PART 3

Read the text below and answer questions 27–40.

THE BIOLOGY OF SLEEP AND CIRCADIAN RHYTHMS

Sleep is often misunderstood as a simple suspension of waking life, yet in neurobiology it is treated as an actively regulated state produced by interacting control systems. A widely used framework in chronobiology is the "two-process model," in which Process S represents homeostatic pressure for sleep and Process C represents circadian timing. Process S rises with time awake and declines with sleep, reflecting the accumulating biological cost of wakefulness. Process C, by contrast, is an oscillatory signal that modulates arousal across the day and night, promoting wakefulness at some times and sleep at others, even when homeostatic pressure is high. The familiar "second wind" in the evening illustrates this interaction: after many waking hours, a person may still experience a temporary increase in alertness because circadian signals can counteract, for a period, the homeostatic drive to sleep. In practice, sleep behaviour emerges from the continuous negotiation between these two processes, shaped by light exposure, social scheduling, and individual variability.

At the centre of circadian timing is the suprachiasmatic nucleus (SCN), a small cluster of neurons in the hypothalamus that functions as the body's master pacemaker. The SCN coordinates rhythms in temperature, hormone secretion, metabolism, and autonomic physiology so that internal processes align with the external day–night cycle. Its timing is not fixed; rather, it is adjusted by environmental cues known as Zeitgebers, with light being the most potent. Specialized retinal cells send signals to the SCN that can reset its phase, effectively "teaching" the clock what time it is. This explains why bright light in the morning can advance the circadian phase, making sleepiness arrive earlier the following night, whereas exposure to bright, blue-rich light late in the evening can delay the phase and shift sleep later. Importantly, the circadian system does not merely dictate when people feel sleepy; it orchestrates a broad timetable for physiological readiness, meaning that circadian disruption can influence mood, performance, and metabolic regulation even when total sleep time appears adequate.

Homeostatic regulation of sleep is often explained through changes in neuromodulators that track wakefulness. Among the best-known is adenosine, which accumulates in the brain during prolonged waking and is associated with the subjective sensation of "sleep pressure." As wakefulness continues, adenosine signalling increases, biasing neural networks toward reduced arousal; during sleep, these levels decline, contributing to the restoration of alertness. This chemistry also clarifies why caffeine is effective as a

stimulant. Rather than “removing” the underlying need for sleep, caffeine acts primarily as a receptor antagonist, blocking adenosine receptors and dampening the perception of pressure. As a result, a person may feel more awake while the biological debt continues to accrue. When the caffeine effect fades, rebound tiredness can occur because the homeostatic drive has been postponed rather than resolved. In this way, everyday choices—especially timing and quantity of stimulants—can change how Process S is experienced without changing the fundamental physiology that generates it.

Sleep itself is not uniform but organised into a characteristic architecture. Across a typical night, the brain cycles through non-rapid eye movement (NREM) and rapid eye movement (REM) sleep in repeating sequences. Within NREM sleep, deep slow-wave sleep is distinguished by high-amplitude, low-frequency delta waves, and it is often associated with physical restoration, immune regulation, and aspects of memory consolidation. Lighter NREM stages show features such as sleep spindles—brief bursts of rhythmic activity—which are frequently discussed in relation to learning and synaptic plasticity. REM sleep, characterised by vivid dreaming, heightened cortical activity, and muscle atonia, appears to play a role in emotional processing and the integration of new information into existing networks. The distribution of stages is not random: slow-wave sleep tends to dominate earlier in the night, while REM episodes become longer and more frequent toward morning. This patterned cycling suggests that the benefits of sleep are not delivered by a single mechanism, but by multiple neurophysiological states that contribute in different, partly complementary ways.

Research on sleep function increasingly extends beyond simple “restoration.” In experimental settings, performance on certain tasks improves when sleep follows training, implying that memory traces can be stabilised or reorganised overnight. A related line of work examines how sleep may support neural housekeeping. During deep sleep, changes in brain fluid dynamics appear to facilitate the clearance of metabolic by-products, an idea often discussed under the umbrella of the glymphatic system. Although the precise human mechanisms remain under refinement, the broader implication is that sleep may help maintain long-term neural health by regulating the accumulation of waste products that are generated by active metabolism. At the same time, scientists caution against treating this as a settled explanation for complex neurological disease; sleep is likely one factor among many, and findings can vary depending on measurement techniques and the populations studied. Nevertheless, the hypothesis has strengthened the view that deep sleep is not merely a quieter form of wakefulness, but a biologically specialised state with distinct consequences.

When circadian timing and social demands diverge, circadian misalignment can occur. Shift work, transmeridian travel, and early institutional start times may force individuals to sleep at biologically unfavourable hours, thereby creating chronic conflict between internal rhythms and external schedules. The consequences extend beyond transient sleepiness. Misalignment can perturb glucose regulation, alter appetite-related hormones, and influence cardiovascular markers, helping to explain why disrupted sleep schedules are studied in relation to metabolic and mental health risk. However, the strength and direction of these associations can vary with lifestyle, socioeconomic context, and the degree of disruption, so researchers often emphasise probabilistic risk rather than deterministic outcomes. Cognitive effects may also be subtle yet cumulative: attention, reaction time, and emotional regulation can degrade when sleep timing is repeatedly shifted, even if individuals report “getting used” to the schedule. In this sense, circadian disruption is best understood not as a single problem, but as a systems-level strain distributed across multiple physiological domains.

Inter-individual variability further complicates any universal account of “normal” sleep. Genetic influences contribute to chronotype, the tendency to prefer earlier or later sleep timing, so some people are naturally “morning larks” while others are “night owls.” Developmental stage matters as well. Adolescents commonly exhibit a biological phase delay, meaning that circadian signals shift later and sleep pressure may build differently, making it harder to fall asleep early even with strong external demands. In older adulthood, by contrast, sleep may become lighter and more fragmented, with more frequent awakenings and reduced slow-wave sleep. These age-related changes mean that the same schedule can be physiologically suitable for one group yet misaligned for another. Adding to this complexity is the rise of consumer measurement. Wearable devices infer sleep from movement and heart-rate patterns and may estimate REM and deep sleep, which can help users notice trends. Yet these tools are less accurate than clinical sleep studies, particularly for distinguishing stages, and they may inadvertently promote “orthosomnia,” where anxiety about sleep metrics worsens insomnia. Clinicians therefore tend to prioritise daytime function and symptoms, not just numerical readouts.

Interventions typically aim to adjust either circadian timing, homeostatic pressure, or the behaviours that maintain sleep problems. Light management—seeking bright morning light, maintaining consistent wake times, and limiting late-night blue-rich exposure—can help shift the circadian phase. For insomnia, a widely recommended first-line approach is Cognitive Behavioural Therapy for Insomnia (CBT-I), which targets behaviours and beliefs that perpetuate poor sleep through structured techniques such as stimulus control and sleep restriction. Pharmacological aids can play a role in specific contexts, but their effects are not interchangeable. Melatonin, for example, may be useful for certain

circadian rhythm disorders when timed correctly, yet it is not a universal sedative that reliably resolves all sleep difficulties. Overall, the emerging picture is that sleep is governed by a layered regulatory system: a clock that coordinates physiology, a homeostatic drive that tracks wakefulness, and a set of cognitive and environmental factors that can either support or undermine the alignment between biology and daily life.

Questions 27–31

Select the correct answer.

27 What does the passage identify as the main function of the SCN?

A It measures the homeostatic pressure accumulated during wakefulness.
B It coordinates physiological rhythms with the external day–night cycle.
C It delays the circadian phase whenever retinal cells detect bright light.
D It determines the balance of NREM and REM sleep independently of environmental cues.

28 How does caffeine promote alertness according to the passage?

A It lowers the amount of adenosine accumulated during wakefulness.
B It prevents the biological need for sleep from continuing to increase.
C It blocks adenosine receptors, reducing the perceived pressure to sleep.
D It advances the circadian phase by stimulating specialised retinal cells.

29 How are sleep stages distributed across a typical night?

A Slow-wave sleep is concentrated earlier, whereas REM episodes increase toward morning.
B NREM and REM repeat in equal proportions throughout the night.
C Both slow-wave sleep and REM episodes become progressively longer toward morning.
D Sleep spindles replace delta waves as deep sleep becomes dominant later in the night.

30 Why can wearable sleep devices sometimes cause problems?

A Their use prevents people from recognising broad changes in their sleeping patterns.
B Their estimates cause clinicians to disregard daytime symptoms and functioning.
C Their measurements are designed to replace clinical studies when sleep disorders are suspected.
D Their numerical feedback can create anxiety that aggravates insomnia.

31 Which approach is described as a first-line treatment for insomnia?

A Correctly timed melatonin for every form of insomnia
B Cognitive Behavioural Therapy for Insomnia (CBT-I)

C Sleep restriction without addressing beliefs or other sleep-related behaviours
D Light management regardless of the underlying cause of the sleep problem

Questions 32–36

Choose **TRUE** if the statement agrees with the information given in the text, choose **FALSE** if the statement contradicts the information, or choose **NOT GIVEN** if there is no information on this.

32 The passage suggests that sleep results from the interaction of circadian timing and sleep homeostasis.

33 The passage states that light is the strongest cue for resetting the circadian clock.

34 The passage provides exact numerical thresholds for the amount of caffeine that causes rebound tiredness.

35 Adolescents commonly experience a biological shift toward later sleep timing.

36 Melatonin is described as a universal sedative that reliably resolves all sleep difficulties.

Questions 37–40

Complete the summary below. Choose **NO MORE THAN TWO WORDS** from the text for each answer.

Key features of sleep regulation

The circadian clock is coordinated by the **37** ..... and is strongly influenced by light. Sleep pressure increases during wakefulness partly because **38** ..... accumulates in the brain. Sleep may also support brain waste clearance during **39** ..... For insomnia, a recommended first-line approach is **40** .....

Answer Key

27. B
28. C
29. A
30. D
31. B
32. TRUE
33. TRUE

34. NOT GIVEN

35. TRUE

36. FALSE

37. suprachiasmatic nucleus

38. adenosine

39. deep sleep

40. CBT-I