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University of Akureyri

Environmental and Individual Correlates of Depressive Symptoms in Iceland

The Role of Season, Environmental Mood Responses and Circadian Preference

Lada Zelinski

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Doctoral thesis

Psychology

Doctoral committee

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Dedicated to my aunt Sweta, the strongest person I have ever known.

Acknowledgments

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Abstract

This dissertation examines environmental and individual correlates of depressive symptoms in Iceland, a high-latitude population characterised by extreme seasonal variation in daylight and rapid weather changes. Paper I combines self-report screening for seasonality with semi-structured diagnostic interviews in a nationally representative sample, finding weighted elevated seasonality of 17.86% but a diagnostically confirmed Seasonal Affective Disorder prevalence of only 2.24%. This pattern of decline is comparable to that reported in interview-confirmed studies from lower-latitude populations, suggesting that screening instruments alone may inflate apparent latitude effects. Paper II examines subjective mood responses to environmental conditions, objective meteorological variables, and their interaction as correlates of depressive symptom severity, showing that more adverse mood responses to cold weather and reduced daylight are associated with greater symptom severity and that the association between temperature and depressive symptoms depends on individuals' subjective sensitivity to cold. Paper III uses a longitudinal design spanning four seasons to examine circadian preference and energy drink consumption in relation to depressive and insomnia symptoms, finding that evening types report greater baseline symptom burden and that energy drink intake frequency is associated with depressive symptoms specifically during the transition from summer to autumn, particularly among evening types. Taken together, the three papers suggest that clinically diagnosable Seasonal Affective Disorder is comparatively rare, but that seasonal shifts in symptoms are common; that environmental influences on depressive symptoms depend on individual sensitivity rather than exposure alone; and that the relevance of behavioural risk factors, such as energy drink consumption, for depressive symptoms can depend on circadian preference and season. These findings indicate that the relationship between seasonal environmental conditions and depressive symptoms is not uniform across individuals. As climate change continues to alter environmental conditions, understanding these interactions will become increasingly relevant for identifying those most at risk and for developing targeted approaches to prevention and support.

Úrdráttur

Í þessari ritgerð er sjónum beint, í þremur rannsóknum, að umhverfis- og eintaklingsbundnum þáttum í þunglyndiseinkennum á Íslandi -landi sem liggur norðarlega og mikill munur er á dagsbirtu og veðurfari eftir árstíðum. Í fyrstu rannsókninni var notaður skimunarlisti ásamt hálfstöðluðu geðgreiningarviðtali til að meta árstíðabundnar sveiflur í þunglyndiseinkennum og þunglyndisröskun í lagskiptu úrtaki Íslendinga. Vegið algengi árstíðabundins þunglyndis var mun hærra samkvæmt skimunarlista, eða 17,86%, heldur en samkvæmt geðgreiningarviðtali, eða 2,24%, sem er í samræmi við rannsóknir í löndum nær miðbaug og bendir til ofmats á áhrifum hnattlegu landa á þunglyndi, ef sjálfsmatsskimunarlistar eru eingöngu notaðir. Í annarri rannsókninni voru skoðuð tengsl skaps fólks við staðbundið hita- og birtustig og hvort þeir þættir tengdust alvarleika þunglyndiseinkenna. Niðurstöður sýndu að verra skap þegar kalt var í veðri og dagsbirta minni, tengdist alvarlegri einkennum og að tengsl hitastigs og þunglyndiseinkenna var háð (ó)þoli fólks fyrir kaldara hitastigi. Í þriðju rannsókninni var dægursveifluhneigð og neysla orkudrykkja skoðuð í tengslum við einkenni þunglyndis og svefnleysis með árstíðabundnum mælingum í eitt ár. Niðurstöður sýndu að kvöldhneigt fólk hafði meiri þunglyndiseinkenni á grunnlínu að sumri og að neysla orkudrykkja tengdist þunglynlyndiseinkennum þegar tók að hausta, sér í lagi hjá kvöldheigðu fólki. Samantekið, benda niðurstöður þessara þriggja rannsókna til að árstíðabundið þunglyndi, greint sem geðröskun í geðgreiningarviðtali, sé tiltölulega sjaldgæft en að sveiflur í þunglyndiseinkennum eftir árstíðum sé mun algengari þegar miðað er við sjálfsmat fólks. Niðurstöðurnar benda einnig til að áhrif umhverfisþátta á þunglyndiseinkenni séu háð einstaklingsbundnu mati og þoli fólks, frekar en þáttunum einum og sér og að tengsl orkudrykkja við þunglyndiseinkenni séu háð dægursveifluhneigð og árstíðum. Niðurstöður rannsóknaverkefnisins benda til að tengsl árstíðabundninna umhverfisþátta og þunglyndiseinkenna séu háð einstaklingsbundnum tilhneigingum. Loftslagsbreytingar móta umhverfi og aðstæður fólks. Aukinn skilningur á samspili umhverfis og einstaklingsbundinna þátta er því mikilvægur og getur nýst til að afmarka viðkvæma hópa og veita markvissari fræðslu og stuðning.

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List of abbreviations

AMOC	Atlantic Meridional Overturning Circulation
APA	American Psychiatric Association
BIS	Bergen Insomnia Scale
CO	Carbon Monoxide
DASS-21	Depression Anxiety Stress Scales-21
DLMO	Dim-Light Melatonin Onset
DSM-5-TR	Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision
EEG	Electroencephalography
FFQ	Food Frequency Questionnaire
GBD	Global Burden of Disease Collaborative Network
GSS	Global Seasonality Score
HPA	Hypothalamic-Pituitary-Adrenal
ICD-11	International Classification of Diseases, 11th Revision
MDD	Major Depressive Disorder
MEQ	Morningness-Eveningness Questionnaire
MINI	Mini International Neuropsychiatric Interview
NO	Nitric Oxide
NO2	Nitrogen Dioxide
PHQ-9	Patient Health Questionnaire-9
PM	Particulate Matter
PTSD	Post-Traumatic Stress Disorder
SAD	Seasonal Affective Disorder
SCN	Suprachiasmatic Nucleus
SERT	Serotonin Transporter
SHQ	Seasonal Health Questionnaire
SO2	Sulfur Dioxide
SPAQ	Seasonal Pattern Assessment Questionnaire
WHO	World Health Organization

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Overview of original articles

The thesis is based on the following three articles. Hereafter, they will be referred to by their numbers as follows:

I. **Zelinski, L.**, Pálmadóttir, A. D., Birgisdóttir, Á. G., Sesseljuson, L. D., Ólafsson, R. P., Bathke, A. C., & Höller, Y. (2025). Prevalence of seasonal affective disorder and seasonality of mood and behaviour in Iceland – Methodological implications for revisiting the latitude hypothesis. *Journal of Affective Disorders, 398,* 121057. https://doi.org/10.1016/j.jad.2025.121057

II. **Zelinski, L.**, Gunnarsdóttir, H. H., Ólafsson, R. P., & Höller, Y. (2026). When the weather gets colder and days get shorter: Environmental mood responses and their link to depressive symptom severity. *Journal of Environmental Psychology*, *111*, 102990. https://doi.org/10.1016/j.jenvp.2026.102990

III. **Zelinski, L.**, Ólafsson, R. P., & Höller, Y. (2026). Energy drink consumption and depressive symptoms in Iceland: Associations with season and circadian preference. Submitted to *BMC Public Health*.

Paper I and Paper II are open access and made available with permission from the publishers. Paper III was submitted to *BMC Public Health* on June 1st, 2026. Following the first round of peer review, the manuscript is currently under revision.

In addition to the three core papers, the following publications were co-authored during the doctoral period:

Birgisdóttir, Á. G., **Zelinski, L.,** Ólafsson, R. P., & Höller, Y. (2025). Safety threats of seasonal and non-seasonal natural disasters increase disaster anxiety and disaster risk perception. *Scandinavian Journal of Psychology*. https://doi.org/10.1111/sjop.70023

Höller, Y., Birgisdóttir, Á. G., **Zelinski, L.,** Pálmadóttir, A. D., Sesseljuson, L. D., & Ólafsson, R. P. (2026). Seasonal symptoms are related to rumination and habitual negative thinking. Submitted to *Scandinavian Journal of Psychology*; revised version is currently under review.

Höller, Y., **Zelinski, L.**, Sesseljuson, L. D., Pálmadóttir, A. D., Latini, A., Matthews, A., Ásmundsdóttir, Á. M. & Guðmundsson, L.S., & Ólafsson, R. P. (2025). Worries about air pollution from the unsustainable use of studded tires and cruise ships – A preliminary study on the relationship between worries and health complaints due to seasonal pollution. *Sustainability*, *17*(10), 4634. https://doi.org/10.3390/su17104634

Potthoff, J., Ólafsson, R. P., **Zelinski, L.**, & Höller, Y. (2025). Can self-reported seasonality predict prospectively assessed seasonal changes of self-reported mood, food cravings, body weight, insomnia, and physical activity? *Scandinavian Journal of Psychology*. https://doi.org/10.1111/sjop.70016

Declaration of Contribution to the thesis

The following authors have contributed to the main papers and manuscripts for this thesis: Ara Dan Pálmadóttir (ADP), Arne Cornelius Bathke (ACB), Ásta Guðrún Birgisdóttir (ÁGP), Harpa Hlín Gunnarsdóttir (HHG), Lada Zelinski (LZ), Leon Daði Sesseljuson (LDS), Ragnar Pétur Ólafsson (RPÓ), and Yvonne Höller (YH).

Paper I: LZ was responsible for writing the original draft, visualization, methodology, formal analysis, and data curation. ADP, ÁGB, and LDS contributed to investigation and data curation. RPÓ contributed to conceptualization, methodology, investigation, data curation, project administration, supervision, and critical revision of the manuscript. ACB contributed to validation and formal analysis. YH obtained ethical approval and contributed to conceptualization, methodology, investigation, data curation, formal analysis, validation, project administration, funding acquisition, resources, supervision, and critical revision of the manuscript. All authors read and approved the published version of the manuscript.

Paper II: LZ was responsible for conceptualization, methodology, formal analysis, visualization, writing the original draft, and critical revision of the manuscript. HHG contributed to conceptualization, methodology, and critical revision of the manuscript. RPÓ contributed to supervision and critical revision of the manuscript. YH obtained ethical approval and contributed to conceptualization, methodology, funding acquisition, supervision, and critical revision of the manuscript. All authors read and approved the published version of the manuscript.

Paper III: LZ was responsible for conceptualization, methodology, formal analysis, visualization, writing the original draft, and critical revision of the manuscript. RPÓ contributed to supervision and critical revision of the manuscript. YH obtained ethical approval and contributed to conceptualization, methodology, data collection, funding acquisition, supervision, and critical revision of the manuscript.

1 Introduction

1.1 Major Depressive Disorder

1.1.1 Prevalence

Major depressive disorder (MDD) is a prevalent mental disorder, affecting an estimated 236 million individuals, or approximately 3% of the global population, based on 2023 estimates (Global Burden of Disease Collaborative Network [GBD], 2025). In Iceland, the prevalence was higher, with approximately 16,553 individuals affected, corresponding to approximately 4.5% of the population (GBD, 2025). Although the prevalence of MDD varies across countries, global estimates suggest that differences between countries grouped by income level are relatively small, with prevalence generally being higher in high-income countries than in low- and middle-income countries (GBD, 2025; Marx et al., 2023).

Beyond differences across countries, prevalence estimates may also vary substantially depending on the assessment method used (Moffitt et al., 2010). Retrospective surveys, which rely on individuals recalling previous depressive episodes, suggest a lifetime prevalence of MDD of approximately 10.6% (Scott et al., 2018). However, longitudinal prospective studies that repeatedly assess participants over time have reported lifetime prevalence estimates approximately twice as high as those obtained through retrospective assessments, suggesting that retrospective methods may underestimate true prevalence due to recall bias and underreporting (Moffitt et al., 2010). In addition, prevalence estimates depend on the time frame examined, with point prevalence (i.e., the proportion of individuals currently affected) being considerably lower than lifetime estimates, as shown by a systematic review of 116 studies, which reported a global point prevalence of 4.7% (Ferrari et al., 2013).

1.1.2 Diagnosis

MDD is diagnosed according to the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition Text Revision (DSM-5-TR) (American Psychiatric Association [APA], 2022) or the International Classification of Diseases, 11th Revision (ICD-11) (World Health Organization [WHO], 2019). Both systems require the presence of at least five symptoms over a two-week period that represent a change from prior functioning and cause clinically significant distress or impairment. Symptoms span three clusters: emotional symptoms such as depressed mood and anhedonia, neurovegetative symptoms such as sleep disturbance, fatigue, and appetite changes, and neurocognitive symptoms such as impaired concentration and psychomotor disturbance. At least one of the two core

emotional symptoms, depressed mood or anhedonia, must be present for diagnosis in both systems (APA, 2022; WHO, 2019). Beyond formal diagnosis, depressive symptoms also occur along a continuum in the general population, and subthreshold presentations are common (Marx et al., 2023).

Categorical diagnostic systems such as the DSM-5-TR and the ICD-11 have well-recognised limitations, including difficulties distinguishing subthreshold from clinical presentations and substantial heterogeneity within diagnostic categories (Marx et al., 2023). Subthreshold presentations, while not meeting diagnostic criteria for MDD, are associated with meaningful functional impairment and an elevated risk of subsequently developing MDD (Cuijpers & Smit, 2004). Together, clinical and subthreshold presentations contribute to substantial individual and societal burden, including reduced quality of life (Brenes, 2007; Papakostas et al., 2004), increased healthcare utilisation (Beiser et al., 2019), and considerable economic costs (König et al., 2020). Although categorical diagnosis remains a practical necessity in clinical settings, dimensional approaches are more appropriate for research purposes, as they capture symptoms across the full severity spectrum, including aforementioned subthreshold cases that would otherwise go undetected (Goldberg, 2000). Depressive symptom severity is commonly assessed using such measures, with the Patient Health Questionnaire (PHQ-9; Kroenke et al., 2001) among the most widely used self-report measures in research and clinical settings (Costantini et al., 2021; von Glischinski et al., 2021), given its brevity, ease of administration, and free availability.

1.1.3 Etiology

The etiology of MDD is multifactorial, involving the interplay of biological, psychological, and environmental factors. The vulnerability-stress model (Ingram & Luxton, 2005; Zubin & Spring, 1977) proposes that pre-existing vulnerabilities, whether genetic, psychological, or biological, interact with environmental stressors to produce depressive episodes. At the biological level, the pathophysiology of depression involves multiple interacting mechanisms, including dysregulation of neurotransmitter systems, particularly serotonin, noradrenaline, and dopamine (Nutt, 2008), structural and functional brain changes such as hippocampal atrophy and altered activity in prefrontal and limbic regions (Campbell & MacQueen, 2004), hypothalamic-pituitary-adrenal (HPA) axis dysregulation (Mello et al., 2003), immune and inflammatory processes (Blume et al., 2011; Kiecolt-Glaser & Glaser, 2002), gut-brain axis signalling (Foster & Neufeld, 2013; Patel et al., 2025), genetic (e.g., Bondy, 2007; Nabeshima & Kim, 2013) and epigenetic (Park et al., 2019) factors.

Beyond biological mechanisms, several non-biological risk factors for MDD have been identified, spanning demographic, psychological, sociocultural, and behavioural domains, as summarised in Marx et al. (2023). Demographic risk

factors include female sex (Kuehner, 2017) and younger and adolescent age (Zisook et al., 2007). At the psychological level, neurotic and introvert personality profiles (Jylhä & Isometsä, 2006), negative core beliefs and ruminative thinking patterns (Beck & Bredemeier, 2016; Nolen-Hoeksema, 2000), and a history of childhood trauma (Kuzminskaite et al., 2022), among others, are associated with increased vulnerability. Behavioural and health factors including physical inactivity (Pearce et al., 2022), unhealthy diet (Li et al., 2017), and substance use (Wilkinson et al., 2016) further contribute to risk. At the sociocultural level, factors such as low socioeconomic status (Everson et al., 2002; Lorant et al., 2003), unemployment (Amiri, 2022; Zuelke et al., 2018), and lack of perceived social support (Grav et al., 2012) are implicated.

This etiological complexity is reflected in considerable variability in symptom profiles, severity, and course across individuals (Cai et al., 2020; Milaneschi et al., 2020). Alongside these factors, physical environmental conditions, including air (Borroni et al., 2022) and noise pollution (Seidler et al., 2017), limited green space (Z. Liu et al., 2023), and exposure to natural disasters and extreme weather (Beaglehole et al., 2018), have emerged as a further source of risk and are examined in more detail in the following section.

1.2 Environmental Correlates of Depression

1.2.1 Air Pollution

Air pollution is increasingly recognised as an important environmental risk factor for depression (Borroni et al., 2022). Relevant air pollutants include gaseous pollutants such as nitrogen dioxide (NO2), nitric oxide (NO), sulfur dioxide (SO2), and carbon monoxide (CO), as well as particulate matter (PM). PM is commonly classified by size into the coarse fraction (PM10, particles between 2.5 and 10 μm in diameter), the fine fraction (PM2.5, particles up to 2.5 μm), and the ultra-fine fraction (particles up to 0.1 μm), with smaller particles generally considered more harmful due to their ability to penetrate more deeply into the respiratory system (Hamanaka et al., 2025). A large prospective cohort study using UK Biobank data, with a follow-up period of approximately 11 years, found that exposure to PM2.5, NO2, and NO was associated with an increased risk of developing depression (Yang et al., 2023). This finding is broadly supported by a systematic review and meta-analysis of 39 studies, which found that long-term exposure to PM2.5 and NO2, as well as short-term exposure to PM10, PM2.5, NO2, SO2, and CO, was associated with an increased risk of depression (Borroni et al., 2022).

Inhaled air pollutants have been shown to induce oxidative stress, which is thought to underlie several pollutant-related health effects (Kelly, 2003). PM has additionally been shown to activate microglia, the brain's resident immune cells,

triggering neuroinflammation (Han et al., 2022). Dopaminergic neurons appear particularly vulnerable to this oxidative stress, with diesel exhaust particles (a subtype of PM) shown to trigger microglia-mediated release of reactive oxygen species that selectively harm these neurons (Block et al., 2004). As loss of dopaminergic neurons reduces dopamine availability, this pathway may be relevant to the monoamine-deficiency theory of depression, which implicates reduced dopamine activity in depressive symptoms (Hasler, 2010).

1.2.2 Limited Greenspace

In addition to air pollution, a lack of green space has been proposed as a risk factor for depression, with a meta-analysis finding that higher green space exposure was associated with a lower risk of depression and anxiety (Z. Liu et al., 2023). Consistent with this finding, a 10-year longitudinal study of 2.3 million adults in Wales reported that higher levels of residential greenness were associated with lower odds of subsequent development of depression or anxiety, with the beneficial effects more pronounced among individuals living in more socioeconomically deprived areas (Geary et al., 2023). Beyond its independent association with depression, green space may also modify the adverse effects of air pollution. For example, a US cohort study examining residential air pollution and green space jointly found that lower green space exposure was associated with a stronger adverse effect of air pollution on depressive and anxiety symptoms, suggesting that green space may buffer against the adverse effects of air pollution (Werder et al., 2024). Similar to the findings of Geary et al. (2023), these associations varied by socioeconomic deprivation, with the strongest effects observed among populations with lower socioeconomic status (Werder et al., 2024).

Exposure to natural environments is thought to facilitate recovery from stress and mental fatigue more effectively than built environment (Kaplan, 1995; Markevych et al., 2017; Ulrich et al., 1991). Consistent with this, exposure to natural environments has been shown to enhance heart rate variability, a marker of reduced physiological stress, relative to comparable activities in non-natural settings (Mygind et al., 2021). Exposure to green space may also benefit mental health by reducing exposure to environmental stressors such as air pollution, excessive noise, and urban heat (Markevych et al., 2017). Green space may further support mental health indirectly by facilitating physical activity and social interaction (Markevych et al., 2017), both of which are independently associated with a lower risk of depression (Pearce et al., 2022; Santini et al., 2015).

1.2.3 Noise Pollution

Evidence linking noise pollution to depression is less consistent than for air pollution and lack of greenspace, though associations have been reported (Seidler et al., 2017). Noise pollution refers to unwanted or excessive sound in the

environment arising primarily from road and air traffic, and has been recognised as a growing global health concern (Slabbekoorn, 2019). A large case-control study of individuals living in the Frankfurt, Germany airport region found that exposure to road traffic, aircraft, and railway noise was each associated with increased odds of a new clinical depression diagnosis, with combined exposure to all three sources above 50 dB yielding the highest risk estimate (Seidler et al., 2017). Notably, the association between aircraft noise and depression peaked at moderate exposure levels and declined at higher levels, which the authors suggested may reflect active noise-coping strategies such as soundproofing or moving away among the most heavily exposed individuals (Seidler et al., 2017). Findings are not entirely consistent, however, as a study comparing residents living near an airport in Sardinia, Italy, with a matched control sample found an increased frequency of anxiety disorders among noise-exposed individuals but no significant association with depression (Hardoy et al., 2005). Sleep may represent one pathway through which noise affects mental health. For example, Synga et al. (2014) found that road traffic noise was associated with psychological distress only among individuals reporting poor sleep quality, suggesting that sleep disruption, a well-established risk factor for depression (Tsuno et al., 2005), may be a key mechanism linking noise exposure to mental health outcomes.

At the physiological level, one proposed mechanism involves cortisol secretion. Noise annoyance has been shown to be positively associated with salivary cortisol levels following occupational noise exposure (Yaghoubi et al., 2020), suggesting that HPA axis activation may be a pathway through which noise exposure contributes to depression.

1.2.4 Natural Disasters and Extreme Weather

In addition to environmental stressors that operate through ongoing exposure, episodic events such as natural disasters (e.g., tsunamis, floods, earthquakes, landslides, and volcanic eruptions) and extreme weather events (e.g., heatwaves) can also substantially affect depression risk (Beaglehole et al., 2018; Cianconi et al., 2020; Tang et al., 2014). For example, a meta-analysis found that individuals exposed to natural disasters had significantly higher rates of depression and post-traumatic stress disorder (PTSD) compared with pre-disaster rates and non-exposed controls (Beaglehole et al., 2018). Depression prevalence following natural disasters has been estimated to range from 5.8% to over 50% in adults, with risk factors including bereavement, injury, loss of employment or property, and prior trauma (Tang et al., 2014). While the frequency of geophysical events, such as earthquakes and volcanic eruptions, has remained relatively stable, climate-related events, including storms, floods, heatwaves, and wildfires, have accounted for the majority of the increase in disaster frequency observed over recent decades, representing nearly 80% of this rise (Leaning & Guha-Sapir, 2013). These climate-related events have also been linked to increased rates of

depression, anxiety, and PTSD (Cianconi et al., 2020). One proposed pathway underlying these associations involves chronic activation of the stress response system, as prolonged trauma exposure can disrupt HPA axis functioning (Lawton et al., 2023).

1.3 Seasonal Light as an Environmental Correlate of Depression

Of the environmental variables examined in relation to depression, seasonal changes and reduced light exposure are among the most consistently associated with depressive episodes, as exemplified by Seasonal Affective Disorder (SAD) (APA, 2022; Rosenthal et al., 1984; WHO, 2019).

1.3.1 The Concept and Classification of SAD

Seasonal changes in mood and behaviour were first systematically described by Norman E. Rosenthal and colleagues (1984), who introduced the term SAD to describe a recurrent form of depression with a predictable seasonal pattern. This pattern most commonly involves episodes that emerge in autumn or winter and remit in spring or summer (Rosenthal, Sack, et al., 1984), although a summer pattern with onset and remission at opposite times of year can also occur (Wehr et al., 1991). In the DSM-5-TR, SAD is classified as a seasonal pattern specifier applicable to MDD and bipolar disorder, requiring a regular temporal relationship between the onset of depressive episodes and a specific season, with remission occurring as the season changes (APA, 2022). The ICD-11 similarly incorporates seasonal pattern as a qualifier for both depressive and bipolar episodes (WHO, 2019). Both systems further specify that seasonal episodes must substantially outnumber non-seasonal episodes over the individual's lifetime, and that the pattern should not be better accounted for by seasonally linked psychosocial stressors, such as seasonal unemployment (APA, 2022; WHO, 2019).

The most widely used screening instrument for SAD is the Seasonal Pattern Assessment Questionnaire (SPAQ), a self-report measure that asks respondents to retrospectively estimate seasonal changes in mood, sleep length, appetite, weight, energy level, and social activity over the past year, as well as the degree to which these changes are problematic (Rosenthal, Bradt, et al., 1984). A Global Seasonality Score (GSS) is derived from these items, with higher scores reflecting more pronounced seasonal changes across the assessed domains (Rosenthal, Bradt, et al., 1984). The items cover both mood and the reverse vegetative symptoms commonly associated with SAD, including hypersomnia, hyperphagia, and weight gain (Tam et al., 1997).

1.3.2 Light Exposure and Biological Mechanisms

Reduced light exposure during autumn and winter is considered the most consistently implicated environmental factor in the winter pattern of SAD (Rosenthal, Sack, et al., 1984), while the summer pattern has been linked to excessive heat and humidity (Morrissey et al., 1996). As research on the winter pattern far outnumbers that on the summer pattern, possibly reflecting its much higher prevalence to date (K. Kim et al., 2025; Magnusson, 2000; Magnusson & Partonen, 2005), the mechanisms discussed below primarily relate to winter-pattern SAD.

Light is detected through the retina by intrinsically photosensitive retinal ganglion cells (Berson et al., 2002; Hattar et al., 2002), which contain melanopsin and transmit signals via the retinohypothalamic tract to the suprachiasmatic nucleus (SCN) of the hypothalamus (Lucas et al., 2012). The SCN functions as the brain's primary circadian pacemaker, synchronising internal biological rhythms with the external light-dark cycle (Lucas et al., 2012). It regulates the secretion of melatonin, a hormone produced by the pineal gland that signals darkness to the body, with secretion suppressed during light exposure and elevated at night (Reiter, 1993). In the absence of external light cues, the SCN continues to oscillate but drifts to a period slightly longer than 24 hours (Czeisler et al., 1999).

Light exposure serves not only to anchor the daily rhythm but also to calibrate the circadian system to seasonal changes in the light-dark cycle (Reiter, 1993). According to the phase-shift hypothesis, reduced light exposure in winter disrupts this calibration, causing internal circadian rhythms to become misaligned with the external light-dark cycle, a process proposed to underlie the depressive symptoms observed in SAD (Lewy et al., 1984, 1987, 2007; Lewy & Sack, 1988). Consistent with this, morning light exposure, which advances the circadian phase and thereby corrects circadian delay, has shown greater antidepressant efficacy than evening light (Lewy et al., 1998). Indeed, bright light therapy, which involves daily exposure to high-intensity artificial light, has demonstrated efficacy in reducing depressive symptoms in SAD and is considered a first-line treatment (Chen et al., 2024; Golden et al., 2005; Partonen & Lönnqvist, 1998). However, not all individuals exposed to reduced light in winter develop depressive symptoms. Genetic variation in circadian clock genes may partly account for this differential susceptibility, with case-control studies identifying associations between winter depression and variants in the NPAS2 and CRY2 genes (Johansson et al., 2003; Lavebratt et al., 2010; Partonen et al., 2007).

Beyond circadian mechanisms, reduced light exposure in winter has been linked to several additional biological pathways implicated in depressive symptoms. Altered serotonergic function is one such pathway (Mc Mahon et al., 2016; Praschak-Rieder et al., 2008; Tyrer et al., 2016). Early neuroimaging evidence suggested that individuals with SAD exhibit higher winter serotonin transporter (SERT) availability than healthy controls, potentially increasing

serotonin reuptake and reducing synaptic serotonin availability (Praschak-Rieder et al., 2008). Subsequent longitudinal studies further demonstrated that seasonal regulation of SERT is altered in SAD and that greater winter SERT availability is associated with more severe depressive symptoms (Mc Mahon et al., 2016; Tyrer et al., 2016). Reduced light exposure has also been linked to changes in cortisol secretion, with human cortisol levels highest in winter and lowest in summer, closely tracking the timing of sunrise (Hadlow et al., 2014). Finally, reduced light exposure in winter also lowers cutaneous vitamin D synthesis, and vitamin D has separately been implicated in monoaminergic pathways relevant to depression (Geng et al., 2019).

1.3.3 Evaluating the Evidence of SAD

Despite the biological evidence outlined in Section 1.3.2 and SAD being recognised as a clinical diagnosis in major diagnostic systems (APA, 2022; WHO, 2019), questions have been raised about the epidemiological evidence supporting it (Hansen et al., 2008; Traffanstedt et al., 2016). For example, drawing on population studies conducted in northern Norway, Hansen and colleagues (2008) compared the prevalence of depression during the winter dark period with rates previously reported in populations at substantially lower latitudes and found no elevated prevalence (see Section 1.3.5 for a fuller discussion of the latitude hypothesis). They further reviewed existing validity studies of the SPAQ, identifying psychometric limitations including low test-retest reliability, low sensitivity, and poor predictive validity, and therefore questioned the clinical validity of SAD diagnoses derived from the SPAQ alone (Hansen et al., 2008). Traffanstedt et al. (2016) extended this critique using a large population-representative US sample (N = 34,294) in which current depressive symptoms were assessed with the Patient Health Questionnaire-8, a self-report measure of current depressive symptom severity consistent with DSM criteria. Controlling for known demographic correlates of depression (age, sex, race/ethnicity, educational level, marital status, and employment status), Traffanstedt et al. (2016) found no association between depression and season, latitude, or sunlight exposure, concluding that the evidence does not support the validity of a seasonal modifier in MDD. The authors acknowledged, however, that they could not rule out the possibility that SAD exists at a very low base rate, noting that very rare conditions are difficult to detect reliably in population-based studies, regardless of the instruments used (Traffanstedt et al., 2016).

These findings have been challenged in several published responses (Winkler et al., 2017; Young, 2017), with researchers arguing that the conclusions may reflect methodological limitations rather than the absence of SAD. A central criticism raised by both Winkler et al. (2017) and Young (2017) concerns the reliance on cross-sectional designs, in which participants were assessed only once, making it impossible to capture the recurrent within-person pattern of seasonal

onset and remission that defines SAD. Winkler et al. (2017) estimated that, given symptoms are typically present for only three to four months of the year, the majority of individuals with SAD would not be symptomatic at the time of a randomly timed cross-sectional assessment, with detection rates likely even lower among treated patients. Young (2017) further argued that the depression measures used in studies cited as evidence against SAD omitted reverse vegetative symptoms such as hypersomnia and hyperphagia, which are considered hallmark features of the disorder. Winkler et al. (2017) additionally pointed to several epidemiological studies employing DSM-based diagnostic questionnaires, which found SAD prevalence rates of around 2% in the general population, and argued that the conclusions of Traffanstedt et al. (2016) overlooked a substantial number of studies from multiple research centres in which patients met diagnostic criteria for SAD.

Taken together, these findings suggest that the debate surrounding SAD concerns not only the epidemiological evidence for its existence but also the methodological approaches used to identify it.

1.3.4 Defining and Measuring SAD

One of the methodological challenges in SAD research concerns the distinction between measuring seasonality and diagnosing a depressive disorder. Although the SPAQ is the most widely used instrument in studies of SAD (Akkaoui & Geoffroy, 2025), it does not assess whether an individual meets DSM-5-TR criteria for MDD or bipolar disorder with a seasonal pattern; rather, it measures the extent to which six domains of functioning change across the seasons. A notable limitation is that a high GSS can be obtained based solely on seasonal changes in vegetative symptoms, without mood change as a required component, whereas DSM-5-TR and ICD-11 criteria require depressed mood or anhedonia as a core symptom (APA, 2022; WHO, 2019). Consequently, the SPAQ measures a broader construct of seasonality than that defined by established diagnostic systems. In response to this limitation, several alternative instruments have been developed (for a review, see Akkaoui & Geoffroy, 2025), among them the Seasonal Health Questionnaire (SHQ), a diagnostically oriented screening instrument (Thompson & Cowan, 2001). Rather than deriving a continuous seasonality score, it screens first for depression and then determines whether depressive episodes are confined to a particular season, recur across consecutive years, and occur more frequently than episodes outside that season (Thompson & Cowan, 2001). However, this makes the SHQ longer and more complex to administer than the SPAQ, likely explaining why the SPAQ remains the dominant instrument in SAD research despite its conceptual limitations (Akkaoui & Geoffroy, 2025; Thompson & Cowan, 2001).

The conceptual distinction between seasonality and depressive disorder is also reflected in the dual vulnerability model, first proposed by Young et al. (1991)

and subsequently extended by Lam et al. (2001), which helps explain why instruments assessing seasonality alone may not align with diagnostic criteria for SAD. According to the model, individuals with high sensitivity to seasonal changes but little vulnerability to depression may experience primarily vegetative symptoms without meeting the criteria for a major depressive episode, representing subsyndromal SAD (Lam et al., 2001). Conversely, those with greater vulnerability to depression but low sensitivity to seasonal changes are expected to develop non-seasonal depression (Lam et al., 2001). Clinical SAD emerges when both vulnerabilities are sufficiently elevated, while the relative balance between seasonal and depressive vulnerability may influence whether symptoms fully remit during summer or persist across seasons (Lam et al., 2001)

1.3.5 The Latitude Hypothesis

The latitude hypothesis proposes that the prevalence of SAD increases with distance from the equator because seasonal variation in daylight becomes more pronounced at higher latitudes (Rosenthal, Sack, et al., 1984). However, testing this hypothesis has proven challenging because prevalence estimates vary according to the methods used to assess SAD. Studies employing both the SPAQ and DSM-consistent diagnostic criteria consistently report substantially lower prevalence estimates once diagnoses are confirmed by interview (Eagles et al., 1999; Levitt et al., 2000; Michalak et al., 2001).

Evidence supporting the latitude hypothesis has accumulated over several decades (K. Kim et al., 2025; Magnusson, 2000; Rosen et al., 1990). In an early systematic review, Magnusson (2000) concluded that SAD generally appeared more prevalent at higher northern latitudes, while noting considerable variation across ethnic groups and that the overall effect of latitude was probably small. For example, Magnusson and Stefansson (1993) found a SPAQ-based SAD prevalence of only 3.8% in Iceland despite its high latitude. Nonetheless, a quantitative synthesis of studies using the SPAQ found a significant positive association between latitude and SAD prevalence in general population samples, whereas findings from studies of specific subpopulations were less consistent (Michalak & Lam, 2002). More recently, a systematic review and meta-analysis restricted to SPAQ-based studies reported that the prevalence of SAD increased by approximately 0.2 percentage points per degree of latitude (K. Kim et al., 2025).

However, not all studies have observed this relationship (Levitt & Boyle, 2002; Mersch et al., 1999). Levitt and Boyle (2002), using structured diagnostic interviews across populations spanning eight degrees of latitude, found no evidence that SAD prevalence increased with latitude. Notably, when seasonality was assessed using the SPAQ, higher latitude was associated with lower rather than higher seasonality scores. Mersch et al. (1999) found a significant positive correlation between SAD prevalence and latitude in North American studies but

not in European studies, and concluded that the overall influence of latitude on prevalence appeared modest, suggesting that other factors, including climate, sociocultural, and genetic influences, may play a more important role.

Taken together, previous findings suggest that, although the latitude hypothesis is biologically plausible, empirical support remains inconsistent. Methodological differences in the assessment of SAD are likely to contribute to this variability and should therefore be considered when interpreting findings across studies.

1.3.6 Rationale for Paper I

Taken together, the literature reviewed in Section 1.3 indicates that SAD is supported by biological evidence, yet epidemiological findings remain inconsistent, largely reflecting differences in study design and measurement. Prevalence estimates vary considerably depending on whether SAD is assessed using screening instruments such as the SPAQ or structured diagnostic interviews. The evidence for a latitude gradient is strongest in studies relying on questionnaire-based measures of seasonality and weaker or absent in studies using diagnostic interviews, further underscoring the importance of distinguishing between elevated seasonality and clinically diagnosed SAD. Paper I of the present dissertation addresses this directly by combining initial SPAQ screening with subsequent diagnostic assessment using the Mini International Neuropsychiatric Interview (MINI; Sheehan et al., 1998), a structured interview based on DSM criteria. This two-stage approach yields a more conservative and clinically grounded prevalence estimate than studies relying on screening instruments alone and situates this estimate within an Icelandic population characterised by marked seasonal variation in daylight.

1.4 Individual Differences in Depression Vulnerability

Not all individuals exposed to the same environmental conditions develop depressive symptoms (Ingram & Luxton, 2005). Individual differences in sensitivity to meteorological and seasonal conditions may partly account for why some individuals are more vulnerable to depression than others (Mersch et al., 2004; Sigmon et al., 2010). Beyond environmental sensitivity, differences in circadian preference and behavioural factors such as energy drink consumption may further shape depressive and sleep outcomes, particularly in the context of seasonal variation (Au & Reece, 2017; Carneiro et al., 2026; Friborg et al., 2012; Nadeem et al., 2021; Zhao et al., 2025). The following sections examine these individual-level factors, which form the basis for Papers II and III of the present dissertation.

1.4.1 Weather and Seasonal Light Sensitivity

Latitude is often used as an indicator of seasonal changes in light, but actual sunlight exposure can also vary due to factors such as weather (e.g., cloud cover), geographical features (e.g., mountains), and urban environments (e.g., building density and resulting shading). Although weather and season are closely related, they represent distinct aspects of environmental exposure. Weather refers to short-term meteorological conditions, whereas season reflects longer-term cyclical changes, including photoperiod. For example, Reykjavík, Iceland (64°*N*), and Anchorage, Alaska (61°*N*) experience comparable seasonal variation in daylight because of their similar latitudes, yet their weather patterns differ substantially. Despite its high latitude, Iceland has a relatively mild climate due to the North Atlantic Current, whereas Alaska experiences colder temperatures and greater seasonal temperature variability because of stronger continental and Arctic influences. Weather and seasonal light exposure should therefore be considered as related but distinct environmental factors when examining their associations with depression.

Thus far, environmental conditions have received increasing attention as potential factors associated with depression, possibly reflecting growing concern about the mental health impact of climate change and extreme weather (Barkin et al., 2021; Cruz et al., 2020). Among these environmental factors, daylight exposure has received particular attention for its proposed physiological pathway to mood, involving mechanisms such as sunlight's role in regulating serotonin activity, as previously discussed in relation to SAD (see Section 1.3.2). Notably, this pathway has also been observed in healthy individuals (Lambert et al., 2002). In support of this, Lambert et al. (2002) used internal jugular venous sampling to estimate the turnover of the neurotransmitters serotonin, noradrenaline, and dopamine in the brain of healthy men across different seasons. Of these, only serotonin turnover varied significantly, being lowest in winter and directly related to the duration of bright sunlight on the day of measurement, with higher turnover on bright days than on overcast days regardless of the time of year, while no associations were found for weather variables, inlcuding temperature, rainfall, or atmospheric pressure (Lambert et al., 2002).

Evidence for the association between depression and environmental conditions has been examined across a range of variables. For instance, depression has been associated with reduced global radiation or sunlight exposure (e.g., S. Y. Kim et al., 2021; Molin et al., 1996; Raza et al., 2024) and overcast conditions (Xu et al., 2020). Associations have also been reported for temperature, with warmer temperatures linked to lower depressive symptom levels in some studies (e.g., Brazienė et al., 2022; Molin et al., 1996), whereas exposure to extreme cold has been associated with greater depressive symptom severity among rural older adults in China (C. Liu et al., 2025). Higher rainfall has also been linked to elevated depressive symptoms among older adults in Ireland (O'Hare et al., 2016),

and associations with wind speed, atmospheric pressure, and humidity have similarly been reported (Brazienė et al., 2022). However, findings are not always consistent. For instance, one study found no association between depression scores and cloud cover, rainfall, or atmospheric pressure (Molin et al., 1996), and another found that temperature, sunshine, and rainfall did not explain the seasonal variation in depression and sad mood in a large sample of over 14,000 Dutch participants (Huibers et al., 2010). Inconsistency has also been observed within individual studies, with one study reporting that higher daily sunlight was associated with an increased number of depression diagnoses in the short term, whereas higher average sunlight over the preceding 30 days was associated with a decreased number of diagnoses, suggesting that the association between sunlight and depression may depend on the exposure period examined (Son & Shin, 2021) Overall, current evidence suggests that associations between objective environmental conditions and depression are heterogeneous and may depend on individual and contextual factors.

A first explanation for these inconsistent findings may be that exposure to environmental conditions is not uniform across individuals. For example, the effects of temperature and sunlight on mood have been found to be stronger among individuals who spend more time outdoors, and the direction of these associations may vary across seasons (Keller et al., 2005; Kööts et al., 2011). Consistent with this, a longitudinal study in Sweden found that individuals who spent at least 1 hour per day outdoors in daylight during winter months were less likely to report depressive symptoms than those with less daylight exposure (Raza et al., 2024).

Another explanation may be that individuals differ substantially in their emotional responses to environmental conditions. For example, using repeated assessments of daily mood and objective weather data collected across 30 days, Klimstra et al. (2011) identified four distinct patterns of weather reactivity among adolescents and their mothers: Summer Lovers and Summer Haters showed opposite mood responses to sunshine, temperature, and precipitation, Rain Haters were selectively affected by precipitation alone, and Unaffected individuals showed no meaningful response to any of these conditions. Although average associations between weather conditions and mood were weak, this reflected opposing, variably specific subgroup patterns rather than a genuine absence of reactivity. Consistent with this interpretation, Zhang et al. (2025) found that the indirect relationship between environmental conditions and physical activity through depression severity was small when estimated across the whole sample, but considerably larger once participants were grouped according to their individual weather–depression association profiles. Increases in temperature and day length were associated with higher depressive symptoms in one subgroup but lower depressive symptoms in another, producing opposing indirect effects on physical activity (Zhang et al., 2025). Together, previous findings (Klimstra et al., 2011; Zhang et al., 2025) suggest that heterogeneity in environmental sensitivity

may partly explain the inconsistency of previous associations between environmental conditions and depression, as opposing individual effects can cancel out when averaged.

These individual differences in responsiveness to weather and light conditions reflect what can be called environmental sensitivity. However, in the literature, they are frequently referred to as weather sensitivity (e.g., von Mackensen et al., 2005). Self-reported weather sensitivity is common in the general population, with representative epidemiological surveys in Germany and Canada finding that 54.5% and 61% of respondents, respectively, reported that weather affected their health, with depression among the most frequently reported symptoms (von Mackensen et al., 2005). Weather sensitivity is also commonly reported among individuals with specific chronic health conditions, including osteoarthritis (L. Wang et al., 2023), fibromyalgia (Hagglund et al., 1994), and coronary artery disease (Martinaitiene & Raskauskiene, 2021), conditions that have been associated with elevated rates of depressive symptoms (Aguglia et al., 2011; Fonseca-Rodrigues et al., 2022; Rudisch & Nemeroff, 2003; Vishne et al., 2008)

Environmental sensitivity, however, has been operationalised inconsistently across studies. Some researchers define it as a general self-reported tendency to perceive weather-related symptom changes, assessed via single-item or brief questionnaires asking whether weather affects one's health in general (Martinaitiene & Raskauskiene, 2021; von Mackensen et al., 2005), whereas others quantify it as the correlation between daily symptom severity (e.g., pain) and objective weather conditions (L. Wang et al., 2023). A third approach assesses subjective emotional responses to specific weather and daylight conditions, distinguishing between adverse, favourable, and neutral mood responses to environmental factors such as cold, warmth, rainfall, or reduced daylight (Mersch et al., 2004). Using this approach, individuals diagnosed with SAD have been found to report more favourable mood responses to hot and sunny weather, and more adverse mood responses to cold and cloudy weather, than controls (Mersch et al., 2004; Sigmon et al., 2010). This condition-specific approach acknowledges that individuals may respond differently to specific weather and daylight conditions rather than exhibiting a uniform sensitivity to all environmental conditions.

1.4.2 Rationale for Paper II

The literature reviewed in Section 1.4.1 indicates that inconsistent findings regarding weather, daylight, and depression may partly reflect individual differences in environmental responsiveness, which has been operationalised inconsistently across studies and has rarely been examined alongside objective meteorological data. Moreover, previous research (Mersch et al., 2004; Sigmon et al., 2010) has focused mainly on SAD rather than depressive symptom severity itself, and has largely relied on samples from temperate latitudes, such as the

Netherlands (approximately 52°*N*) and the northeastern United States (approximately 44.5°*N*), where seasonal changes in daylight and weather tend to be closely synchronised.

Paper II of the present dissertation addresses this research gap by adopting a condition-specific approach to environmental sensitivity, examining subjective mood responses to specific weather and daylight conditions using the corresponding SPAQ items. By integrating subjective environmental sensitivity with objective environmental exposure in an Icelandic population, Paper II aims to provide a more comprehensive understanding of how each factor, independently or in combination, contributes to depressive symptom severity.

1.4.3 Chronotype and Circadian Preference

Human physiology follows clear daily cycles, such as those in core body temperature and hormonal secretion (Dibner & Schibler, 2015). While the circadian system is universal, its timing is not; it differs considerably between individuals. This variation is captured by the concept of chronotype, an individual's inherent inclination toward earlier or later sleep timing and peak alertness, independent of external constraints such as work or school schedules (Roenneberg et al., 2003). It is commonly described along a continuum ranging from morning types (also referred to as early chronotypes, or "larks"), who wake early and feel most alert earlier in the day, to evening types (late chronotypes, or "owls"), who prefer later sleep and wake times and feel most alert later in the day, with the majority of individuals falling somewhere in between as intermediate types (Horne & Östberg, 1976; Roenneberg et al., 2003).

Chronotype differs by sex, with men tending toward later chronotypes than women (Adan & Natale, 2002; Fischer et al., 2017; Randler, 2011). Cross-sectional studies also indicate that chronotype shifts later during adolescence and early adulthood before advancing progressively earlier with age (Fischer et al., 2017; Randler, 2011). At the individual level, however, chronotype is highly stable, with 63% of adults retaining their category over a six-year follow-up and morning types proving more stable (68% retained) than evening types (44% retained) (Koskenvuo et al., 2007). Another study showed that across a decade of mid-adulthood, most adults likewise displayed no change, and shifts between definite evening and morning types were very rare (0.5%) (Merikanto et al., 2024).

While environmental influences, such as social schedules, can shape the timing of sleep and wakefulness (Roenneberg et al., 2003; Walch et al., 2016), individual differences in chronotype are also substantially influenced by genetics, with twin studies estimating heritability at approximately 50% (Hur et al., 1998; Koskenvuo et al., 2007). At the biological level, circadian rhythms are not generated by the SCN alone, as individual cells throughout the body maintain self-sustained oscillations of their own, kept in phase by this central pacemaker (Dibner & Schibler, 2015), which is itself entrained to the external day primarily by light (see

Section 1.3.2). These cellular clocks rely on a feedback loop in which two proteins, CLOCK and BMAL1, activate the Period (PER1, PER2) and Cryptochrome (CRY1, CRY2) genes, and the resulting proteins accumulate and switch those same genes back off, producing a cycle of roughly 24 hours (simplified for clarity here; for a more detailed account of the molecular mechanism, see Dibner & Schibler, 2015). The near-normal distribution of chronotype in the population (Chelminski et al., 1997; Roenneberg et al., 2007) suggests, however, that its genetic basis extends beyond these core clock genes to a polygenic architecture, in which many genes each contribute a small, additive effect alongside environmental factors (Landolt & Dijk, 2017).

Several approaches exist to quantify chronotype. Some rely on direct physiological markers, such as dim-light melatonin onset (DLMO) (e.g. Burgess & Eastman, 2005), while others use behavioural proxies, such as actigraphy-derived sleep midpoints (e.g. Schneider et al., 2022). A widely used approach, especially in large-scale research, relies on self-report questionnaires, which are quick to administer and do not require specialized equipment or laboratory conditions, making them inexpensive. The most widely used instrument for this purpose is the Morningness–Eveningness Questionnaire (MEQ), developed by Horne and Östberg (1976). The MEQ consists of 19 items assessing, among other aspects, preferred sleep and wake times, timing of mental and physical activities, subjective alertness and fatigue, ease of waking, and self-perceived morningness or eveningness (Horne & Östberg, 1976). These items are combined into a composite score that classifies respondents along the morningness–eveningness continuum into five categories, ranging from definite evening through intermediate to definite morning types (Horne & Östberg, 1976). This measure is hereafter referred to as circadian preference. Although self-reported circadian preference is not synonymous with chronotype, MEQ scores were validated against circadian variation in body temperature (Horne & Östberg, 1976) and correlate with actigraphy-derived sleep timing (Schneider et al., 2022), supporting the use of MEQ as a proxy.

The relevance of assessing circadian preference extends beyond describing individual differences in sleep timing and daily activity patterns, as circadian preference has consistently been associated with a range of health and psychological outcomes. In particular, eveningness has been linked to poorer mental health, with meta-analytic evidence indicating higher levels of depressive symptoms among evening types (Au & Reece, 2017) and an elevated likelihood of insomnia symptoms (Zhao et al., 2025). Eveningness has also been associated with less favourable health behaviours, including greater alcohol use and smoking (Gariépy et al., 2019; Urbán et al., 2011; Wittmann et al., 2010), poorer diet quality (Fleig & Randler, 2009; Gariépy et al., 2019), and lower physical activity (Urbán et al., 2011).

1.4.4 Rationale for Paper III

One health behaviour associated with eveningness, and the focus of paper III, is energy drink consumption. Evening types consume energy drinks more frequently than morning types (Ágoston et al., 2019; Gariépy et al., 2019; Jason et al., 2014; Siudej & Malinowska-Borowska, 2021). Energy drinks have become one of the fastest-growing beverage categories, marketed on claims of enhanced alertness and performance (Bleakley et al., 2022; Reissig et al., 2009). Although caffeine itself can temporarily reduce fatigue and improve attention (Kløve & Petersen, 2025), there is little evidence that energy drinks confer benefits beyond this, or that their non-caffeine ingredients contribute meaningfully (Heneghan et al., 2012; McLellan & Lieberman, 2012). Their use is concentrated among adolescents and young adults (Aonso-Diego et al., 2024). Sex differences in consumption are less consistent, with some studies reporting higher use among boys (Carneiro et al., 2026; Granda et al., 2024; Silva-Maldonado et al., 2022) and others finding little difference or higher use among girls (Choi et al., 2025; Malinauskas et al., 2007). Unlike coffee, which is typically consumed early and habitually, energy drink intake tends to occur at irregular or later times of day (Benson et al., 2019; Malinauskas et al., 2007). Frequent energy drink intake has also been linked to poorer sleep quality and shorter sleep duration (Carneiro et al., 2026), as well as increased insomnia risk (Nadeem et al., 2021), with later-day consumption particularly implicated (Stefansdottir et al., 2026), as well as to elevated depressive symptoms (Azagba et al., 2014; Kaur et al., 2020; Lund-Iversen et al., 2025), although findings are not entirely consistent (Hofmeister et al., 2010) and derive largely from adolescent and student samples.

Taken together, these overlapping associations suggest that evening types may represent a group of particular interest, as they are more likely to experience disturbed sleep and low mood while also being more likely to consume energy drinks, a behavior independently associated with both outcomes. These vulnerabilities may be further amplified during autumn and winter, when reduced daylight delays circadian timing and is associated with poorer sleep and lower mood, particularly at higher latitudes (Friborg et al., 2012; Golder & Macy, 2011; Pallesen et al., 2001), as in the Icelandic setting of the present work. However, these factors have largely been examined separately. It therefore remains unclear whether the associations between eveningness and insomnia or depressive symptoms are partly explained by energy drink consumption, and whether any such associations are specific to energy drinks, which differ from coffee in both composition and patterns of use (Aranda & Morlock, 2006; Benson et al., 2019; Malinauskas et al., 2007; Reissig et al., 2009). Paper III of the present dissertation addresses this research gap by examining energy drink intake frequency, circadian preference, and season in relation to insomnia and depressive symptoms, using coffee intake as a comparison source of caffeine.

1.5 Iceland as a Natural Laboratory

The three studies comprising this dissertation were conducted in Iceland, which provides a particularly informative setting for examining environmental contributions to depressive symptoms. Located at approximately 65°*N*, Iceland exposes its population to extreme seasonal variation in daylight, ranging from approximately four hours at the winter solstice to near-continuous daylight in summer, alongside rapid and unpredictable weather changes throughout the year. This combination of pronounced photoperiod fluctuation and meteorological variability creates naturalistic conditions under which the role of these environmental factors in depressive symptoms can be examined in ways that would be difficult to replicate in more temperate regions.

1.6 Aims of the Present Dissertation

The present dissertation examined environmental and individual correlates of depressive symptoms in Iceland across three studies. Papers I and II drew on the same dataset collected from an Icelandic sample, while Paper III drew on an independent longitudinal dataset collected across four seasons.

Paper I aimed to provide a timely and accurate estimate of the prevalence of elevated seasonality and of clinically diagnosed SAD in Iceland, using a two-stage design in which participants were first screened with the SPAQ, and those with elevated seasonality were subsequently invited to complete a semi-structured diagnostic interview. This methodological approach was motivated by the possibility that reliance on screening instruments alone may have inflated prevalence estimates in prior research, potentially contributing to the latitude effect observed in SAD prevalence.

Paper II examined whether subjective mood responses to environmental conditions, objective meteorological variables, and their interaction are independently or jointly associated with depressive symptom severity in a population exposed to pronounced seasonal and meteorological variability, building on earlier work conducted in more temperate regions. Specifically, it investigated whether self-reported mood responses to temperature and daylight, objective weather variables including temperature, humidity, and wind speed, and the interaction between these subjective and objective domains each explain variability in depressive symptom severity.

Paper III investigated the associations between energy drink intake frequency, circadian preference, and season in relation to depressive and insomnia symptoms, using coffee intake as a caffeine comparison condition, across both cross-sectional and longitudinal assessments. Although each of these factors has been individually linked to sleep and mood, they have not previously been examined together, and Paper III aimed to address this gap by testing whether they interact,

such that evening types with frequent energy drink intake during the low-light seasons show the greatest symptom burden.

Taken together, the three papers address three questions that matter for understanding seasonal depressive burden: how many people are affected (Paper I), under which environmental conditions symptoms emerge (Paper II), and who is most vulnerable (Paper III). By combining these perspectives, the dissertation aims to investigate how depressive symptoms can arise from the interplay of individual and environmental factors.

2 Methods

This dissertation draws on two independent datasets from Iceland, both collected via online surveys. The first dataset (Papers I and II) was collected using a cross-sectional design with a random sample drawn from the national registry, supplemented by a convenience sample. The second dataset (Paper III) was collected using a longitudinal design, with measurements taken at four time points over one year. Comprehensive methodological details are provided in each of the three papers included in this dissertation. For an overview of the study design and data collection procedures see Figure 2.1.

2.1 Ethical Approval

Ethical approval was obtained from the National Bioethics Committee of Iceland for the research underlying this dissertation. Papers I and II fall under approval VSN-22-058, and Paper III falls under approval VSN-21-088. All research was conducted in accordance with the Declaration of Helsinki. All researchers signed non-disclosure agreements. Participants in both datasets provided written informed consent prior to participation, having received information about the study purpose, procedures, and their right to withdraw at any time without consequence.

2.2 Dataset 1: Cross-Sectional Study (Papers I and II)

2.2.1 Sampling Strategy and Rationale

A random sample of 15,000 adults was drawn from the Icelandic National Registry in May 2022. Random sampling was chosen to support prevalence estimates that could be corrected for differential response rates across demographic subgroups, as described in Paper I.

In addition to the random sample, a convenience sample was recruited via a dedicated study website and a paid Facebook advertisement, primarily to increase the number of participants with elevated seasonality available for follow-up assessment beyond the scope of this thesis. For Paper I, only participants from the randomly drawn registry sample were included because of the study‘s epidemiological purpose. Paper II, by contrast, uses the full combined sample, including convenience participants, to maximise statistical power. The two papers, therefore, draw on different analytic samples.

2.2.2 Recruitment

Recruitment took place between May 2022 and December 2024. Trained research staff contacted individuals from the random sample by phone where numbers were available, inviting them to complete an online survey. Non-responders were contacted a second time; email was used where phone contact was unsuccessful,

and social media was used where neither was possible. Postal invitations were piloted but discontinued due to low response rates. Full procedural details and a participant flow diagram are provided in Paper I.

2.2.3 Measures

Participants completed an online survey via Microsoft Forms that collected demographic information and included the SPAQ (Rosenthal, Bradt, et al., 1984) and the PHQ-9 (Kroenke et al., 2001). The GSS of the SPAQ was used to screen for elevated seasonality in Paper I, and the weather and daylight items of the SPAQ were additionally used in Paper II to assess subjective mood responses to specific environmental conditions. The PHQ-9 was used across both papers to assess depressive symptom severity. Both instruments have been validated in Icelandic samples with full psychometric details reported in the respective papers.

2.3 Dataset 2: Longitudinal Study (Paper III)

2.3.1 Design and Rationale

A prospective longitudinal design with four repeated measurement points was used, with data collection timed to each meteorologically defined season: summer (June–August), autumn (September–November), winter (December–February), and spring (March–May), with summer 2021 serving as the baseline.

2.3.2 Recruitment

Participants were recruited via email and social media, with invitations distributed to companies, organisations, and students and staff at the University of Akureyri. Follow-up invitations and phone reminders were used to support retention, and participants who completed all four assessments were entered into a draw for gift certificates.

2.3.3 Measures

At each seasonal wave, participants completed an online survey via Microsoft Forms. The instruments used in the analyses were the MEQ (Horne & Östberg, 1976), the depression subscale of the Depression Anxiety Stress Scales-21 (DASS-21; (Lovibond & Lovibond, 2011), the Bergen Insomnia Scale (BIS; Pallesen et al., 2008), and a Food Frequency Questionnaire (FFQ; Harris et al., 2007; Þorgeirsdóttir et al., 2011) assessing coffee and energy drink intake frequency. Full descriptions of each instrument, including scoring procedures and reliability estimates across the four seasonal assessments, are provided in Paper III.

2.4 General Statistical Principles

Statistical analyses across all papers were conducted in R (version 4.4.1; R Core Team, 2021) except for matching participants to registry data in Paper I, which additionally used MATLAB (The MathWorks Inc., 2024). All tests used a nominal significance threshold of $\alpha = .05$. Given the multiple comparisons across all papers, *p*-values were interpreted against a corrected significance threshold. The specific correction procedure is described in each paper.

2.5 Paper-Specific Methodological Features

While the core design and measures of each dataset are described in Sections 2.1 to 2.4, three methodological features that distinguish the papers are briefly summarised here

2.5.1 Paper I: Clinical Diagnosis and Statistical Procedures

Participants from Dataset 1 who screened positive for elevated seasonality (GSS ≥11) were invited to take part in a clinical diagnostic interview by phone. The interview used the mood disorder module of the MINI (Sheehan et al., 1998) to assess current and past depressive episodes and was supplemented with additional questions to determine whether episodes followed a seasonal pattern. This allowed SAD to be diagnosed according to DSM-IV criteria.

Since response rates to both the initial screening and the follow-up interview varied across sociodemographic strata, prevalence estimates were weighted to correct for this non-randomness. The random sample was stratified into 80 strata based on sex, region, and age group. Within each stratum, the observed number of participants with elevated seasonality was adjusted by the stratum's participation proportion, and these weighted values were summed across strata and divided by the total sample size to yield the overall prevalence estimate of elevated seasonality. An analogous procedure, additionally accounting for participation in the MINI interview within each stratum, was used to estimate the prevalence of SAD. Full mathematical details of this weighting procedure are provided in Paper I.

Associations between sociodemographic variables and seasonality in Paper I were examined using Spearman's rank correlations for continuous variables and $\chi 2$-tests for categorical variables.

2.5.2 Paper II: Weather Data and Statistical Procedures

Survey responses from Dataset 1 were linked to objective meteorological data obtained from the Icelandic Meteorological Office, matched to each participant's reported municipality and survey completion date. Weather variables, including temperature, relative humidity, and wind speed, were averaged over the 14 days preceding each participant's survey date. Because this linkage required a valid

location record, participants who did not provide residence information or who could not be matched to a weather station within 20 km were excluded from the Paper II analytic sample. Paper II used multiple linear regression, adjusting for age and sex, to examine subjective environmental mood responses and objective weather conditions as predictors of depressive symptoms, including their interaction.

2.5.3 Paper III: Longitudinal and Interaction Analyses

In addition to cross-sectional baseline analyses using multiple linear regression, Paper III used linear mixed-effects models, with a random intercept per participant to account for the repeated-measures structure of the data. These models examined associations between energy drink and coffee intake frequency and depressive and insomnia symptoms across all four seasonal waves, including interactions between beverage intake, season, and morningness-eveningness type. All models adjusted for age and gender.



Figure 2.1. Overview of the study design and data collection procedures for Datasets 1 and 2, underlying Papers I–III

3 Summary of Main Findings and Discussion

3.1 Overview of Aims and Approach

This dissertation examined environmental and individual correlates of depressive symptoms in Iceland, a high-latitude population characterised by extreme seasonal variation in daylight and rapid weather changes throughout the year. Across three papers, drawing on two independent datasets, the work moved from (1) establishing the epidemiological prevalence of elevated seasonality and SAD, to (2) examining how subjective environmental mood sensitivity and objective environmental conditions relate to depressive symptom severity, to (3) examining circadian preference and energy drink consumption as individual-level factors associated with depressive and insomnia symptoms across the seasons.

In Paper I, a methodological concern in the SAD literature was addressed, concerning the possibility that reliance on self-report screening instruments alone, without subsequent diagnostic confirmation, may have inflated prevalence estimates and contributed to inconsistent support for the latitude hypothesis. By combining SPAQ screening with semi-structured diagnostic interviews in a nationally representative random sample, a more conservative and clinically grounded estimate of SAD prevalence in Iceland was obtained.

In Paper II, it was examined whether subjective environmental mood sensitivity, objective meteorological variables, or their interaction better account for variation in depressive symptom severity. This approach addressed prior evidence that associations between weather and mood are often weak or inconsistent when estimated across whole samples, yet become more apparent once individual differences in environmental mood sensitivity are taken into account.

In Paper III, this focus on individual variation was extended by examining a different vulnerability marker, circadian preference, alongside a related health behaviour, energy drink consumption, in relation to depressive and insomnia symptoms across all four seasons. It was tested whether evening-type individuals, already known to show elevated depressive symptoms and poorer sleep, were particularly susceptible to the combined influence of season and frequent energy drink intake.

As the individual papers already present detailed results, discuss these findings in relation to the relevant literature, and address their study-specific limitations, the following sections focus primarily on the broader implications of each paper within the framework established in the dissertation's introduction. The papers are first considered individually before their relevance, limitations, and directions for future research are considered in relation to the dissertation as a whole.

3.2 Paper I: The Latitude Hypothesis Revisited

Section 1.3.5 outlined the latitude hypothesis, the proposal that SAD prevalence increases with distance from the equator due to more pronounced seasonal variation in daylight. Support for this hypothesis has been inconsistent, and one recurring source of disagreement is that studies vary considerably in how SAD is assessed, with SPAQ-based studies generally showing stronger support for a latitude gradient than studies using diagnostic interviews (K. Kim et al., 2025; Levitt & Boyle, 2002; Mersch et al., 1999; Michalak & Lam, 2002). Iceland occupies an unusual position in this literature. Despite its high latitude, Magnusson and Stefansson (1993) reported a notably low SPAQ-based SAD prevalence of only 3.8%, a finding that appears inconsistent with a straightforward latitude effect. Paper I revisited this question using a two-stage design, in which participants scoring above the SPAQ screening threshold underwent a subsequent diagnostic interview, allowing screening-based and diagnostically confirmed prevalence estimates to be examined within the same study.

Weighted SPAQ-based elevated seasonality in the present sample was 17.86% (95% CI [15.93, 19.97]), considerably higher than the earlier Icelandic estimate and exceeding SPAQ-only estimates reported from other high-latitude regions such as Alaska (5.8%; Levine, 1995) and Greenland (6.9-11.5%; Kegel et al., 2009) though differing cutoffs across studies warrant caution when interpreting these comparisons (see also Section 3.6). Considered in isolation, our screening findings are broadly consistent with the expectation that populations living at extreme latitudes experience substantial seasonal symptomatology. However, when participants with elevated GSS underwent diagnostic interview, only 2.24% (95% CI [0.69, 6.98]) met criteria for SAD, an approximately eightfold reduction that closely parallels the screening-to-diagnosis reductions reported in interview-confirmed studies conducted at lower latitudes, including Scotland (9.1% to 3.5%; Eagles et al., 1999), North Wales (5.3% to 2.4%; Michalak et al., 2001), and Canada (5.0% to 2.9%; Levitt et al., 2000). This suggests that the apparent latitude gradient observed in the SPAQ-only literature reviewed in Section 1.3.5 may be substantially attenuated, or may not hold at all, when diagnostic confirmation is incorporated, offering one possible explanation for why studies relying on different assessment methods have reached different conclusions about the latitude hypothesis.

Another possible explanation for previous inconsistent latitude findings concerns differences between vegetative and affective components of seasonal symptoms. A large Finnish study (N = 5,749) found no overall support for the latitude hypothesis, as neither the GSS nor the Beck Depression Inventory sumscore was associated with latitude (Grimaldi et al., 2009). However, additional item-level analyses in the same study revealed a more nuanced pattern, with some vegetative symptoms (i.e., sleep duration and weight and appetite variation) being more common among individuals living at northern than southern

latitudes, whereas affective symptoms (e.g., mood and social activity) did not differ by latitude (Grimaldi et al., 2009). These findings suggest that latitude may be more strongly associated with vegetative than affective seasonal symptoms. If so, future studies evaluating the latitude hypothesis may need to distinguish not only between seasonality and clinically significant SAD, but also between vegetative and affective symptoms.

The findings from Paper I also contribute to the debate outlined in Section 1.3.3 concerning whether SAD exists as a distinct clinical phenomenon at all (Hansen et al., 2008; Traffanstedt et al., 2016), or whether null findings instead reflect the limitations of cross sectional, screening based designs (Winkler et al., 2017; Young, 2017). Winkler et al. (2017) argued that because SAD symptoms are typically present for only three to four months of the year, a single, randomly timed assessment is likely to miss cases of SAD regardless of the instrument used. Paper I's two-stage design addresses part of this critique by not relying on SPAQ scores alone to establish a diagnosis. It does not, however, fully address Winkler and colleagues‘ (2017) concern because only participants who exceeded the GSS screening threshold underwent a diagnostic interview. If the initial screening measure failed to identify individuals with SAD, they would not have proceeded to the diagnostic interview, potentially resulting in an underestimate of prevalence. With this limitation in mind, the low but non-zero diagnostically confirmed prevalence observed here (2.24%) is more consistent with the interpretation that SAD is a genuine but comparatively rare condition than with the view that SAD does not exist as a distinct clinical phenomenon.

Although the diagnostic findings of Paper I suggest that clinically confirmed SAD is relatively uncommon, the SPAQ data highlight a broader issue that extends beyond the latitude hypothesis, namely that seasonal symptoms themselves are widespread. In Paper I, nearly one in five participants (i.e., 17.86%) reported elevated seasonality overall, with mood (17.27%), energy level (11.85%), and sleep (9.97%) being the most commonly affected domains, followed by social activity (8.45%), appetite (4.77%), and weight (3.97%). In addition, 87.12% of respondents reported feeling worst during late autumn and winter. The substantial discrepancy between elevated seasonality and diagnostically confirmed SAD observed in the present study suggests that focusing only on diagnosable SAD may underestimate the extent and broader public health relevance of seasonal symptoms.

The demographic correlates of elevated seasonality provide further context for this finding. The GSS was significantly higher among women, younger individuals, those not working full time, and those without a partner or children. This pattern overlaps with established demographic risk factors for depression more broadly (Amiri, 2022; Kuehner, 2017; Zisook et al., 2007; Zuelke et al., 2018; see Section 1.1.3), suggesting that elevated seasonality may be more common among groups already at greater risk of depression.

3.3 Paper II: Individual Variation in Environmental Mood Responses

Section 1.4.1 highlighted that individuals may differ not only in the strength but also in the direction of their depressive symptom response to the same environmental conditions, with opposing subgroup patterns potentially cancelling out at the population level (Klimstra et al., 2011; Zhang et al., 2025). Such heterogeneity in individual responses to environmental conditions may help explain the inconsistent associations reported between objective environmental conditions and depressive symptoms across previous studies (Brazienė et al., 2022; Huibers et al., 2010; Molin et al., 1996; O'Hare et al., 2016; Son & Shin, 2021). However, environmental sensitivity has been operationalised inconsistently across studies (Martinaitiene & Raskauskiene, 2021; Mersch et al., 2004; von Mackensen et al., 2005; L. Wang et al., 2023). The findings of Paper II suggest that assessing mood responses to specific weather and daylight conditions can provide important insights into individual differences in depressive symptom severity, as environmental sensitivity appears to vary across conditions rather than reflecting a uniform response. More adverse mood responses to cold weather and reduced daylight were associated with higher depressive symptom severity. In contrast, more adverse responses to dry weather were associated with lower depressive symptom severity. Although unexpected, this latter finding may reflect variation in how individuals experienced and interpreted dry weather conditions (i.e., dry cold versus dry heat). No significant associations were observed for the remaining subjective mood responses or for objective measures of temperature, humidity, and wind speed after correction for multiple comparisons.

A key finding of Paper II was the interaction between subjective mood responses to cold weather and objectively measured temperature. Temperature was not uniformly associated with depressive symptoms across the sample; rather, the association depended on how individuals reported responding to cold conditions. Among individuals with adverse mood responses to cold, higher temperatures were associated with fewer depressive symptoms, whereas among those with favourable mood responses to cold, higher temperatures were associated with more depressive symptoms. Taken together, the absence of an overall association between temperature and depressive symptoms in this study may reflect the presence of opposing associations among individuals with different environmental mood responses. This interpretation is consistent with previous work highlighting individual heterogeneity and potential cancelling-out processes (Klimstra et al., 2011; Zhang et al., 2025) and suggests that variability in environmental responses may be one factor contributing to inconsistent associations between objective environmental conditions and depressive symptoms across previous studies. Comparable patterns have also been reported beyond depressive symptoms. In a large cohort of approximately 6000 individuals with chronic pain, temperature showed only a modest overall association with

pain severity, but this reflected opposing subgroup patterns, with some individuals experiencing greater pain at lower temperatures and others at higher temperatures (Yimer et al., 2022). This suggests that heterogeneous, individual-level responses to environmental conditions may be a more general phenomenon, extending beyond depressive symptoms to other health outcomes.

Overall, Paper II suggests that associations between environmental conditions and depressive symptoms are unlikely to be fully understood by examining objective exposures alone. Instead, individual differences in environmental mood responses appear to shape the relationship between specific environmental conditions and depressive symptoms. These findings provide one possible explanation for inconsistent weather–depression associations in previous research and highlight the importance of considering both environmental exposures and individual responses when investigating the relationship between environment and depressive symptoms.

3.4 Paper III: Converging Vulnerabilities in Evening-Type Individuals

Sections 1.4.3 and 1.4.4 highlighted that eveningness, a circadian preference for later sleep and activity, is consistently associated with higher depressive and insomnia symptoms (Au & Reece, 2017; Zhao et al., 2025) and less favourable health behaviours, including more frequent energy drink consumption (Ágoston et al., 2019; Siudej & Malinowska-Borowska, 2021), which has itself been associated with poorer sleep and elevated depressive symptoms (Azagba et al., 2014; Carneiro et al., 2026; Kaur et al., 2020; Nadeem et al., 2021). However, these overlapping associations have largely been examined separately. Paper III addressed this gap by examining whether the associations between energy drink and coffee intake frequency and depressive and insomnia symptoms varied by both circadian preference and season.

The findings provide further support for circadian preference as an important correlate of mental health. At baseline in summer, evening types reported higher depressive and insomnia symptoms than both intermediate and morning types, suggesting that eveningness represents more than a preference for later sleep and wake timing. Energy drink intake frequency was also associated with greater insomnia severity at this stage, whereas coffee intake frequency showed no comparable association. This distinction aligns with evidence from Ágoston and colleagues (2019), who argued that caffeine-containing beverages should not be considered interchangeable. In their study, total caffeine intake was not associated with circadian preference, whereas analyses of individual beverages showed associations between eveningness and energy drink and cola consumption, with coffee consumption showing no association (Ágoston et al., 2019). Furthermore, energy drink consumption mediated the association between eveningness and

caffeine use disorder (Ágoston et al., 2019). As discussed in Section 1.4.4, one possible explanation is that coffee and energy drinks may differ in their timing and context of consumption. Coffee is often consumed habitually and earlier in the day, whereas energy drinks may be consumed more irregularly or later in the day (Benson et al., 2019; Malinauskas et al., 2007), potentially increasing their impact on sleep.

In the present study, however, neither beverage was associated with depressive symptoms at baseline. This suggests that the relationship between caffeine-containing beverages and mood may differ from their relationship with insomnia symptoms. While energy drink consumption may contribute to insomnia symptoms through sleep disruption, associations with depressive symptoms may depend on aspects of consumption not captured by a cross-sectional measure of recent intake frequency alone. Although the present study assessed energy drink intake frequency over the previous three months, this measure did not indicate whether the pattern reflected a stable habitual behaviour or a recent change in consumption (e.g., increased or decreased intake during periods of stress or poor sleep).

The longitudinal analyses suggested that the relationship between energy drink intake and depressive symptoms was context dependent. Although no baseline association with depressive symptoms was observed, a positive association between energy drink intake frequency and depressive symptoms emerged in autumn, indicating that this relationship became stronger during the transition from summer to autumn. This seasonal increase was attenuated among intermediate and morning types compared with evening types, suggesting that eveningness may increase susceptibility to the effects of energy drink consumption during periods of seasonal change. Coffee intake frequency showed no comparable seasonal association with depressive symptoms. Instead, depressive symptoms increased from summer to autumn overall, with this seasonal increase being weaker among intermediate types. No similar seasonal or circadian preference-related pattern was observed for insomnia, indicating that this contextual sensitivity was specific to the association between energy drink intake and depressive symptoms rather than reflecting a general seasonal effect on all symptoms.

Together, these findings suggest that circadian preference contributes not only to overall vulnerability to depressive and insomnia symptoms but also to the conditions under which energy drink consumption may become relevant. The fact that the association between energy drink intake and depressive symptoms emerged specifically in autumn, rather than emerging in winter, suggests that it may be the rate of change in daylight, rather than the absolute amount of daylight, that is most relevant. Daylight decreases at its fastest rate of the year around the autumn equinox, pointing to circadian adjustment demands rather than sustained low-light exposure itself as a plausible explanation for this seasonal pattern. This

rate of change is particularly pronounced in Iceland, where daylight ranges from near-continuous in summer to as little as four hours around the winter solstice (see Section 1.5), and may pose a greater challenge for evening types, whose endogenous rhythms are already more likely to be misaligned with social schedules.

3.5 Relevance and Broader Implications

Beyond each paper's contribution to specific debates in the SAD, environment–depression, and chronotype literatures, the combined findings of this dissertation may also contribute to understanding how climatic and seasonal environmental conditions shape depressive symptoms, a question that is becoming increasingly important as the climate changes.

While excessive heat has been proposed as a contributing factor to the summer pattern of SAD (Morrissey et al., 1996), little is known about whether rising global temperatures are associated with increasing prevalence of the summer pattern of SAD over time. Nevertheless, accumulating evidence suggests that elevated temperatures are associated with poorer mental health outcomes (Mullins & White, 2019; Nori-Sarma et al., 2022; F. Wang et al., 2026). For example, periods of extreme heat have been associated with increased emergency department visits across several mental health conditions, including mood disorders (Mullins & White, 2019; Nori-Sarma et al., 2022). Likewise, summer temperatures above the seasonal average have been associated with greater depressive symptoms (F. Wang et al., 2026). Climate change may also affect environmental conditions relevant to the development of winter-pattern SAD. As temperatures rise, precipitation increasingly falls as rain instead of snow (Trenberth, 2011). This could matter for winter light levels, since snow has a substantially higher albedo, the fraction of incident sunlight a surface reflects, than snow-free tundra (Coakley, 2003). Consequently, reduced snow cover during the low-light winter period could diminish one of the few natural sources of additional ambient light during the darkest months, particularly in high-latitude regions, thereby increasing vulnerability to winter-pattern SAD.

Importantly, the environmental consequences of climate change are unlikely to be uniform across regions. Iceland represents a particularly interesting case, as climate-related environmental changes may not simply reflect gradual warming. Iceland is located in a region of the North Atlantic strongly influenced by the Atlantic Meridional Overturning Circulation (AMOC), a major ocean circulation system that redistributes heat from lower latitudes (Johns et al., 2011). Through its connection with the Gulf Stream, the AMOC helps maintain Iceland's relatively mild oceanic climate and reduces the annual temperature range compared with other locations at similar latitudes.

Yet the circulation system that contributes to Iceland's mild climate may not remain stable. Recent observations have provided evidence consistent with changes in AMOC strength, including a persistent "cold blob" south of Greenland and Iceland, a region that has cooled even as much of the globe has warmed since the nineteenth century (Rahmstorf et al., 2026). Future weakening of the AMOC has been linked to continued climate change, partly through increased freshwater input from Greenland ice melt, potentially leading to more severe winters and more frequent, intense storms across the North Atlantic and northwestern Europe (Bakker et al., 2016; Drijfhout et al., 2025; Van Westen et al., 2024; van Westen & Baatsen, 2025). Using statistical early-warning signals derived from sea surface temperature records, the estimated timing of a potential AMOC tipping point has been placed between 2057 and 2065 (95% CI: 2037–2109), suggesting that substantial weakening could occur within this century, although the timing remains uncertain (Ditlevsen & Ditlevsen, 2023). These changes could influence environmental conditions relevant to depressive symptoms in ways that are not yet fully understood, underscoring the importance of considering not simply warmer temperatures in isolation but how multiple changing conditions, including daylight, snow cover, temperature, and weather variability, interact with individual vulnerability.

The findings of this dissertation contribute to understanding the relationship between (seasonal) environmental conditions and depressive symptoms. In particular, the three papers address questions that may become increasingly important as climate change alters seasonal environmental conditions: how many people are affected, under which specific environmental conditions symptoms emerge, and who is most vulnerable. Paper I addressed the first question, showing that while diagnostically confirmed SAD is comparatively rare, seasonal shifts in symptoms are far more widespread, affecting close to one in five people in the Icelandic population. Paper II addressed the second question, demonstrating that the relationship between environmental conditions and depressive symptoms is shaped by individuals' subjective mood responses to those conditions. Paper III extended this individual-differences perspective by examining circadian preference as a further vulnerability marker, showing that the association between energy drink intake frequency and depressive symptoms was observed only among evening types and only during a specific seasonal transition (summer to autumn). Overall, the findings of this dissertation suggest that the associations between seasonal environmental conditions and depressive symptoms are not uniform across individuals. Rather, vulnerability appears to emerge through the interaction of specific seasonal environmental conditions and individual characteristics. As climate change alters not only average temperatures but also broader environmental conditions, understanding these interactions may become increasingly important for identifying populations at greater risk and informing future approaches to prevention and support.

If elevated seasonality is common but diagnosable SAD is relatively rare, approaches that address common seasonal symptoms in the general population may complement support for individuals with SAD. As discussed in Section 1.3.2, bright light therapy, also known as phototherapy, is well established as an effective treatment for winter-pattern SAD and may represent a promising approach for addressing elevated seasonality. Given that elevated seasonality often reflects atypical symptoms, such as increased sleep, appetite, and low energy, interventions that specifically target these symptoms may be particularly relevant. In one early study, individuals with subsyndromal SAD (i.e. elevated seasonality) responded favourably to bright light exposure, whereas individuals with no seasonality symptoms, exposed to the same light treatment, showed no such response (Kasper et al., 1989). Combining bright light exposure with physical exercise may further extend these benefits, an effect that does not appear to depend on a prior history of seasonal symptoms. Evidence from randomized research conducted among working-age volunteers recruited through Finnish occupational health services suggests that physical exercise combined with morning bright light exposure produces greater improvement than exercise alone, particularly for atypical symptoms such as carbohydrate craving and prolonged sleep (Leppämäki et al., 2002a, 2002b; Partonen et al., 1998).

Because mood responses to cold weather and reduced daylight exposure were associated with depressive symptom severity, interventions could be targeted specifically at these conditions. One potentially relevant target is outdoor physical activity, as both daylight exposure (Burns et al., 2021) and physical exercise (Singh et al., 2023) are independently associated with improved mood. However, this presents a practical challenge because the very conditions associated with poorer mood, such as cold and reduced daylight, may also make outdoor activity less appealing or accessible. A recent survey of older adults across the Nordic Arctic, including Iceland, found that icy or slush-covered surfaces, rainfall, and darkness were among the most reported barriers to outdoor mobility (Emelyanova et al., 2026). For individuals whose mood is especially sensitive to cold or darkness, encouraging indoor social or physical activities on days when these conditions are expected, for example, by checking weather forecasts in advance and planning accordingly, may therefore be a more realistic and effective strategy than promoting outdoor activity indiscriminately. These results raise broader questions about how changing environmental conditions may influence the feasibility and effectiveness of interventions targeting seasonal vulnerability. Although the present dissertation did not find a significant association between adverse mood responses to heat and depressive symptom severity, this may reflect Iceland's specific climate. As temperatures continue to rise in many parts of the world, sensitivity to heat may become a more important consideration for future intervention strategies. These changing conditions also highlight the potential role of the built environment in supporting physical activity across a range of weather

conditions. Designing environments that reduce weather-related barriers to physical activity may help maintain opportunities for movement even under adverse conditions.

Given that circadian preference was associated with symptom severity and appeared to shape how the association between energy drink intake and depressive symptoms varied by season, interventions targeting circadian alignment may represent another potential strategy for reducing seasonal vulnerability. An example of this approach comes from a randomized controlled trial in which evening-type individuals followed a three-week intervention targeting earlier sleep and wake times, increased morning light exposure, earlier and more regular meal timing, restricted afternoon caffeine intake, and where applicable, exercise scheduled in the morning (Facer-Childs et al., 2019). This intervention produced an approximately 2-hour circadian phase advance, as verified by actigraphy and biological markers, including DLMO. Depression and stress scores fell significantly following the intervention, and this improvement occurred without any corresponding reduction in sleep duration (Facer-Childs et al., 2019). Although the intervention combined multiple behavioural targets and therefore cannot determine the specific contribution of each component, it demonstrates that circadian timing can be shifted through coordinated changes to daily routines. Among these behavioural targets, meal timing may be particularly relevant because it represents a non-light cue that can influence circadian regulation. While the central circadian pacemaker in the SCN is primarily synchronised by light exposure, peripheral clocks located in organs such as the liver, pancreas, and gastrointestinal tract are also influenced by behavioural cues, including the timing of food intake (Cagampang & Bruce, 2012). Adjusting meal timing could therefore be a potential route to influence circadian alignment without relying solely on light exposure, particularly for evening-type individuals whose internal timing may be misaligned with social and environmental schedules. This suggests that relatively simple behavioural adjustments may offer one avenue for supporting circadian alignment and reducing symptom burden among individuals whose vulnerability appears to depend on circadian preference.

Taken together, these findings suggest that addressing seasonal vulnerability may require approaches that move beyond uniform recommendations and instead consider how specific environmental exposures interact with individual characteristics and behavioural patterns. As climate change alters the conditions under which these vulnerabilities may be expressed, identifying these interactions will become increasingly important for developing targeted, context-sensitive approaches to prevention and support.

3.6 Limitations

A key limitation of this dissertation is the observational nature of the included studies. Except for Paper III, which employed a longitudinal design, the studies were cross-sectional, meaning that the direction of the identified associations cannot be firmly established. For example, depressive symptoms may alter how individuals perceive and report their sensitivity to environmental changes, as well as influence sleep timing and health behaviours, such as energy drink consumption, rather than these factors solely contributing to depressive symptoms. Although the longitudinal design of Paper III provides some evidence regarding temporal precedence, it remained observational in that it did not involve active intervention or manipulation. Therefore, causal relationships cannot be conclusively established, even within this design.

Beyond the observational design, a further limitation is that all three papers relied on results of self-report measures, which are open to several biases. While the SPAQ allows seasonal symptoms to be assessed efficiently in large samples without assuming that seasonal changes will manifest in a particular direction, it relies on retrospective self-report, which may introduce recall bias, as respondents must reconstruct the whole year and may be influenced by particularly salient or recent episodes (Horwitz et al., 2023). In addition, its items directly reference seasonal variation, which may prompt respondents to answer based on their own expectations about how the seasons affect them rather than their actual symptom experience (Øverland et al., 2020). Furthermore, although participants were randomly sampled, individuals who perceive their mood as seasonal may have been more inclined to respond to a study on seasonality, potentially leading to an overestimation of the prevalence of elevated seasonality. Together with recall and expectation effects, this may have affected the reported seasonality in Paper I, although the magnitude of these effects cannot be estimated from the present data.

Differences in cutoff criteria used to classify elevated seasonality may further limit comparability across studies and contribute to variation in reported prevalence estimates. The sensitivity and specificity of the SPAQ relative to clinical diagnosis of SAD also remain debated, with estimates varying across populations. In an Icelandic sample, Magnusson (1996) reported high sensitivity (94%) but lower specificity (73%), whereas Mersch et al. (2004) reported high specificity (94%) but limited sensitivity (44%) in another population. The limited sensitivity reported by the latter study may be particularly relevant for Paper I, as it suggests that some individuals with SAD may have been missed during screening, given that participants were recruited continuously across seasons rather than exclusively during periods when seasonal symptoms are typically most pronounced. The use of the SPAQ may also have influenced the difference observed in Paper I between screening-based and diagnostically confirmed prevalence, as more diagnostically oriented instruments, such as the SHQ (Thompson & Cowan, 2001; see Section 1.3.4), might have produced closer

estimates. Nevertheless, the SPAQ was selected because it is the most widely used instrument in SAD research (Akkaoui & Geoffroy, 2025) and allows efficient assessment in large samples.

A separate limitation is that not all participants with elevated seasonality completed the subsequent diagnostic interview, and systematic differences between those who did and those who did not participate cannot be ruled out. For example, individuals who did not complete the interview may have been experiencing more severe depressive symptoms, which could have affected their likelihood of participation. However, PHQ-9 scores assessed at screening did not differ significantly between interview completers and non-completers. Given the time interval between screening and the diagnostic interview, depressive symptom severity may nevertheless have differed by the time participants were invited to the interview.

Although the MEQ is a widely used and validated measure of circadian preference (see Section 1.4.3), it provides a less direct assessment of circadian timing than objective biological markers such as DLMO or actigraphy-derived measures. Likewise, the assessment of environmental exposure in Papers II and III relied on season and meteorological variables rather than participants' actual light exposure. Individual differences in time spent outdoors, occupational schedules, and use of artificial lighting may result in substantial variation in light exposure despite similar environmental conditions. Future studies combining questionnaire-based measures with objective assessments, including DLMO, actigraphy, and personal light exposure monitoring, would provide a more comprehensive characterization of environmental and circadian influences on mental health.

Finally, the findings should be interpreted in light of the Icelandic setting, which may limit their generalizability to other populations. Iceland's high latitude produces substantial seasonal variation in daylight, while the North Atlantic Current moderates its climate, resulting in relatively mild temperatures and a narrower annual temperature range than might otherwise be expected. Consequently, the observed relationships may reflect the combination of pronounced daylight variation and relatively moderate temperatures despite substantial weather variability, which may not generalize to other high-latitude regions. Iceland's demographic and built-environment characteristics may also influence how individuals experience seasonal changes. Iceland's relatively small population, at approximately 396,500 inhabitants (Statistics Iceland, 2026), may limit the generalizability of these findings to larger and more diverse populations. Additionally, approximately 94% of the population lives in urban areas, with nearly two-thirds residing in the Greater Reykjavík region (Statistics Iceland, 2025). This urban context may reduce direct exposure to adverse weather conditions, as features such as well-insulated buildings, widespread heating systems, reliable transportation, and maintained urban infrastructure may buffer

the effects of seasonal environmental variability. Moreover, rural residents may experience greater direct exposure to seasonal and meteorological variation, as they are more likely to work in weather-dependent sectors such as agriculture and fishing and to spend more time engaged in outdoor activities than urban residents (Matz et al., 2015). Seasonal experiences are shaped by cultural norms and the meanings attached to different times of year. In Iceland, winter is associated with established traditions and forms of social engagement, including the Christmas season and New Year celebrations, which facilitate social contact during periods of reduced daylight. Notably, these traditions are concentrated in December. This is consistent with the findings of Paper I, which showed that participants most commonly reported feeling worst and reducing social activity during January and February rather than November and December, after the main period of holiday celebrations and social gatherings had ended.

3.7 Future Directions

One important direction for future research is to examine whether differences in SAD prevalence across countries spanning different latitudes reflect true epidemiological variation or methodological differences. Applying the same two-stage screening-and-interview design and cutoff criterion as in Paper I would address the limitation that the case for an assessment-method explanation currently rests largely on comparisons across separate studies, where differences in sampling, instruments, cutoff criteria, and diagnostic procedures may confound interpretation. By controlling for these methodological differences, this approach could determine whether apparent latitude effects reflect genuine epidemiological variation or methodological artifacts.

Another important direction concerns the generalizability of environmental sensitivity findings across different climates. The findings of Paper II may not generalize beyond the specific environmental conditions present in Iceland. For example, the observed association between subjective and objective cold-weather exposure could reflect a genuine relationship, or it could instead result from cold temperature being confounded with daylight availability, since both decrease together across the Icelandic winter. In climates where these variables do not covary in the same way, such as equatorial regions where daylight length remains nearly constant year-round, or regions with wet and dry seasons rather than four distinct seasons, this relationship may not hold. Future research applying the same condition-specific mood response measures across countries with different climates would help clarify whether this interaction reflects a generalizable pattern of environmental sensitivity or is limited to Iceland's specific environmental context.

A further direction involves moving beyond environmental exposures alone to identify the individual characteristics that determine who is most affected by

them. The findings of Paper III suggest that such characteristics may influence both overall vulnerability to depressive symptoms and the conditions under which specific behavioural and environmental factors become relevant. Extending this individual-differences perspective, Hjordt and Stenbæk (2019) found that trait Sensory Processing Sensitivity, assessed during symptom-free periods in summer, predicted the severity of depressive symptoms among individuals with SAD the following winter, suggesting that heightened sensitivity to environmental and internal stimuli may represent a vulnerability marker for more severe seasonal mood disturbance. Beyond psychological traits, future research could also investigate neurobiological markers that may contribute to differences in seasonal vulnerability. Electroencephalography (EEG) is a potential approach, as EEG measures have been widely studied in relation to depressive disorders (de Aguiar Neto & Rosa, 2019) and have also been shown to be relevant to seasonal symptom patterns (Passynkova & Volf, 2001; Theódórsdóttir & Höller, 2023). Previous research has identified differences in neural activity among individuals with elevated seasonality, including alterations in frequency-band activity and asymmetry (Passynkova & Volf, 2001). Integrating EEG measures with assessments of seasonality, chronotype, and psychological vulnerability factors could help clarify the neural correlates of seasonal vulnerability and why individuals exposed to comparable seasonal environmental conditions differ in their psychological responses.

Finally, the findings of this dissertation align with recent calls to recognize seasonal timing as an important source of variation in psychological phenomena, rather than a consideration limited to SAD (Hohm et al., 2024). Across the three papers, the findings highlight the importance of considering both individual characteristics and the seasonal context in which psychological processes are examined. Yet, definitions of "season" vary considerably across studies and are often left unspecified altogether (Øverland et al., 2020). Future studies should therefore routinely report not only the season but also the specific time-period definitions used to operationalize it (e.g., calendar months or solstice-based boundaries). Where feasible, studies should incorporate designs spanning multiple seasons to test whether psychological outcomes vary across time. In addition, despite its limitations, brief measures such as the SPAQ, including its weather and daylight items, could help characterize individual differences in seasonality and clarify when seasonal timing may influence psychological outcomes.

4 Conclusion

This dissertation examined how seasonal and environmental conditions relate to depressive symptoms in Iceland, moving from population-level prevalence to individual variation in environmental sensitivity and circadian preference. Taken together, the three papers suggest that clinically diagnosable SAD is comparatively rare, but that seasonal shifts in symptoms are common; that environmental influences on mood depend on individual sensitivity rather than exposure alone; and that the relevance of behavioural risk factors, such as energy drink consumption, for depressive symptoms can depend on circadian preference and season. These findings indicate that the relationship between seasonal environmental conditions and depressive symptoms is not uniform across individuals. As climate change continues to alter environmental conditions, understanding these interactions will become increasingly relevant for identifying those most at risk and for developing targeted approaches to prevention and support.

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Research paper

Prevalence of seasonal affective disorder and seasonality of mood and behaviour in Iceland – Methodological implications for revisiting the latitude hypothesis

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ABSTRACT

Background: **Seasonal Affective Disorder (SAD)** is thought to be more prevalent at higher latitudes, yet early research in Iceland reported a surprisingly low prevalence. Prior studies were based on markedly different methodological approaches. We provide an update on prevalence of SAD and seasonal fluctuations in Iceland and propose that distinction between these two conditions is crucial to clarify the relation between prevalence and latitude.

Methods: We randomly selected a representative sample of 15,000 Icelandic citizens from the population registry. Among them, 9319 could be contacted and invited to complete the Seasonal Pattern Assessment Questionnaire (SPAQ). Participants with high Global Seasonality Score (GSS $\geq$ 11) were invited to complete a semi-structured clinical interview via phone to obtain a clinical diagnosis of SAD.

Results: Among 1384 participants (response rate = 14.85 %) the weighted prevalence of elevated seasonality was 17.86 %. Seasonality was more pronounced among younger individuals, women, students, unemployed individuals, single participants, and those without children. It correlated moderately with depressive symptoms as assessed with the Patient Health Questionnaire. Of the high scorers, 111 completed the clinical interview, among which 18 met diagnostic criteria for SAD. This resulted in a weighted prevalence of 2.24 %.

Limitations: Response rates were rather low. Only a subset of participants with elevated seasonality completed the clinical interview.

Conclusions: SAD remains relatively rare in Iceland, but seasonal mood and behaviour fluctuations are as common as in other areas at high latitude, underscoring the need to distinguish between subjective seasonality and clinical SAD.

1. Introduction

Seasonal Affective Disorder (SAD) is a clinical condition characterized by a seasonal pattern of recurrent depressive episodes, typically emerging in the fall or winter and fully remitted in the spring or summer (Rosenthal et al., 1984). Risk factors include young age (Höller et al., 2021; Rastad et al., 2005), being a woman (Jepson et al., 1999), and a family history of depression (Allen et al., 1993). In the current Diagnostic and Statistical Manual of Mental Disorders (i.e., DSM-5-TR), SAD is not classified as its own diagnostic entity but instead a "seasonal pattern" specifier is applied to either recurrent major depressive disorder or bipolar disorder (American Psychiatric Association [APA], 2022). The primary diagnostic criteria include experiencing major depressive episodes that have a seasonal pattern for at least two consecutive years, and the seasonal depressive episodes must significantly outnumber any non-seasonal depressive episodes experienced over the individual's lifetime (APA, 2022).

Beyond clinically diagnosed SAD, seasonal changes also produce fluctuations in mood and behaviour across the general population (Kasper et al., 1989). These non-clinical seasonal variations are

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commonly measured with self-report instruments such as the Seasonal Pattern Assessment Questionnaire (SPAQ) (Rosenthal, 1984). The SPAQ measures changes across multiple domains including mood, energy, social activity, sleep, appetite, and weight, producing a Global Seasonality Score (GSS), with scores of 11 or higher indicating elevated seasonality, which can be an indicator for probable SAD (Rosenthal, 1984; Kasper et al., 1989; Melrose, 2015).

A long-standing hypothesis suggests that SAD and elevated seasonality become more prevalent at higher latitudes (e.g., Rosenthal et al., 1984; Potkin et al., 1986; Kegel et al., 2009). A comprehensive review of epidemiological studies reported prevalence rates from 0 % to 9.7 % (Magnusson, 2000) with higher rates more commonly observed at northern latitudes (Levine, 1995; Konradsen, 1995; Kegel et al., 2009). Yet findings from Iceland challenge this latitude hypothesis as an epidemiological study from the early 1990s reported a surprisingly low SPAQ-derived elevated seasonality prevalence of just 3.8 % (Magnusson and Stefansson, 1993). Similarly, a prevalence of 1.2 % was found among descendants of Icelandic emigrants in Canada, leading to speculation about possible genetic adaptations (Magnússon and Axelsson, 1993).

These differences raise broader questions about methodology in epidemiological studies, particularly the reliance on the SPAQ as the sole assessment tool (Mersch et al., 1999a; Eagles et al., 1999; Magnusson and Partonen, 2005). While the SPAQ is effective in accurately identifying those without SAD, it was found to frequently overestimate the prevalence of SAD (Blazer et al., 1994; Michalak et al., 2001; Levitt et al., 1997, 2000). For instance, several studies reviewed by Magnusson (2000), as well as the study by Kegel et al. (2009), relied exclusively on SPAQ screening without clinical verification. In contrast, studies combining SPAQ screening with structured clinical interviews, reported lower and likely more accurate prevalence rates; in Scotland estimates decreased from 9.1 % to 3.5 % (Eagles et al., 1999), and in Canada from 5 % to 2.9 % (Levitt et al., 2000). Screening tools, such as the SPAQ, rely on subjective evaluations and cannot assess impairment or diagnostic criteria rigorously. Therefore, they should be complemented by objective and standardized diagnostic interviews that are based on formal diagnostic criteria (Meesters and Gordijn, 2016; Mersch et al., 2004).

Crucially, there is a significant conceptual difference between seasonal fluctuations in mood and behaviour as measured by the SPAQ and clinically significant SAD. While the DSM focuses strictly on diagnosable recurrent depressive episodes, the SPAQ assesses a broader range of seasonal symptom patterns across several domains, all weighed equally in the GSS. Thus, in line with the dual vulnerability model (Young, 1999), an individual may for example show substantial seasonal variation in energy or appetite without meeting the threshold for a depressive episode. Consequently, elevated seasonality represents a broader construct that can consist largely of vegetative changes in the absence of mood complaints (Young, 1999). This discrepancy might explain inconsistent findings related to overall prevalence of SAD and the latitude hypothesis, which several studies have failed to confirm (Levitt and Boyle, 2002; Brancaleoni et al., 2009; Mersch et al., 1999a). We could speculate that observed geographic variation may reflect methodological inconsistencies rather than real epidemiological trends.

Iceland represents an excellent case study to challenge the latitude hypothesis. In Iceland, there is currently no estimate of SAD prevalence based on clinical interviews. While earlier data suggest a low prevalence of elevated seasonality (Magnusson and Stefansson, 1993), a more recent convenience sample with $N = 402$ participants in Iceland found that over 10 % of participants had a GSS of 11 or higher (Höller et al., 2021).

The present study aimed at providing a timely and accurate estimate of the prevalence of elevated seasonality, as assessed by the SPAQ, as well as the prevalence of clinically diagnosed SAD in Iceland. This data can add to a better understanding of potential methodological bias that gave rise to the latitude hypothesis.

2. Methods

2.1. Ethical approval

The National Bioethics Committee of Iceland approved the study (VSN-22-058, 22.03.2022), which adhered to the Declaration of Helsinki. All researchers signed a non-disclosure agreement, and participants gave written informed consent prior to participation.

2.2. Random sample population

A random sample of 15,000 residents of Iceland was drawn from the Icelandic National Registry in May 2022. Information on full name, home address including region of Iceland, age, and biological sex was provided for the whole sample. The random sample had an almost perfectly balanced proportion of women ($N = 7501$) and men ($N = 7499$) and a representative dispersion of age ranging from 18 to 65 years ($M = 40.66$, $SD = 13.29$), divided into five groups: 18–30, 31–40, 41–50, 51–60, and 61–65 years.

The random sample had a representative dispersion of residence of all eight regions of Iceland with the majority of the sample located in the Capital region (66.43 %), followed by the Northeastern region (7.49 %), Southern region (7.17 %) and Southern peninsula (7.17 %), Western region (4.46 %), Eastern region (3.43 %), Northwestern region (2 %), and the Westfjords (1.86 %).

See Table S1 for demographic breakdowns by age group, sex, and region for the total sample.

2.2.1. Contactable portion of random sample population

Phone numbers were available for 58.21 % of the random sample, and email addresses for 5.15 %. All individuals with an email address also had a phone number.

In total, 9319 individuals were contacted, including 8731 via available contact information and 588 via social media (see Section 2.3). The contacted sample differed significantly from the total random sample by sex ($\chi^2(1, N = 9319) = 42.63$, $p < .001$), age group ($\chi^2(4, N = 9319) = 412.53$, $p < .001$) and residence region ($\chi^2(7, N = 9319) = 62.88$, $p < .001$). Response rates and chi-square tests by age group, sex, and region are presented in Table S2.

2.3. Participant recruitment and filtering

Participant recruitment occurred between May 2022 and December 2024. Individuals from the random sample were contacted by trained research staff via phone when numbers were available. If reached, participants were invited to complete an online survey and asked whether they preferred the link via email or text message. Those without access to an electronic device were offered to complete the survey by phone, with the caller recording their responses. Non-responders to the initial call were contacted a second time. If an email address was available but phone calls were unsuccessful, an email with study details and the survey link was sent. When neither phone nor email contact was possible, participants were sought via social media (Facebook and/or Instagram). Postal invitations were tested among 200 individuals but discontinued due to low response rates.

In connection with the present epidemiologic study, a longitudinal study was conducted to recruit a larger sample of individuals with elevated seasonality. Recruitment for the longitudinal study was conducted via a link on the study webpage and social media, including a paid Facebook advertisement between November 2023 and March 2024. A filtering procedure was applied before including participants in the epidemiologic sample. Specifically, a participant was retained only if at least three self-reported details (e.g., name, age, and address) matched registry data. Participants who could not be matched to the randomly drawn sample based on this criterion were excluded from prevalence estimates.

A flowchart that illustrates the recruitment and sampling process of the study can be found in Fig. 1.

2.4. Measurements

2.4.1. Survey instruments

The online survey was administered via Microsoft Forms and included the following items, with mandatory questions marked by an asterisk: informed consent agreement*, full name, home address, town*, e-mail address, phone number, gender identity, age, educational level, occupational status, relationship status, whether they have kids, whether they live in Iceland, whether they speak Icelandic fluently, and whether they were born in Iceland. The survey also included the Icelandic version of the SPAQ (Magnusson, 1996) and the Patient Health Questionnaire-9 (PHQ-9; Icelandic version described in Kristófersdóttir et al., 2023).

2.4.2. Seasonal pattern assessment questionnaire

We screened for elevated seasonality using the SPAQ (Rosenthal, 1984). The questionnaire includes a six-item self-report scale for characterizing seasonal patterns in sleep length, social activity, mood, weight, appetite, and energy level. Each item response is on a five-point Likert scale. The sum of the score of all items represents the GSS. In addition, the SPAQ includes questions about the month(s) in which specific symptoms on these subscales are experienced and about changes in food preferences. The questionnaire also includes a question about the extent to which seasonal changes are considered a problem. A GSS of $\geq$11 is considered as an indication of elevated seasonality (Kasper et al., 1989). While the full Kasper criteria also consider whether seasonal changes are experienced as a moderate to disabling problem (Kasper et al., 1989), this study used only the GSS cutoff to determine elevated seasonality. Using the GSS cutoff alone captured a broader pool of participants with elevated seasonal patterns, consistent with the SPAQ's role as a screening tool, while leaving clinical interpretation of perceived problems for the diagnostic interview stage.

The SPAQ has good internal consistency (Cronbach's α =0.81) and test-retest reliability (r = 0.76) (Young et al., 2003). The Icelandic version has been shown to have good sensitivity (94 %) and specificity (73 %) (Magnusson, 1996). In the present study Cronbach's α for the SPAQ was 0.87.

2.4.3. Patient Health Questionnaire-9

The PHQ-9 is a self-report scale that assesses the severity of nine core symptoms of depression within the past two weeks, as defined by the DSM-IV (APA, 1994). It has high internal consistency (Cronbach's α = 0.86–0.89) and test-retest reliability ($r = 0.84$) (Kroenke et al., 2001). A recent systematic review indicated that the sensitivity of the PHQ-9 ranged from 0.37 to 0.98, while its specificity ranged from 0.42 to 0.99 (Costantini et al., 2021). The Icelandic version of the PHQ-9 has good internal consistency and good convergent and discriminant validity (Björnsson et al., 2018; Kristófersdóttir et al., 2023). In the present study Cronbach's α for the PHQ-9 was 0.89.

2.5. Clinical diagnostic interview

Participants with elevated seasonality (GSS $\geq$ 11) were called via phone to obtain a clinical interview. In the interview we assessed depression history (current, past) using the mood disorder module of the semi-structured MINI-International Neuropsychiatric Interview (MINI; Sheehan et al., 1998) according to the diagnostic criteria defined in DSM-IV (APA, 1994). Diagnostic criteria for MDD, Dysthymia, and Bipolar I and II disorders were assessed in the interview. The Icelandic version was administered, for which adequate validity has been demonstrated (Hjartarson et al., 2022; Kristjánsdóttir et al., 2013; Ólafsson et al., 2020). It includes questions to assess the number of past depressive episodes and time of depression onset and remission. In the present study, we added questions evaluating whether episode onset and remission systematically varied by season during the past two years, independent of season-specific circumstances. Winter was defined as November–February and summer as May–August. Participants also indicated whether they believed most of their lifetime episodes followed a seasonal or nonseasonal pattern, coded as *Yes* (majority probably/definitely seasonal), *No* (majority nonseasonal), or *Unclear* (uncertain/unable to estimate). MDD with seasonal pattern was defined as season-specific onset and remission of two episodes during the past two years, with no nonseasonal episodes during that period, and a prior episode history—if any—also judged to be seasonal (*Yes* code). While this definition of SAD reflects DSM-IV criteria, it remains highly consistent with the DSM-5-TR definition, as only minimal text changes were introduced. Each interview was administered via phone either by a licensed clinical psychologist or by a clinical psychology graduate student at the University of Iceland who received prior training and supervision in the administration of the MINI. Interviews lasted approximately 1 to 1.5 h

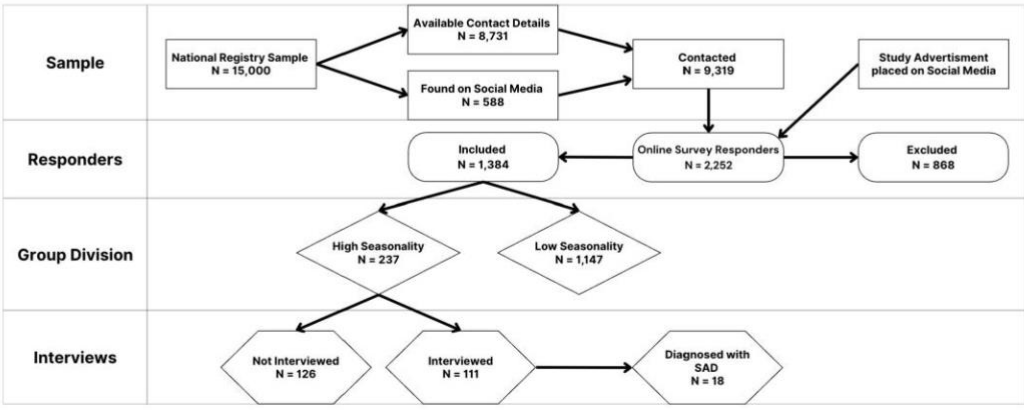


Fig. 1. Participant flow from initial contact through survey inclusion, seasonality-based group assignment, and progression to interview-based Seasonal Affective Disorder (SAD) diagnosis.

and were audiotaped.

2.6. Data analysis and statistical methods

Statistical analyses were performed using R/R-Studio (v4.4.1; R Core Team, 2021) and MATLAB (The MathWorks Inc., 2024).

Missing data in the SPAQ responses were addressed using a mode imputation approach. To minimize data loss while maintaining response integrity, missing values were replaced with the most frequently occurring value (i.e., mode) within each respondent's available responses, provided that no more than two items were missing.

To examine significant associations between sociodemographic variables and seasonality, Spearman's rank correlation tests were used for continuous variables and $\chi 2$-tests were used for categorical variables.

The proportion of participants with elevated seasonality was calculated as a weighted estimate that compensated for non-randomness in response rates. To this end, the random sample of 15,000 residents (N) was stratified into 80 strata based on sex (2 categories), region (8 categories), and age group (5 categories). Within each stratum, the number of participants with elevated seasonality (γR_i) was determined. The number of respondents in each stratum (R_i) was considered relative to the total sample size of that stratum (N_i). This proportion (θ_i) was calculated as:

$$\theta_i = \frac{R_i}{N_i} \tag{1}$$

To obtain a weighted estimate, the number of participants with elevated seasonality in each stratum was divided by the participation proportion in that stratum. These weighted values were summed across all strata and divided by N to yield the final prevalence estimate of seasonal fluctuations in mood and behaviour:

$$\frac{1}{N} \sum_{i=1}^{S} \frac{\gamma R_i}{\theta_i} \tag{2}$$

Analogously we calculated of the prevalence of SAD. First, for each stratum the number of participants with elevated seasonality who agreed to participate in a MINI interview (M_i) was considered relative to the total number of participants with elevated seasonality in each stratum. This proportion (δ_i) was calculated as:

$$\delta_i = \frac{M_i}{\gamma R_i} \tag{3}$$

Then, for each stratum the number of participants who met the diagnostic criteria for SAD based on the MINI (γD_i) was divided by the product of the proportion of participants within that stratum and the proportion of participants of that stratum who had elevated seasonality and participated in a MINI interview. The results were summed across strata and divided by N to yield the overall prevalence rate of SAD in Iceland:

$$\frac{1}{N} \sum_{i=1}^{S} \frac{\gamma D_i}{\theta_i \times \delta_i} \tag{4}$$

All statistical analyzes were considered significant at $p < .05$ (two-tailed tests). For post hoc pairwise comparisons, only results that survived Bonferroni correction for multiple comparisons are reported.

3. Results

3.1. Response rates

Of the 9319 individuals contacted, 2252 responded to the survey. Of these, 845 could not be matched to the registry sample using the filtering procedure described in the Methods. For these participants, it was assumed that they were not part of the random sample and, therefore, were excluded from the prevalence analyses presented hereafter. Results on the entire recruited sample before filtering are given in *Supplementary Section B*.

Five participants who reported not residing in Iceland were excluded from the current analysis. Of the remaining 1402 online survey responders, 1321 participants completed all items of the SPAQ, leaving 81 participants with at least one missing response. Following the mode imputation procedure outlined in the Methods, the number of incomplete cases was reduced from 81 to 18. Evaluation of the GSS distribution indicated that mode imputation did not meaningfully alter variance (see Supplementary Section C).

The final sample for analysis consisted of $N = 1384$ participants, aged between 18 and 69 years (response rate = 14.85 %). Response rates and chi-square tests which compared age group, sex, and region between responders and non-responders are presented in Table S2.

There was a significant difference in the response rates between women and men ($\chi^2(1, N = 9319) = 57.55$, $p < .001$). Women were more likely (17.42 %) to respond to the survey than men (11.79 %).

There was a significant difference in the response rates between age groups ($\chi^2(4, N = 9319) = 125.27$, $p < .001$). Pairwise comparisons of response rates between age groups revealed several statistically significant differences. Participants aged 31–40 had significantly higher response rates (12.41 %) than those aged 18–30 (8.21 %) ($p < .001$). Similarly, individuals aged 41–50 had significantly higher response rates (15.17 %) than those aged 18–30 ($p < .001$). Individuals in the 51–60 age group had significantly higher response rates (18.86 %) than those in the 18–30 ($p < .001$), 31–40 ($p < .001$), and 41–50 ($p = .012$) groups. The oldest group (61–65 years) had significantly higher response rates (20.72 %) than all younger age groups (*all* $p < .001$), except for the 51–60 group.

There was a significant difference in the response rates between regions of Iceland $\chi^2(7, N = 9319) = 128.41$, $p < .001$). Response rates ranged from a low of 12.07 % in the Capital region to a high of 23.56 % in the Northeastern region. Pairwise comparisons of response rates between regions can be found in the Supplement.

Of the analyzed sample, 25.51 % completed the questionnaire in spring (March–May), 47.69 % in summer (June–August), 12.5 % in fall (September–November), and 14.31 % in winter (December–February). The distribution of response rates across seasons significantly differed from a uniform distribution ($\chi^2(3, N = 1384) = 434.91$, $p < .001$). Response rates were significantly higher in summer compared to all other seasons (all $p < .001$), and higher in spring compared to fall and winter (both $p < .001$).

3.2. Prevalence of symptoms for SAD

Elevated seasonality (GSS ≥ 11) was found in $N = 237$ (17.12 %) and the weighted prevalence rate was 17.86 % (95 % CI [15.93, 19.97]). The season during which participants completed the SPAQ did not affect the prevalence of elevated seasonality ($\chi^2(3, N = 1384) = 5.01$, $p = .17$).

The median GSS was 4 (mean = 5.48, SD = 4.8, range = 0–24). Most people reported at least marked changes in mood (17.27 %), followed by energy level (11.85 %), sleep (9.97 %), and social activity (8.45 %). Fewer participants reported at least marked changes on the subscales appetite (4.77 %) and weight (3.97 %).

According to a separate item on perceived problems due to seasonal changes, 15.46 % of participants found such changes problematic, with 1.08 % describing them as severe or disabling. Among those with elevated seasonality, 48.95 % reported seasonal changes as problematic, including 5.49 % who rated them as severe or disabling.

On average, participants reported sleeping the longest during winter ($M = 7.58$, $SD = 1.18$) and the shortest during summer ($M = 7.03$, $SD = 0.96$), while spring ($M = 7.19$, $SD = 0.98$) and fall ($M = 7.38$, $SD = 0.99$) were in between these numbers.

Overall, 45.3 % of participants reported seasonal changes in food preferences. On average, participants indicated an annual weight fluctuation of 2.91 kg (SD = 2.08).

Of the survey responders 87.12 % reported feeling worst in the late fall and winter months and 2.45 % reported to feel worst in the summer months. More information on the pattern of seasonal changes by month can be seen in Fig. 2.

3.3. Analysis of factors associated with seasonality

Descriptive statistics and associations between demographic variables and seasonality symptoms, as measured by the GSS, are presented in Table 1.

Sex was significantly associated with seasonality symptoms ($\chi^2(1, N = 1384) = 23.21, p < .001, V = 0.13$); women had a significantly higher GSS than men.

Age group was associated with seasonality symptoms ($\chi^2(4, N = 1384) = 53.12, p < .001, V = 0.20$). Individuals aged 18–30 were significantly more likely to report a higher GSS than those aged 41–50, 51–60, and 61–65 (all $p < .001$). Participants aged 31–40 also had significantly higher GSS than those aged 41–50 ($p = .043$), as well as 51–60 and 61–65 (both $p < .001$).

Occupational status was associated with seasonality symptoms ($\chi^2(3, N = 1336) = 30.71, p < .001, V = 0.15$). Participants who were not working reported significantly higher GSS than those working full-time ($p < .001$) or part-time ($p = .022$). Similarly, students had higher GSS than full-time workers ($p = .019$). Single individuals ($\chi^2(1, N = 1377) = 7.08, p = .008, V = 0.07$) and those without kids ($\chi^2(1, N = 1374) = 14, p < .001, V = 0.1$) had higher GSS compared to people in relationships and those with kids.

There was a moderate positive correlation between PHQ-9 sum scores and GSS ($r(1304) = 0.57, p < .001$, 95 % CI [0.53, 0.61]).

Educational level, region of residence and whether the person was born in Iceland were not significantly associated with seasonality symptoms.

3.4. Clinical interviews and prevalence of SAD

Out of 237 participants who had a GSS of 11 or higher, $N = 111$ agreed to participate in the clinical interview. Between interviewed and not interviewed participants, there was no significant difference in age ($W = 6407, p = .266$), sex ($\chi^2(1) = 0.01, p = .92$), GSS ($W = 6064, p = .075$), and PHQ-9 scores ($W = 5234.5, p = .114$).

Following the interviews, $N = 18$ (all women) were diagnosed with SAD according to diagnostic criteria. After weighting this number against the number of participants interviewed from the registry sample and the stratification approach described in the methods, the prevalence rate of SAD in Iceland was estimated at 2.24 % (95 % CI [0.69, 6.98]).

Group comparisons among interviewed participants revealed that those meeting criteria for SAD were significantly more often women ($\chi^2(1) = 4.502, p = .034$) than those who did not get a clinical diagnosis of SAD. However, these two groups did not differ by age ($W = 874.5, p = .767$), GSS ($W = 649.5, p = .131$), and PHQ-9 scores ($W = 614.5, p = .302$).

A significantly higher proportion of women were found in the group meeting criteria for SAD compared to those diagnosed with recurrent Major Depressive Disorder without Seasonal Pattern ($\chi^2(1) = 4.492, p = .034$). No significant differences were observed between the groups in age ($W = 473, p = .952$), GSS ($W = 396, p = .332$), and PHQ-9 scores ($W = 391.5, p = .556$).

In contrast, participants diagnosed with SAD had significantly higher GSS ($W = 105, p = .027$) and PHQ-9 scores ($W = 36.5, p < .001$) and the group had a higher proportion of women ($\chi^2(1) = 6.872, p = .009$) as compared to those who had never experienced a depressive episode. However, the two groups had a similar age distribution ($W = 237, p = .098$). Descriptive statistics for all groups are presented in Table 2.

4. Discussion

In the present study, the weighted prevalence of SPAQ-derived elevated seasonality was 17.86 %, notably higher than the 3.8 % reported in the only previous epidemiological study conducted in Iceland (Magnusson and Stefansson, 1993). Prevalence varied across demographic groups, with younger individuals, women, students, unemployed individuals, single participants, and those without children reporting elevated seasonality. Most remarkably, when semi-structured diagnostic interviews were conducted with a subset of participants, only 2.24 % met the DSM criteria for SAD, resulting in a substantial gap between self-reported seasonal symptoms and clinical diagnosis. In the following, we discuss these results in the Icelandic and global context of research in SAD and seasonal symptoms.

Several factors may explain the elevated prevalence of seasonal symptoms observed in our sample as compared to an earlier study which was based on a similar self-report screening (Magnusson and Stefansson, 1993). First, public awareness of mental health issues has grown substantially in recent decades. A meta-analysis by Schomerus et al. (2012) documented rising mental health literacy across Western countries, including greater recognition of depression and broader acceptance of treatment. This increased familiarity with psychological terminology may lead individuals to label seasonal fluctuations as indicative of a disorder, even when symptoms are subclinical. Second, lifestyle changes may have contributed to the findings. Modern life is increasingly spent indoors (Klepeis et al., 2001), reducing exposure to natural daylight during winter, a suspected key factor in SAD (Magnusson, 2000; Raza et al., 2024). This tendency may have been amplified by the widespread adoption of remote work and increased screen time following the COVID-19 pandemic (Giuntella et al., 2021). It is important to note that the earlier Icelandic study (Magnusson and Stefansson, 1993) did not find significant sex or age-related differences in response rates, while our study had a higher response rate among women and older adults which are in general more likely to complete surveys (Lallukka et al., 2020; Gigliotti and Dietsch, 2014; Rastad et al., 2005). Moreover, while our study used only the GSS ≥ 11 cutoff to identify elevated seasonality, the earlier Icelandic study additionally required that seasonal changes be experienced as at least a moderate problem and that symptoms were worst in January and/or February (Magnusson and Stefansson, 1993). Using only the GSS ≥ 11 cutoff allowed us to capture a broader group of participants with elevated seasonality than the previous study. However, prior work suggests that the SPAQ has limited sensitivity for DSM-defined SAD (Mersch et al., 2004), meaning that some individuals with clinically relevant seasonal patterns may fall below this threshold. As a result, our prevalence estimate should be considered a conservative rather than a precise estimate of true prevalence.

Our findings align with earlier research stating that self-report tools like the SPAQ tend to overestimate the prevalence of SAD compared to diagnostic interviews (Eagles et al., 1999; Levitt et al., 2000). Our results highlight the importance of using clinical interviews when prevalence of SAD is to be assessed, especially when comparing findings across different countries or populations. Such methodological considerations also need to be taken into account when reviewing data supporting or questioning the latitude hypothesis. Our SPAQ-based findings on elevated seasonality align with studies from other high-latitude regions, such as Alaska, Norway, and Greenland, where prevalence was assessed solely via the SPAQ (Levine, 1995; Konradsen, 1995; Kegel et al., 2009). Crucially, however, the addition of diagnostic interviews in our study revealed substantially lower SAD prevalence, consistent with other high-latitude studies that applied similar interview-based methods (Eagles et al., 1999; Levitt et al., 2000). Based on our data we suggest that much of the apparent variation in SAD prevalence across latitudes may stem from differences in assessment tools rather than geographic effects. To clarify the relationship between latitude and SAD, future research should prioritize standardized diagnostic procedures across regions.

Furthermore, the large discrepancy between the prevalence of

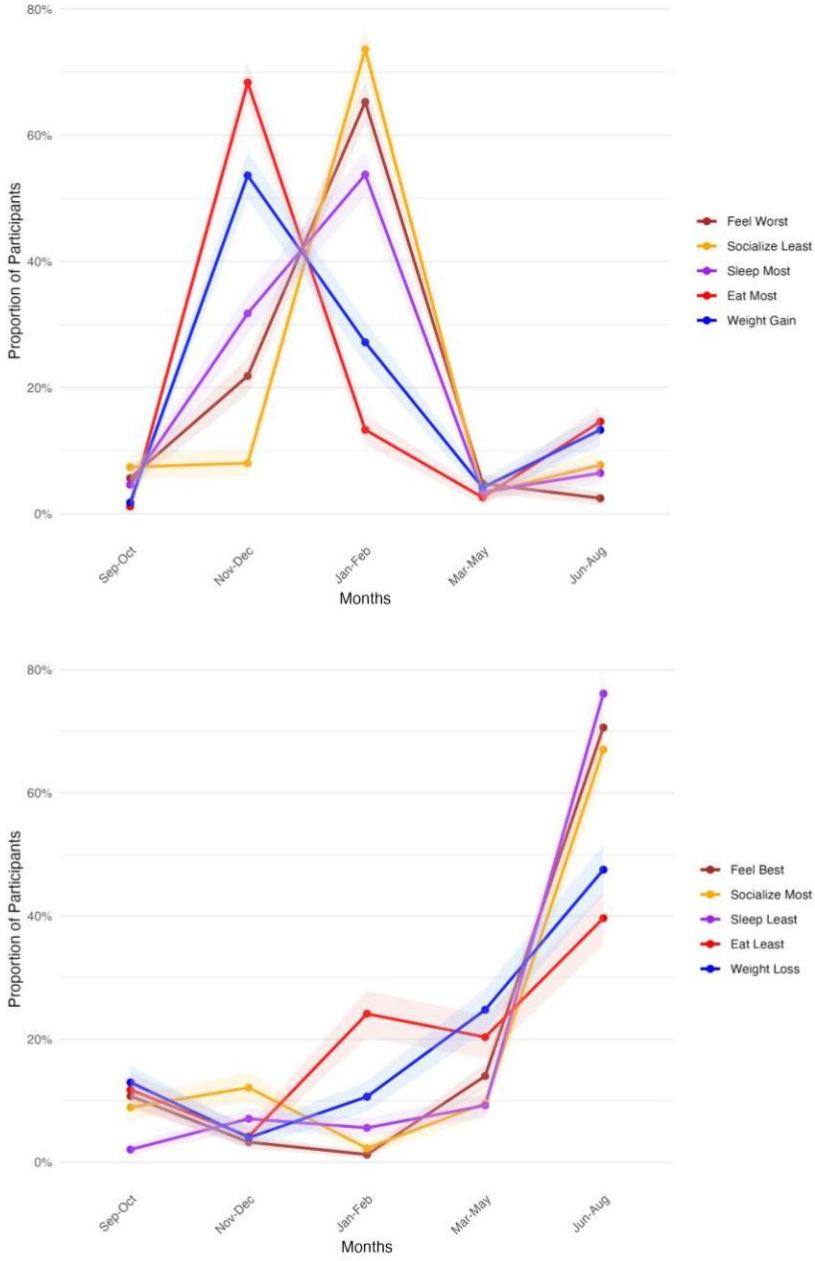


Fig. 2. Pattern of reported seasonal changes by month.
Lines represent the percentage of participants reporting seasonal changes, and shaded areas indicate 95 % confidence intervals.

Table 1
Demographic characteristics and their associations with seasonality symptoms.

	N	GSS Median	χ^2 Test	Effect Size
Sex				
Women	882 (63.73 %)	5	χ^2 (1, N = 1384) = 23.21, $p < .001$	*0.13*
Men	502 (36.27 %)	3		
Age Group				
18–30	134 (9.68 %)	8	χ^2 (4, N = 1384) = 53.12, $p < .001$	*0.20*
31–40	265 (19.15 %)	6		
41–50	340 (24.57 %)	5		
51–60	415 (29.99 %)	3		
61–65	230 (16.62 %)	3		
Occupational Status				
Full-time	992 (74.25 %)	4	χ^2 (3, *N* = 1336) = 30.71, $p < .001$	*0.15*
Part-time	117 (8.76 %	5		
Unemployed	162 (12.13 %)	7		
Student	65 (4.87 %)	6		
Relationship Status				
Relationship/Married/ Cohabitation	1093 (83.95 %)	4	χ^2 (1, *N* = 1377) = 7.08, $p = .008$	0.07
Single/Divorced/ Widowed	209 (16.05 %)	5		
Kids				
Yes	1165 (84.36 %)	4	χ^2 (1, *N* = 1374) = 14, $p < .001$	0.1
No	216 (15.64 %)	6		
Highest Educational Level			χ^2 (2, *N* = 1376) = 1.12, $p = .57$	0.03
Higher Education	740 (53.78 %)	4		
Secondary Education	445 (32.34 %)	4		
Primary Education	191 (13.88 %)	4		
Region				
Capital	710 (51.3 %)	4	χ^2 (7, *N* = 1384) = 3.05, $p = .88$	0.05
Northeastern	197 (14.23 %)	4		
Southern	141 (10.19 %)	4		
Southern peninsula	93 (6.72 %)	4		
Western	93 (6.72 %)	5		
Eastern	70 (5.06 %)	3		
Northwestern	50 (3.61 %)	5		
Westfjords	30 (2.17 %)	3		
Born in Iceland			χ^2 (1, *N* =	0.06
Yes	1323 (96.08 %)	4	1377) = 3.73, $p = .05$	
No	54 (3.92 %)	5		

Note. GSS = Global Seasonality Score; Effect sizes are reported using Cramer's V. N reflects the number of participants within each subgroup.

Table 2
Demographic and clinical characteristics by interview status and diagnostic group.

	N (% Women)	M Age (SD)	Mdn GSS	Mdn PHQ-9
Not Interviewed	126 (76.98 %)	41.9 (11.4)	13	9
Interviewed:	111 (78.38 %)	43.7 (12.9)	14	10
MDD recurrent, with Seasonal Pattern	18 (100 %)	42.8 (12)	15	11
MDD recurrent, without Seasonal Pattern	52 (73.08 %)	43 (13)	14	10
MDD single episode	5 (80 %)	39.4 (12.7)	13	12
Bipolar I or II	8 (87.5 %)	41.1 (14.4)	14	13
Dysthymia	7 (100 %)	38.4 (13.8)	14	13
Never experienced a depressive episode	20 (60 %)	49.6 (11.8)	12.5	5

Note. Symptomatology could not be evaluated for one interviewed participant. *N = sample size; M = mean; SD = standard deviation; Mdn = median; GSS = Global Seasonality Score; PHQ-9 = Patient Health Questionnaire 9-item; MDD = Major Depressive Disorder.*

elevated seasonality based on the SPAQ and the much lower prevalence of clinically diagnosed SAD suggests that seasonality may not reflect a single, uniform condition. While some individuals experience full depressive episodes, others may experience primarily vegetative symptoms without marked affective disturbances. This pattern was discovered previously and has given rise to the dual vulnerability model, suggesting that SAD emerges when physiological sensitivity (e.g., to reduced daylight) interacts with psychological predispositions (e.g., rumination) (Young et al., 1991, 2008). Prospective research suggests that vegetative symptoms often precede mood changes (Whitcomb-Smith et al., 2014), raising the question of whether such symptoms serve as early indicators or reflect a separate subclinical seasonal response. Understanding this distinction may improve early detection and allow for more targeted interventions.

In addition to symptom heterogeneity, our results highlight demographic and psychosocial factors associated with elevated seasonality. Consistent with earlier research, younger individuals and women reported elevated seasonality (Magnusson, 2000; Mersch et al., 1999b). Students also reported elevated seasonality, in line with previous findings (Fonte and Coutinho, 2021). We further observed elevated seasonality among unemployed individuals, consistent with Mersch et al. (1999b), who found greater unemployment and sick leave in participants with increased seasonality symptoms. Single individuals and those without children also reported elevated seasonality. A large Norwegian study similarly found that being unmarried and childless was linked to greater risk of SAD, possibly due to reduced social support (Bjorvatn et al., 2020). As our data are correlational, it remains unclear whether these factors contribute to elevated seasonality or result from it. Nevertheless, these findings highlight the importance of psychosocial characteristics in identifying individuals who may benefit from early monitoring or preventive strategies.

5. Limitations

While our study provides insight into methodological issues in the long-debated relationship between SAD and latitude, several limitations should be considered when interpreting the results.

Firstly, the low response rate of 14.85 % increases the risk of non-response bias, as individuals who chose not to participate may differ from those who responded. In our sample, women and older adults were overrepresented, while younger men responded at notably lower rates. Although weighted analyses were applied to correct for varying response rates across strata, this differential response pattern may nevertheless have influenced prevalence estimates. In particular, since younger men show lower levels of seasonality, their under-representation may have inflated overall prevalence. This issue is especially relevant given that only a subset of respondents with elevated seasonality completed diagnostic interviews, and all participants diagnosed with SAD were female, again suggesting a possible sex-related limitation in generalizability. Moreover, the small number of SAD cases limits the precision of our estimates, resulting in wide confidence intervals.

Secondly, our interviews focused specifically on assessing SAD and did not include a full evaluation of other psychiatric conditions that are sometimes comorbid with SAD, such as generalized anxiety disorder (Levitt et al., 1993) or alcohol use disorder (Morales-Muñoz et al., 2017). We also did not include measures of potential psychological moderators, such as rumination or chronotype (Rohan et al., 2003; Höller et al., 2021; Bjorvatn et al., 2020), nor did we examine potential moderators or mediators of differences in seasonality across specific participant groups (e.g., women, students). Future research should explore these factors to better understand underlying mechanisms and individual differences in vulnerability to general seasonality versus clinical SAD.

Thirdly, methodological constraints need to be considered. The cross-sectional design restricts inferences about the temporal progression of symptoms. Despite the benefits of phone-based interviews, such as facilitating data collection in rural areas, they have disadvantages, including the absence of non-verbal cues (Stephens, 2007) and the difficulty of establishing rapport (Glogowska et al., 2011). It should also be noted that the SPAQ is a retrospective self-report instrument, which may limit the precision of the data due to recall bias (Wicki et al., 1992). Finally, a limitation of this study is the use of a GSS cut-off of $\geq$11. Applying an alternative threshold, such as the Kasper criteria (Kasper et al., 1989), could have altered the number of participants classified as experiencing elevated seasonality, potentially affecting the results.

6. Conclusion

In conclusion, the present study was the first epidemiological investigation in Iceland to use semi-structured clinical interviews to diagnose SAD. This methodological choice is a central strength, as it enabled a more objective and reliable assessment than self-report measures alone. According to our data, clinical SAD was rather rare in Iceland, but a significant portion of people reported seasonal symptoms affecting their wellbeing. Integrating standardized diagnostic criteria allowed for clinical prevalence estimates and revealed meaningful discrepancies between reported symptoms and clinical diagnoses. As diagnostic methods strongly shape prevalence outcomes, future epidemiological studies on seasonality and SAD, and specifically the relation between latitude and these two overlapping phenomena should continue to incorporate validated interviews to enhance diagnostic precision and support informed public health planning. Treatment and prevention plans might be set out to target the two phenomena of SAD and seasonality differentially.

CRediT authorship contribution statement

Lada Zelinski: Writing – original draft, Visualization, Methodology, Formal analysis, Data curation. **Ara Dan Pálmadóttir:** Investigation, Data curation. **Ásta Guðrún Birgisdóttir:** Investigation, Data curation. **Leon Daði Sesseljuson:** Investigation, Data curation. **Ragnar Pétur Ólafsson:** Writing – review & editing, Supervision, Project administration, Methodology, Investigation, Data curation, Conceptualization. **Arne Cornelius Bathke:** Validation, Formal analysis. **Yvonne Höller:** Writing – review & editing, Validation, Supervision, Resources, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation.

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Declaration of competing interest

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jad.2025.121057.

Data availability

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

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When the weather gets colder and days get shorter: Environmental mood responses and their link to depressive symptom severity

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ABSTRACT

As weather patterns become increasingly variable, understanding how weather and daylight influence mood has become more important, but empirical evidence remains inconclusive. This inconsistency may stem from individual differences in sensitivity to environmental conditions, as well as differences in actual environmental exposure. Yet little is known about how subjective sensitivity and objective weather variables contribute independently or interactively to depressive symptoms. To address this research gap, we examined these associations in Iceland, where pronounced seasonal and meteorological variability provides a particularly unique setting. We collected data from 2018 Icelandic adults in an online survey assessing demographics, subjective mood responses to environmental conditions using the Seasonal Pattern Assessment Questionnaire, and depressive symptom severity via the Patient Health Questionnaire-9. These responses were linked to 14-day averages of objective weather measurements, including temperature, humidity, and wind speed. Results showed that more adverse mood responses to cold weather and reduced daylight were associated with higher depressive symptom severity (both $p < .001$), whereas adverse responses to dry weather were associated with lower depressive symptom severity ($p = .004$). Those reporting adverse responses to cold weather experienced fewer depressive symptoms at higher temperatures ($p = .003$). Our findings underscore the importance of considering both individual differences in sensitivity to environmental conditions as well as actual environmental exposure when evaluating self-assessed depressive symptom severity. Brief, low-cost assessment of environmental mood responses may therefore offer a practical tool for identifying vulnerable individuals, an issue of increasing importance as global climate change intensifies weather variability.

1. Introduction

Despite its intuitive appeal and prevalence in everyday language (e.g., "feeling under the weather"), scientific evidence for a consistent relationship between environmental conditions and mood has been inconclusive (e.g., Denissen et al., 2008; Huibers et al., 2010; Keller et al., 2005; Kööts et al., 2011). Environmental conditions include both short-term weather patterns (e.g., temperature, precipitation) and broader seasonal phenomena such as daylight duration (i.e., photoperiod), which are often conflated but reflect distinct aspects of environmental exposure. Researchers have examined how weather and seasonal conditions are associated with a range of outcomes, including life satisfaction (Kämpfer & Mutz, 2011; Schmiedeberg & Schröder, 2014), daily mood and affect (Denissen et al., 2008; Keller et al., 2005; Klimstra et al., 2011; Kööts et al., 2011), job satisfaction (Venz & Pundt, 2021), and the severity of depressive symptoms (Huibers et al., 2010; Molin et al., 1996; O'Hare et al., 2015), among others.

Findings focusing on the relationship between depressive symptoms and objective environmental conditions, such as temperature, precipitation, and light exposure, have been mixed (Huibers et al., 2010; Molin et al., 1996; O'Hare et al., 2015). For instance, one study found no evidence that daily temperature, sunshine duration, or rainfall were significantly associated with either major depression or sad mood (Huibers et al., 2010). Despite observing seasonal patterns in mood, the researchers concluded that objective weather conditions did not account for these changes (Huibers et al., 2010). In contrast, evidence from a longitudinal follow-up of individuals with winter depression over multiple years indicated that greater sunlight exposure, longer daylight, and higher temperatures were significantly associated with lower depression scores, while no associations were found for cloud cover, rainfall, or

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atmospheric pressure (Molin et al., 1996). Increased sunshine and temperature have also been linked to reduced depressive symptom levels among older adults in Ireland; however, in this case, higher rainfall predicted elevated symptoms (O'Hare et al., 2015). Together, these findings suggest that certain environmental variables, particularly light and temperature, may be associated with depressive symptom severity in specific populations, although the influence of other weather-related factors remains less consistent.

According to some studies, both season and time spent outdoors influence how temperature and light affect mood (Keller et al., 2005; Kööts et al., 2011). It has been shown that individuals who spent more time outdoors showed stronger associations between temperature and negative affect, and between sunlight and positive affect, with physical activity noted as a possible explanation for the sunlight–positive affect link (Kööts et al., 2011). Evidence from the United States across several geographic regions indicated that higher temperature and, additionally, barometric pressure were associated with improved mood valence (i.e., greater positive relative to negative affect) and enhanced cognition (working memory capacity and openness to new information), but only among individuals who spent more time outside and only during spring (Keller et al., 2005). Interestingly, during summer, higher temperatures were associated with lower mood, particularly among participants living in southern regions where heat tends to be more intense and uncomfortable (Keller et al., 2005).

An often-overlooked factor in environment-related mood variation is how individuals perceive and respond to specific environmental conditions. In a study of adolescents and their mothers, the average association between weather and mood was weak (Klimstra et al., 2011). However, latent class analysis revealed four distinct weather reactivity types: Unaffected, Summer Lovers, Summer Haters, and Rain Haters (Klimstra et al., 2011). While the Unaffected group showed no meaningful weather–mood links, the remaining participants experienced substantial but divergent effects, including stronger negative responses to specific weather elements, such as precipitation (Klimstra et al., 2011). These findings suggest that individuals' adverse and favourable environmental mood responses moderate weather–mood associations and that such variability can obscure group-level effects (Klimstra et al., 2011). Comparisons of self-reported mood responses to cold, hot, sunny, and cloudy weather across women with heightened sensitivity of mood and behaviour to seasonal variation, those with non-seasonal depression, and controls showed that the seasonally sensitive group reported stronger adverse mood responses to cold and cloudy weather, as well as stronger favourable mood responses to sunny weather (Sigmon et al., 2009). Another study found that individuals with winter depression reported stronger favourable responses to summer-like weather and light conditions (e.g., sun, heat, dryness, increased daylight) and stronger adverse responses to winter-like conditions (e.g., cold, grey, reduced daylight) as compared to individuals with non-seasonal depression and controls (Mersch et al., 2004). In summary, subjective environmental mood responses may account for meaningful individual differences in mood variability and should be considered alongside objective meteorological factors.

While previous studies have provided valuable insights into adverse and favourable environmental mood responses (Mersch et al., 2004; Sigmon et al., 2009, 2010), their focus has primarily been on seasonal mood and behaviour variation, with limited examination of depressive symptom severity. Other studies have examined the relation between objective environmental conditions and affective state (Huibers et al., 2010; Molin et al., 1996; O'Hare et al., 2015), but did not sufficiently account for individual differences in mood responses to weather and light. Moreover, prior research was conducted in temperate regions, such as the Netherlands (approximately 52°N; Mersch et al., 2004) and the northeastern United States (approximately 44.5°N; Sigmon et al., 2009, 2010), where seasonal shifts in daylight and weather follow relatively synchronized patterns.

To address these gaps, we need to determine how individuals' adverse and favourable mood responses to environmental factors, including both weather and daylight, and objectively measured weather conditions, independently and in combination, contribute to depressive symptom severity, particularly in populations exposed to extreme weather conditions and pronounced seasonal variability. Iceland provides a compelling setting to extend prior work. Located near the Arctic Circle (approximately 65°N), it experiences extreme photoperiod fluctuations, from near-total darkness in winter to continuous daylight in summer. While temperatures remain relatively moderate due to the oceanic climate, rapid weather changes and diffuse seasonal boundaries are common. These unique characteristics make Iceland an especially informative setting for investigating how subjective and objective environmental factors relate to depressive symptom severity across the general population.

Building on earlier work conducted in more temperate regions (Mersch et al., 2004; Sigmon et al., 2009, 2010), this study aims to clarify whether and how these two domains, subjective environmental mood responses to weather and daylight, and external exposure to different weather conditions, are independently or interactively associated with depressive symptom severity in a population exposed to pronounced seasonal and meteorological variability. Specifically, we address the following research questions: (1) Are self-reported mood responses to specific environmental conditions, in particular temperature and daylight, associated with depressive symptom severity? (2) Are the objective weather variables, temperature, humidity, and wind speed, associated with depressive symptom severity? (3) Do interactions between individuals' mood responses to environmental conditions and actual weather conditions further explain variability in depressive symptom severity? Corresponding to these research questions, we hypothesize that (1) individuals' self-reported mood responses to environmental conditions will be associated with depressive symptom severity, with adverse responses related to higher symptom severity and favourable responses related to lower symptom severity; (2) objective weather variables will be associated with depressive symptom severity; (3) there will be a significant interaction between subjective environmental mood responses and objective weather conditions in their association with depressive symptom severity.

2. Material and methods

Ethical approval

The National Bioethics Committee of Iceland approved the study (VSN-22-058, March 22, 2022). The study adhered to the Declaration of Helsinki. Written informed consent was obtained from all participants, and all researchers signed non-disclosure agreements.

2.1. Sample and data overview

The present study was based on a random sample of 15,000 adults drawn from the Icelandic National Registry. Recruitment occurred between May 2022 and December 2024. In addition to the random sample, participants were recruited via a dedicated study website and a paid Facebook advertisement. Participants completed the survey online or by phone.

Afterwards, survey responses were temporally and geographically matched to weather data derived from local weather stations collected during the same period.

Additional details on recruitment procedures are provided in Supplementary Methods S1.

2.2. Participants and final analytic sample

A total of 2532 participants completed the online survey. Of these, 46 individuals completed the questionnaire more than once, and only their most recent responses were retained, while earlier entries were

excluded. Additionally, 16 participants were excluded for indicating that they did not reside in Iceland, and 8 participants were excluded for reporting an age below 18 years.

Of the remaining 2462 participants, 2258 completed all items of the Patient Health Questionnaire-9 (PHQ-9; Kroenke et al., 2001), while 204 provided incomplete responses. After applying the mode imputation procedure described in section 2.5, 67 participants with incomplete PHQ-9 data were excluded, resulting in 2395 participants with available PHQ-9 scores. Among these, 125 participants provided missing responses for the Seasonal Pattern Assessment Questionnaire (SPAQ; Rosenthal, 1984) items assessing environmental mood responses and were therefore excluded, leaving 2270 participants with complete PHQ-9 and SPAQ data.

Objective weather data could be linked for 2024 participants based on available location information and proximity to a weather station; the remaining 246 participants were excluded.

Of the 2024 participants with complete weather information and survey data, 675 (33.35%) identified as men, 1343 (66.35%) as women, 2 (0.1%) as non-binary, and 4 (0.2%) did not report their gender. Because the number of participants identifying as non-binary was too small for meaningful statistical analysis, they, along with those with missing gender information, were excluded from the analyses.

This resulted in a final sample of 2018 participants. The mean age of the final sample was 46.03 years (SD = 12.39), with a median of 47 years and a range of 18 to 80 years. Descriptive statistics for the sample are presented in Table S1.

Participants took part in the study at different times of the year, with participation occurring most frequently in summer (n = 651), followed by winter (n = 603), spring (n = 489), and autumn (n = 275).

2.3. Survey instruments

The online survey was administered using Microsoft Forms and included informed consent, full name, home address, municipality of legal residence, email address, phone number, gender, age, level of education, employment status, relationship status, parental status, whether the respondent currently resides in Iceland, fluency in Icelandic, and whether they were born in Iceland. The survey also incorporated validated Icelandic versions of the SPAQ (Magnusson, 1996) and the PHQ-9 (described in Kristófersdóttir et al., 2023).

2.3.1. Seasonal Pattern Assessment Questionnaire (SPAQ)

The SPAQ is a self-report instrument used to assess seasonal variation in mood and behaviour (Rosenthal, 1984). It contains a subscale consisting of nine items concerning weather and light variables: cold, warm, rainy, sunny, dry, cloudy, windy, long days (increased daylight), and short days (reduced daylight). These items measure how participants experience the effects of different environmental conditions on their mood and energy. Responses are given on a seven-point Likert scale, where 1 indicates feeling extremely low and unenergetic, 4 indicates no effect, and 7 indicates a very favourable effect on mood and energy. For analysis, items were reverse-coded so that higher values indicated stronger adverse mood responses to environmental conditions. Each of the nine items was treated as a separate variable, and no composite or aggregated score was derived.

2.3.2. Patient Health Questionnaire-9 (PHQ-9)

The PHQ-9 was used to assess symptoms of depression (Kroenke et al., 2001) as it is a widely used self-report instrument that evaluates the presence and severity of nine core depressive symptoms over the previous two weeks, consistent with DSM-IV diagnostic criteria (American Psychiatric Association, 1994). Responses are provided on a four-point Likert scale, from 0 indicating "Not at all" to 3 indicating "Nearly every day". Based on the sum of all items, a total PHQ-9 score is calculated, ranging from 0 to 27, with higher scores indicating greater depression severity. The following classification is frequently used to distinguish depression severity: minimal (PHQ $\leq$4), mild (4 $\geq$ PHQ $\leq$9), moderate (10 $\geq$ PHQ $\leq$14), moderately severe (15 $\geq$ PHQ $\leq$19), and severe (20 $\geq$ PHQ) (Kroenke et al., 2001).

The PHQ-9 has demonstrated high internal consistency (Cronbach's α ranging from 0.86 to 0.89) and strong test-retest reliability (r = 0.84; Kroenke et al., 2001). Sensitivity estimates ranging from 0.37 to 0.98 and specificity estimates from 0.42 to 0.99 have been reported in a systematic review (Costantini et al., 2021). The Icelandic version has shown robust internal consistency and strong convergent and discriminant validity (Björnsson et al., 2018; Kristófersdóttir et al., 2023). In the present study, Cronbach's α for the PHQ-9 was 0.9.

2.3.3. Objective weather variables derived from local weather stations

Objective weather data were obtained freely from the Icelandic Meteorological Office (Icelandic Meteorological Office, 2025). These datasets included daily averages for key meteorological variables, such as air temperature (°C), relative humidity (%), and wind speed (m/s), recorded across a wide range of monitoring stations throughout Iceland.

Weather records were linked to participants based on their reported municipality or nearest available location, within a maximum distance of 20 km. The final dataset included observations from 62 places, ensuring broad geographic coverage. Notable sampling densities were seen in Reykjavík (n = 501), Akureyri (n = 261), Garðabær (n = 189), and Kópavogur (n = 143). Additional coverage spanned a diverse range of rural and coastal regions, including Ísafjörður (n = 35), Egilsstaðir (n = 66), Hveragerði (n = 27), and Vestmannaeyjar (n = 28), among others. A full list of weather station locations and the number of participants per station is provided in Table S2, accompanied by a map in Fig. S1 for geographical reference. On average, participants were located 5.5 km from the nearest weather station (range: 0.2 km – 20 km).

For each participant, daily weather data were matched to their survey response based on the date of survey completion. Averages of weather variables were then calculated from the 14 days preceding that date.

2.4. Data analysis and statistical methods

Statistical analyses were performed using R (version 4.4.1; R Core Team, 2021). The PHQ-9 total score was calculated for each participant. Missing values in the PHQ-9 were handled using mode imputation. To preserve response integrity while minimizing data loss, missing items were replaced with the respondent's most frequently selected response, provided that no more than two items were missing.

A multiple linear regression model was conducted to investigate whether subjective environmental mood responses and objective weather conditions were associated with psychological outcomes. The model used the PHQ-9 total score as the dependent variable. Explanatory variables included self-reported mood responses to cold, warm, sunny, dry, rainy, and cloudy weather, as well as to reduced daylight and increased daylight, each treated as an independent explanatory variable. Objective weather variables were operationalized as average temperature, relative humidity, and wind speed over the preceding two weeks. To assess potential moderation effects, interaction terms were specified by linking mood responses to cold weather with temperature, mood responses to warm weather with temperature, and mood responses to windy weather with wind speed. Age and gender were included as covariates.

P-values were interpreted using a Bonferroni-corrected significance level to account for multiple comparisons.

Given the inclusion of interaction terms, predictor-level generalized variance inflation factors (GVIFs) were examined (Fox, 2015; Fox & Monette, 1992; Fox & Weisberg, 2018), with scaled GVIF values ranging from 1.04 to 1.98, all below the threshold of 10 (Chatterjee & Price, 1991; Midi & Bagheri, 2010), indicating that multicollinearity was within acceptable limits (see Table S3 for all GVIFs).

3. Results

Analyses were conducted on a final sample of 2018 participants with complete data on all explanatory variables and PHQ-9 scores.

The overall regression model was statistically significant and accounted for approximately 27% of the variance in PHQ-9 scores (adjusted $R^2 = 0.272$, $F(17, 2000) = 45.39$, $p < .001$). Full details of the primary explanatory variables included in the model are presented in Table 1, with significant effects additionally illustrated in Fig. 1.

Greater adverse mood responses to reduced daylight and cold weather were significantly associated with higher depressive symptom severity (both $p < .001$), as measured by higher PHQ-9 total scores. In contrast, adverse mood responses to dry conditions were significantly associated with lower depressive symptom severity, i.e., lower PHQ-9 scores ($p = .001$).

Additionally, a significant interaction was observed between mood responses to cold weather and average fortnightly temperature ($p = .003$). Specifically, individuals reporting stronger adverse responses to cold weather showed a reduction in depressive symptoms as temperatures increased, whereas those reporting favourable responses to cold weather exhibited the opposite pattern, with depressive symptom severity increasing as temperatures rose (see Fig. 2 and Table S4).

Younger age was significantly associated with higher PHQ-9 total scores ($B = -0.10$, $t(2000) = -11.27$, $p < .001$) (see also Fig. S2). Gender was not significantly associated with PHQ-9 scores ($p = .166$) after Bonferroni correction.

A supplementary analysis, including season of participation as a covariate, yielded results consistent with the primary analysis and is reported in Supplementary Results S1.

4. Discussion

4.1. Are self-reported mood responses to specific environmental conditions, in particular temperature and daylight, associated with depressive symptom severity?

In the present study, we found that individual differences in mood responses to specific environmental conditions, including both seasonal factors such as daylight duration and short-term weather patterns such as temperature, significantly explained depressive symptom severity. Importantly, this extends earlier work by showing that subjective environmental mood responses are not only associated with seasonal mood and behaviour variation (Mersch et al., 2004; Sigmon et al., 2009) but also current depressive symptom severity. Moreover, our study additionally accounted for mood responses to reduced daylight, underscoring that variability in how people experience light and temperature contributes meaningfully to mood outcomes.

Consistent with prior work (Mersch et al., 2004; Sigmon et al., 2009), we observed stronger adverse mood responses to cold weather; however, unlike Sigmon et al. (2009), we did not find significant effects for cloudy or sunny conditions. This may reflect Iceland's climate, where overcast conditions are common and direct sunshine is often scarce, potentially reducing variability in these factors compared to other regions. Cold weather and reduced daylight showed particularly strong associations in our analyses. These effects should be interpreted considering their natural co-occurrence during Icelandic winters, where reduced daylight and lower temperatures may jointly exert a stronger adverse impact on mood among vulnerable individuals.

While epidemiological data on environmental mood responses are currently unavailable for Iceland, our findings align with population-based studies from other countries showing that a substantial proportion of individuals consider themselves weather-sensitive (von Mackensen et al., 2005). For example, over 50% of adults in Germany and Canada reported weather sensitivity, with cold weather frequently cited as a trigger for symptoms such as fatigue, sleep disturbance, and depression (von Mackensen et al., 2005). Additional evidence has indicated that subjectively perceived morning weather predicted workplace vigour only among highly weather-sensitive individuals (Venz & Pundt, 2021).

Together, our findings suggest that associations between environmental conditions and depressive symptom severity are condition-specific. Mood responses to cold and dry weather and reduced daylight showed the strongest associations, whereas responses to other weather conditions were not significant. Accordingly, brief, low-cost assessments of individual mood responses to the environment may help identify those at higher risk for depressive symptoms.

Table 1
Explanatory variables of PHQ-9 total score.

Explanatory variable	B	SE	95% CI	β	t	p	p (Bonf.)
Subjective Environmental Mood Responses							
cold weather response	1.45	0.16	[1.13, 1.76]	0.27	8.93	<0.001	<0.001
reduced daylight response	1.01	0.11	[0.80, 1.22]	0.22	9.56	<0.001	<0.001
dry weather response	−0.48	0.12	[-0.71, −0.24]	−0.09	−3.99	<0.001	0.001
warm weather response	0.41	0.17	[0.09, 0.74]	0.09	2.48	0.013	0.227
cloudy weather response	0.26	0.13	[-0.003, 0.51]	0.04	1.94	0.053	0.900
windy weather response	−0.28	0.25	[-0.77, 0.22]	−0.05	−1.09	0.277	1
increased daylight response	0.11	0.08	[-0.04, 0.26]	0.03	1.44	0.151	1
sunny weather response	0.08	0.15	[-0.21, 0.36]	0.16	0.52	0.606	1
rainy weather response	0.02	0.13	[-0.23, 0.26]	0.003	0.13	0.897	1
Objective Weather Variables							
humidity	0.05	0.02	[0.02, 0.08]	0.06	2.86	0.004	0.072
wind	−0.69	0.27	[-1.22, −0.17]	−0.23	−2.58	0.010	0.171
temperature	0.21	0.13	[-0.04, 0.47]	0.18	1.64	0.102	1
Interaction Terms							
cold weather response x temperature	−0.09	0.02	[-0.13, −0.04]	−0.35	−3.79	<0.001	0.003
windy weather response x wind	0.12	0.05	[0.02, 0.22]	0.23	2.29	0.022	0.381
warm weather response x temperature	0.004	0.02	[-0.03, 0.04]	0.01	0.22	0.826	1

Note. Results are from a multiple linear regression model predicting Patient Health Questionnaire-9 (PHQ-9) total scores. Subjective environmental mood responses were assessed using the weather and light items of the Seasonal Pattern Assessment Questionnaire and reflect self-reported changes in mood and energy in response to specific environmental conditions (cold, warm, sunny, dry, rainy, cloudy, and windy weather, as well as reduced and increased daylight). Objective weather variables were derived from data provided by the Icelandic Meteorological Office and represent average temperature (°C), relative humidity (%), and wind speed (m/s) over the 14 days preceding survey completion. Interaction terms between subjective mood responses and objective weather variables were included in the model. The model was adjusted for age and gender, and reported p-values are Bonferroni-adjusted to account for multiple comparisons.

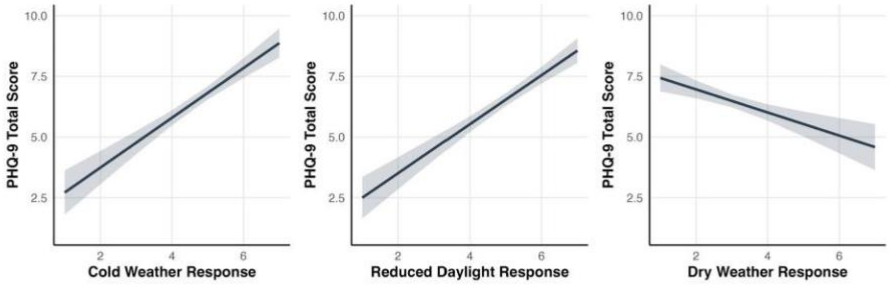


Fig. 1. Associations between environmental mood responses and depressive symptom severity (PHQ-9 total score).
Subjective environmental mood responses were assessed on 7-point Likert scales, reverse-coded so that higher values reflect more adverse mood responses (1 = Very favourable, 2 = Moderately favourable, 3 = Slightly favourable, 4 = No effect, 5 = Slightly adverse, 6 = Moderately adverse, 7 = Very adverse). The plots display estimated Patient Health Questionnaire-9 (PHQ-9) total scores based on self-reported mood responses to cold weather, reduced daylight, and dry weather. Estimates were derived from a linear regression model including interaction terms and are displayed with 95% confidence intervals (shaded areas). Estimated PHQ-9 scores represent model-based means and may include decimal values, although actual scores are integers.

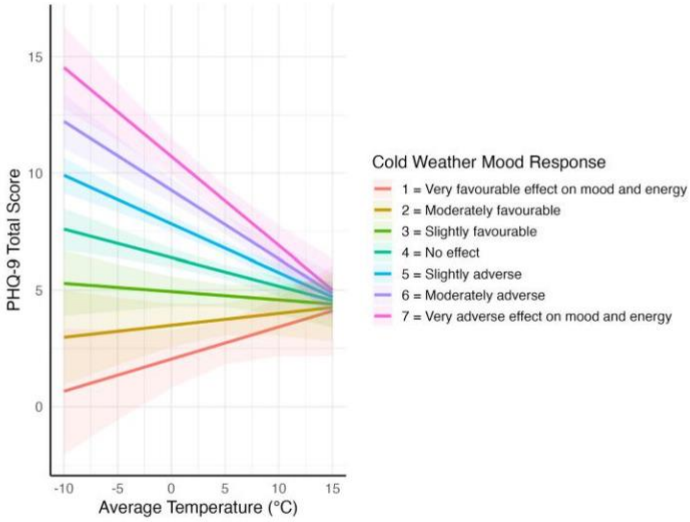


Fig. 2. Interaction between cold weather mood response and objectively measured temperature in estimating depressive symptom severity (PHQ-9 total score).

4.2. Are the objective weather variables, temperature, humidity, and wind speed, associated with depressive symptom severity?

In contrast to our hypothesis that objective weather indicators would be associated with depressive symptom severity, we found no significant effects. This mirrors mixed findings in the literature, with some studies reporting associations (Brazienė et al., 2022; Sigmon et al., 2009) and others finding none (Huibers et al., 2010), suggesting that temperature, wind speed, and humidity may play a limited role after controlling for subjective environmental mood responses and demographic factors. Another possibility is that the use of 14-day average weather conditions may have smoothed over short-term fluctuations that could influence mood, suggesting that finer-grained temporal analyses, such as examining day-to-day variability, might reveal stronger associations, an explanation also put forward by other researchers (Huibers et al., 2010).

4.3. Do interactions between individuals' mood responses to environmental conditions and actual weather conditions further explain variability in depressive symptom severity?

In our study, a significant interaction between cold weather mood responses and temperature indicated that weather influenced mood more strongly among individuals with either adverse or favourable responses to cold. Those with more adverse responses reported higher depressive symptoms at lower temperatures and fewer symptoms as temperatures rose, whereas those with favourable responses showed the opposite pattern. By contrast, the cold response–temperature interaction was minimal among individuals reporting no mood response to cold. This crossover effect, in which individuals with opposing responses exhibit opposite mood patterns as temperature changes, supports the validity of self-reported environmental mood responses and suggests

that people are often aware of how weather conditions affect their mood.

Our finding aligns with the differential susceptibility model, which posits that certain individuals are more emotionally responsive to environmental influences, exhibiting greater vulnerability under adverse conditions and greater benefit under favourable ones (Belsky & Pluess, 2009).

Overall, our results contrast with a previous study, which found that higher temperatures, in general, have protective effects on depressive symptoms (Brazienė et al., 2022). Instead, our findings highlight that the psychological impact of temperature is not determined solely by the environment, but by how it is subjectively experienced. Building on the observed interaction between temperature and individual mood responses, our findings are particularly relevant given the severe climatic changes currently underway. Global climate change, with increasing weather variability and extremes, is likely to disproportionately affect individuals who are sensitive to weather and light conditions, placing additional psychological strain on these vulnerable groups.

4.4. Unexpected findings and significant covariates

An unexpected finding was that mood worsening in response to dry weather was significantly associated with lower depressive symptom severity. One potential explanation is that this association may be confounded by other environmental factors that tend to co-occur with dry weather, such as increased sunlight, clearer skies, or more opportunities for outdoor activity, which are generally linked to improved mood (McMahan & Estes, 2015; Ryan et al., 2010). It is also possible that participants interpreted "dry weather" differently, such as experiencing dry cold (e.g., clear, crisp winter days without precipitation) versus dry heat (e.g., hot, arid conditions with low humidity), which could affect mood in distinct ways.

Demographic covariates also contributed to variability in mood outcomes, independent of subjective environmental mood responses and recent weather. Younger age was associated with higher levels of depressive symptom severity. Younger individuals also report higher seasonal fluctuation in mood and behaviour across multiple countries (Magnusson, 2000), including Iceland (Höller et al., 2021; Magnusson & Stefansson, 1993; Zelinski et al., 2025). Although we did not examine interactions between demographic factors and environmental mood responses or conduct subgroup analyses, existing research suggests that age is associated with such responses in mood-related outcomes (Dam et al., 1998; Kööts et al., 2011). For instance, in a large Danish study, younger participants reported stronger adverse responses to reduced daylight, whereas older adults reported more favourable effects from dry air (Dam et al., 1998). These findings underscore the importance of considering both individual mood responses and demographic characteristics when investigating variability in mood.

4.5. Limitations and future research

Several limitations should be considered when interpreting the present findings, including uncertainty around actual weather exposure, Iceland-specific environmental factors, and potential constraints on generalizability. Although we used a 14-day average of weather conditions at participants' places of residence to provide a more stable estimate of typical environmental exposure, we cannot be certain where participants were during that period, whether indoors or outdoors, in transit, in another region, or outside the country. This uncertainty limits the precision of our weather exposure estimates, though averaging across two weeks likely helped reduce the impact of short-term location changes or daily fluctuations. While Iceland's highly variable weather and extreme seasonal daylight fluctuations offer a unique setting for studying mood–environment dynamics, these same features may also limit the extent to which the findings can be applied. For example, the psychological impact of a sunny day may be more pronounced in Iceland, where overcast conditions are frequent, than in regions with more stable or sunnier climates.

To improve external validity, future research should aim to replicate these findings across a range of locations, extended timeframes, and all seasons. Although our study includes data collected across all seasons, the cohort design limits the ability to directly observe individual changes across seasons. Therefore, longitudinal studies tracking the same individuals over time would be better suited to capture the effects of seasonal mood variations. Furthermore, future research could benefit from incorporating data on time spent outdoors, physical activity, sleep, and personality traits to clarify if weather-related changes in mood can be explained by factors beyond individuals' subjective responses to weather and light conditions. Poor weather has been shown to reduce participation in outdoor and physically active leisure, promoting instead more sedentary, home-based behaviours (Spinney & Millward, 2011). Additional evidence indicates that sleep and activity partially mediate the relationship between maximum temperature and mood in individuals with bipolar disorder, suggesting that behavioural rhythms may play a meaningful, though not exclusive, role in shaping weather-related affective outcomes (Bullock et al., 2017). Moreover, prior research suggests that individual differences such as personality traits like neuroticism and extraversion (Kööts et al., 2011), as well as time spent outdoors (Keller et al., 2005; Kööts et al., 2011) may moderate the effects of weather on mood. These findings imply that environmental mood responses may reflect not only internal emotional reactivity but also co-occur with weather-related behavioural changes that influence mood.

5. Conclusions

The present study provides novel evidence that individual differences in mood responses to specific environmental conditions, particularly cold weather, dry weather, and reduced daylight, are significantly associated with variability in depressive symptom severity. Moreover, our findings extend previous research (e.g., Mersch et al., 2004; Sigmon et al., 2009, 2010) by showing that the association between environmental mood responses and depressive symptoms is shaped by actual weather conditions. Most interestingly, a significant interaction between subjective responses to cold weather and measured temperature showed that temperature influenced depressive symptoms differently across individuals. Those reporting adverse responses had higher depressive symptoms at lower temperatures, which decreased as temperatures rose, whereas those reporting favourable responses showed the opposite pattern, with symptoms increasing as temperatures rose. Individuals reporting no mood response showed only minimal changes.

These findings emphasize that environmental exposures and subjective experiences jointly shape vulnerability to depression, highlighting the complex interplay between people and their environments. Such insights are particularly relevant in the context of global climate change, where increasing weather variability and extremes may disproportionately affect those most sensitive to environmental conditions. Brief, low-cost assessments of environmental mood responses could therefore serve as valuable tools for identifying individuals at heightened risk for depressive symptoms.

CRediT authorship contribution statement

Lada Zelinski: Writing – review & editing, Writing – original draft, Visualization, Methodology, Formal analysis, Conceptualization. **Harpa Hlín Gunnarsdóttir:** Writing – review & editing, Methodology, Conceptualization. **Ragnar Pétur Ólafsson:** Writing – review & editing, Supervision. **Yvonne Höller:** Writing – review & editing, Supervision, Methodology, Funding acquisition, Conceptualization.

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Declaration of competing interest

The authors have no competing interests to declare.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jenvp.2026.102990.

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Article III

Energy Drink Consumption and Depressive Symptoms in Iceland: Associations with Season and Circadian Preference

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Abstract

Background: Energy drinks are among the fastest-growing segments of the global beverage market, and their consumption is increasingly recognised as a public health concern, especially given their associations with sleep disturbances and depressive symptoms. These outcomes are also shaped by circadian preference and seasonal variation in daylight, with evening types reporting higher levels of insomnia and depressive symptoms as well as more frequent energy drink consumption. Yet no study has examined how energy drink intake, circadian preference, and seasonal variation jointly relate to insomnia and depressive symptoms across seasons, or whether such associations are specific to energy drinks rather than coffee. Iceland, with its extreme seasonal variation in daylight, offers a unique setting to study these associations.

Methods: An Icelandic adult sample (N = 281) completed self-report measures across four seasons, comprising the Food Frequency Questionnaire (energy drink and coffee intake frequency), the Morningness–Eveningness Questionnaire (circadian preference), the Bergen Insomnia Scale, and the Depression Anxiety Stress Scales depression subscale. Cross-sectional associations were examined using multiple linear regression and longitudinal associations were examined using linear mixed-effects models.

Results: Cross-sectionally, energy drink intake frequency was positively associated with insomnia (p = .023), whereas no such association emerged for coffee. Longitudinal analyses revealed a significant three-way interaction, whereby the association between energy drink intake frequency and depressive symptoms was amplified in autumn and was strongest among evening types compared to morning (p = .032) and intermediate types (p = .013), a pattern again absent for coffee.

Conclusions: Evening types who frequently consume energy drinks may represent a particularly vulnerable subgroup, especially as daylight rapidly decreases in autumn. These findings underscore the importance of considering circadian preference and seasonal context when examining the health effects of energy drink consumption, and support calls for greater public health attention to this group.

Keywords:

Energy drinks; Circadian preference; Insomnia; Depression; Seasonal variation

1. Background

Energy drink intake has become increasingly popular, largely driven by advertising that promotes enhanced mental and physical performance and active lifestyles (1). This marketing has contributed to their position as one of the fastest-growing segments of the beverage industry with rapid market expansion since the late 20th century (2,3). Energy drinks vary in composition, with caffeine content ranging from approximately 50 mg to over 500 mg per can or bottle (3). Beyond caffeine as the main ingredient, they typically include sugar or sweeteners, taurine, water-soluble B vitamins (e.g., riboflavin, pyridoxine, and nicotinamide), and compounds such as glucuronolactone and herbal extracts (e.g., ginseng, guarana) (3,4) Notably, certain herbal extracts, such as guarana, contribute additional caffeine that is not required to be explicitly declared on product labels, potentially leading to an underestimation of the total caffeine content per serving (5).

Coffee is a traditional source of caffeine, typically consumed early in the day (6). In contrast, energy drinks are frequently consumed at more variable times throughout the day (6) and in different contexts, often to support academic work or in nightlife settings where they are frequently mixed with alcohol (7). Energy drink intake is also more prevalent among adolescents and young adults (8), which may be partly explained by age-based differences in perceptions. For example, one study found that younger participants framed the consumption of energy drinks in positive terms (e.g., a “kick” or “hit”), whereas older participants emphasized health concerns and described energy drinks as a “last resort” (9). Besides young age, energy drink intake has been associated with male gender (10–13). These demographic patterns mirror marketing strategies that construct the public image of energy drinks around themes of extreme sport, masculinity, and risk-taking, while promoting claims of enhanced alertness, physical performance, and cognitive functioning (1,13). However, empirical support for the claimed performance-enhancing and cognitive benefits of energy drinks remains limited (14). Beyond the effects of caffeine, which can temporarily reduce fatigue and increase attention (15), there is little evidence from randomized, placebo-controlled studies that energy drinks confer additional benefits (16).

In addition to demographic factors, individual differences in circadian preference were reported to play an important role in energy drink intake patterns (17–19). Circadian preference, as a primary proxy of chronotype, refers to individual differences in preferred timing of sleep and daily activities, and is commonly assessed using the Morningness–Eveningness Questionnaire (MEQ) to derive classifications of morning, intermediate, and evening types (20). Specifically, individuals with an evening type (i.e., a preference for later sleep–wake timing) are more likely to consume energy drinks than those with a morning type (17–19). One possible explanation is that evening types are more likely to experience misalignment between their biological rhythms and socially imposed schedules, a phenomenon known as social jetlag (21), which may increase reliance

on stimulants (22). Consistent with this, eveningness has also been associated with a range of less favourable lifestyle behaviours, such as alcohol use, smoking, lower physical activity, and unhealthier diet (22–25), and has further been linked to higher levels of depressive symptoms (26) and insomnia (27).

Similarly, energy drink intake has been linked to a growing range of negative health effects, particularly sleep disturbances (10,28). In a recent systematic review, it was found that energy drink use in adolescents is consistently associated with poorer sleep quality and shorter sleep duration (10).

Further evidence suggests that the timing of consumption may also play an important role, with later-day energy drink intake being particularly associated with reduced sleep duration among Icelandic adolescents (29), a population that tends to shift toward eveningness during adolescence (30). This is consistent with experimental evidence showing that caffeinated energy drinks can directly disrupt sleep, as demonstrated in a double-blind, placebo-controlled trial in which athletes consuming caffeinated energy drinks experienced more side effects, including insomnia, than those receiving a decaffeinated placebo (28). Importantly, this relationship may be bidirectional, as insufficient sleep can increase reliance on caffeinated beverages to counteract daytime sleepiness, while caffeine intake can further disrupt subsequent sleep, thereby reinforcing a self-perpetuating cycle (31).

While the association between energy drink intake and sleep is relatively well established, consistent with the well-documented effects of caffeine, research has also linked energy drinks to depressive symptoms (32–35). For instance, one study among Canadian high school students found that energy drink users reported a higher risk of depressive symptoms than non-users, with the greatest risk observed among frequent consumers (32). Also, in a large cross-sectional study of 18–24-year-old university students in Norway, a higher frequency of energy drink intake was associated with increased depressive symptoms in both men and women, with sleep duration partially mediating the association (35). Further longitudinal evidence of the energy drink-depression association revealed that young adult men transitioning from non-use to energy drink use over two years showed increases in depressive symptoms, whereas no such changes were observed in women (34). Despite several studies reporting positive associations, findings are not entirely consistent. For example, one study reported higher depression scores among regular energy drink users at one of three university sites, while no significant differences were observed at the remaining sites (33). Importantly, much of the evidence linking energy drink intake to sleep and depressive outcomes is derived from adolescent and university student samples, potentially limiting its generalisability to other age groups and underscoring the need for research in more diverse populations.

Besides behavioural factors such as energy drink intake, environmental influences may also play an important role in shaping sleep and mood outcomes.

Seasonal influences are often underappreciated, despite evidence that they affect a wide range of affective, cognitive, and behavioural processes (36). Research shows that depressive symptoms vary across seasons, with reduced daylight during autumn and winter associated with lower positive affect (37) and the presence of depressive symptoms in non-clinical populations (38,39). In more severe cases, this seasonal pattern may manifest as Seasonal Affective Disorder (40). Sleep patterns also fluctuate seasonally, with reduced light exposure linked to delayed sleep timing (41), longer sleep duration (42), as well as greater difficulty initiating sleep and increased daytime impairment (43). Notably, these effects appear stronger in higher-latitude regions. For instance, one study reported minimal seasonal variation in insomnia and mood in Ghana, compared to moderate-to-strong effects in Norway, suggesting that greater variation in daylight amplifies seasonal sensitivity (41).

Taken together, prior findings highlight the frequency of energy drink intake as an important factor associated with sleep and mood, in addition to environmental influences such as seasonal variation (37,43) and individual differences in circadian preference or morningness–eveningness (26,27). Notably, individuals with an evening preference may be particularly vulnerable in this context, as they are more prone to experience social jetlag (21) and to consume energy drinks more frequently (17,18). This suggests that eveningness and energy drink consumption may jointly relate to sleep and mood, a relationship that may be further exacerbated during periods of reduced daylight, such as autumn and winter, when circadian rhythms are more easily disrupted and disturbances in sleep and mood become more prevalent (38,39,43).

However, existing research has largely examined these factors in isolation. There is a lack of studies investigating the combined effects of energy drink intake, circadian preference, and seasonal variation on sleep and mood outcomes. Moreover, it remains unclear whether observed associations are attributable to caffeine consumption in general or to specific sources, such as energy drinks, which contain additional stimulants and additives beyond caffeine (3,4), compared with coffee. Importantly, these sources also differ in their typical patterns of use, with energy drinks more often consumed at irregular or later times of day, and coffee typically consumed earlier and in more habitual patterns (6,7). As such, comparing these sources may help clarify whether associations with sleep and mood reflect general caffeine effects or are more pronounced for energy drinks specifically.

The aim of the present study was to investigate the associations between energy drink intake frequency, circadian preference (i.e, Morningness-Eveningness-Type), and seasonal variation in relation to depressive and insomnia symptoms, while using coffee intake as a comparison source of caffeine. Iceland provides a particularly informative setting for examining these relationships. Due to its high latitude (approximately 65°*N*), the country experiences extreme

seasonal variation in daylight, with about 4 hours at the winter solstice and up to 24 hours during summer. This pronounced fluctuation provides a natural setting to investigate how seasonal changes in light exposure interact with circadian preference and behavioural factors, such as energy drink intake frequency, to shape sleep and mood outcomes.

The following hypotheses were formulated: (H1) Energy drink intake frequency will be positively associated with insomnia and depressive symptoms, whereas no such associations are expected for coffee intake frequency. (H2) Circadian preference will be associated with insomnia and depressive symptoms, such that individuals with an evening preference will report higher symptom levels than those with intermediate or morning preference. (H3) There will be a three-way interaction between energy drink intake frequency, season, and circadian preference in predicting depressive and insomnia symptoms, such that the highest symptom levels are expected among evening types with more frequent energy drink intake during autumn and winter; no comparable interaction effects are expected for coffee intake frequency. Given prior evidence linking age and sex to both energy drink consumption (8,10) and mental health outcomes (44–46), all analyses controlled for these variables as covariates.

The present study includes both cross-sectional analyses at baseline (summer) assessment and longitudinal analyses across all four seasons, enabling examination of both concurrent associations and seasonal variation in these relationships over time.

2. Methods

2.1 Ethical Approval

This study was approved by the National Bioethics Committee of Iceland (approval number VSN-21-088). All researchers signed agreements to ensure the confidentiality of personal data. Participants gave informed consent before participation and after receiving information about the study's purpose, procedures, and their right to withdraw at any time.

2.2 *Recruitment*

Participants were recruited via email and social media. Invitations were distributed to companies, organizations, and students and staff at the University of Akureyri. Recruitment materials included an informational letter and a YouTube video describing the study.

2.3 Longitudinal Study Design and Procedure

The present study employed a prospective longitudinal design with repeated measurements over one calendar year. Data were collected in each meteorologically defined season: summer (June–August), autumn (September–November), winter (December–February), and spring (March–May). Data collection began in summer 2021, which served as the baseline measurement point. At each new measurement period, participants received follow-up

invitations and reminder messages to support continued participation. To further improve retention, non-respondents were contacted by phone when possible. To encourage sustained engagement across all four waves, participants who completed every assessment were entered into a draw for gift certificates.

2.4 Survey Instruments

At each seasonal assessment, participants completed an online survey in Icelandic via Microsoft Forms, which included demographic questions and nine standardized questionnaires. For the present analysis, only data from the Depression Anxiety Stress Scales-21 (DASS) (47), the Bergen Insomnia Scale (BIS) (48), the Food Frequency Questionnaire (FFQ) (49,50), and the Morningness-Eveningness Questionnaire (MEQ) (20) were used.

2.4.1 Food Frequency Questionnaire (FFQ)

Coffee and energy drink intake frequency were assessed using the FFQ, which included 20 items covering various food and drink groups. The FFQ was adapted from the AGES-Reykjavík Study (49) and the Icelandic National Nutritional Survey 2010–2011 (50) and assesses intake frequency over the past three months. Frequency of coffee and energy drink intake was reported on a 7-point scale (1 = less than once per month; 2 = 1–3 times per month; 3 = 1–3 times per week; 4 = 4–7 times per week; 5 = 2–3 times per day; 6 = 4–5 times per day; 7 = more than 5 times per day), with participants responding separately for each of these two beverages.

2.4.2 Depression Anxiety Stress Scales (DASS)

Mood was assessed using the DASS-21 (47), a brief self-report measure consisting of 21 items designed to evaluate symptoms of the three subscales: depression, anxiety, and stress. Each subscale includes seven items, and participants indicated how much each statement described their experiences during the past week using a 4-point Likert scale ranging from 0 (did not apply to me at all) to 3 (applied to me very much or most of the time), with higher scores reflecting more severe symptoms.

The current study employed the Icelandic version of the DASS (as used in (51)). Previous research has shown that this version demonstrates good internal consistency with Cronbach's alpha subscale values ranging from 0.90 to 0.96 (51). In the present study, only the depression subscale was included in the analyses. The depression subscale demonstrated good internal consistency across all four measurement periods (see Table 1).

2.4.3 Bergen Insomnia Scale (BIS)

Sleep quality was measured using the BIS (48), a self-report instrument measuring subjective insomnia symptoms. Each of the six items on the BIS is rated on an 8-point Likert scale, indicating the number of days per week (0 to 7) during the past month that specific sleep difficulties occurred. Scores from all items are summed to produce a total score ranging from 0 to 42, with higher scores reflecting more severe insomnia symptoms.

The Icelandic version of the BIS was employed, which had been previously translated and validated through a translation and back-translation procedure, and demonstrated acceptable internal consistency (Cronbach's alpha = .76; (51)). The BIS demonstrated good internal consistency across all four time points (see Table 1).

2.4.4 Morningness-Eveningness Questionnaire (MEQ)

The MEQ (20) is a 19-item self-report measure designed to assess individual differences in circadian preference, often used to estimate chronotype. Depending on the item, responses are provided on a 4- or 5-point scale, with some items presented as visual analogue scales. Total scores range from 16 to 86, with lower scores indicating a stronger evening preference and higher scores reflecting a stronger morning preference. The MEQ total score demonstrated acceptable internal consistency across all four measurement periods (see Table 1), and the original MEQ has shown good psychometric properties, with reliability coefficients between 0.78 and 0.86 (52–54) and test–retest correlations of 0.88 over three months (55) and 0.89 over two months (54).

Based on total scores, individuals can be categorized into five types: 16–30 = Definite Evening; 31–41 = Moderate Evening; 42–58 = Intermediate (Neither); 59–69 = Moderate Morning; and 70–86 = Definite Morning. For the present study, these categories were collapsed into three circadian preference groups by combining the definite and moderate types, resulting in morning, intermediate, and evening types. Analyses were conducted using these three categories rather than the continuous MEQ total score.

The Icelandic version used in the present study had been previously developed through a translation and back-translation process and demonstrated acceptable internal consistency (Cronbach's alpha = .72; (51)).

Table 1 Cronbach's Alpha Values for Study Measures Across Seasons

Measure	Summer	Autumn	Winter	Spring
DASS-21 Depression	0.89	0.92	0.88	0.90
Bergen Insomnia Scale	0.79	0.83	0.82	0.80
Morningness-Eveningness Questionnaire	0.79	0.78	0.77	0.79

2.5 Data Analysis

Statistical analyses were performed using R (version 4.4.1; (56)). The DASS depression subscale, the BIS total score, and the MEQ total score were calculated for each participant.

Two sets of analyses were conducted. First, participants who completed the baseline (summer) assessment were analysed using multiple linear regression models implemented in base R. Separate models were fitted for each dependent variable (depressive symptoms and insomnia symptoms), with coffee and energy

drink intake frequency entered simultaneously as predictors in each model. Circadian preference, age, and sex were included as covariates. Scale scores were treated as continuous variables, consistent with common practice in the field.

Second, all available data from participants were included, noting that some participants missed one or more follow-up assessments, with some rejoining the study at a later time point. Data were analysed using linear mixed-effects models with a random intercept for each participant to account for repeated measurements. Models were fitted using maximum likelihood estimation using the R lme4 package, and t-tests were based on Satterthwaite's method. Separate models were fitted for each dependent variable and each caffeinated beverage. These models included interactions among beverage intake frequency, season, and circadian preference, with age and sex as covariates, allowing us to examine longitudinal effects.

P-values were interpreted against Benjamin–Hochberg corrected significance thresholds to control for multiple comparisons. A priori power analyses using the R pwr package indicated that a minimum sample of 172 participants was required to detect a medium effect ($f^2 = 0.15$; (57)) with 80% power and $\alpha = .05$.

3. Results

3.1 Baseline (Summer) - Sample Characteristics

A total of 282 participants completed the online survey at baseline in the summer, of which 223 (79.08%) identified as women, 58 (20.57%) as men, and 1 (0.35%) as non-binary. Because the number of participants identifying as non-binary was too small to permit meaningful statistical analysis, they were treated as missing in the regression models. The mean age at study entry was 44.53 years ($SD = 12.98$), with a median age of 45 years and a range of 19 to 75 years.

3.2 Cross-Sectional Associations between Caffeine Intake Frequency, Insomnia, and Depressive Symptoms in Summer

Both multiple regression models based on the summer data were statistically significant, including the depression model ($F(6, 256) = 7.55$, $p < .001$, adjusted $R^2 = .13$), and the insomnia model ($F(6, 256) = 4.37$, $p < .001$, adjusted $R^2 = .07$).

In summer, the frequency of energy drink intake was significantly associated with higher BIS scores, indicating more severe insomnia symptoms with more frequent energy drink intake (see *Fig. 1*). Coffee intake frequency was not significantly associated with BIS scores.

Neither the frequency of energy drink intake nor the frequency of coffee intake was significantly associated with DASS depression scores.

Circadian preference (MEQ) was significantly associated with both depression and insomnia. Specifically, individuals with morningness preference and those with an intermediate preference reported lower DASS depression and BIS scores than those with eveningness preference. Younger age was associated with higher

depression scores but was not significantly related to insomnia. Gender was not significantly associated with either depression or insomnia.

All associations between caffeine intake, covariates, and DASS depression and BIS scores in summer are presented in Table 2.

Table 2 Estimates for Associations Between Caffeine Intake Frequency, Depression, Insomnia, and Covariates in Summer

Explanatory Variable	B	SE	β	95% CI	t	p	p (BH)
DASS Depression							
FFQ Energy Drink	0.39	0.21	0.12	[-0.01, 0.24]	1.84	.067	.094
FFQ Coffee	-0.16	0.12	-0.08	[-0.21, 0.04]	-1.32	.187	.218
MEQ (Morning Type)	-2.84	0.87	-0.71	[-1.14, -0.28]	-3.26	.001	.004
MEQ (Intermediate Type)	-1.99	0.75	-0.50	[-0.87, -0.13]	-2.64	.009	.021
Age	-0.05	0.02	-0.17	[-0.30, -0.04]	-2.51	.013	.022
Gender (Women)	0.22	0.57	0.05	[-0.23, 0.34]	0.38	.705	.705
BIS							
FFQ Energy Drink	1.25	0.50	0.16	[0.03, 0.29]	2.49	.013	.023
FFQ Coffee	-0.12	0.29	-0.03	[-0.15, 0.10]	-0.41	.686	.686
MEQ (Morning Type)	-7.31	2.04	-0.81	[-1.25, -0.36]	-3.58	<.001	.001
MEQ (Intermediate Type)	-4.58	1.77	-0.51	[-0.89, -0.12]	-2.59	.010	.023
Age	0.05	0.05	0.07	[-0.07, 0.20]	0.96	.338	.394
Gender (Women)	2.59	1.34	0.29	[-0.01, 0.58]	1.93	.054	.076

Note. BIS = Bergen Insomnia Scale; DASS = Depression, Anxiety, and Stress Scales; FFQ = Food Frequency Questionnaire; MEQ = Morningness–Eveningness Questionnaire. *P*-values adjusted using Benjamin-Hochberg (BH) correction.

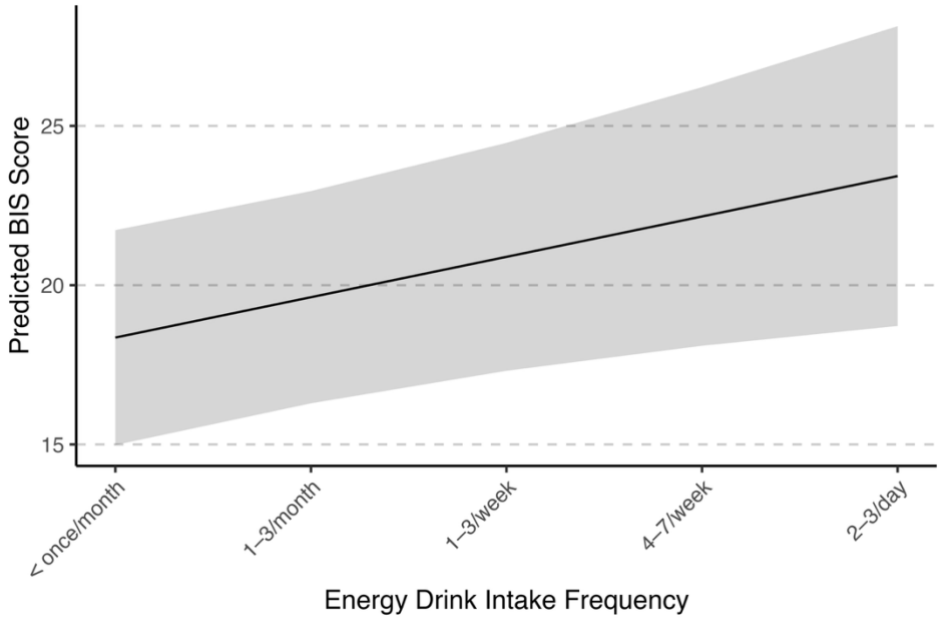


Fig 1. Association Between Energy Drink Intake Frequency and Insomnia Symptoms in Summer. Predicted insomnia symptoms (BIS scores) are shown across energy drink intake frequency, controlling for coffee intake frequency, age, gender, and circadian Preference (Morningness-Eveningness-Type). Shaded areas represent 95% confidence intervals from the linear regression model.

3.3. Longitudinal - Sample Characteristics

Of the 281 participants at baseline in summer, 245 (87.19%) completed follow-up 1 in autumn, 117 (41.64%) follow-up 2 in winter, and 191 (67.97%) follow-up 3 in spring. Longitudinal analyses included all participants, including those with only baseline data.

No significant seasonal differences were found for beverage (i.e., energy drink, coffee) intake frequency, psychological outcome measures (i.e., DASS depression, BIS), circadian preference (i.e., MEQ), and gender (all $p > .05$; see Table 3). Age differed significantly across seasons ($p < .001$; see Table 3).

Table 3 Caffeinated beverage intake frequency, DASS depression, BIS, MEQ-type, Age, and Gender across Seasons

Study Variables	Summer (N = 281)	Autumn (N = 245)	Winter (N = 117)	Spring (N = 191)	Test Statistic
DASS Depression	2 (0-21)	2 (0-21)	3 (0-17)	2 (0-21)	F(3,560.49) =0.760, p =.517
BIS	13 (0-42)	13 (0-38)	15 (0-40)	12 (0-42)	$F(3, 552.1) = 0.482$, $p = .695$
FFQ Energy Drink	1 (1-5)	1 (1-5)	1 (1-5)	1 (1-5)	$F(3, 505.15) = 1.08$, $p = .356$
FFQ Coffee	5 (1-7)	5 (1-7)	4 (1-7)	5 (1-7)	$F(3, 516.84) = 2.60$, $p = .052$
MEQ	E = 31, I = 183, M = 67	E = 25, I = 171, M = 49	E = 11, I = 83, M = 23	E = 17, I = 126, M = 48	$\chi^2(3) = 4.63$, $p = .201$
Age	44 (19-75)	45 (19-76)	43 (20-75)	47.5 (20-75)	$F(3, 542.04) = 148.47$, $p < .001$
Gender	Men = 58, W = 223	Men = 53, W = 192	Men = 23, W = 94	Men = 44, W = 147	$\chi^2(3) = 0.62$, $p = .892$

Note. DASS = Depression, Anxiety, and Stress Scales; FFQ = Food Frequency Questionnaire; MEQ = Morningness–Eveningness Questionnaire; E = Evening type, I = Intermediate type; M = Morning type; W = Women. Values are reported as median (range) for continuous/ordinal measures and counts for categorical measures. Statistical comparisons across seasons were performed using ANOVA for continuous/ordinal variables (DASS depression, BIS, FFQ Energy Drink, FFQ coffee) and chi-square tests for categorical variables (MEQ, Gender).

3.4 Longitudinal findings

3.4.1 Depression

Significant results after correction for multiple comparisons are presented in Table 4, whereas all tested effects, including nonsignificant ones, are reported in Tables S1 and S2.

Energy Drink:

After adjusting for multiple comparisons, a significant two-way interaction effect between energy drink intake frequency and season (autumn vs. summer) was found (p = .008), indicating that, for the reference group (evening type), the positive association between energy drink intake frequency and depression was stronger in autumn than in summer.

Significant three-way interactions emerged between energy drink intake frequency, season (autumn), and circadian preference. Specifically, these interactions were significant for both the intermediate (p = .013) and morning (p

= .032) preference groups, indicating that the autumn-specific increase in the association between energy drink intake and depression differed by circadian preference. Specifically, the positive association observed in autumn among evening types was significantly attenuated in both intermediate and morning types (see *Fig. 2* and *Fig. S1* for all-season comparisons).

To explore the temporal direction of this association, post-hoc correlational analyses were conducted. Summer depressive symptoms were not significantly associated with change in energy drink intake frequency from summer to autumn (r = .02, p = .781), nor was summer energy drink intake frequency significantly associated with change in depressive symptoms from summer to autumn (r = .11, p = .101).

Coffee:

After adjusting for multiple comparisons, a significant main effect of the autumn season was found (p = .047), indicating that participants in the reference group (evening type) reported higher depression in autumn than in summer. Additionally, a significant two-way interaction effect between season (autumn) and intermediate type was observed (p = .047), such that the association between autumn and depressive symptoms was weaker among intermediate types than evening types (see *Fig. S2*).

Age:

A significant main effect of age was observed across both the energy drink model and the coffee model, indicating that higher age was associated with lower depression scores (p < .001).

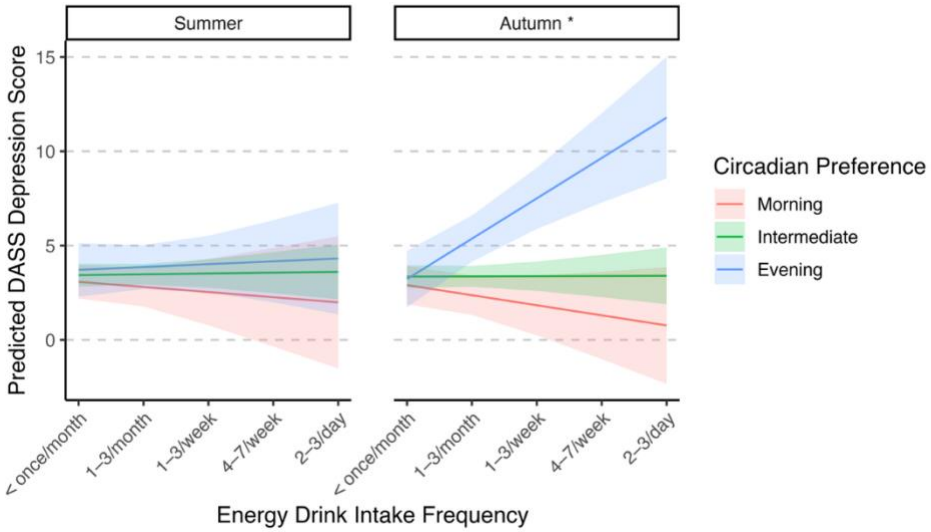


Fig. 2 Predicted DASS Depression scores across energy drink intake frequencies for Summer (reference) and Autumn, separated by Circadian Preference (Morningness-Eveningness-Type). The asterisk denotes a statistically significant three-way interaction, indicating that the positive association between energy drink intake frequency and depression was strongest among evening types and attenuated in intermediate and morning types in autumn. Shaded areas represent 95% confidence intervals. Predicted scores are shown controlling for age and gender.

3.4.2 Insomnia Symptoms

All tested effects, including nonsignificant ones, are reported in Tables S3 and S4.

Across both the energy drink model and the coffee model, no significant main or interaction effects were observed for insomnia symptoms with respect to caffeinated beverage intake frequency, season, or circadian preference after correction for multiple comparisons (all adjusted $p \geq .05$). However, a significant main effect of gender was observed across both the energy drink model ($p = .009$) and the coffee model ($p = .004$), indicating that a higher insomnia score was found in women compared to men (see Table 4).

Table 4. Caffeine Intake Frequency, Covariates, and Their Associations with Depression and Insomnia

Explanatory Variable	*B*	*SE*	*β*	*95% CI*	*t*	*p*	*p (BH)*
DASS Depression ~ **FFQ Energy Drink** × Season × Circadian Preference + Age + Gender							
*Energy Drink * Autumn*	1.95	0.58	0.56	[0.23, 0.89]	3.34	.001	.008
*Energy Drink * MEQ (Morning Type) * Autumn*	-2.24	0.81	-0.64	[-1.10, -0.18]	-2.75	.006	.032
*Energy Drink * MEQ (Intermediate Type) * Autumn*	-1.98	0.64	-0.57	[-0.93, -0.21]	-3.11	.002	.013
Age	-0.09	0.02	-0.27	[-0.37, -0.17]	-5.45	< .001	< .001
DASS Depression ~ **FFQ Coffee** × Season × Circadian Preference + Age + Gender							
Season (Autumn)	4.12	1.53	0.27	[-0.08, 0.62]	2.70	.007	.047
*Autumn * MEQ (Intermediate Type)*	-4.56	1.68	-0.31	[-0.69, 0.08]	-2.71	.007	.047
Age	-0.09	0.02	-0.28	[-0.37, -0.18]	-5.58	< .001	< .001
BIS ~ **FFQ Energy Drink** × Season × Circadian Preference + Age + Gender							
Gender Woman	4.07	1.14	0.43	[0.19, 0.67]	3.57	< .001	.009
BIS ~ **FFQ Coffee** × Season × Circadian Preference + Age + Gender							
Gender Woman	4.20	1.15	0.44	[0.21, 0.68]	3.66	< .001	.004

Note. DASS = Depression, Anxiety, and Stress Scales; FFQ = Food Frequency Questionnaire; MEQ = Morningness–Eveningness Questionnaire. *P*-values adjusted using Benjamin-Hochberg (BH) correction.

4. Discussion

The present study investigated the associations between energy drink intake frequency, circadian preference (Morningness-Eveningness-Type), and seasonal variation in relation to insomnia and depressive symptoms in Iceland, using coffee intake frequency as a comparison caffeine source.

The findings provided partial support for the hypotheses. Energy drink intake frequency was associated with insomnia, but not depressive symptoms, cross-sectionally, and eveningness was consistently linked to both outcomes. Most notably, longitudinal analyses revealed that the association between energy drink

intake frequency and depressive symptoms was amplified in autumn and was strongest among evening types, a pattern that did not emerge for coffee.

4.1 Cross-Sectional Associations between Energy Drink Intake Frequency, Insomnia, and Depression (H1)

In the cross-sectional analyses, energy drink intake frequency was positively associated with insomnia symptoms, whereas no association emerged for coffee. Given that both beverages contain caffeine, this pattern suggests that the sleep-related effects are not attributable to caffeine alone but may reflect factors more specific to energy drinks, including their additional stimulants and higher sugar content (3,4), as well as their tendency to be consumed later in the day (6,7). Our findings are also supported by a study in university students, which reported that high-frequency energy drink users had later bedtimes, greater difficulty falling asleep, and more all-nighters than less frequent consumers, whereas such differences were not observed between high-frequency and low-frequency coffee drinkers (58). The present finding extends this association between energy drinks and insomnia beyond student and athlete samples (10,28) to a broader adult age range (i.e., 19 to 76 years) in a high-latitude population experiencing almost continuous daylight at the time of assessment.

Contrary to our hypothesis, energy drink intake frequency was not significantly associated with depressive symptoms in the cross-sectional analysis. This is inconsistent with prior studies reporting positive associations between energy drink consumption and depression in adolescent and young adult samples (32,35). A potential explanation for the absence of a cross-sectional association between energy drink intake frequency and depression, in contrast to insomnia, may relate to the differing timescales over which these outcomes develop. Insomnia may be a relatively immediate response to caffeine intake, as experimental evidence shows that caffeine can disrupt sleep even when consumed up to six hours before bedtime (59), suggesting that such an association could be detectable even at a single time point. Depression may instead develop indirectly over time, with repeated nights of caffeine-disrupted sleep representing one potential pathway, consistent with longitudinal evidence showing that sleep disturbances may precede the onset of depression (60), making such an association less likely to be detectable at a single time point.

4.2 Cross-Sectional Associations between MEQ-Type, Insomnia, and Depression (H2)

In line with our second hypothesis, circadian preference was significantly associated with both insomnia and depressive symptoms in the cross-sectional analyses. Individuals with greater eveningness reported higher levels of insomnia and depressive symptoms, with morning types showing the lowest symptom levels and intermediate types falling in between. To our knowledge, this is one of the first demonstrations of these associations under conditions of nearly continuous daylight. The present findings add to a substantial body of literature linking

eveningness to poorer sleep (27) and elevated depressive symptoms (26). Notably, standardised beta coefficients were larger for circadian preference than for energy drink intake frequency in both models, suggesting that it may be a more powerful correlate of sleep and mood disturbance. Furthermore, circadian preference, as an indicator of chronotype, remained a significant predictor of both outcomes after controlling for age, which is relevant given that chronotype shifts toward morningness with age (61), suggesting that circadian preference may have an independent association with insomnia and depressive symptoms.

Notably, circadian preference did not emerge as a significant main effect in the longitudinal analyses after correction for multiple comparisons, suggesting that it may not independently predict changes in symptoms across seasons. Rather than acting as a stable risk factor over time, it may function primarily as a moderator, interacting with behavioural factors such as energy drink intake under specific seasonal conditions, a possibility supported by the longitudinal findings.

4.3 Three-Way Interactions: Season, Energy Drink Intake Frequency, and MEQ-Type (H3)

The longitudinal analyses provided partial support for our third hypothesis. In contrast to the cross-sectional findings, no effects on insomnia symptoms emerged in either the energy drink or the coffee model. As discussed in section 4.1, insomnia may represent a more immediate response to energy drink intake, potentially making it less sensitive to the seasonal and chronotype-dependent patterns captured in the longitudinal analyses. In contrast, a significant three-way interaction indicated that the association between energy drink intake frequency and depressive symptoms differed by season and circadian preference. Specifically, in autumn, the positive association between energy drink intake frequency and depressive symptoms was strongest among evening types.

The finding that the interaction emerged specifically during autumn rather than winter is particularly noteworthy. One possible interpretation of this pattern is that the rate of change in daylight may matter more than the absolute amount. In Iceland, the contrast between the near-continuous daylight of summer and the low daylight levels of winter means that the autumn equinox marks a particularly sharp environmental transition, which may place greater demands on circadian adaptation than would be experienced at lower latitudes. Evening types, whose circadian rhythms are already misaligned with social schedules (21), may be particularly vulnerable to this rapid photoperiod transition. The present findings suggest that frequent energy drink intake may be associated with greater depressive symptoms among evening types during this period. A review identified two mechanisms that may underlie this association, namely self-medication, whereby individuals with low mood use energy drinks as a short-term boost, and sleep dysregulation, whereby energy drink consumption and sleep disturbance may mutually reinforce one another, though the direction of this relationship remains unclear (62). Our post-hoc correlational analyses did not support a self-

medication account, as summer depressive symptoms were not significantly associated with change in energy drink intake frequency from summer to autumn, nor was the reverse direction significant. Therefore, causal conclusions cannot be drawn from the present data.

In the coffee model, depressive symptoms were higher in autumn than in summer, with intermediate types showing a weaker seasonal effect than evening types. However, coffee intake frequency did not differentially predict depressive symptoms across chronotypes, as observed for energy drinks. The absence of a comparable effect for coffee may partly reflect the complex and dose-dependent relationship between caffeine and mood, with low to moderate intake associated with mood-enhancing effects and higher intake linked to anxiogenic effects (63). Such mood-enhancing effects in habitual consumers may, however, partly reflect the offsetting of withdrawal symptoms rather than a direct stimulant effect (64,65). Another explanation for the divergent findings may lie in differences in which energy drinks and coffee are typically consumed. Coffee is commonly consumed in social settings, such as work breaks and shared meals, whereas energy drinks are consumed across a wider range of contexts, including solitary, fatigue-driven situations and social settings, such as parties, where they are often mixed with alcohol (7). One study that used ecological momentary assessment in naturalistic settings found that the association between caffeinated beverage consumption and positive affect was moderated by social context, with mood-enhancing effects being stronger when consumed alone, though the study did not distinguish between caffeinated beverages in the analyses (66). Given the varied contexts in which energy drinks are consumed, future research directly assessing consumption context alongside beverage type would help clarify whether situational differences contribute to the divergent effects on mood across Morningness-Eveningness-Types and seasons. Taken together, the present findings suggest that evening types may represent a particularly vulnerable subgroup and given existing calls for greater regulatory oversight of energy drink marketing and consumption (67,68), targeted public health attention may be warranted, particularly during the autumn period.

4.4 Limitations and Strengths

Several limitations of the present study warrant consideration. First, energy drink and coffee intake were assessed solely by frequency, without accounting for serving size, timing, or other dietary sources of caffeine (e.g., tea), meaning that two participants with the same frequency score may have differed substantially in their actual caffeine exposure. Although the data included information on both beverages, sample sizes within consumer subgroups (i.e., exclusive energy drink consumers, exclusive coffee drinkers, concurrent consumers, and non-consumers) were too small to permit meaningful comparisons, particularly when further stratified by circadian preference. Future studies should therefore recruit larger samples to better isolate source-specific effects and examine whether energy

drink-specific effects would survive when total daily caffeine intake is held constant. Second, the present study relied on self-reported measures, which are susceptible to recall bias and, in the case of beverage intake, may not accurately capture habitual consumption patterns. Future studies could employ ecological momentary assessment of beverage intake to obtain more precise and temporally accurate exposure data. Third, the study did not control for other substances commonly co-consumed with energy drinks, such as alcohol, tobacco, and cannabis (69), whose independent effects on sleep, mood, and mental health could confound the observed associations. Future studies should adjust for these co-occurring behaviours to better isolate effects attributable to energy drink intake specifically. Fourth, the study cannot establish causal direction. Energy drink consumers have been found to report greater familial vulnerability to depression, anxiety, and substance use problems compared to non-consumers (70), and it is further possible that the effects observed here are attributable to personality characteristics associated with caffeine use, such as risk-taking behaviour (13) rather than to beverage intake itself. Together, these considerations suggest that observed associations may partly reflect pre-existing individual differences rather than a direct effect of consumption. Longitudinal designs that experimentally manipulate energy drink intake and assess psychological vulnerability and relevant personality traits at baseline are needed to establish the direction of these associations. Finally, the sample was recruited via convenience sampling at an Icelandic university, which may have skewed the demographic composition towards students and university staff, and generalisability to populations at lower latitudes or different cultural contexts may therefore also be limited.

Despite these limitations, the present study has several strengths. The four-season longitudinal design represents an advance over purely cross-sectional approaches, and the inclusion of coffee as a comparison caffeine source allows for a more nuanced interpretation of energy drink-specific effects than studies examining energy drinks in isolation. The use of a general population sample spanning a broad adult age range further extends findings beyond the student and athlete samples that dominate the energy drink literature. Finally, the Icelandic setting, with its extreme seasonal variation in daylight, provides a naturalistic context for examining circadian and seasonal effects that would be difficult to replicate elsewhere.

5. Conclusions

Longitudinal analyses revealed that the association between energy drink intake frequency and depressive symptoms was amplified in autumn and was strongest among evening types, a pattern absent for coffee. Energy drink intake frequency was also associated with insomnia cross-sectionally, an effect not observed for coffee or in the longitudinal analyses. Whether these effects reflect the specific composition of energy drinks, their patterns of use, or pre-existing individual

differences remains to be determined. Our findings highlight the importance of considering these factors jointly rather than in isolation, suggesting that evening types who consume energy drinks frequently may represent a particularly vulnerable subgroup, especially as daylight rapidly decreases in autumn. The four-season longitudinal design embedded in a high-latitude setting, with its natural variation in light exposure, enabled the detection of these dynamic associations that would likely have been invisible in a single-wave study. Future research should employ longitudinal designs with objective measures of caffeine exposure to establish causal direction and clarify the mechanisms underlying these associations. In the meantime, the present findings support calls for greater public health attention to energy drink consumption among individuals with an evening circadian preference.

List of Abbreviations
BIS: Bergen Insomnia Scale
DASS: Depression Anxiety Stress Scales
FFQ: Food Frequency Questionnaire
MEQ: Morningness–Eveningness Questionnaire

Declarations
Ethics approval and consent to participate: This study involved human participants, all of whom provided informed consent prior to participation. Ethical approval was granted by the National Bioethics Committee of Iceland (approval number VSN-21-088). The study was conducted in accordance with the Declaration of Helsinki.
Clinical trial number: Not applicable.
Consent for publication: Not applicable.
Availability of data and materials: The datasets generated and analysed during the current study are not publicly available due to information that could compromise the privacy of research participants but are available from the corresponding author upon reasonable request.
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Authors' contributions: LZ conceptualised the study, developed the methodology, conducted the formal analysis, and drafted the manuscript. R.P.Ó. and YH supervised the project and contributed to critical revision of the manuscript. YH also contributed to conceptualisation, methodology, and secured funding. All authors read and approved the final manuscript.

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Supplement

Energy Drink Consumption and Depressive Symptoms in Iceland: Associations with Season and Circadian Preference

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Table S1.

Estimates for Associations Between Energy Drink Intake Frequency, Depressive Symptoms and Covariates

Explanatory Variable	**B**	**SE**	**β**	**95% CI**	**t**	**p**	**p (BH)**
DASS Depression ~ FFQ Energy Drinks × Season × Circadian Preference + Age + Gender							
Main Effects							
FFQ Energy Drinks	0.18	0.44	0.05	[-0.20, 0.30]	0.41	.682	.928
Season (Autumn)	-2.31	1.31	0.21	[-0.14, 0.56]	-1.77	.078	.338
Season (Winter)	0.39	2.04	0.31	[-0.18, 0.81]	0.19	.847	.928
Season (Spring)	0.21	1.45	0.13	[-0.27, 0.53]	0.14	.886	.928
MEQ (Morning Type)	-0.13	1.25	-0.20	[-0.55, 0.14]	-0.13	.917	.928
MEQ (Intermediate Type)	-0.10	1.08	-0.08	[-0.37, 0.22]	-0.10	.927	.928
Age	-0.09	0.02	-0.27	[-0.37, -0.17]	-5.45	< .001	< .001
Gender (Women)	0.71	0.47	0.17	[-0.05, 0.39]	0.71	.134	.388
2-Way Interaction Effects: FFQ Energy Drinks (EDs) * Season							
*EDs * Autumn*	1.95	0.58	0.56	[0.23, 0.89]	3.34	.001	.008
*EDs * Winter*	0.56	0.95	0.16	[-0.37, 0.69]	0.59	.556	.928
*EDs * Spring*	0.20	0.66	0.06	[-0.31, 0.43]	0.31	.760	.928
2-Way Interaction Effects: FFQ Energy Drinks (EDs) * Circadian Preference							
*EDs * MEQ (Morning Type)*	-0.44	0.64	-0.13	[-0.49, 0.23]	-0.69	.494	.928
*EDs * MEQ (Intermediate Type)*	-0.13	0.48	-0.04	[-0.31, 0.23]	-0.28	.782	.928
2-Way Interaction Effects: Season * Circadian Preference							
*Autumn * MEQ (Morning Type)*	2.47	1.60	-0.29	[-0.72, 0.15]	1.55	.122	.388
*Autumn * MEQ (Intermediate Type)*	2.33	1.42	-0.22	[-0.60, 0.16]	1.64	.101	.376
*Winter * MEQ (Morning Type)*	-2.30	2.35	-0.30	[-0.90, 0.30]	-0.98	.328	.775
*Winter * MEQ (Intermediate Type)*	1.16	2.14	-0.12	[-0.66, 0.41]	0.54	.589	.928
*Spring * MEQ (Morning Type)*	-0.76	1.77	-0.15	[-0.64, 0.34]	-0.43	.668	.928
*Spring * MEQ (Intermediate Type)*	-0.25	1.55	-0.03	[-0.46, 0.40]	-0.16	.875	.928
3-Way Interaction Effects: FFQ Energy Drinks (EDs) * Circadian Preference * Season							
*EDs * MEQ (Morning Type) * Autumn*	-2.24	0.81	-0.64	[-1.10, -0.18]	-2.75	.006	.032
*EDs * MEQ (Intermediate Type) * Autumn*	-1.98	0.64	-0.57	[-0.93, -0.21]	-3.11	.002	.013
*EDs * MEQ (Morning Type) * Winter*	0.64	1.18	0.18	[-0.48, 0.85]	0.55	.586	.928
*EDs * MEQ (Intermediate Type) * Winter*	-1.02	0.99	-0.29	[-0.85, 0.27]	-1.03	.304	.775

*EDs * MEQ (Morning Type) * Spring*	0.09	0.98	0.03	[-0.52, 0.57]	0.09	.928	.928
*EDs * MEQ (Intermediate Type) * Spring*	0.06	0.72	0.02	[-0.39, 0.42]	0.09	.928	.928

Note. DASS = Depression, Anxiety, and Stress Scales; FFQ = Food Frequency Questionnaire; MEQ = Morningness–Eveningness Questionnaire. *P*-values adjusted using Benjamin-Hochberg (BH) correction.

Table S2.

Estimates for Associations Between Coffee Intake Frequency, Depressive Symptoms and Covariates.

Explanatory Variable	**B**	**SE**	**β**	**95% CI**	**t**	**p**	**p (BH)**
DASS Depression ~ FFQ Coffee × Season × Circadian Preference + Age + Gender							
Main Effects							
FFQ Coffee	-0.16	0.28	-0.08	[-0.34, 0.18]	-0.58	.561	.858
Season (Autumn)	4.12	1.53	0.27	[-0.08, 0.62]	2.70	.007	.047
Season (Winter)	2.70	1.88	0.10	[-0.41, 0.61]	1.43	.152	.440
Season (Spring)	-0.04	1.60	0.09	[-0.32, 0.49]	-0.03	.978	.994
MEQ (Morning Type)	-1.65	1.42	-0.25	[-0.60, 0.09]	-1.17	.244	.528
MEQ (Intermediate Type)	-0.93	1.17	-0.13	[-0.44, 0.17]	-0.80	.427	.788
Age	-0.09	0.02	-0.28	[-0.37, -0.18]	-5.58	< .001	< .001
Gender (Women)	0.57	0.47	0.14	[-0.09, 0.36]	1.20	.233	.528
2-Way Interaction Effects: FFQ Coffee (C) * Season							
*C * Autumn*	-0.76	0.36	-0.37	[-0.70, -0.03]	-2.15	.032	.120
*C * Winter*	-0.58	0.48	-0.28	[-0.73, 0.17]	-1.21	.226	.528
*C * Spring*	0.10	0.38	0.05	[-0.31, 0.41]	0.28	.783	.925
2-Way Interaction Effects: FFQ Coffee (C) * Circadian Preference							
*C * MEQ (Morning Type)*	0.15	0.33	0.07	[-0.24, 0.39]	0.50	.646	.925
*C * MEQ (Intermediate Type)*	0.10	0.29	0.05	[-0.23, 0.32]	0.33	.745	.925
2-Way Interaction Effects: Season * Circadian Preference							
*Autumn * MEQ (Morning Type)*	-4.49	1.93	-0.34	[-0.77, 0.09]	-2.33	.020	.106
*Autumn * MEQ (Intermediate Type)*	-4.56	1.68	-0.31	[-0.69, 0.08]	-2.71	.007	.047
*Winter * MEQ (Morning Type)*	-1.66	2.39	-0.17	[-0.77, 0.43]	-0.69	.489	.794
*Winter * MEQ (Intermediate Type)*	-1.54	2.05	0.005	[-0.54, 0.55]	-0.75	.455	.788
*Spring * MEQ (Morning Type)*	-0.02	2.07	-0.14	[-0.62, 0.33]	-0.01	.994	.994
*Spring * MEQ (Intermediate Type)*	0.12	1.77	-0.03	[-0.46, 0.41]	0.07	.944	.994
3-Way Interaction Effects: FFQ Coffee (C) * Circadian Preference * Season							
*C * MEQ (Morning Type) * Autumn*	0.79	0.44	0.38	[-0.04, 0.79]	1.79	.074	.239
*C * MEQ (Intermediate Type) * Autumn*	0.84	0.39	0.40	[0.03, 0.77]	2.15	.032	.120
*C * MEQ (Morning Type) * Winter*	0.24	0.58	0.12	[-0.43, 0.66]	0.41	.681	.925
*C * MEQ (Intermediate Type) * Winter*	0.40	0.52	0.19	[-0.30, 0.68]	0.77	.443	.788
*C * MEQ (Morning Type) * Spring*	-0.15	0.47	-0.07	[-0.52, 0.38]	-0.31	.753	.925

*C * MEQ (Intermediate Type) * Spring*	-0.06	0.41	-0.03	[-0.42, 0.36]	-0.15	.883	.994

Note. DASS = Depression, Anxiety, and Stress Scales; FFQ = Food Frequency Questionnaire; MEQ = Morningness–Eveningness Questionnaire. *P*-values adjusted using Benjamin-Hochberg (BH) correction.

Table S3.

Estimates for Associations Between Energy Drink Intake Frequency, Insomnia and Covariates

Explanatory Variable	B	SE	β	95% CI	t	p	p (BH)
BIS ~ FFQ Energy Drinks × Season × Circadian Preference + Age + Sex							
Main Effects							
FFQ Energy Drinks	1.83	0.99	0.23	[-0.01, 0.48]	1.86	.063	.400
Season (Autumn)	-0.24	2.83	0.07	[-0.26, 0.40]	-0.08	.933	.958
Season (Winter)	6.09	4.43	0.22	[-0.26, 0.69]	1.38	.169	.400
Season (Spring)	5.05	3.14	0.19	[-0.19, 0.57]	1.61	.108	.400
MEQ (Morning Type)	-0.48	2.76	-0.28	[-0.61, 0.06]	-0.17	.861	.956
MEQ (Intermediate Type)	1.13	2.37	-0.14	[-0.42, 0.15]	0.48	.633	.783
Age	-0.02	0.04	-0.03	[-0.13, 0.08]	-0.52	.603	.783
Gender (Women)	4.07	1.14	0.43	[0.19, 0.67]	3.57	< .001	.009
2-Way Interaction Effects: FFQ Energy Drinks (E) * Season							
*E * Autumn*	0.54	1.26	0.07	[-0.25, 0.38]	0.43	.670	.792
*E * Winter*	-2.48	2.06	-0.31	[-0.83, 0.30]	-1.21	.228	.492
*E * Spring*	-2.00	1.44	-0.25	[-0.61, 0.10]	-1.39	.164	.400
2-Way Interaction Effects: FFQ Energy Drinks (E) * Circadian Preference							
*E * MEQ (Morning Type)*	-1.29	1.41	-0.16	[-0.51, 0.19]	-0.92	.360	.595
*E * MEQ (Intermediate Type)*	-1.49	1.05	-0.19	[-0.45, 0.07]	-1.42	.156	.400
2-Way Interaction Effects: Season * Circadian Preference							
*Autumn * MEQ (Morning Type)*	2.90	3.44	0.06	[-0.36, 0.47]	0.84	.400	.595
*Autumn * MEQ (Intermediate Type)*	-0.16	3.06	-0.05	[-0.41, 0.31]	-0.05	.957	.958
*Winter * MEQ (Morning Type)*	-7.43	5.10	-0.16	[-0.73, 0.41]	-1.46	.145	.400
*Winter * MEQ (Intermediate Type)*	-5.20	4.66	-0.13	[-0.64, 0.38]	-1.12	.265	.492
*Spring * MEQ (Morning Type)*	-3.20	3.82	-0.13	[-0.59, 0.34]	-0.84	.404	.595
*Spring * MEQ (Intermediate Type)*	-5.45	3.36	-0.21	[-0.62, 0.20]	-1.62	.105	.400
3-Way Interaction Effects: FFQ Energy Drinks (E) * Circadian Preference * Season							
*E * MEQ (Morning Type) * Autumn*	-1.45	1.77	-0.18	[-0.62, 0.26]	-0.82	.412	.595
*E * MEQ (Intermediate Type) * Autumn*	-0.20	1.38	-0.03	[-0.37, 0.32]	-0.15	.882	.956
*E * MEQ (Morning Type * Winter*	3.62	2.57	0.46	[-0.18, 1.10]	1.41	.159	.400

*E * MEQ (Intermediate Type) * Winter*	2.44	2.16	0.31	[-0.23, 0.85]	1.13	.259	.492
*E * MEQ (Morning Type) * Spring*	1.22	2.11	0.16	[-0.37, 0.68]	0.58	.562	.769
*E * MEQ (Intermediate Type) * Spring*	2.14	1.55	0.27	[-0.12, 0.66]	1.38	.169	.400

Note. BIS = Bergen Insomnia Scale; FFQ = Food Frequency Questionnaire; MEQ = Morningness–Eveningness Questionnaire. *P*-values adjusted using Benjamin-Hochberg (BH) correction.

Table S4.

Estimates for Associations Between Coffee Intake Frequency, Insomnia and Covariates

Explanatory Variable	B	SE	β	95% CI	t	p	p (BH)
BIS ~ FFQ Coffee × Season × Circadian Preference + Age + Sex							
Main Effects							
FFQ Coffee	0.73	0.60	0.16	[-0.09, 0.40]	1.23	.221	.646
Season (Autumn)	0.59	3.18	0.03	[-0.29, 0.36]	0.18	.854	.925
Season (Winter)	5.41	3.94	-0.03	[-0.50, 0.44]	1.37	.170	.646
Season (Spring)	-0.53	3.35	0.15	[-0.22, 0.53]	-0.16	.875	.925
MEQ (Morning Type)	0.63	3.03	-0.29	[-0.54, 0.04]	0.21	.837	.925
MEQ (Intermediate Type)	0.82	2.49	-0.25	[-0.54, 0.04]	0.33	.741	.925
Age	-0.05	0.04	-0.07	[-0.17, 0.03]	-1.30	.194	.646
Gender Woman	4.20	1.15	0.44	[0.21, 0.68]	3.66	< .001	.004
2-Way Interaction Effects: FFQ Coffee (C) * Season							
*C * Autumn*	-0.07	0.74	-0.01	[-0.32, 0.29]	-0.09	.925	.925
*C * Winter*	-1.45	1.01	-0.31	[-0.73, 0.11]	-1.44	.150	.646
*C * Spring*	0.50	0.79	0.11	[-0.22, 0.43]	0.63	.527	.925
2-Way Interaction Effects: FFQ Coffee (C) * Circadian Preference							
*C * MEQ (Morning Type)*	-0.87	0.71	-0.18	[-0.48, 0.11]	-1.22	.223	.646
*C * MEQ (Intermediate Type)*	-0.81	0.62	-0.17	[-0.43, 0.09]	-1.30	.194	.646
2-Way Interaction Effects: Season * Circadian Preference							
*Autumn * MEQ (Morning Type)*	-0.44	4.03	0.03	[-0.36, 0.43]	-0.11	.912	.925
*Autumn * MEQ (Intermediate Type)*	-0.48	3.51	-0.01	[-0.37, 0.34]	-0.14	.891	.925
*Winter * MEQ (Morning Type)*	-3.96	5.00	-0.04	[-0.59, 0.51]	-0.79	.429	.925
*Winter * MEQ (Intermediate Type)*	-4.42	4.30	0.13	[-0.37, 0.63]	-1.03	.304	.790
*Spring * MEQ (Morning Type)*	-3.12	4.34	-0.13	[-0.57, 0.31]	-0.72	.472	.925
*Spring * MEQ (Intermediate Type)*	-1.00	3.70	-0.16	[-0.56, 0.24]	-0.27	.787	.925
3-Way Interaction Effects: FFQ Coffee (C) * Circadian Preference * Season							
*C * MEQ (Morning Type) * Autumn*	0.19	0.92	0.04	[-0.34, 0.42]	0.21	.834	.925
*C * MEQ (Intermediate Type) * Autumn*	0.09	0.82	0.02	[-0.32, 0.36]	0.11	.914	.925
*C * MEQ (Morning Type) * Winter*	0.91	1.22	0.19	[-0.31, 0.70]	0.74	.457	.925
*C * MEQ (Intermediate Type) * Winter*	1.44	1.08	0.31	[-0.15, 0.76]	1.33	.184	.646

*C * MEQ (Morning Type) * Spring*	0.49	0.99	0.10	[-0.31, 0.52]	0.49	.623	.925
*C * MEQ (Intermediate Type) * Spring*	-0.13	0.87	-0.03	[-0.39, 0.33]	-0.15	.880	.925

Note. BIS = Bergen Insomnia Scale; FFQ = Food Frequency Questionnaire; MEQ = Morningness–Eveningness Questionnaire. *P*-values adjusted using Benjamin-Hochberg (BH) correction.

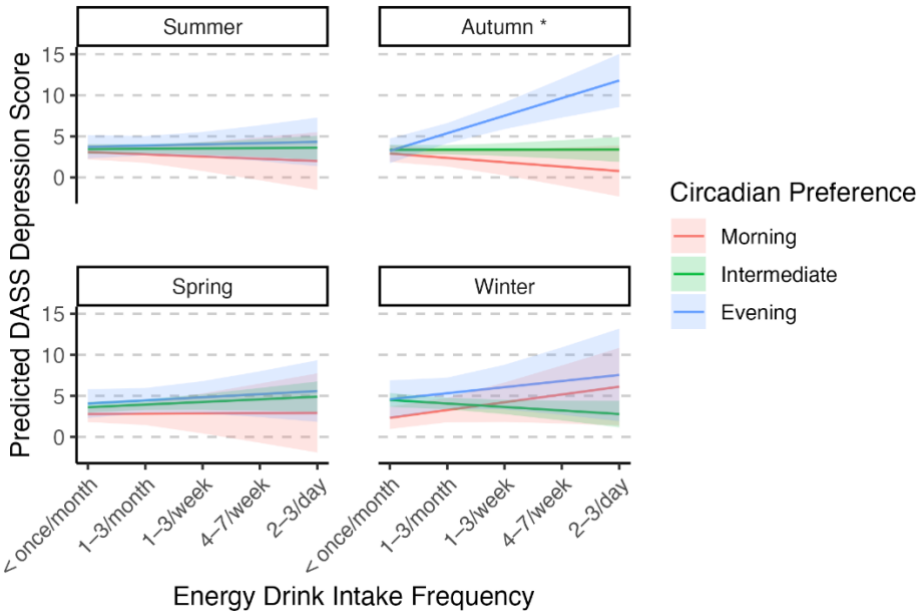


Fig. S1

Predicted DASS Depression scores across energy drink intake frequencies for all seasons, separated by Circadian Preference (Morningness-Eveningness-Type)

Note. Shaded areas represent 95% confidence intervals. Predicted scores are shown controlling for age and gender.

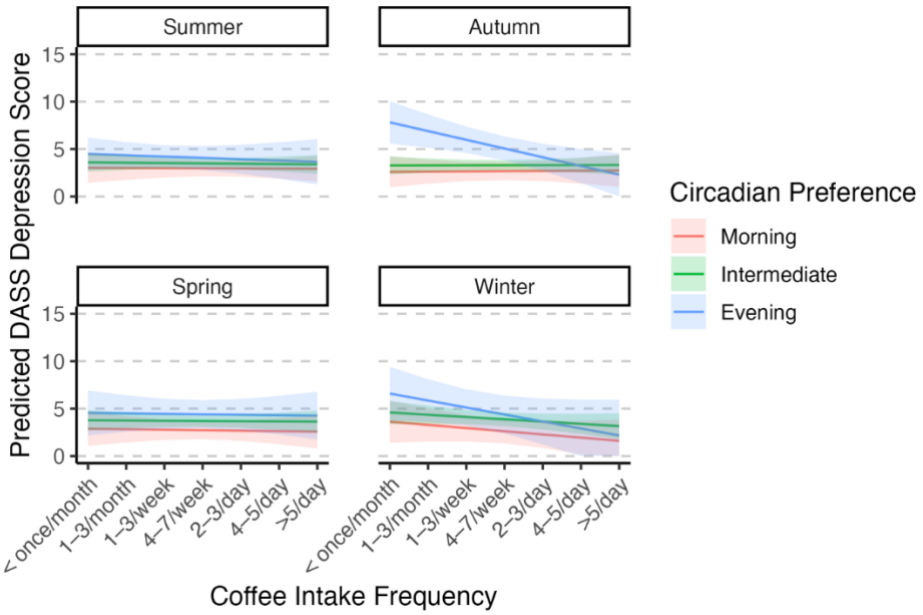


Fig. S2

Predicted DASS Depression scores across coffee intake frequencies for all seasons, separated by Circadian Preference (Morningness-Eveningness-Type)

Note. Shaded areas represent 95% confidence intervals. Predicted scores are shown controlling for age and gender.