

A GENERAL THEORY OF BEHAVIOUR

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SLEEPING, INSOMNIA, WORK-LIFE BALANCE

We must not look upon science as a 'body of knowledge,'
but rather as a system of hypotheses.

Karl Raimund Popper

SLEEPING

One of the greatest mysteries is why humans need to spend so much time sleeping. When we sleep well we are refreshed, feel good and perform well. When we sleep badly with unwanted awakenings, we are 'out of sorts', grumpy or miserable and our ability to perform is diminished. Why these phenomena happen in the way that they do is almost a complete puzzle. One theory suggests that sleep enables the brain to reset neuronal plasticity to a regulated set range.¹ Many theories have been proposed, but none is generally accepted.² One thing that we do know is that a *balance* between waking and sleeping is a prerequisite for a healthy and happy life. Nature

¹Griffith, L.C. and Rosbash, M. (2008). Sleep: Hitting the reset button. *Nature Neuroscience*, 11(2), 123; Vyazovskiy, V.V., Cirelli, C., Pfister-Genskow, M., Faraguna, U. and Tononi, G. (2008). Molecular and electrophysiological evidence for net synaptic potentiation in wake and depression in sleep. *Nature Neuroscience*, 11(2), 200.

²Krueger, J.M., Frank, M.G., Wisor, J.P. and Roy, S. (2016). Sleep function: Toward elucidating an enigma. *Sleep Medicine Reviews*, 28, 46–54.



has done her best to help us because sleep happens to be pleasurable, while fatigue remains displeasurable for as long as it lasts. Sleep onset occurs when the displeasure of excessive waking becomes maximal and the level of mesolimbic dopamine is at a minimum.³

Sleep is intimately related to time – the ‘right’ amount at the ‘right’ time is necessary to protect mental functioning from becoming degraded. Temporal regulation of sleep is governed by an interplay of homeostatic and circadian processes. A two-process model was proposed by the Hungarian-Swiss pharmacologist Alexander A. Borbély in 1982.⁴ The model holds that sleeping and waking are governed by the following:

1. A homeostatic process that builds pressure for sleep in wakefulness as a function of time spent awake and dissipates during sleep. Sleep homeostasis is an innate regulatory process that maintains the amount of sleep at a set range. Loss of sleep results in a compensatory increase in sleep whereas excess sleep results in compensatory reduction in sleep. Sleep homeostasis produces sleep propensity or the urge to fall asleep, also termed ‘sleep pressure’ or ‘sleep debt’. Contributing to sleep drive is the build-up of sleep-modulating substances (i.e., sleep factors) during the waking period in multiple brain regions, e.g. adenosine, nitric oxide, tumor necrosis factor alpha, interleukin 1.⁵
2. A circadian process that modulates sleep pressure entrained to the time of day (the CLOCK system).

The two-process model successfully predicts a host of neurobehavioural functions related to sleep and waking. For example, waking neurobehavioural functions are predicted by the arithmetic difference between the homeostatic pressure for sleep and the circadian pressure for wakefulness (see Figure 9.1).

³Rial, R.V., Canellas, F., Gamundí, A., Akaârîr, M. and Nicolau, M.C. (2018). Pleasure: The missing link in the regulation of sleep. *Neuroscience & Biobehavioural Reviews*, 88, 141–154.

⁴Borbély, A.A. (1982). A two process model of sleep regulation. *Human Neurobiology*, 1(3), 195–204.

⁵Thakkar, M.M., Sharma, R. and Sahota, P. (2015). Alcohol disrupts sleep homeostasis. *Alcohol*, 49, 299–310.

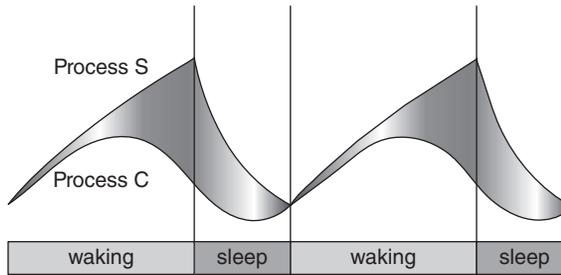


Figure 9.1 The two-process model of Alexander A. Borbély. Sleep and wakefulness occur at the peak and trough of two additive processes, S (Homeostasis) and C (circadian process, or 'CLOCK').

In addition to the two processes identified by Borbély, a third process of wilful behaviour is also a significant controlling element in determining the context and timing of sleep and waking. One's ability to choose when to sleep, whether or not to use sleeping aids or medicines, the time and method of our awakening, and if and when we might try to take a nap or two are all part of the balancing act we produce in our lives. The BCS sets rest-related and sleep-related goals and therefore plays a crucial role in regulating the pattern, quality and quantity of sleep. Thus, the *General Theory* postulates a three-process model of sleeping and waking (see Figure 9.2).

Principle XVII (Sleeping and Waking): Sleeping and waking are controlled by Type I homeostatic sleep pressure and the circadian CLOCK in coordination with the BCS and Type II homeostasis. Any of these processes can override any other, but increased sleep pressure, in combination with the CLOCK, ultimately will always produce sleeping.

The BCS with Type II homeostasis controls when and where we sleep, what we do before we go to sleep, and any habits that provide the context and conditions for sleep. If necessary, demanding goal-setting by the BCS can act to limit the duration of sleep by overriding Type I homeostatic pressure and the circadian CLOCK, often to the detriment of cognitive functioning, subjective well-being and physical health. For example, in writing a report with a deadline, a stressed manager might be forced to 'skip' several hours sleep. Ultimately, in combination with the CLOCK, Type I homeostatic sleep pressure can become overwhelming and sleeping begins.

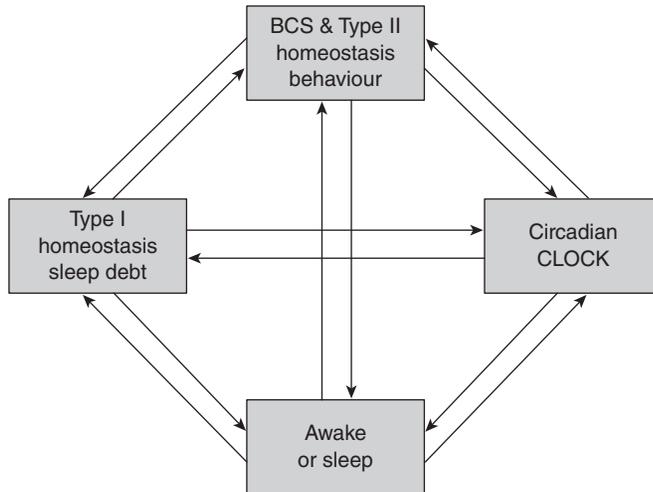


Figure 9.2 The three-process model of waking and sleeping. The 'Awake or sleep' process acts as a flip-flop switch. The two halves of the switch strongly inhibit each other, creating a feedback loop that is stable in only two states so that intermediate states of sleep and wakefulness are very brief.⁶

From the perspective of the *General Theory*, a host of behaviours can significantly influence sleeping and waking. Here, we briefly review the role of habits, alcohol and devices. It is established that sleep is necessary for mental rejuvenation, providing the ultimate reset function. Yet epidemiological studies indicate that human sleep quality is decreasing worldwide and that sleep hygiene is quite low on the list of pressing public health issues. One example is the prevalence of 'social jet lag' caused by regular awakenings at earlier than optimum times. As many as 80% of the general population use alarm clocks on workdays, cutting their needed sleep in the mornings in order to be at work on time.⁷

⁶It has been suggested that neurones containing orexin or melanin concentrating hormone reciprocally regulate wake and sleep. See: Brown, R.E., Basheer, R., McKenna, J.T., Strecker, R.E. and McCarley, R.W. (2012). Control of sleep and wakefulness. *Physiological Reviews*, 92(3), 1087–1187; Konadhode, R.R., Pelluru, D. and Shiromani, P.J. (2015). Neurones containing orexin or melanin concentrating hormone reciprocally regulate wake and sleep. *Frontiers in Systems Neuroscience*, 8, 244.

⁷Roenneberg, T., Kantermann, T., Juda, M., Vetter, C. and Allebrandt, K.V. (2013). Light and the human circadian clock. *Handbook of Experimental Pharmacology*, 217, 311–331.

Lost sleep on workdays is compensated on free days and weekends by longer sleeps. Sleeping during different sleep windows on workdays and on free days correlates positively with increased prevalence of smoking, obesity, depression and cardiovascular problems.⁸

Subjective well-being (SWB) is significantly reduced among people with social jet lag. However, SWB resets to the set range once the jet lag has abated. [AP 063]

The homeostatic Process S produces increased sleep pressure during waking (a saturating exponential function) and dissipation during sleep (a decreasing exponential function). Individual variability in homeostatic responses to sleep and sleep deprivation suggest a moderate form of trait variability.⁹ Social jet lag brings tiredness, irritability and impaired concentration. Food habits tend to fall into disorder as do any restrained addictions. Individuals normally display stability in their preferences for sleeping and waking up, sleep duration, sleep depth and sleep structure.

Alcohol and marihuana are used as sleep aids by a significant number of individuals.¹⁰ Through its effects on adenosine, chronic alcohol use can produce severe disruption to sleep.¹¹ This disruption is indicated by changes in the proportions of NREM and REM phases of sleep. Increased NREM sleep following acute alcohol consumption may be produced by the inhibition of wake-promoting neurones through activation of GABA_A receptors. At the same time, decreased REM sleep may be due to the activation of GABA_B receptors and/or inhibition of kainate receptors on brainstem cholinergic cells. In rats, 6 weeks of chronic exposure to alcohol induces desynchrony of circadian rhythms

⁸Roenneberg, T. (2013). Chronobiology: The human sleep project. *Nature*, 498(7455), 427.

⁹Rusterholz, T., Tarokh, L., Van Dongen, H. and Achermann, P. (2017). Interindividual differences in the dynamics of the homeostatic process are trait-like and distinct for sleep versus wakefulness. *Journal of Sleep Research*, 26(2), 171–178.

¹⁰Goodhines, P.A., Gellis, L.A. and Park, A. (2018). Alcohol and marijuana use for sleep aid in college students: A daily diary investigation. *Sleep*, 41, A76; Brower, K.J., Aldrich, M.S., Robinson, E.A, Zucker, R.A. and Greden, J.F. (2001). Insomnia, self-medication, and relapse to alcoholism. *American Journal of Psychiatry*, 158, 399–404.

¹¹Martindale, S.L., Hurley, R.A. and Taber, K.H. (2017). Chronic alcohol use and sleep homeostasis: Risk factors and neuroimaging of recovery. *The Journal of Neuropsychiatry and Clinical Neurosciences*, 29(1), A6-5.

(e.g., core body temperature, activity level, hormonal secretion) and a phase shift of peripheral circadian clocks.¹²

Regular ingestion of a moderate amount of alcohol before bedtime produces decreased sleep latency, increased NREM sleep, increased total sleep time, and reduced or fragmented REM sleep that leads to decreased sleep efficiency followed by rebound increases of REM sleep on subsequent nights. There is a positive association between daily alcohol use and sleep duration and a negative association with sleep quality but with large individual differences. During acute withdrawal from regular alcohol use, sleep latency increases, total sleep decreases, NREM sleep decreases, REM episodes increase in frequency, and NREM-REM cycles are shortened, but with no increase in total REM duration.¹³

The chronic use of alcohol disorganizes the homeostatic sleep system, sleep stage transitions and circadian rhythms. [AP 064]

Using light-emitting devices such as e-readers, smart phones, tablets and TVs in the few hours immediately prior to sleeping can perturb the natural activity of Processes S and C, causing significant decreases in both sleep quality and time. Sex, age, chronotype and occupation can all influence sleep-wake processes at multiple levels. In response to industrial work pressures, many people have sleep-wake cycles that are out of synch with nature's 24-hour LD cycle. Using a TV, computer, gaming console, tablet, mobile phone or audio player in bed before going to sleep is associated with insomnia, daytime sleepiness, morningness or chronotype.¹⁴ Reading light-emitting eBooks before bedtime produces unintended biological and behavioural impacts. Compared to reading a light-reflecting printed book, reading an eBook leads to decreased subjective sleepiness, decreased EEG delta/theta activity, and suppression of the late evening rise of pineal melatonin secretion during the time that the book was

¹²Guo, R., Simasko, S.M. and Jansen, H.T. (2016). Chronic alcohol consumption in rats leads to desynchrony in diurnal rhythms and molecular clocks. *Alcoholism: Clinical and Experimental Research*, 40, 291–300.

¹³Lydon, D.M., Ram, N., Conroy, D.E., Pincus, A.L., Geier, C.F. and Maggs, J.L. (2016). The within-person association between alcohol use and sleep duration and quality in situ: An experience sampling study. *Addictive Behaviors*, 61, 68–73.

¹⁴Fossum, I.N., Nordnes, L.T., Storemark, S.S., Bjorvatn, B. and Pallesen, S. (2014). The association between use of electronic media in bed before going to sleep and insomnia symptoms, daytime sleepiness, morningness, and chronotype. *Behavioural Sleep Medicine*, 12(5), 343–357.

being read.¹⁵ Also, sleep latency is lengthened and sleep propensity, REM sleep propensity and morning alertness are impaired. From the three-process model it can be assumed that the arousal, interest and affect generated by the activity with the device is overriding Processes S and C, causing a build-up of sleep debt.

The use of light-emitting devices prior to bedtime is associated with decrements in sleep time, sleep quality and daytime waking performance. [AP 065]

We turn now to the multifaceted issue of insomnia.

INSOMNIA

Insomnia is a prevalent sleep disorder with approximately 30% of people reporting insomnia symptoms and 20% meeting diagnostic criteria.¹⁶ For people who suffer from it, the inability to sleep is persistent. Morin et al.'s 3-year longitudinal study in Quebec with 388 adults with insomnia found that 46% reported insomnia persisting over the entire 3-year study.¹⁷ Difficulty in sleeping is related to hyperarousal, anxiety and depression, all having genetic components. Insomnia increases with age. Whole body VO_2 is found to be elevated in people with insomnia compared to controls, and people with chronic insomnia have relatively high metabolic rates.¹⁸ People with insomnia typically have a high set range of arousal that persists over the complete 24-hour cycle, with increased wake drive both at night and during the day.¹⁹ The high set range is determined genetically, epigenetically and by early life experience. People with insomnia have prolonged sleep latencies about one standard deviation above the mean of the population during the day, despite having disrupted

¹⁵Chang, A.M., Aeschbach, D., Duffy, J.F. and Czeisler, C.A. (2015). Evening use of light-emitting eReaders negatively affects sleep, circadian timing, and next-morning alertness. *Proceedings of the National Academy of Sciences*, 112(4), 1232–1237.

¹⁶Roth, T., Roehrs, T. and Pies, R. (2007) Insomnia: Pathophysiology and implications for treatment. *Sleep Medicine Reviews*, 11, 71–79.

¹⁷Morin, C.M., Bélanger, L., LeBlanc, M., Ivers, H., Savard, J., Espie, C.A., Merette, C., Baillargeon, L. and Grégoire, J.P. (2009). The natural history of insomnia: A population-based 3-year longitudinal study. *Archives of Internal Medicine*, 169(5), 447–453.

¹⁸Bonnet, M.H. and Arand, D.L. (1995). 24-hour metabolic rate in insomniacs and matched normal sleepers. *Sleep*, 18(7), 581–588.

¹⁹Stepanski, E., Zorick, F., Roehrs, T. and Roth, T. (2000). Effects of sleep deprivation on daytime sleepiness in primary insomnia. *Sleep*, 23(2), 215–219.

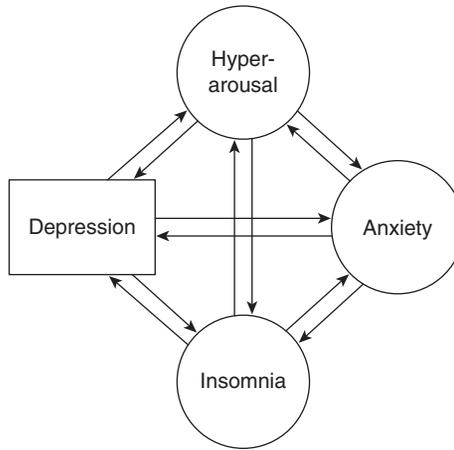


Figure 9.3 Model of insomnia. The model is a simplified version of a more complex model with multiple causal determinants of insomnia.

and shortened nocturnal sleep the previous night.²⁰ ACTH and cortisol secretions are significantly higher in insomniacs, compared with normal controls.²¹

The three-process model of waking and sleeping (see Figure 9.2) holds that sleeping and waking are mediated by a balance between homeostatic, circadian and behavioural determinants. A few main determinants of insomnia are specified in a simple model shown in Figure 9.3.

Physiologically, people with insomnia have a high set range of arousal, a condition of 'hyperarousal'.²² **[AP 066]**

²⁰Roehrs, T., Gumenyuk, V., Drake, C. and Roth, T. (2014). Physiological correlates of insomnia. In V. Kumari, P. Bob and N.N. Boutros (eds), *Electrophysiology and Psychophysiology in Psychiatry and Psychopharmacology*. Champaign, OH: Springer. pp. 277–290

²¹Vgontzas, A.N., Bixler, E.O., Lin, H.M., Prolo, P., Mastorakos, G., Vela-Bueno, A., Kales, A. and Chrousos, G.P. (2001). Chronic insomnia is associated with nyctohemeral activation of the hypothalamic-pituitary-adrenal axis: Clinical implications. *The Journal of Clinical Endocrinology & Metabolism*, 86(8), 3787–3794.

²²Altena, E., Chen, I.Y., Daviaux, Y., Ivers, H., Philip, P. and Morin, C.M. (2017). How hyperarousal and sleep reactivity are represented in different adult age groups: Results from a large cohort study on insomnia. *Brain Sciences*, 7(4), 41.

Psychologically, people with insomnia and hyperarousal are expected to react more strongly to stressful events and have higher scores on depression and anxiety. [AP 067]

Psychological interventions to improve the sleep function and reduce hyperarousal of people with insomnia are expected to be more effective and less harmful than any pharmaceutical approach.²³ [AP 068]

Since the time of Hippocrates it has been suggested that sleep helps recuperation from illnesses, infectious diseases and surgery. Sleep loss adversely affects the immune system. People with primary insomnia show nocturnal elevations of circulating levels of norepinephrine and a reduction of NK activity.²⁴ Adults who report having 5 or fewer hours of sleep a night are 2.5 times more likely to have diabetes, compared to people who sleep 7–8 hours per night. People who sleep 6 hours a night are also significantly more likely to have obesity than their peers who sleep longer.²⁵

We now turn to a related issue about ‘balance’ – ‘work–life balance’.

WORK-LIFE BALANCE

‘Stress’ is our response to pressures from situations or life events. In a UK survey of 4169 adults in 2018, three-quarters reported having at some point over the past year felt so stressed that they were overwhelmed or unable to cope.²⁶ Many possible reasons for this high figure include the fact that, to an increasing extent, people are taking work calls and checking work emails outside of working hours, and taking large chunks of work home with them to do in the evenings and weekends. There is evidence that it is becoming increasingly difficult for working people to maintain a work–life balance that is beneficial to themselves and their families. ‘Work–life balance’ is a form of homeostasis, but

²³Qaseem, A., Kansagara, D., Forciea, M.A., Cooke, M. and Denberg, T.D. (2016). Management of chronic insomnia disorder in adults: A clinical practice guideline from the American College of Physicians. *Annals of Internal Medicine*, 165(2), 125–133.

²⁴Irwin, M., Clark, C., Kennedy, B., Gillin, J.C. and Ziegler, M. (2003). Nocturnal catecholamines and immune function in insomniacs, depressed patients, and control subjects. *Brain, Behaviour, and Immunity*, 17(5), 365–372.

²⁵Altevogt, B.M. and Colten, H.R. (eds) (2006). *Sleep Disorders and Sleep Deprivation: An Unmet Public Health Problem*. Washington, DC: National Academies Press.

²⁶Mental Health Foundation (2018). *Stress: Are We Coping?* London: Mental Health Foundation.

the term is a little simplistic. It is no easy matter to compare the 'apples' of work with the 'barrel of fruit' that is all of the non-work-related aspects of life. We must break the barrel down into categories, e.g. family, friends, interests, habits, addictions, to reveal significant complexity. However, the *General Theory* holds that all of life consists of a balancing act of homeostasis: then, like a skilled accountant working on a set of accounts, the theory is put to work and, voilà: we have a solution (see Figure 9.4).

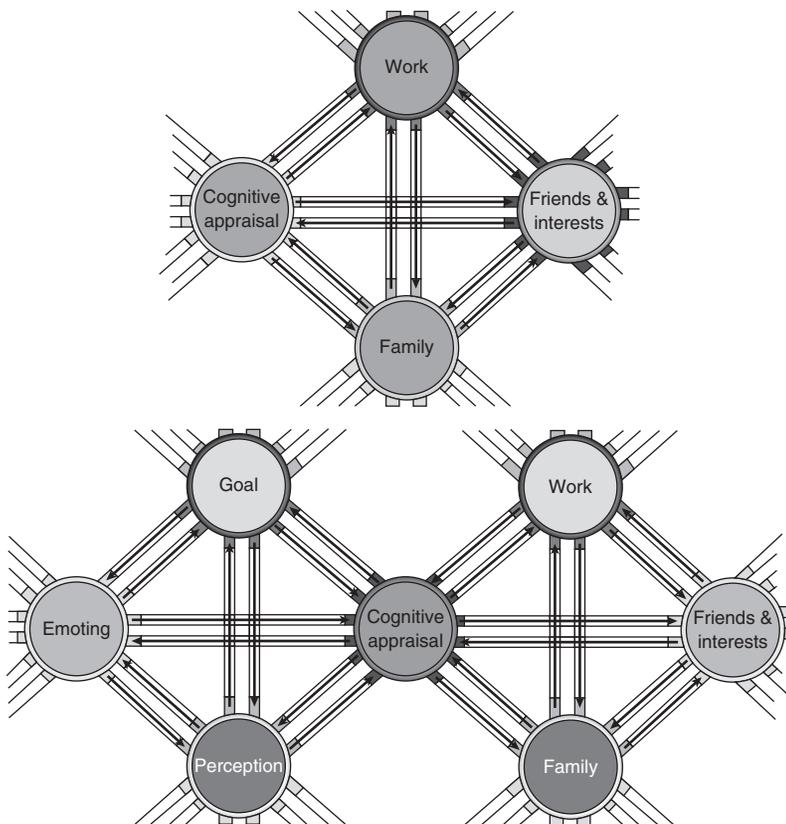


Figure 9.4 Upper panel: A model of work-life balance with four domains: cognitive appraisal, friends and interests, family, and work. Lower panel: The model of work-life balance (right) interfaced with the model of emoting (left) showing cognitive appraisal and six processes within a lattice structure.

Balancing is carried out across the board, both between and within domains. Within the family, multiple goals, interests and tasks all need to be attended to, all of which consume energy from a limited resource. At work there are multiple goals, personnel, organizational levels, tasks and procedures to be 'juggled' within a limited set of resources. As noted, to an increasing extent the boundary between work and life is blurring and work can intrude into non-work domains. Among friends and interests, yet another set of balances must be achieved to optimize one's resources to maximize the benefits to one's affiliation needs. The balancing and juggling continues unabated across all areas of life, at every twist and turn, and there can be no end to it.

The Law of Conservation of Energy holds that the total energy of an isolated system remains constant. One can imagine the existence of a parallel law of conservation of 'mental energy' in which there is a fixed quantity of 'mental energy' for the body and brain to carry out all necessary functions within any one 24-hour cycle. One measure of the energy expenditure by the entire system is the rate of metabolism of the brain.²⁷ The balances that are struck between different areas of life are a necessary consequence of the psychological version of the Law of Conservation of Energy:

Principle XVIII (Law of Conservation of Energy): In any 24-hour circadian cycle there is a fixed quantity of energy to expend across life goals and domains as behaviour, affect and cognition.

In any 24-hour cycle, there is a continuous homeostatic balancing of energy between different goal domains, including work, family, friends and interests. **[AP 069]**

In illness, the quantity of energy that is available for the attainment of goal domains is depleted, resulting in abnormally high levels of fatigue. **[AP 070]**

It is erroneous to assume that the depletion of energy that occurs in an illness is anything but real. **[AP 071]**

²⁷Benton, D., Parker, P.Y. and Donohoe, R.T. (1996). The supply of glucose to the brain and cognitive functioning. *Journal of Biosocial Science*, 28, 463–479; Fairclough, S.H. and Houston, K. (2004). A metabolic measure of mental effort. *Biological Psychology*, 66, 177–190; Gailliot, M.T., Baumeister, R.F., DeWall, C.N., Maner, J.K., Plant, E.A., Tice, D.M. and Brewer, L.E. (2007). Self-control relies on glucose as a limited energy source: Will-power is more than a metaphor. *Journal of Personality and Social Psychology*, 92(2), 325.

Baumeister proposes a resource model of self-control in which restraint uses a significant amount of energy from the total amount that is available, so that engaging self-control eventually creates a state of 'ego depletion'.²⁸ Gailliot and Baumeister argue that self-control failures are more likely when glucose is low or cannot be mobilized effectively to the brain (i.e., when insulin is low or insensitive). When glucose is restored to a sufficiently high level, self-control is typically improved.²⁹ Energy usage can be assumed to vary across tasks and functions.

Acts of self-control deplete particularly large amounts of energy, which can be measured as glucose metabolism. [AP 072]

In balancing energy across life goals, transfers of energy occur between one goal domain and another. In our model of work–life balance (see Figure 9.3), there is a continuous process of time and energy distribution between and within goal domains from the finite amounts of time and energy that are available. Yet if demands continually grow, stress will be an inevitable outcome.

CONCLUSIONS

1. Sleeping and waking are controlled by sleep pressure from Type I homeostasis, Type II homeostatic behaviour and the circadian CLOCK.
2. Many people with insomnia have a high set range of arousal or 'hyperarousal'.
3. Homeostatic balancing of time and energy occurs both between and within different goal domains, including work, family, friends and interests.

²⁸Muraven, M. and Baumeister, R.F. (2000) Self-regulation and depletion of limited resources: Does self-control resemble a muscle? *Psychological Bulletin*, 126, 247–259.

²⁹Gailliot, M.T. and Baumeister, R.F. (2018). The physiology of willpower: Linking blood glucose to self-control. In R.F. Baumeister (ed.), *Self-Regulation and Self-Control*. London: Routledge. pp. 137–180.