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Outpaced by Industry: Industrial Environments Reduce Endurance, With Implications for Evolutionary Fitness

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ABSTRACT

Objectives: Physical function—the capacity to perform tasks requiring endurance and/or strength—is a key determinant of fitness that has directly influenced *Homo sapiens'* survival, reproduction and health throughout our evolutionary journey. However, the last 200–300 years of global industrialization has transformed human habitats at an unprecedented rate and may now be compromising key functions that underpin our fitness (*Environmental Mismatch Hypothesis)*. Although industrialization has delivered a range of benefits, it has simultaneously introduced novel environmental challenges (e.g., air pollution, microplastics) and reduced contact with beneficial aspects of nature (e.g., phytoncides). While negative effects of industrialization have been demonstrated for other determinants of fitness, its impact on physical function remains almost completely unexplored.

Materials and Methods: We conducted a randomized, counterbalanced crossover study to determine whether brief exposure to an industrialized environment would impair endurance performance relative to a forest environment (used as a proxy for non-industrial ancestral conditions). Twenty-five healthy adults (19 females, 6 males) completed two test sessions, each involving a 90 min environmental exposure followed by a standardized laboratory cycling test of endurance.

Results: Endurance performance was significantly reduced following industrial exposure (time-to-exhaustion: 13.5 ± 0.9 min) compared to forest exposure (14.6 ± 1.0 min; $p = 0.007$). Industrial exposure also worsened mood and led to volitional exhaustion at a lower perceived exertion, while cardiorespiratory markers recorded during the endurance test (e.g., $\dot{V}O$) did not differ significantly between conditions.

Conclusions: These results suggest that acute exposure to industrialized environments may reduce physical capacity, with potential consequences for evolutionary fitness.

Research Highlights

- Industrialization has transformed human habitats, introducing novel environmental challenges and reducing contact with nature.
- A 90 min industrial exposure reduced cycling time-to-exhaustion by ~7.5%, supporting the Environmental Mismatch Hypothesis.
- Industrial exposure worsened mood and caused participants to stop at a lower perceived exertion.

1 | Introduction

1.1 | Physical Function as an Evolutionary Asset

Physical function—the capacity to perform tasks requiring endurance and/or strength—is a fundamental determinant of survival, reproduction, and therefore, evolutionary fitness. Across the hominin lineage, selective pressures appear to have favored endurance capacity in particular. Unlike other primates, *Homo sapiens* evolved a unique capacity for sustained endurance activity, supported by a suite of anatomical and physiological adaptations that enhanced locomotor efficiency, strength, stabilization, and thermoregulation (Bramble and Lieberman 2004; Carrier 1984; Lieberman, Bramble, et al. 2006).

Throughout human evolution, behaviors such as hunting, scavenging, and seasonal mobility—which were essential for acquiring food and water and navigating changing climatic conditions—demanded prolonged physical effort over long distances and likely provided key selective contexts for the evolution of human endurance (Bramble and Lieberman 2004). Hunting, for example, provided access to calorie-dense animal foods critical for survival and provisioning, with endurance running emerging as a central component of hunting success in some human groups. Persistence hunting, documented among the !Kung and San of the Kalahari, the Tarahumara of Northern Mexico, and the Aché of Paraguay, involves pursuing prey over long distances, sometimes up to 35 km (Liebenberg 2006; Pennington 1963), until the animal is exhausted or hyperthermic. This strategy illustrates how, at least in warm climates, endurance capacity could yield high caloric returns at a relatively low metabolic cost (Liebenberg 2006; Lieberman, Bramble, et al. 2006), thereby favoring adaptations for efficient endurance walking and running (Lieberman, Raichlen, et al. 2006; Schapera 1930; Steyn 1984). Endurance capacity was complemented by strength, which facilitated weapon use and load carrying (Apicella 2014).

Endurance capabilities may also have been reinforced through social and reproductive mechanisms (Zahavi 1975). Physically demanding activities such as hunting may have served as reliable signals of underlying desirable traits—including endurance, strength, intelligence, and altruism—enhancing social status, mate attraction, and reproductive success (Hawkes 1993; Kaplan and Hill 1985; Longman 2019; Longman et al. 2015; Marlowe 2004). Moreover, recent archaeological (Lacy and Ocobock 2024) and physiological (Ocobock and Lacy 2024) evidence suggests that females contributed substantially to hunting as well as extractive foraging, gathering, and long-distance mobility while carrying loads or infants—activities that would have imposed considerable endurance and strength demands that could have driven physiological adaptation (Stock and Pfeiffer 2001; Wall-Scheffler 2022). Thus, endurance capacity appears to have emerged as a key determinant of both survival and reproductive success that shaped the physiology of human males and females.

Endurance capacity remains a critical determinant of health in contemporary populations. As a reflection of the integrated functioning of multiple physiological systems—including the musculoskeletal, respiratory, cardiovascular, psychoneurological, endocrine and metabolic systems—cardiovascular fitness strongly predicts physical capacity and morbidity and mortality (Harber et al. 2017; Ortega et al. 2008). Muscular strength also remains a strong independent predictor of mortality risk (García-Hermoso et al. 2018). Together, endurance and strength represent measurable markers of evolutionary fitness that continue to shape health outcomes in modern industrialized populations.

1.2 | Is the Industrialization of Human Habitats Impairing Fitness?

For most of the human evolutionary journey, a range of natural environments defined the ecological parameters within which selection shaped our biology. Over the last ~6–7 million years, hominins adapted to diverse climatic and ecogeographical challenges, and in the last 100,000 years *Homo sapiens'* remarkable biological and cultural adaptability enabled expansion into nearly all terrestrial biomes (Wells and Stock 2007). Although human-induced alterations to the terrestrial biosphere have been evident for over 10,000 years, the pace and scale of environmental change accelerated dramatically with the Industrial Revolution (Ellis 2011). While our species has adapted to a wide range of ecological conditions throughout its evolutionary journey, the speed of recent change may be exceeding our biological capacity for adaptation, with implications for long-term health and evolutionary fitness (Longman and Shaw 2026).

Industrialization has fundamentally transformed *Homo sapiens'* primary habitat, driving mechanized production, fossil fuel dependence, rural-to-urban migration and extensive environmental degradation (Erb et al. 2018; Rockström et al. 2009; Steffen et al. 2011). Today, most of the global population lives in industrialized urban areas (UN 2018) that differ fundamentally from ancestral environments. To one extent or another, urban areas lack natural features that support health and function while simultaneously imposing novel stressors, such as air, noise and light pollution, and microplastic contamination (Fleming et al. 2011; Haahtela 2019; Rook 2012, 2013; Scudellari 2017; Smith et al. 2019).

The concept of evolutionary mismatch describes the adverse consequences arising from an imbalance between an organism and its environment (Gluckman and Hanson 2006; Lieberman 2014; Lloyd et al. 2011; Nesse and Williams 1996). The evolutionary mismatch framework has been productively applied to understanding contemporary patterns of

chronic disease, particularly by linking modern human behaviors to disease risk (Gluckman et al. 2019, 2020; Godfrey et al. 2007; Gurven and Lieberman 2020; Lea et al. 2023; Low et al. 2019). Established applications link nutritional changes to obesity and metabolic disorders (Gluckman et al. 2019; Godfrey et al. 2007; Raubenheimer et al. 2012; Simpson and Raubenheimer 2005), increased physical inactivity to cardiovascular, musculoskeletal, and cognitive diseases (Lieberman 2015; Raichlen et al. 2017; Raichlen and Alexander 2017), increased chronic psychosocial stress to immune dysregulation and mental health conditions (Brenner et al. 2015; Li et al. 2018; Jiaqing et al. 2021), and microbial depletion to increased autoimmune and allergic disease burden (Björkstén 2012; Haahtela et al. 2013; Matricardi 2010; Rook 2012, 2013).

Research considering the influence of urbanization and industrialization on human biology has largely focused on how environmental transformation affects health. This work has explored the consequences of urban living for infectious and chronic disease patterns (McMichael 2001; Schell and Ulijaszek 1999), the effects of urban environments on child growth and development (Schell et al. 2012; Tanner 1987), and biosocial perspectives on health in contemporary urban settings (Dorsey 2025; Schell 2025). This body of research has established that environmental mismatch—particularly the shift from rural to urban living—can have important consequences for human biology.

Building on this foundation, we recently proposed the *Environmental Mismatch Hypothesis* (Longman and Shaw 2026), which extends prior work in several key respects. First, it provides a systematic comparison of ancestral and contemporary habitats. Second, rather than focusing on health outcomes, it examines impacts on markers of evolutionary fitness. Third, it advances a dual mechanism emphasizing both novel environmental stressors and loss of contact with essential natural features. Finally, it positions physical habitat transformation along an industrialization continuum rather than treating environmental change as a binary rural–urban distinction.

In the 21st century, human environments lie along an industrialization continuum, ranging from minimally altered ecosystems to highly modified megacities. Truly non-industrialized habitats—widespread before the advent of agriculture ~12,000 years ago—are now exceedingly rare; even remote regions contain microplastics in soil, air and water (Goudie 2018; Rillig 2012). At the opposite end, populous cities such as Tokyo or Delhi are dominated by concrete, steel and plastic, with few remaining natural features (Bettencourt 2013; Elhacham et al. 2020). Most habitats fall between these extremes, combining natural and synthetic elements, and most people live much closer to the industrialized end of this continuum. As of 2018, over half the global population resided in urban areas, a figure projected to rise to 68% by 2050 (UN 2018). In parallel, daily life has shifted indoors, with individuals in developed countries spending over 90% of their time inside buildings or vehicles (Baczynska et al. 2019; Khajehzadeh and Vale 2017; Matz et al. 2014). Consequently, most people inhabit environments lacking natural features that support health and function, while being exposed to novel environmental challenges such as air pollution, noise and light pollution, and pervasive microplastic accumulation (Fleming et al. 2011; Haahtela 2019; Rook 2012, 2013; Scudellari 2017).

Testing the *Environmental Mismatch Hypothesis* requires experimentation that assesses whether exposure to industrialized environments compromises key biological functions central to evolutionary fitness. To date, research that has considered the effect of industrialization on physical performance has been limited and largely observational. However, these studies are cross-sectional and vary in design and the level of control for confounders. To address this gap, we conducted a randomized crossover study that examined whether brief exposure to an industrialized environment impaired endurance performance relative to a natural environment. Based on the *Environmental Mismatch Hypothesis* (Longman and Shaw 2026), we hypothesized reduced endurance performance following industrial exposure.

2 | Materials and Methods

2.1 | Participants

All procedures were approved by the Loughborough University Ethics Review Sub-Committee (Application No: 2022–6225-8343) and adhered to relevant guidelines and regulations. Written informed consent was obtained from all participants. Testing took place in Leicestershire, UK.

Thirty-six healthy adults were recruited from Leicestershire, UK. Exclusion criteria were: hypertension (blood pressure > 140/90 mmHg), blood donation in the past 3 months, diagnosed mood disorders (e.g., depression, anxiety), medical conditions that restricted physical activity, amenorrhea, smoking, excessive alcohol consumption (> 14 units/week), pregnancy or lactation. Participants were instructed to refrain from physical activity for 24 h prior to participating, consume the same food before both sessions and to avoid alcohol or other recreational substances the evening prior to testing sessions.

All participants completed both experimental conditions. Ultimately, 25 participants were included in the analyses of endurance performance. Specifically, only participants who reached a rating of perceived exertion (RPE) of at least 15 out of 20 (i.e., "hard" or greater) during the cycle test were included. This threshold was selected to increase confidence that the task was performed to near exhaustion, as required by the study design. Participants who did not meet this criterion were excluded to avoid confounding effects associated with submaximal effort (this did not alter the main results).

Participant descriptive statistics, including age, height, body mass, and BMI, are displayed in Table 1.

To minimize the effects of hormonal fluctuation throughout the menstrual cycle and to reduce potential confounding effects of cycle-related hormonal fluctuations, female participants were tested during menstruation. Baseline salivary oestradiol levels

did not differ between forest and industrial sessions; a linear mixed effects model (fixed effects: environment, trial order; random effect: participant ID) showed no difference in baseline oestradiol (Forest estimated marginal mean = 1.7 pg/mL, standard error = 0.1 pg/mL; Industrial = 1.5 pg/mL, standard error = 0.1 pg/mL; estimated difference = −0.1 pg/mL, standard error = 0.1 pg/mL, 95% confidence interval [−0.4, 0.1], $p = 0.343$).

2.2 | Experimental Protocol

Participants first attended a familiarization session, during which time they were introduced to all aspects of the experimental protocol.

A randomized counterbalanced crossover design was employed. A list of randomized numbers was produced by an online randomization generator. The numbers were split evenly between the 2 intervention groups to ensure equal distribution of the group. On each test day, participants arrived at the National Centre for Sports and Exercise Medicine (NCSEM), Loughborough University, at 08:30. After a 10 min seated rest to establish a physiological baseline, pre-trial measurements were taken (described below). Participants were then driven approximately 8 min to one of the two test sites: The Outwoods ancient woodland (forest) or Loughborough town centre (industrial setting). See Figures 1 and 2 below and Supporting Information 1 for a detailed description of the test sites.

TABLE 1 | Participant descriptive statistics.

	Male ($n = 6$) Mean (SD)	Female ($n = 19$) Mean (SD)
Age (years)	27.3 (1.8)	30.0 (4.2)
Height (cm)	176.8 (7.4)	166.3 (10.0)
Body mass (kg)	69.3 (10.1)	65.9 (11.6)
BMI (kgm^{-2})	22.1 (2.3)	24.0 (4.7)

At the test sites, participants completed an individual 90 min guided environmental exposure protocol designed to facilitate sensory engagement with the surroundings (Figure 1). Participants visited four nearby locations ("nodes") within each environment, spending 20 min at each (10 min seated on a chair and 10 min lying on a mat). The nodes were separated by short walks of approximately one to 2 min. At each node, participants received written prompts encouraging engagement with the environment through hearing, smell, sight and touch.

At the third node, participants were offered a snack (Graze bar; 30 g, 535 kJ/128 kcal, 6.3 g fat [3.6 g saturates], 14 g carbohydrates [3.8 g sugar], 4.8 g fiber, 1.8 g protein, 0.06 g salt). Snack consumption was kept consistent between testing sessions.

Following the environmental exposure protocol, participants were driven back to the NCSEM for post-trial testing (described below).

2.3 | Measures

Measures taken during endurance test after environmental exposure:

1. Physical performance

Endurance performance was assessed using a maximal ramp-incremental cycling test on an upright ergometer (Lode BV, Groningen, Netherlands). The test began with 3 min at 50 W, followed by a stepwise increase of 25 W every 3 min until volitional exhaustion. Participants maintained a cadence of 80 rpm (±5 rpm), and the test was terminated when the cadence dropped below 70 rpm for more than 5 s.

2. Cardiorespiratory Performance

Pulmonary gas exchange was measured continuously during the endurance test using a Cortex Metalyzer3B cardiopulmonary test system (Cortex Biophysik, Leipzig, Germany).

FIGURE 1 | Forest (The Outwoods; left) and industrial (Loughborough town centre; right) test sites.

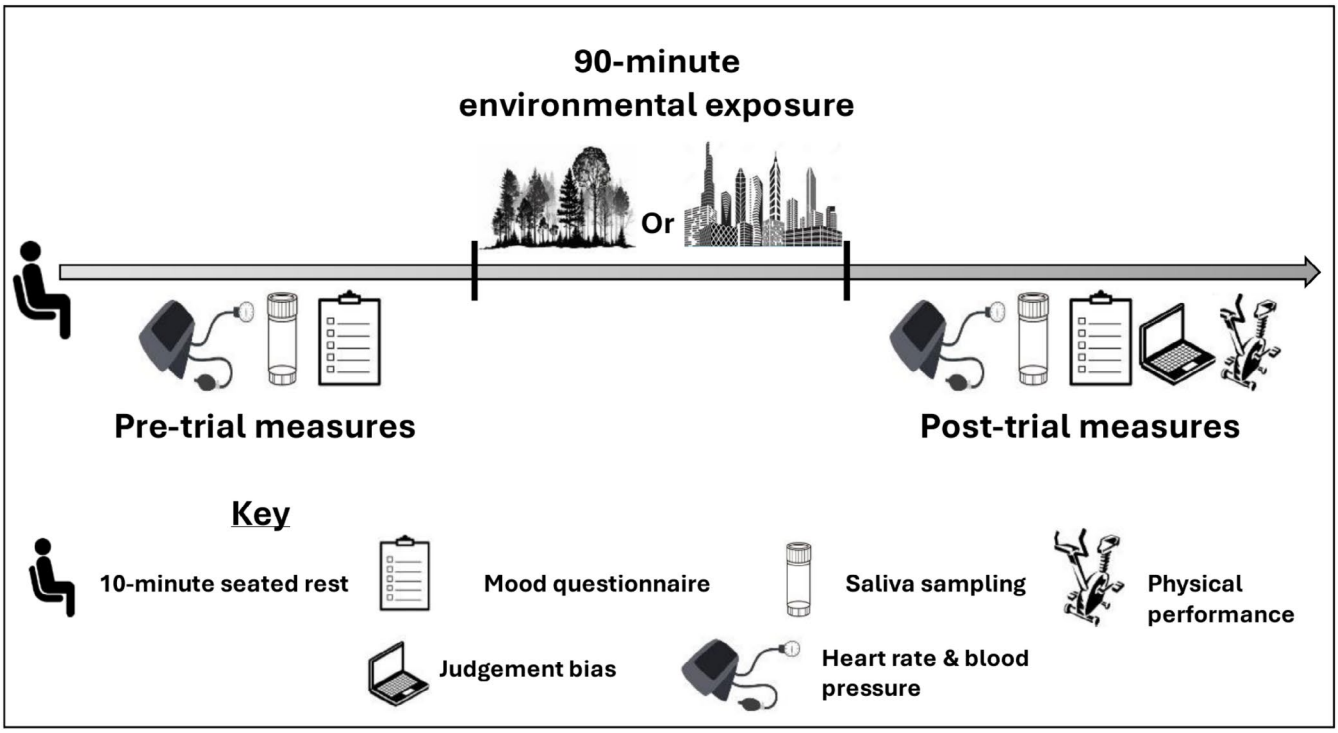

FIGURE 2 | Overview of the experimental protocol. Baseline assessments included psychophysiological stress (heart rate, blood pressure, salivary cortisol) and mood. Post-exposure assessments repeated these measures and added a computerized judgment bias task and a maximal ramp-incremental cycling test of physical performance.

3. Exertion

Ratings of perceived exertion (RPE) were recorded verbally at the start of each new stage using the 6–20 Borg Scale (Borg 1998).

Measurements taken before and after environmental exposure:

4. Physiological biomarkers

Resting heart rate and blood pressure were measured in a seated position using a sphygmomanometer (M6, Omron, Kyoto, Japan) on the non-dominant upper arm (three measurements were taken and the data averaged).

Saliva samples were collected using the passive drool method and Saliva Collection Aids (Salimetrics LLC., Ely, UK), then stored at −80°C. Salivary cortisol concentrations were analyzed in duplicate using a commercially available ELISA kit (Salimetrics LLC., Ely, UK). Samples with a coefficient of variation exceeding 10% were re-analyzed.

5. Psychology

Participants completed a printed version of the Abbreviated Profile of Mood States (POMS) questionnaire (Grove and Prapavessis 1992; McNair et al. 1992), which assessed seven subscales (Tension, Anger, Fatigue, Depression, Confusion, Vigor, and Esteem Related Affect) that were aggregated into a Total Mood Disturbance (TMD) score.

Participants also completed a computerized judgment bias task (developed on Gorilla Experiment Builder, https://gorilla.sc/) (Davidson et al. 2025; Iigaya et al. 2016). After the environmental exposure, participants were required to determine whether visual stimuli (Gabor patches) were tilted to the left or right. The stimuli used were either unambiguous—the degree of tilt from vertical allowed easy classification, or ambiguous—the degree of tilt from vertical made the orientation difficult to classify. Correct classification of one orientation (e.g., left) was associated with a higher nominal reward (£4) while correct classification of the alternate orientation (e.g., right) was associated with a lower nominal reward (£1). Incorrect classification resulted in no reward (£0). The task included 80 trials (balanced for direction bias) and participants were incentivized with a £10 voucher for the highest score. An 'optimistic' decision is operationally defined as classification of a stimulus in the direction associated with the higher reward outcome, with 'optimistic' decision-making in judgment bias tasks associated with a more positive mood (Neville et al. 2020).

6. Environmental characterization

To characterize the forest site, and any differences between forest nodes, we conducted a survey of the species composition of the plots by carrying out plant community surveys at three times throughout the study period. We surveyed each plot on 23rd May, 10th August, and 14th October 2023. We used a line transect approach because this enables coverage of a large area and was repeatable between each survey. This approach can miss rarer species, but for the purposes of this study, we considered the dominant species to be the most important because these are the species that will contribute to participants' experience of the woodland. For each survey, we laid out two 20 m transects in a cross pattern, so that each transect was oriented East to West or North to South, and the centre was located in the centre of the

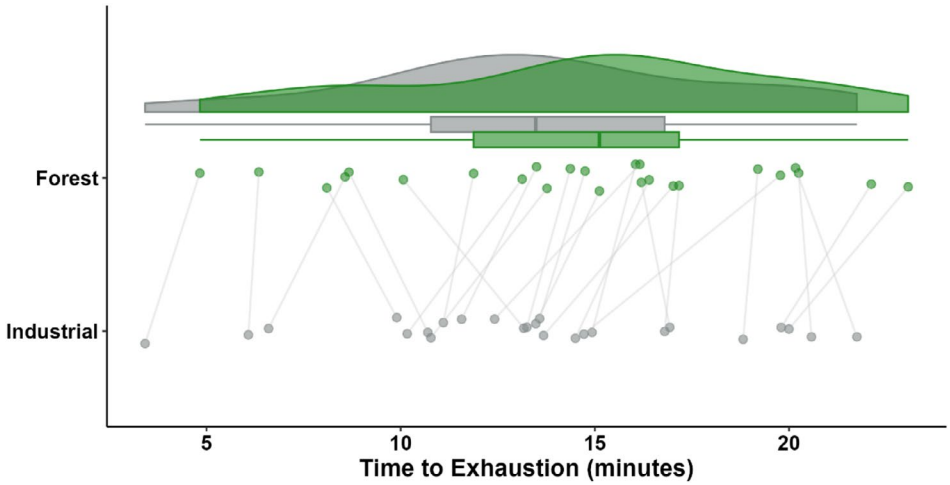

FIGURE 3 | Differences in cycling endurance performance (time to exhaustion, measured in minutes) following exposure to the industrial and forest environments. Time to exhaustion was shorter after visiting the industrial site relative to visiting the forest site. Industrial: Gray; Forest: Green. Raincloud plot code from (Allen et al. 2021).

study plots. We used a vertical densiometer (Geographic Resource Solutions) to survey the species present in the canopy, sub canopy, and on the ground for each point at 1 m intervals along the transects, giving a total of 41 points. Data were converted to percent cover by dividing the number of hits by 41.

To characterize the volatile organic compounds (VOCs) in the air of urban and forest environments, we set up sampling locations at 10 cm, 1 m and 2 m in each referred to as low (L), medium (M), and high (H) in SI Table S1. All air samples were collected through PTFE tubing onto a 6 mm OD stainless steel cartridge (PerkinElmer, Waltham, MA, USA) packed with an adsorbent matrix (200 mg Tenax TA 60/80 and 100 mg Carbotrap 20/40) at a flow rate of 0.2 L min^{-1} using a high precision handheld pump (SKC Ltd, Blandford Forum, UK). Samples were collected for 20 min to obtain a 4000 mL sample. The cartridges with the air samples were analyzed at UKCEH on a state of the art Agilent 8890 GC System 5977B with HES MSD Turbo (Agilent Santa Clara, California, USA) and Markes UNITY-ULTRA-XR thermodesorption unit (Markes, Cardiff, UK). We used reference standards for >40 monoterpenes that we can quantify to a very low detection limit (ppt level).

2.4 | Statistical Analyses

Analyses were conducted using the Statistical Package for the Social Sciences (SPSS) v.25. Consistent with guidance advising against binary null-hypothesis testing based on arbitrary *p* value cut-offs (Muff et al. 2022), we do not classify results as "significant" or "non-significant." Rather than applying a $p<0.05$ threshold, we interpret effect estimates, confidence intervals, and effect sizes to characterize the magnitude and uncertainty of the observed effects.

Primary analyses used linear mixed effects models to evaluate the effect of environment (industrial vs. forest) and, for variables measured multiple times, time (pre vs. post environmental exposure) on outcome variables relating to endurance performance, cardiorespiratory and effort outcomes, measured during the endurance test and physiological and psychological outcomes, measured pre- and post-environmental exposure. Detailed site descriptions and weather conditions are available in (including Figure S1 and Table S1) and 2 (Table S2). Trial order was controlled for to account for potential order effects. Participant ID was included as a random effect to account for repeated measures and within-subject variability. All models specified a normal probability distribution with an identity link function. Estimated marginal means (EMMs) were calculated to obtain covariate-adjusted condition means.

Model residuals were assessed for normality (Shapiro–Wilk tests, histograms and Q-Q plots), homoscedasticity (residual vs. predicted scatterplots) and outliers (residual boxplots).

Effect sizes (Cohen's dz) were calculated from EMMs and their standard errors. Condition-specific SDs were estimated by multiplying the SE of the EMM by $\sqrt{n}$, and dz was computed as the difference between adjusted means of the two conditions divided by the pooled SD.

3 | Results

Results are presented in three domains: (1) endurance performance, (2) cardiorespiratory and effort outcomes, measured during the endurance test, and (3) physiological and psychological outcomes, measured pre- and post-environmental exposure. Detailed site descriptions and weather conditions, as well as further model statistics, are available in (Table S2) and (Table S3 through S11), respectively.

3.1 | Endurance Performance

There was strong evidence for an effect of environment on endurance performance, with time to exhaustion being shorter following exposure to the industrialized environment (13.5 ± 0.9 min) compared with the forest environment (14.6 ± 1.0 min, $F(1,47)=8.092$, $p=0.007$). The effect size was small (dz = 0.2). See Figure 3 and Table 2.

TABLE 2 | Summary of key outcome measures following exposure to forest and industrial environments.

Domain	Measure	Forest (EMM ± SE)	Industrial (EMM ± SE)	Estimated difference (95% CI)	*p*	Effect size (*d*)
Endurance performance	Time to exhaustion (min)	14.6 ± 1.0	13.5 ± 0.9	−1.1 [−1.9, −0.3]	0.007	0.2
Cardiorespiratory & effort measures during endurance test	Heart rate; last 30 s (bpm)	177.0 ± 3.0	172.3 ± 2.7	−4.6 [−9.3, 0.2]	0.051	0.3
	RPE at exhaustion	17.7 ± 0.3	16.9 ± 0.3	−0.8 [−1.4, −0.2]	0.007	0.5
Physiological & psychological measures pre- and post-environmental exposure	Heart rate	60.9 ± 2.2 ➔68.9 ± 2.2	60.0 ± 2.2 ➔72.2 ± 2.2	Forest: 8.0 [4.5, 11.4] Industrial: 12.1 [8.6, 15.5]	0.099	—
	Systolic blood pressure	112.2 ± 1.8 ➔115.2 ± 1.8	112.7 ± 1.8 ➔115.2 ± 1.8	Forest: 3.0 [0.2, 5.9] Industrial: 2.5 [−1.6, 6.5]	0.234	—
	Diastolic blood pressure	70.3 ± 1.2 ➔72.7 ± 1.2	112.7 ± 1.8 ➔115.2 ± 1.8	Forest: 2.4 [−0.2, 5.0] Industrial: 2.4 [−0.2, 5.0]	0.420	—
	Cortisol (nmol/L)	23.6 ± 3.6 ➔6.3 ± 1.2	22.7 ± 3.0 ➔15.7 ± 3.1	Forest: −17.3 [−24.4, −10.2] Industrial: −7.0 [−15.1, 1.2]	0.061	Forest: 1.3 Industrial: 0.5 Difference: 0.7
Psychological	Mood (POMS Total Mood Disturbance)	98.1 ± 3.4 ➔86.2 ± 3.7	94.4 ± 2.8 ➔98.7 ± 2.3	Forest: −11.9 [−19.1, −5.8] Industrial: 4.3 [−0.4, 8.9]	<0.001	Forest: 0.7 Industrial: 0.3 Difference: 1.1
	Judgment bias (proportion of 'optimistic' responses)	0.59 ± 0.03	0.52 ± 0.02	−0.07 [−0.13, −0.01]	0.026	0.6

Note: Values are estimated marginal means (EMMs±SE) derived from linear mixed effects models. For time to exhaustion, heart rate and endurance performance and exertion, models included environment and trial order as fixed effects and participant ID as a random effect. For physiological and psychological measures, models additionally included time (pre/post exposure) and environment × time interactions. Reported *p* values correspond to the fixed effect of environment (or environment × time where applicable). Effect sizes (Cohen's d) are shown for significant or near-significant effects.

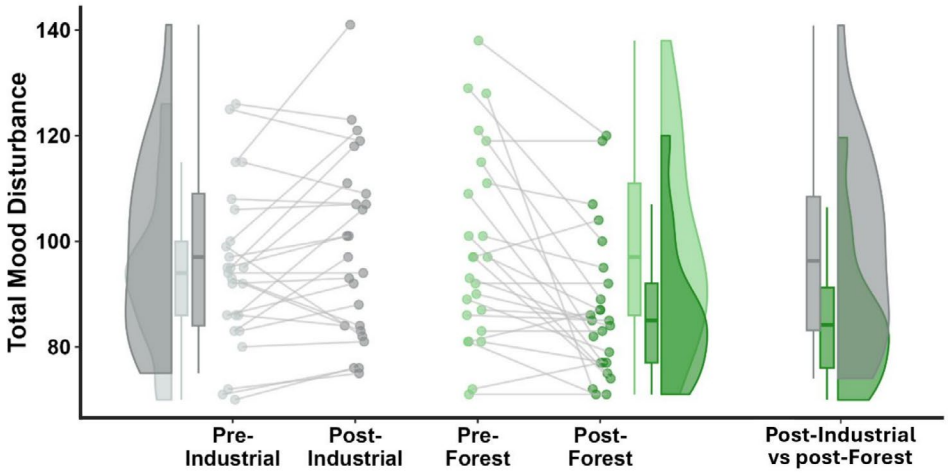

FIGURE 4 | Differences in mood (Total Mood Disturbance—a higher score indicates a worse mood) before and after exposure to the industrial and forest environments. There was strong evidence of divergent effects of exposure to the industrial and forest sites on participants' mood, whereby visiting the industrial site worsened mood relative to visiting the forest site. Pre-Industrial: Light gray, post-industrial: Dark gray, pre-forest: Light green, post-forest: Dark green. Raincloud plot code from (Allen et al. 2021).

3.2 | Cardiorespiratory and Effort Measures, Captured During the Endurance Test

There was weak evidence for an effect of environment on heart rate during the final 30 s of the endurance test, with a lower heart rate following exposure to the industrialized environment compared with the forest environment ($F(1,39) = 4.061$, $p = 0.051$; Table 2). The effect was small (Cohen's $d = 0.3$). There was no evidence for an effect of environment on any of the other cardiorespiratory measures (see Table S11). There was strong evidence for decreased perceived exertion at volitional exhaustion following exposure to the industrialized environment, compared with the forest environment ($F(1,47) = 8.000$, $p = 0.007$; Table 2), with a medium effect (Cohen's $d = 0.5$).

3.3 | Physiological and Psychological Measures Pre- and Post-Environmental Exposure

3.3.1 | Physiological

There was no evidence of environment*time interactions for heart rate ($F(1,72) = 2.790$, $p = 0.094$), systolic blood pressure ($F(1,72) = 1.440$, $p = 0.234$) or diastolic blood pressure ($F(1,72) = 0.657$, $p = 0.420$). There was weak evidence for an effect of environment on salivary cortisol, whereby cortisol decreased less following industrial exposure ($F(1,87) = 3.591$, $p = 0.061$).

3.3.2 | Mood (POMS)

There was strong evidence of an environment*time interaction for mood, which worsened after visiting the industrial location relative to the forest ($F(1,95) = 14.011$, $p < 0.001$; Table 2; Figure 4), with a large effect (Cohen's $d = 1.1$). While pre-session mood did not differ between environments, there was strong evidence of poorer post-session mood following industrial exposure compared with forest exposure (b = 12.5, SE = 3.4, t(95) = 3.671, $p < 0.001$, 95% CI [5.8, 19.3]). There was strong evidence that mood improved following the forest visit ($b = -11.9$, SE = 3.1, $t(72) = -3.890$, $p < 0.001$, 95% CI [−18.0, −5.8]) but not after the industrial site visit ($b = 4.3$, SE = 3.1, $t(73) = 1.401$, $p = 0.165$, 95% CI [−1.8, 10.4]).

3.3.3 | Optimism (Judgment Bias)

There was moderate evidence of an effect of environment on judgment bias, with lower 'optimism' observed following exposure to the industrialized environment compared with the forest ($F(1,45) = 5.288$, $p = 0.026$; Table 2; Figure 5). The effect was medium (Cohen's $d = 0.6$).

For each of the variables assessed above, additional analyses showed that the effect of the industrial environment on outcome variables was not significantly moderated by participant sex, age, or BMI.

4 | Discussion

Endurance performance, as measured by time to exhaustion, was reduced following exposure to an industrial environment relative to a forest. This finding is notable given that performance was assessed in a controlled laboratory setting two hours after exposure, indicating the environmental impact persisted beyond immediate contact.

Our results demonstrate that even brief exposure to industrialized environments acutely impairs endurance performance, suggesting that such settings may constrain a biological capacity with deep evolutionary roots. This has two important implications: first, environmental industrialization may be undermining evolutionary fitness in modern populations. Aerobic capacity and strength are key contemporary indicators of resilience, long-term health and disease risk in both men and women (Silverman and Deuster 2014), directly affecting survival—a core component of evolutionary fitness. Physical function also

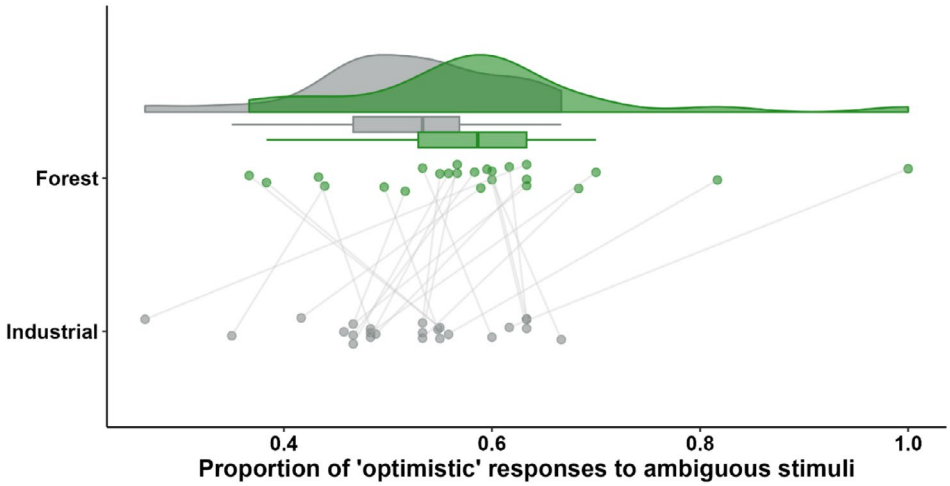

FIGURE 5 | 'Optimism' following exposure to the industrial and forest environments. The proportion of optimistic responses was lower after visiting the industrial site relative to visiting the forest site. Industrial: Gray; Forest: Green. Raincloud plot code from (Allen et al. 2021).

influences reproductive success through multiple pathways. It serves as a sexually selected trait: male muscularity, for example, signals mate quality (Durkee et al. 2019) and correlates positively with number of sexual partners and offspring in societies lacking widespread contraception (Frederick and Haselton 2007; Lidborg et al. 2022), while hunting ability signals underlying desirable traits and enhances reproductive success (Longman et al. 2015). Physical fitness also contributes to social status, which in turn affects mating opportunities across both traditional and industrialized societies (Hopcroft 2006; Nettle and Pollet 2008). Research considering the link between female physical fitness and sexual selection, however, remains limited (Hönekopp et al. 2004). While existing studies suggest that physical fitness may improve pregnancy outcomes for women (Hönekopp et al. 2004) and body shape and size have been linked to reproductive success in females (Jasieńska et al. 2004), the role of physical fitness in female sexual selection remains underexplored, warranting further investigation. Second, these findings raise concerns for athletic and occupational contexts where small decrements in endurance have meaningful consequences.

The environmental mismatch hypothesis (Longman and Shaw 2026) provides a plausible ultimate explanation: our biology evolved in ancestral natural environments and is poorly suited to the novel challenges posed by industrial settings. However, the proximate mechanisms explaining how performance is impaired remain less clear. One plausible pathway involves physiological disruption. Air pollution, for example, is known to impair pulmonary and cardiovascular function, thereby reducing exercise capacity (Chen and Kan 2008; Cutrufello et al. 2011; Rundell and Caviston 2008). Local emissions data confirm markedly higher concentrations of fine and ultrafine particles at the industrial test site used in this study relative to the forest site (UK Emissions Interactive Map 2025), raising the possibility that pollutant inhalation contributed to the reduced endurance performance observed here. There was, however, no apparent effect of environmental exposure on cardiorespiratory measures, which may reflect the timing of measurement (2 h post-exposure) rather than the absence of acute physiological responses.

The pattern of results points more strongly toward psychological mechanisms, which may have acted in concert with physiological mechanisms. Self-reported mood and mood as assessed via 'optimism' in a judgment bias task were significantly lower following the industrial site visit than after the forest visit, whereas physiological stress markers (blood pressure, heart rate variability, heart rate and cortisol) did not differ between conditions. These findings align with our previous experimental work demonstrating that natural environments enhance psychological outcomes while physiological stress markers show less consistent responses (Longman, Todorova, et al. 2025; Longman, Van Hedger, et al. 2025; Todorova et al. 2023). During the cycling test, ratings of perceived exertion at test termination were significantly lower after industrial exposure relative to the forest, while cardiorespiratory variables showed no consistent differences between environments. Taken together, these results suggest that exposure to the industrial environment diminished tolerance for discomfort and/or the subjective perception of effort, relative to exposure to the forest environment. Thus, psychological mechanisms, including both perceptual and affective pathways, may have contributed to impaired performance, although we cannot rule out physiological contributions that may have operated through pathways not captured by our measurement protocol. Future research should aim to disentangle the relative contributions of psychological and physiological mechanisms by combining environmental exposure with in situ testing, as well as a comprehensive assessment of environmental variables in both environments (e.g., air quality, noise, temperature and light) during field experiments and the measurement of a broader suite of biological and psychological measures.

The findings from this study have broad interdisciplinary relevance. From a biological anthropology perspective, industrialization and urban migration can be viewed as large-scale natural experiments, offering insight into how *Homo sapiens* adapt to novel habitats to maintain health and, ultimately, evolutionary fitness. This perspective not only sheds light on the biological consequences of recent environmental industrialization, but

also on the adaptive mechanisms that have enabled our species to thrive across diverse ecological settings over the past 300,000+ years.

The implications of this work are also forward-facing. Industrialization has largely been driven by short-term financial priorities, with limited consideration of its long-term effects on human or ecosystem health (Whitmee et al. 2015). Although industrialization and urbanization have delivered substantial social and economic benefits (Glaeser 2011), they have also caused severe ecological degradation, contributing to climate disruption, freshwater scarcity, biodiversity loss, pollution, declining soil microbial health, and reduced food security (Lovins et al. 2018). These interlinked crises now threaten population health and may reverse recent global health gains (Romanelli et al. 2015).

Our findings therefore add urgency to the need for environmental strategies with dual conservation and human health goals (Ford et al. 2015). One promising avenue is the ecosystem services framework, which emphasizes the benefits humans derive from healthy natural ecosystems, including food, materials, flood protection, climate regulation and tourism (Bennett et al. 2009; Daily and Matson 2008). However, human health benefits remain underrepresented within this framework (Ford et al. 2015). The present findings add to growing evidence that natural environments support human health (Bratman et al. 2021; Jimenez et al. 2021) and reinforce the argument that ecosystem services extend beyond economic or material value to include human biological function and wellbeing (Hernández-Blanco et al. 2022). While we have purposefully emphasized the evolutionary implications of our findings by framing natural environments as the ancestral baseline against which industrialized settings are compared, the results equally support a complementary public health interpretation. Given that aerobic fitness is a key indicator of resilience, long-term health and disease risk in contemporary populations (Silverman and Deuster 2014), access to natural environments may enhance physical capacity with important implications for urban planning and health promotion strategies.

This study, which is the first to experimentally assess the effect of exposure to industrial vs. natural environments on physical function, has several notable strengths. The randomized, counterbalanced crossover experimental design enhanced internal validity and statistical power, while the inclusion of a one-month washout period helped minimize order effects. Female participants were tested during menstruation, reducing the potential influence of hormonal fluctuations on key outcome variables (Armour et al. 2020). Finally, all data collection occurred within a three-month period (June–August 2022), limiting the influence of seasonal variation.

The study's participants were predominantly young adults (aged 25–44 years), with limited ethnic and cultural diversity and a skewed sex distribution (19 females, 6 males), which constrains generalisability. Although endurance performance was measured in a highly controlled laboratory setting, this protocol introduced a delay between environmental exposure and performance assessment which may have diluted immediate environmental effects—potentially leading to an underestimation of the negative impact of industrial exposure and/or the beneficial effects of nature exposure. Additionally, limitations regarding the reproducibility of endurance tests to exhaustion have been highlighted (Jeukendrup et al. 1996). Future research would benefit from assessing more ecologically valid indicators of endurance performance with a finite goal (e.g., outdoor running over a particular distance) and muscular strength, ideally within the testing environment itself, to better capture the real-world effects of industrial versus natural settings on physical function. In addition, future work should build upon these findings by recruiting larger and more diverse samples.

In summary, this study provides novel experimental evidence that even brief exposure to an industrialized environment impairs endurance performance relative to a forest environment. The effect appears to be at least partly psychologically mediated. These results support aspects of the Environmental Mismatch Hypothesis (Longman and Shaw 2026) and suggest that ongoing environmental industrialization may be constraining human physical capacity and, in turn, may undermine evolutionary fitness.

Author Contributions

Daniel P. Longman: conceptualization, methodology, formal analysis, resources, data curation, writing – original draft, writing – review and editing, project administration, funding acquisition, visualization, supervision. **Yvanna Todorova:** methodology, investigation, data curation, writing – review and editing, visualization. **Stephen J. Bailey:** methodology, writing – review and editing. **Nicolette C. Bishop:** writing – review and editing, supervision. **Molly Davidson:** methodology, writing – review and editing. **Julia Drewer:** writing – review and editing, methodology. **Ciara Dwyer:** methodology, investigation, writing – review and editing. **Lewis J. James:** writing – review and editing, supervision. **Yanzhe Li:** investigation, writing – review and editing. **Adelina Lintuluoto:** writing – review and editing, formal analysis. **Jonathan Millett:** methodology, investigation, writing – review and editing. **Vikki Neville:** methodology, writing – review and editing. **Fatin Sabrina Nor Azian:** investigation, writing – review and editing. **Matthew Putland:** investigation, writing – review and editing. **Toby Roberts:** methodology, writing – review and editing. **Mate Szazvai:** investigation, writing – review and editing. **Colin N. Shaw:** conceptualization, methodology, writing – review and editing, funding acquisition, visualization.

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Ethics Statement

All procedures were approved by the Loughborough University Ethics Review Sub-Committee (Application No: 2022–6225-8343) and adhered to relevant guidelines and regulations. Written informed consent was obtained from all participants.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

Data is available from the Loughborough University Research Repository. https://doi.org/10.17028/rd.lboro.30913409

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Supporting Information

Additional supporting information can be found online in the Supporting Information section. **Figure S1:** Species composition of ground flora and canopy tree species for the four study locations (plots) in Outwoods, Leicestershire. Presented is the percentage cover of plant species at three different survey dates in 2023. **Table S1:** Monoterpenes in ng L^{-1} from Outwoods and City sites. NA non detectable concentration (< LOD). **Table S2:** Meteorological data for the test days. The industrialized site had significantly higher average temperature, average windspeed and illuminance, and a non-significant trend for lower average humidity than the forest site. There was no significant difference in the number of days of rain. **Table S3:** Results of linear mixed-effects models examining the effects of environment and time on time to exhaustion. Linear mixed-effects model including fixed effects of Environment (forest, city) and Order (Forest-City vs. City-Forest), with

a random intercept for participant ID. The intercept represents the estimated mean time to exhaustion at the reference condition (Environment = Forest; Order = City-Forest). Positive coefficients indicate a higher time to exhaustion relative to the reference category. *p* values correspond to tests of fixed effects. Random-effects estimates represent the variance and standard deviation of participant-level intercepts and residual error. The full model (fixed and random effects) explained 92% of the total variance in time to exhaustion (conditional $R^2 = 0.919$) (1). **Table S4:** Results of linear mixed-effects models examining the effects of environment and time on maximal heart rate. Linear mixed-effects model including fixed effects of Environment (forest, city) and Order (Forest-City vs. City-Forest), with a random intercept for participant ID. The intercept represents the estimated mean maximal heart rate at the reference condition (Environment = Forest; Order = City-Forest). Positive coefficients indicate a higher maximal heart rate relative to the reference category. *p* values correspond to tests of fixed effects. Random-effects estimates represent the variance and standard deviation of participant-level intercepts and residual error. The full model (fixed and random effects) explained 76% of the total variance in maximal heart rate (conditional $R^2 = 0.764$) (1). **Table S5:** Results of linear mixed-effects models examining the effects of environment and time on maximal RPE. Linear mixed-effects model including fixed effects of Environment (forest, city) and Order (Forest-City vs. City-Forest), with a random intercept for participant ID. The intercept represents the estimated mean RPE at the reference condition (Environment = Forest; Order = City-Forest). Positive coefficients indicate a higher RPE relative to the reference category. *p* values correspond to tests of fixed effects. Random-effects estimates represent the variance and standard deviation of participant-level intercepts and residual error. The full model (fixed and random effects) explained 57% of the total variance in RPE (conditional $R^2 = 0.572$) (1). **Table S6:** Results of linear mixed-effects models examining the effects of environment and time on cortisol. Linear mixed-effects model including fixed effects of Environment (forest, city), Time (pre, post), the Environment × Time interaction and Order (Forest-City vs. City-Forest), with a random intercept for participant ID. The intercept represents the estimated mean cortisol at the reference condition (Environment = Forest; Time = Pre-exposure; Order = City-Forest). Positive coefficients indicate a higher cortisol relative to the reference category. *p* values correspond to tests of fixed effects. Random-effects estimates represent the variance and standard deviation of participant-level intercepts and residual error. The full model (fixed and random effects) explained 60% of the total variance in cortisol (conditional $R^2 = 0.598$) (1). **Table S7:** Results of linear mixed-effects models examining the effects of environment and time on heart rate. Linear mixed-effects model including fixed effects of Environment (forest, city) and Order (Forest-City vs. City-Forest), Time (pre, post), the Environment × Time interaction and Order (Forest-City vs. City-Forest), with a random intercept for participant ID. The intercept represents the estimated mean heart rate at the reference condition (Environment = Forest; Time = Pre-exposure; Order = City-Forest). Positive coefficients indicate a higher heart rate relative to the reference category. *p* values correspond to tests of fixed effects. Random-effects estimates represent the variance and standard deviation of participant-level intercepts and residual error. The full model (fixed and random effects) explained 75% of the total variance in heart rate (conditional $R^2 = 0.753$) (1). **Table S8:** Results of linear mixed-effects models examining the effects of environment and time on systolic blood pressure. Linear mixed-effects model including fixed effects of Environment (forest, city), Time (pre, post), the Environment × Time interaction and Order (Forest-City vs. City-Forest) and Order (Forest-City vs. City-Forest), with a random intercept for participant ID. The intercept represents the estimated mean systolic blood pressure at the reference condition (Environment = Forest; Time = Pre-exposure; Order = City-Forest). Positive coefficients indicate a higher systolic blood pressure relative to the reference category. *p* values correspond to tests of fixed effects. Random-effects estimates represent the variance and standard deviation of participant-level intercepts and residual error. The full model (fixed and random effects) explained 68% of the total variance in systolic blood pressure (conditional $R^2 = 0.680$) (1). **Table S9:** Results of linear mixed-effects models examining the effects of environment and time on diastolic blood pressure. Linear mixed-effects model including fixed effects of Environment (forest, city), Time (pre, post), the Environment × Time interaction and Order (Forest-City vs. City-Forest) and Order (Forest-City vs. City-Forest), with a random intercept for participant ID. The intercept represents the estimated mean diastolic blood pressure at the reference condition (Environment = Forest; Time = Pre-exposure; Order = City-Forest). Positive coefficients indicate a higher diastolic blood pressure relative to the reference category. *p* values correspond to tests of fixed effects. Random-effects estimates represent the variance and standard deviation of participant-level intercepts and residual error. The full model (fixed and random effects) explained 46% of the total variance in diastolic blood pressure (conditional $R^2 = 0.462$) (1). **Table S10:** Results of linear mixed-effects models examining the effects of environment and time on TMD. Linear mixed-effects model including fixed effects of Environment (forest, city), Time (pre, post), the Environment × Time interaction and Order (Forest-City vs. City-Forest) and Order (Forest-City vs. City-Forest), with a random intercept for participant ID. The intercept represents the estimated mean TMD at the reference condition (Environment = Forest; Time = Pre-exposure; Order = City-Forest). Positive coefficients indicate a higher TMD (and hence a poorer mood) relative to the reference category. *p* values correspond to tests of fixed effects. Random-effects estimates represent the variance and standard deviation of participant-level intercepts and residual error. The full model (fixed and random effects) explained 60% of the total variance in TMD (conditional $R^2 = 0.596$) (1). **Table S11:** Results of linear mixed-effects models examining the effects of environment and time on judgment bias. Linear mixed-effects model including fixed effects of Environment (forest, city) and Order (Forest-City vs. City-Forest), with a random intercept for participant ID. The intercept represents the estimated mean judgment bias at the reference condition (Environment = Forest; Time = Pre-exposure; Order = City-Forest). Positive coefficients indicate a higher judgment bias (and hence a poorer mood) relative to the reference category. *p* values correspond to tests of fixed effects. Random-effects estimates represent the variance and standard deviation of participant-level intercepts and residual error. The full model (fixed and random effects) explained 25% of the total variance in judgment bias (conditional $R^2 = 0.251$) (1). **Table S12:** Results of linear mixed-effects models examining the effects of environment on the cardiorespiratory response to the endurance test.