which is a physiological rhythm and has a period shorter than 24 h, running throughout the 24 h with four cycles during the day and five at night [47].

2.3. Development across the Lifespan

In the first few weeks of life, sleep can total up to 16 h in a day. By about six weeks of life, the infant is more awake during the day and sleeps more at night. By four months of age, most infants sleep most of the night. At this stage of development, infants have three distinct sleep stages: active sleep, quiet sleep, and intermediate sleep. In the first year of life, sleep duration averages 14 h in a day and by six months of age, infants are generally sleeping predominantly at night (see [48]). In early childhood, the stages of sleep are the same as in adults, although the length each one lasts is different. Prior to the onset of puberty, children sleep about 9–11 h per day, almost exclusively at night (see [48]). At puberty, both sleep onset and natural awakenings are delayed. In the transition from adolescence to adulthood, the length of various NREM stages change, resulting in lighter sleep. The delayed onset of sleep and wakening associated with puberty subsides in adulthood and a 90 min sleep cycle of NREM-to-REM stages is established with all sleep stages represented (see [48]). There is a small reduction in REM sleep in early- and mid-adulthood. Older age is associated with earlier sleep onset and poorer sleep quality (see [42]; see also [49] for summary of ontogeny of functions at specific developmental stages).

Sleep disturbances can affect any age group and are not a normal part of ageing (see [50]). However, sleep problems are common among older people because medical conditions and changes in social engagement, lifestyle, and living environment associated with ageing can contribute to sleep problems [51]. Artificial light and mistimed light, associated with the modern environment, affects both circadian rhythms and sleep–wake cycles [52][53].

There are some sex differences in human sleep. Females report poorer sleep quality and a greater risk of developing some specific sleep disorders such as insomnia than males [54][55]. Males, on the other hand, have a greater risk of developing some other sleep disorders such as obstructive sleep apnoea than females [54][55]. Variations in hormones, physical and mental condition, social roles, and ageing are among the factors that explain this sex difference. Sleep disturbances are common during menstruation, pregnancy, and menopause (see [54][56]). Later sleep timing associated with puberty starts earlier in females, because, on average, they reach puberty earlier than males.

2.4. Functions

Two lines of theory outline the functions of sleep: restorative theories and adaptive theories [57]. Restorative theories suggest that sleep serves a number of functions including energy restoration, metabolic regulation, thermoregulation, boosting the immune system, brain and body detoxification, brain maturation, circuit reorganisation, and synaptic optimisation [58]. It is essential for many vital functions including physiological, somatic, and neuroanatomical development, energy conservation, brain waste clearance, modulation of immune responses, cognition, performance, vigilance, disease response, and psychological state [43]. Long-term sleep loss and sleep disorders have been associated with a number of deleterious health effects including cancer [59], hypertension, type 2 diabetes, obesity, depression, heart attack, and stroke [60]. Common sleep disorders involve, or include respiratory disorders of sleep, insomnia, hypersomnia, parasomnia, circadian rhythm disorders, and sleep movement disorders [61].

Non-rapid eye movement sleep is associated with immune system function by playing a role in the formation of immunological memory [62] and supporting the immune system’s ability to anticipate infectious threats from injury [63]. Slow cortical oscillations in NREM sleep facilitate restoration and repair of the body and the nervous system—the latter probably by enabling information processing, synaptic plasticity, and prophylactic cellular maintenance [64]. This also facilities memory processing. The three stages of NREM sleep are associated with a progressive deactivation of a select group of neurons in the brain structures that are reactivated during REM sleep [65]. It is for this reason that REM sleep has been dubbed “paradoxical sleep”. High levels of brain metabolic demand and attenuation of homeostatic regulation make it unclear how REM sleep can be adaptive in the broader context of sleep [66].

Adaptive theories suggest that animals sleep to avoid danger. At first glance, it appears that, during sleep, an individual is largely non-responsive to environmental stimuli, placing them at risk of harm from the social and physical environment. This is true, but it must be considered in the context of trade-offs and our evolutionary history. The result of trade-offs (see

[67]) indicates that the benefits of sleep outweigh any immediate costs to survival. It should also be noted that sleep evolved long ago in our evolutionary history, and we most likely sleep at night because this is the time when resource collection is at its lowest and is the period in which the greatest risk of predation existed during our evolutionary history (see [57]). There is empirical support for the notion that a sleep strategy is adaptive in most contexts [68].

2.5. Evolutionary History

We know relatively little about the evolution of NREM and REM sleep, or sleep generally [28]. Sleep involving NREM and REM sleep is ubiquitous among placental (*Eutheria*) and marsupial (*Marsupialiformes*) mammals and birds [42]. Sleep is common in reptiles, amphibians, and fish [29]. Behaviour analogous to sleep has been identified in numerous invertebrates and lower vertebrates [58], suggesting that it is evolutionarily old. Non-rapid eye movement sleep and REM sleep are thought to have evolved as a differentiation of a single, phylogenetically older sleep state [69]. REM sleep, or a precursor state with aspects of REM sleep, may have originated in reptiles [70][71]. The presence of both types of sleep in birds and mammals is probably the result of parallel evolution [42].

3. Romantic Love and Sleep Variations

To our knowledge, there are seven studies that have empirically investigated sleep in people experiencing romantic love [35][36][37][38][72][73][74]. These studies have investigated adolescents and young adult females and males in Iran, German-speaking countries, and Finland. Measures of romantic love include individual questions about love status or a variation of the Yale–Brown Obsessive Compulsive Scale [75], which measures the intensity of romantic love. Sleep is generally measured by self-report questionnaires. However, one study [74] used an accelerometer, although this was an average of 7.2 months after participants self-reported being in love. Aspects of sleep for which data have been collected include sleep onset latency, sleep duration, wake after sleep onset (WASO), sleep quality, and restoring sleep, although several other related factors have also been investigated (i.e., concentration during the day, tiredness during the day, and mood). Kuula and colleagues [74] investigated the clock times at which individuals slept. Studies used parametric and nonparametric tests to identify relationships between romantic love and sleep features.

The sum of evidence is mixed regarding the effect of romantic love on sleep. There appears to be an age-related effect; some self-reported sleep features are associated with romantic love in young adults, but not adolescents, although this could be the result of the methods employed in these studies. The two studies investigating sleep variations in young adults measured the association of romantic love intensity and sleep features, whereas the majority of studies investigating adolescents simply grouped participants according to the presence or absence of romantic love, so the intensity of romantic love would have been variable in these groups. One study [73] was a longitudinal study of adolescents that found no relationship between sleep features and either the onset or extinction of romantic love. These findings suggest that factors that influence sleep (e.g., developmental stage) may also moderate the effect of romantic love on sleep. **Table 1** presents a summary of the evidence supporting the influence of particular sleep features in adolescents and young adults who were experiencing romantic love.

Table 1. Sleep variations in people experiencing romantic love (evidence from at least two studies).

	Adolescents	Young Adults	Studies
Sleep onset latency	**-**	**Shorter**	**[37][38]; see also [35][36][72][73]**
Sleep duration	**Shorter**	**-**	**[72][74] *; see also [35][36][37][38][73]**
WASO	**-**	**Fewer**	**[37][38]; see also [35][36]**
Sleep quality	**-**	**Better**	**[37][38]; see also [72] and [35][36][37][73][74]**
Restoring sleep		**Increased**	**[37][38]**

Notes. Refs. [37][38] investigated the intensity of romantic love; restoring sleep has not been investigated in adolescents. Ref. [72] was the only study of adolescents that found a significant association with sleep quality; WASO = wake after sleep onset; * = [74] Females only; - = non-significant association.

The evidence is mixed on the effect of romantic love on sleep onset. Sleep onset latency appears to be affected by romantic love in young adults but not adolescents. None of the studies investigating self-reported sleep onset latency in adolescents experiencing romantic love [35][36][72] found a significant difference in self-reported sleep onset latency. Falling in love and falling out of love were not associated with differences in self-reported sleep onset latency [73]. Two studies [37]

[38] did, however, find that a greater intensity of romantic love was associated with shorter self-reported sleep onset latency in young adults. Kuula and colleagues [74] found that female adolescents who were in love and in a relationship had the latest self-reported sleep midpoint among any group in their sample, suggesting that sleep onset may be delayed for this group.

The evidence is mixed about the effect of romantic love on sleep duration. Brand and colleagues [72] found shorter self-reported sleep duration in adolescents experiencing romantic love compared to controls and shorter sleep duration with greater intensity of romantic love. Kuula and colleagues [74] found that adolescent females experiencing romantic love self-reported shorter sleep duration than the controls. All other studies that investigated sleep duration [35][36][37][38][73] found no significant effect.

The evidence is mixed about the effect of romantic love on WASO. Wake after sleep onset appears to be reduced by romantic love in young adults but not adolescents. Studies that investigated self-reported number of WASO in adolescents found no significant effect of romantic love [35][36]. The two studies on young adults [37][38], however, found that the intensity of romantic love was negatively associated with self-reported number of bouts of WASO.

The evidence is mixed regarding the effect of romantic love on sleep quality. Brand and colleagues [72] found a significant effect of romantic love on sleep quality. In that study, adolescents who were in love reported better sleep quality than the controls (although it is important to note that the study excluded participants that might meet the criteria for a psychiatric disorder). The remaining adolescent studies [35][36][73][74] found no significant effect of romantic love on sleep quality. Both studies of young adults [37][38], however, found that the intensity of romantic love was associated with better sleep quality, as measured by the Insomnia Severity Index [76], and more restoring sleep [37][38].

Psychopathological Symptoms Associated with Sleep Variations

Just as developmental stage may play a role in moderating the relationship between romantic love and sleep, other psychological factors may influence the effect of romantic love on sleep features. Symptoms of hypomania, depression, and anxiety appear to be associated with specific sleep variations in people experiencing romantic love. This is relevant because certain symptoms of hypomania [37][38], depression symptoms [37][38][74], and anxiety symptoms [35][37][38][73][74][77] are associated with romantic love. Sleep variations associated with these symptoms in people experiencing romantic love may simply be the consequence of psychopathology. However, we think that it is also possible that these symptoms may be caused by romantic love, and any relationship between symptoms of psychopathology and sleep variations may be indirectly caused by romantic love.

Two studies of young adults [37][38] found that specific constellations of hypomanic symptoms are associated with the intensity of romantic love, and that each of these constellations is associated with different sleep variations and other symptoms of psychopathology in young adults experiencing romantic love. Active/elated hypomania symptoms were associated with shorter sleep onset latency, shorter sleep duration, fewer WASO, better sleep quality, and increased restoring sleep. Irritable/risk-taking hypomanic symptoms were associated with longer sleep onset latency, more WASO, and worse sleep quality [37][38]. One of those studies [37] found that irritable/risk-taking symptoms were associated with shorter sleep duration.

The results of both studies in young adults [37][38] found that depressive symptoms were associated with longer sleep onset latency, shorter sleep duration, more WASO, poorer sleep quality, and decreased restoring sleep. One study in adolescents [35] found that increased depressive symptoms were associated with worse sleep quality and fewer WASO, while another [74] found that increased depressive symptoms were associated with later sleep timing, shorter sleep duration, and worse sleep quality. Sleep variations and associated tiredness and fatigue are measured in some measures of depressive symptoms (i.e., BDI; BDI-II), meaning that the association may be inflated.

Both studies in young adults [37][38] found that anxiety symptoms were also associated with longer sleep onset latency, shorter sleep duration, more WASO, and poorer sleep quality. One of these studies [38] found that anxiety was associated with decreased restoring sleep. Kuula and colleagues [74] found that anxiety symptoms were associated with later sleep timing, shorter sleep duration, and poorer sleep quality in adolescent females and males. There are numerous mechanistic similarities between romantic love, hypomania, depression, and anxiety (see [1] for review). Bajoghli and colleagues [35] found that different components of anxiety (i.e., trait anxiety and state anxiety) were associated with variations in sleep onset latency, WASO, and sleep quality in adolescents.

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