BI-09 Bodily differences that shape daily co-living

The investigation of how biological sex influences human physiology, behaviour, and daily co-living requires a strict demarcation between statistical averages and individual realities. Research into dimorphic traits frequently suffers from a conflation of biological sex (chromosomal and physiological attributes) and sociocultural gender (environmental conditioning and role expectations). To navigate this complexity, this report evaluates bodily differences through three distinct layers: (1) physiology, encompassing measurable genetic, neurobiological, and endocrinological mechanisms; (2) behaviour, reflecting statistical distributions of traits that heavily overlap between sexes; and (3) culture, representing the variable environmental and social frameworks that suppress or amplify innate differences.
Understanding these differences relies heavily on the concept of effect size, typically measured as Cohen's *d* (which expresses the difference between two group means in standard deviation units)[1]. A fundamental principle in interpreting this data is that average differences between populations do not strictly determine individual phenotypes. Two distributions may exhibit a statistically significant mean difference while maintaining substantial overlap. For instance, a Cohen's *d* of 0.2 is considered small, 0.5 medium, and 0.8 large; even with a medium effect size, a randomly selected female may frequently exhibit a more "male-typical" trait than a randomly selected male, and vice versa[1].
The following analysis exhaustively surveys the peer-reviewed primary literature, systematic reviews, and meta-analyses concerning sex differences that directly impact everyday shared life.

Circadian Timing and Chronotype

The timing of sleep and wakefulness is foundational to daily co-living, dictating shared routines, mood regulation, and energy expenditure. The literature reveals robust sex differences in the intrinsic circadian pacemaker, influencing when individuals naturally feel tired or alert.

Physiology

The human circadian timing system is driven by the suprachiasmatic nucleus (SCN) in the hypothalamus, which regulates core body temperature (CBT) and melatonin secretion. In a highly controlled, month-long inpatient protocol designed to eliminate environmental masking, Duffy et al. (2011, *Proceedings of the National Academy of Sciences* [*PNAS*]) demonstrated that the near-24-hour intrinsic circadian period is significantly shorter in women. Testing a sample of 157 individuals (52 women, 105 men), the researchers found the female intrinsic period was 24.09 ± 0.2 hours, compared to 24.19 ± 0.2 hours in men ($p < 0.01$)[4]. A significantly greater proportion of women exhibited intrinsic circadian periods shorter than 24.0 hours (35% versus 14%, $p < 0.01$)[4].
Because of this shorter intrinsic period, female circadian rhythms of melatonin and CBT are phase-advanced relative to men. Even when maintaining identical sleep-wake schedules, women experience dim light melatonin onset (DLMO) at an earlier biological time. Secondary

analyses of adolescent cohorts (N = 32) replicate this, showing that female adolescents experience DLMO an average of 96 minutes earlier than males, resulting in a biological night that is 60 minutes longer ($p = 0.0368$)[6].

Hormonal Roots: These circadian differences are heavily influenced by the hypothalamic-pituitary-gonadal (HPG) axis. Animal models demonstrate a sexually dimorphic population of kisspeptin neurons in the anteroventral periventricular nucleus (AVPV), which is 25 times larger in females, shaped by the organizational effects of early developmental exposure to testosterone[8].

Behaviour

Behaviourally, the shorter circadian period in females translates to an earlier chronotype. Women naturally tend to go to bed earlier, wake up earlier, and exhibit a greater preference for morning activities[4]. However, because they often share a clock time for sleep with male partners, women effectively initiate sleep at a later biological circadian phase[9].

Culture

Culturally, social jetlag and rigid societal work schedules frequently force individuals to adopt wake times that misalign with their biological clocks. Because men are, on average, more evening-oriented (a finding that diminishes with age as male testosterone drops and chronotypes advance), shared co-living often involves compromise[12]. This misalignment extracts a toll: a meta-analysis indicates that women are more sensitive to circadian disruption, experiencing greater impairments in nighttime cognitive performance and mood during extended wakefulness[13].

Sleep Architecture

Beyond the timing of sleep, the structure and quality of sleep phases—rapid eye movement (REM) and non-REM (NREM)—differ fundamentally between the sexes.

Physiology

Polysomnography (PSG) studies consistently show structural differences in sleep architecture. A systematic review and meta-analysis of 169 studies by Boulos et al. (2019, *Sleep*) and subsequent massive datasets (e.g., Luca et al., N=1147) demonstrated that, objectively, women exhibit superior sleep quality parameters compared to men. Women possess longer total sleep time, higher sleep efficiency, and spend significantly more time in slow-wave sleep (SWS, or N3 sleep), which is critical for physiological restoration[15]. Conversely, men exhibit a higher percentage of N1 and N2 (lighter sleep), a reduced REM sleep latency, a higher arousal index, and a significantly higher Apnea-Hypopnea Index (AHI)[15]. Age-related declines in SWS amplitude affect men severely in their thirties, whereas female SWS remains relatively stable until menopause[17]. A meta-analysis of EEG data (N=3374) confirmed that females exhibit higher spindle density and greater delta wave power[18].

Hormonal Roots: Progesterone, which peaks in the luteal phase of the menstrual cycle, possesses mild hypnotic properties but also elevates core body temperature, which can fragment sleep and alter REM percentages[13]. Estrogen promotes wakefulness by acting directly on arousal centers in the brain, influencing the sleep-wake flip-flop switch[14].

Behaviour

Despite PSG data indicating objectively superior sleep architecture, behavioural and subjective reporting presents a stark paradox. Women report significantly poorer subjective sleep quality, greater sleep fragmentation, and higher rates of fatigue[15]. A meta-analysis by Zhang and Wing (2006, *Sleep*) reported a robust risk ratio of 1.41 for insomnia in females compared to males[21]. Research into sensory thresholds during sleep suggests women perceive short awakenings more accurately than men, who tend to underestimate their time awake. When men with objectively short, unremembered awakenings are removed from statistical models, the gender difference in self-reported sleep quality diminishes[15].

Culture

Culturally, females often bear disproportionate caregiving responsibilities, which fragments sleep and elevates hyperarousal and stress reactivity[23]. The clinical diagnosis of insomnia often conflates physiological hyperarousal with anxiety rooted in social expectations and ambient threat perception.

Thermoregulation and Preferred Ambient Temperature

Disputes over the thermostat are pervasive in co-living environments, rooted deeply in physiological differences regarding metabolic heat production and vasodilation.

Physiology

The foundational critique of modern indoor climate control was published by Kingma and van Marken Lichtenbelt (2015, *Nature Climate Change*). They demonstrated that standard indoor climate regulations—specifically the widely used ANSI/ASHRAE Standard 55—are based on an empirical thermal comfort model developed in the 1960s. The primary variable for metabolic rate in this model (1 *met*, or 58 W/m^2) was calibrated to an average 70 kg male[24]. Testing a sample of 16 females, Kingma and Lichtenbelt showed that the actual resting metabolic rate of females performing light office work is significantly lower, leading the standard to overestimate female metabolic heat production by up to 35%[24].

Furthermore, thermoregulatory cooling mechanisms differ. Gagnon and Kenny (2012) observed that women exhibit lower sudomotor activity (sweat rate) and a lower thermosensitivity of the sweating response compared to men when exercising at a fixed rate of metabolic heat production[28]. Male physiology activates cold output mechanisms (sweating and vasodilation) more aggressively to dissipate heat[29].

Hormonal Roots: Estrogen promotes vasodilation, while progesterone acts on the hypothalamus to elevate the basal body temperature set-point by approximately 0.3°C to 0.5°C during the luteal phase, forcing the female body to defend a higher core temperature and inducing a sensation of cold at lower ambient temperatures[6].

Behaviour

Behaviourally, this translates to distinct thermal preferences. Field and laboratory studies indicate that women generally prefer ambient temperatures around 24°C to 25°C, whereas men prefer temperatures closer to 21°C to 22°C[26]. A meta-analysis by Karjalainen (2011) confirmed

that females are significantly more likely to express thermal dissatisfaction in shared environments (ratio: 1.74, 95% CI: 1.61–1.89), primarily due to overcooling[26].

Culture

The cultural layer heavily confounds this physiological baseline. Leading critics of the Kingma and Lichtenbelt hypothesis, such as Bjarne Olesen (ASHRAE), argue that the physiological differences in metabolism are offset by female skin temperature and evaporative loss[26]. Olesen contends that when male and female subjects are matched for clothing insulation (*clo* values), thermal preferences converge[26]. The cultural expectation that men wear high-*clo* value attire (suits and ties) in summer, while women wear low-*clo* value attire, exacerbates the physiological gap, placing the burden of adaptive actions overwhelmingly on female occupants[33].

Pain Perception and Analgesia

Sex differences in pain sensitivity and analgesic efficacy represent a vital area of dimorphic biology, directly affecting illness management in shared environments.

Physiology

Historically, ascending and descending pain pathways were assumed to be identical across sexes. This was overturned by Sorge et al. (2015, *Nature Neuroscience*), who demonstrated a qualitative sex difference in pain processing[35]. In male mice, chronic mechanical pain hypersensitivity is mediated by microglia in the spinal cord. In female mice, microglia are entirely dispensable; instead, females utilize adaptive immune cells, specifically T lymphocytes, to achieve the same mechanical pain hypersensitivity[35]. This widely replicated finding proves that the cellular mechanism of pain is fundamentally sexually dimorphic[38].

Physiological divergence is also observed in the endogenous opioid system. Gear, Mogil, and others have documented sex differences in analgesia mediated by the kappa opioid receptor (KOR)[39]. In clinical settings, KOR agonists produce significantly greater analgesia in women than in men, while mu-opioid receptor (MOR) agonists display complicated, dose-dependent sexual dimorphism that frequently favours greater efficacy in males in rodent models[41].

Hormonal Roots: The transition from microglial to T-cell pain mediation in females is deeply influenced by the hormonal milieu; testosterone suppresses T-cell infiltration in males, forcing reliance on microglia, while female prolactin and its specific receptors critically mediate female-specific nociception[44].

Behaviour

Statistically, females exhibit greater sensitivity to experimentally induced pain. A meta-analysis by Racine et al. (2012) and Riley et al. (1998, *Pain*) examined cross-sectional studies on pain threshold and tolerance (N = 7,464 for threshold, N = 4,935 for tolerance)[46]. Males demonstrate significantly higher pain thresholds (d = 0.66) and higher pain tolerance (d = 0.79), reporting lower pain intensity (d = -0.58) compared to females[47]. The effect sizes are stimulus-dependent; differences are largest for pressure and electrical stimulation, and smaller for thermal pain[48]. Epidemiologically, women are vastly overrepresented in chronic pain syndromes[44].

Culture

The behavioural reporting of pain is profoundly shaped by culture. Boerner et al. (2018) note that nearly 70% of clinical pain literature conflates biological sex with psychosocial gender[46]. Studies show that individuals who identify with highly masculine traits demonstrate higher pain thresholds (r = 0.17), while adherence to gender stereotypes specific to pain shows a moderate correlation with sensitivity (r = -0.41)[50]. Furthermore, male participants exhibit lower sensitivity when assessed by a female examiner, illustrating a strong cultural masking effect on innate physiological thresholds[51].

Immune Function and Illness Behaviour

When individuals co-habit, the transmission of pathogens is inevitable. Biological sex fundamentally alters the probability of infection, the severity of the illness, and the likelihood of developing autoimmune conditions.

Physiology

Klein and Flanagan (2016, *Nature Reviews Immunology*) detail the conserved phylogenetic advantage of female immunity. Generally, adult females mount vastly stronger innate and adaptive immune responses than males[52]. Genetically, the human X chromosome contains approximately 1,100 genes, many of which are heavily immune-related. While females (XX) undergo random X-chromosome inactivation (XCI) to prevent gene dosage toxicity, approximately 15% of X-linked genes escape inactivation[54]. Toll-like receptor 7 (TLR7), critical for detecting viral nucleic acids, frequently escapes X-inactivation, resulting in higher baseline expression and greater pro-inflammatory cytokine production in female immune cells[53].

Hormonal Roots: Estrogen generally enhances immune activity, whereas testosterone acts as an immunosuppressant[55]. This results in females demonstrating a greater neutralizing antibody seroconversion rate following vaccinations and carrying lower viral loads during acute infections (e.g., HIV, COVID-19)[53].

Behaviour

The behavioural outcome of this physiological variance is a double-edged sword. While males are more susceptible to severe outcomes from viral, bacterial, and fungal infections, and have a twofold higher risk of death from malignant cancers, females suffer overwhelmingly from autoimmune diseases[53]. Approximately 80% of all autoimmune disease cases occur in females, driven by the breakdown of peripheral tolerance and the hyperactivity of T-regulatory cells in response to self-antigens[52].

Culture

Culturally, gender influences the risk of exposure to specific pathogens via occupational sorting, nutritional differences, and healthcare-seeking behaviour. Socialized gender roles can alter the microbiome through early environmental exposures and dietary habits, adding a confounding cultural overlay to intrinsic physiological immunity[52].

Olfaction, Pheromone Signalling, and Sensory Thresholds

Sensory thresholds dictate how individuals perceive their shared physical environment, from food preferences to the perception of ambient odors.

Physiology

At the neurological level, the olfactory bulb (OB) exhibits profound sexual dimorphism. Oliveira-Pinto et al. (2014, *PLOS ONE*) utilized the isotropic fractionator method to count absolute cells in postmortem human OBs (N = 18; 7 men, 11 women). They found that despite similar gross anatomical masses, females possess an average of 6.9 million OB neurons compared to 3.5 million in males—a 49.3% difference in neuronal density[58]. Females also possess 38.7% more non-neuronal (glial) cells in the OB[58]. In gustation, anatomical data (Bartoshuk et al., 1994) revealed that females have a statistically significantly higher density of fungiform papillae and taste buds on the anterior tongue[60].

Hormonal Roots: Olfactory and gustatory acuity fluctuate with the menstrual cycle, peaking when estrogen levels are highest during the late follicular phase, equipping females with heightened environmental detection during periods of peak fertility[61].

Behaviour

Behaviourally, these physiological advantages translate into females demonstrating lower olfactory thresholds, superior odorant identification, and a higher prevalence of genetic "supertasters"[59]. Women consistently rate ambient odors as more intense and are more likely to find body odors unpleasant compared to men[61].

Culture and Controversy: The Pheromone Debate

Despite clear differences in olfactory sensitivity, the existence of behavior-altering human sex pheromones remains highly contested. For decades, the steroids androstadienone (AND, found in male sweat) and estratetraenol (EST, found in female urine) were marketed as putative sex pheromones. In a rigorously controlled, double-blind study published in *Royal Society Open Science*, Hare et al. (2017) definitively failed to replicate previous claims regarding AND and EST[64]. Testing 94 participants across consecutive days, they found that exposure to AND or EST had zero effect on the perception of gender in morphed androgynous faces, nor did it influence ratings of attractiveness or probable unfaithfulness of opposite-sex faces[64]. Leading critics, including Wyatt (2015), note that EST has never even been directly detected in human axillary sweat[67]. The consensus suggests that human intellect largely overrides any subliminal chemosignaling in complex social judgments[64].

Metabolism and Appetite

Metabolism dictates energy requirements, dietary preferences, and physical endurance capabilities, all of which manifest daily in shared meals.

Physiology

During submaximal endurance exercise, female and male metabolisms partition fuel differently. Tarnopolsky (2008, *Medicine and Science in Sports and Exercise*) highlighted that females exhibit a significantly lower Respiratory Exchange Ratio (RER) than males[69]. A lower RER indicates a proportionately higher reliance on lipid (fat) oxidation and a lower reliance on carbohydrate and protein oxidation[69]. Females possess higher intramyocellular lipid content

and demonstrate greater adipocyte lipolysis[71]. Consequently, females are better able to spare muscle glycogen, which delays the onset of peripheral fatigue[72].

Hormonal Roots: This metabolic divergence is heavily mediated by the hormonal influence of 17-beta-estradiol, which upregulates the mRNA abundance for components of fat-oxidation pathways, directly suppressing carbohydrate oxidation in favour of lipid utilization[69].

Behaviour

Following physical exertion, male resting metabolic rate (RMR) remains elevated significantly longer than in females, resulting in greater post-absorptive whole-body lipolysis[73]. This implies female metabolic control returns to baseline homeostasis with greater precision, whereas male metabolism remains perturbed.

Culture and Controversy

While the sex difference in substrate utilization is robust in general populations, it faces critique in athletic contexts. Quittmann et al. argued that when men and women are strictly matched for maximal oxygen uptake (VO2max) and fat-free mass, differences in Maximal Fat Oxidation (MFO) dissipate (d = 0.330, p = 0.432)[74]. However, a systematic review by Cano et al. (2022, N = 8,990 across 36 studies) maintains that absolute MFO remains lower in females, but when adjusting for lean mass, the sexual dimorphism persists largely due to complex age- and obesity-related moderating factors rather than vanishing entirely[72]. Culturally, dietary guidelines frequently default to male-normative caloric and macronutrient baselines, failing to account for female metabolic flexibility[75].

Physical Strength and Endurance

Though not explicitly detailed in the provided snippets, extrapolating from established physiological literature is necessary to complete the requested scope of bodily differences impacting co-living.

Physiology

Males possess, on average, significantly larger cross-sectional muscle fiber areas and higher percentages of fast-twitch (Type II) muscle fibers compared to females. This generates a massive divergence in upper body strength (often yielding effect sizes of d > 2.0) and lower body strength (d > 1.5).

Hormonal Roots: This dimorphism is almost entirely driven by the androgenic surge during male puberty, where high levels of circulating testosterone drive protein synthesis and inhibit protein breakdown, resulting in greater lean muscle mass and bone mineral density.

Behaviour

Behaviourally, males exhibit higher peak force generation and explosive power. However, owing to the metabolic differences discussed previously (higher reliance on lipid oxidation and higher proportions of Type I slow-twitch muscle fibers), females generally exhibit superior fatigue resistance during sustained, submaximal isometric contractions.

Culture

Culturally, shared physical tasks (e.g., lifting heavy objects in a household) typically default to males due to this overwhelming divergence in peak strength, reinforcing traditional gender roles. However, the female advantage in endurance and fatigue resistance is rarely culturally

codified, often remaining invisible in domestic labor expectations.

The Brain Mosaic vs. Dimorphism Controversy

Any discussion of bodily differences ultimately reaches the brain. The debate over whether human brains are fundamentally "male" or "female" represents the most fiercely contested territory in neuroscience.

The Mosaic Hypothesis

Daphna Joel and colleagues (2015, *PNAS*) proposed the "mosaic brain" model. Analyzing MRI scans of over 1,400 brains, Joel's team evaluated regions exhibiting the largest average sex differences. They concluded that while specific regional volumes (e.g., the left hippocampus) show group-level sex differences, a single individual's brain rarely possesses exclusively "male-typical" or "female-typical" features[76]. Up to 53% of brains consist of a heterogeneous mosaic of masculine and feminine traits, with only 0 to 8% showing exclusively uniform sex-typical traits[77]. Joel argues that environmental conditioning constantly remodels the brain, rendering a strict male-female continuum scientifically invalid[78].

The Multivariate Critique

Critics, led by Larry Cahill and Marco Del Giudice, argue that Joel's methodology was mathematically constructed to obscure differences by examining traits in isolation. Del Giudice applied standard multivariate analysis to the exact same dataset. By analyzing the variables collectively, Del Giudice demonstrated that individual men and women could be correctly classified by biological sex 69% to 77% of the time based solely on brain morphology[80].

Furthermore, Ingalhalikar et al. (2014, *PNAS*) utilized diffusion tensor imaging (DTI) on 949 youths to map the structural connectome. They identified massive sex differences in trajectory during adolescence. Males exhibited stronger intra-hemispheric connectivity (front-to-back), hypothesized to optimize the translation of perception into coordinated action. Females exhibited stronger inter-hemispheric connectivity (left-to-right across the corpus callosum), optimizing communication between analytical and intuitive processing modes[81]. This controversy highlights the tension between averages and individuals: while multivariate algorithms can definitively separate male and female brains in *n*-dimensional space, any single morphological trait viewed in isolation will show massive overlap[76].

Summary of Robust Findings

Metric	Key Robust Findings
1. Circadian Rhythm	Females possess a shorter intrinsic circadian period (by ~6 mins) and earlier melatonin onset, driving earlier chronotypes and longer biological nights.
2. Sleep Architecture	Females objectively possess higher sleep efficiency and more slow-wave sleep, yet

	subjectively report significantly more insomnia and poorer sleep quality.
3. Thermoregulation	Females have a lower basal metabolic rate and lower sweat-rate thermosensitivity, resulting in a physiological preference for ambient temperatures ~3°C warmer than males.
4. Pain Processing	Chronic pain hypersensitivity is mediated by microglia in males and T-cells in females; males exhibit larger effect sizes for pain tolerance and threshold.
5. Analgesia	Kappa-opioid receptor (KOR) agonists provide significantly greater and more robust analgesia in females compared to males.
6. Immune Function	Driven by X-chromosome gene escape (TLR7) and estrogen, females mount vastly stronger immune responses to pathogens but suffer from 80% of all autoimmune diseases.
7. Olfaction	Females possess nearly 50% more olfactory bulb neurons than males, translating to lower sensory thresholds and higher acuity for ambient and body odors.
8. Metabolism	Females rely more heavily on fat oxidation (lower Respiratory Exchange Ratio) during endurance tasks, sparing glycogen due to the influence of 17-beta-estradiol.
9. Pheromones	Claims regarding putative human sex pheromones (AND and EST) have failed rigorous replication, showing no effect on gender perception or attractiveness.

10. Brain Morphology	While the brain exhibits a mosaic of overlapping traits individually, multivariate structural connectome algorithms can accurately classify sex in 69–77% of individuals.

Contested or Open Questions

- **The Pheromone Effect:** Do any human chemosignals actually drive complex social or mating behaviour, given the definitive replication failure of AND and EST?
- **Substrate Oxidation:** Does the female metabolic advantage in fat oxidation disappear when male and female athletes are perfectly matched for VO2max and fat-free mass, or does it persist due to intrinsic dimorphism?
- **The Brain Modularity Debate:** Is it more accurate to conceptualize the brain as a highly variable, sex-neutral "mosaic" (Joel) or a dimorphic organ accurately classified via multivariate connectivity algorithms (Cahill/Del Giudice)?
- **The Sleep Paradox:** Why do women report significantly worse sleep quality, higher fatigue, and higher rates of insomnia despite polysomnography data repeatedly showing superior sleep architecture?
- **Analgesic Prescribing:** Should clinical protocols for analgesia (specifically mu vs. kappa opioids) be universally bifurcated by biological sex to optimize pain management?

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Implications for Living Together

Note: Identifying a biological difference does not prescribe a moral or social imperative. The naturalistic fallacy—the assumption that "natural" means "good" or "inevitable"—must be strictly avoided when designing shared spaces or assigning roles.

The implications of these biological differences for couples, families, and organisations suggest that standardizing shared environments based on a single biological normative (historically, the male average) generates structural and interpersonal friction. In organisations, adjusting ambient temperatures or providing individual thermal controls can mitigate the documented 35% metabolic overestimation of female heat production, potentially increasing comfort, productivity, and energy efficiency.

For couples and families, recognizing that chronotypes are driven by intrinsic circadian periods (with females biologically predisposed to earlier biological nights) can depersonalize conflicts over sleep routines and morning obligations. Understanding the dimorphic processing of pain and illness removes the stigma from behavioral differences during sickness; an awareness of female vulnerability to autoimmunity and male susceptibility to acute pathogens should foster tailored, equitable approaches to healthcare within families, moving away from universal analgesic dosing.

Acknowledging disparities in sensory thresholds—such as heightened female olfactory and gustatory acuity—can explain divergent reactions to foods, household odors, or hygiene standards, reframing them as physiological realities rather than arbitrary aesthetic preferences. Furthermore, acknowledging the differing metabolic responses to exercise and fasting can optimize shared nutritional planning without holding both partners to an identical standard of metabolic flexibility. Ultimately, co-living is optimized not by ignoring biological sex differences, but by engineering flexible environments that accommodate overlapping but distinctly divergent physiological needs.

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