



## Psychological models of development of idiopathic environmental intolerances: Evidence from longitudinal population-based data

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### ABSTRACT

The origin of idiopathic environmental intolerances (IEIs) is an open question. According to the psychological approaches, various top-down factors play a dominant role in the development of IEIs. The general psychopathology model assumes a propensity towards mental ill-health (negative affectivity) increases the probability of developing IEIs. The attribution model emphasizes the importance of mistaken attribution of experienced somatic symptoms; thus, more symptoms should lead to more IEIs. Finally, the nocebo model highlights the role of expectations in the development of IEIs. In this case, worries about the harmful effects of environmental factors are assumed to evoke IEIs.

We estimated cross-lagged panel models with latent variables based on longitudinal data obtained at two time points (six years apart) from a large near-representative community sample to test the hypothesized associations. Indicators of chemical intolerance, electromagnetic hypersensitivity, and sound sensitivity fit well under a common latent factor of IEIs. This factor, in turn, showed considerable temporal stability. However, whereas a positive association was found between IEIs and increased somatic symptoms and modern health worries six years later, the changes therein could not be predicted as hypothesized by the three psychological models. We discuss the implications of these results, as well as methodological aspects in the measurement and prediction of change in IEIs.

### 1. Introduction

Idiopathic environmental intolerances (IEIs), such as chemical intolerance, noise sensitivity, and electromagnetic hypersensitivity, are chronic conditions characterized by the presence of non-specific somatic symptoms that decrease individuals' well-being and often considerably impair their everyday functioning. The sufferers attribute the experienced symptoms to low doses of the respective environmental factor that do not evoke symptoms in the majority of the population (Baguley, 2003; Baliatsas et al., 2012a; Dantoft et al., 2015; Dieudonné, 2020; Haanes et al., 2020; Hansson Mild et al., 2006; Kjellqvist et al., 2016). These conditions are self-diagnosed without officially elaborated and generally accepted diagnostic criteria (Baliatsas et al., 2012a; WHO, 2005). As a consequence, their estimated prevalence also varies. The findings of population-based studies indicate a considerable

pervasiveness: annoyance and negative effects attributed to exposure of chemical substances or noise impact approximately one fifth to one third of the population (Björk et al., 2006; Bluhm et al., 2004; Carlsson et al., 2005; Johansson et al., 2005; Nordin et al., 2013b). The prevalence of EHS is somewhat lower, usually in the 5–10% range (Baliatsas et al., 2015; Blettner et al., 2009; Eltiti et al., 2007; Hillert et al., 2002; Huang et al., 2018; Karvala et al., 2018; Tseng et al., 2011).

The background and development of these conditions are not clear. One option, called the bottom-up or biophysical approach, raises the possibility that the impacted population is biologically hypersensitive to certain factors. In other words, it assumes pathophysiological processes in the background (Dieudonné, 2020; Staudenmayer et al., 2003a). Alternatively, the condition could be considered of top-down origin (psychological approach) and could be explained predominantly by psychological factors (Röösli, 2008; Rubin et al., 2011; Staudenmayer

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et al., 2003b; Szemerszky et al., 2010). Overall, evidence supporting the biophysical theory is scarce (Baliatsas et al., 2012b; Dieudonné, 2020; Nordin, 2020; Staudenmayer, 1998, 2006; Staudenmayer et al., 2003a). For example, double-blind provocation studies do not show that individuals with various IELs experience more symptoms when exposed to the respective environmental factor (Das-Munshi et al., 2006; Röösl, 2008; Rubin et al., 2005, 2010; Schmiedchen et al., 2019; Szemerszky et al., 2015b). Also, studies applying ecological momentary assessment (EMA), a method with high external validity, revealed no or only very weak associations between actual exposure to electromagnetic fields and reported symptoms (Bogers et al., 2018; Bolte et al., 2019). Finally, a proportion of the impacted population reports multiple sensitivities and/or changes concerning the environmental factor assumed to cause the symptoms (Nordin et al., 2014; Palmquist et al., 2014). This latter finding makes the existence of specific pathophysiological processes less likely and highlights the possible role of psychological factors. In the study presented in this paper, we focus on the psychological approach, a model that, over the decades, has received more empirical support than the biophysical model. The psychological theory is not a unitary approach to IELs; in fact, it is an umbrella term encompassing several possible top-down mechanisms. The involved mechanisms can be roughly divided into three categories; these will be named general psychopathology model, attribution model, and nocebo model throughout this paper. Although they are not completely separable from one another and are usually combined, we will present and discuss them separately.

The general psychopathology model assumes a mental disorder, or at least a proneness to a mental disorder, in the background. IELs shows comorbidity with several psychological symptoms as well as severe pathological conditions, such as somatoform disorder, depressive disorder, posttraumatic stress disorder, and panic disorder (Gruber et al., 2018; Johansson et al., 2010; Labarge and McCaffrey, 2000; Tseng et al., 2011). Also, the association between negative affectivity and IELs is well-known (Pennebaker, 1994; Watson and Pennebaker, 1989). Also, cross-sectional and longitudinal associations between indicators of negative affectivity and environmental annoyance were reported (Eek et al., 2010; Österberg et al., 2007). The model assumes that severe IELs, in fact, belong to one or more diagnostic categories and that the somatic symptoms are simply concomitants of these conditions, mistakenly ascribed to environmental factors (Baliatsas et al., 2014; Belpomme et al., 2015; Bornschein et al., 2001; Palmquist et al., 2014). If this is the case, high levels of indicators of negative affectivity, such as depression, anxiety, and/or chronic stress, should predict IELs.

Somatic symptoms do not necessarily indicate pathology; almost everyone experiences them daily (Pennebaker, 1982; Rosenzweig et al., 1993). However, the presence and impact of symptoms, also called somatic symptom distress, becomes more severe and chronic for certain individuals (Kroenke et al., 2002; Witthöft et al., 2012, 2016). The attempt to find an appropriate explanation for personally meaningful experiences, such as disturbing and unpleasant somatic symptoms, can be considered a healthy motive (Cioffi, 1991; Kolk et al., 2003; Swartzman and Lees, 1996; Szemerszky et al., 2015a). Thus, the attribution approach emphasizes the crucial importance of attribution in IELs. Such tendency towards attribution has been demonstrated in experimental (Szemerszky et al., 2015a), quasi-experimental (Petrie et al., 2005), and longitudinal studies (Köteles and Simor, 2013). Based on the attribution model, it can be assumed that high levels of somatic symptoms predict IELs.

Finally, expectations of sickness and the affective states associated with these expectations can cause perceived and actual body changes, called the nocebo phenomenon (Hahn, 1985, 1997, 1999). A nocebo approach to non-specific symptoms was also proposed for medication side effects (Barsky et al., 2002) and EHS (Rubin et al., 2008; Szemerszky et al., 2010). From a broader point of view, the predictive processing approach to symptom perception describes a mechanism that can take top-down factors (aka expectations or priors) into account in

the generation of somatic symptoms and IELs (Edwards et al., 2012; Van den Bergh et al., 2017a, 2017b). Modern health worries (MHWs), i.e., the perceived risk posed by modern technologies on health (Petrie et al., 2001), represents a construct that can be conceptualized as the indicator of health-related negative expectations. MHWs are widely available in the mass media (Claassen et al., 2012; Elvers et al., 2009; Faasse et al., 2012; MacKrell et al., 2019), and able to trigger the nocebo mechanism population-wide (Köteles et al., 2016; Witthöft et al., 2018; Witthöft and Rubin, 2013). Taken together, the expectation model forecasts that MHWs predict IELs.

The present study, based on longitudinal data from a near representative, large-scale environmental study (Palmquist et al., 2014), tests the aforementioned hypotheses with respect to three variants of IEL, i.e., chemical intolerance, noise sensitivity, and electromagnetic hypersensitivity. More specifically, we assumed that (1) indicators of negative affectivity (general psychopathology model), (2) somatic symptom distress (attribution model), and (3) MHWs (nocebo model) would be able to predict the temporal changes of IELs.

## 2. Method

### 2.1. Design and study population

We used data from the prospective Västerbotten Environmental Health Study (Palmquist et al., 2014), a questionnaire-based survey focusing on environmental intolerance. Västerbotten is a county in northern Sweden with approximately 260 000 inhabitants with an age and sex distribution similar to the general Swedish population (Statistics Sweden, 2010). The first data collection (T1) was completed in 2010 for which 8600 adults (aged 18–79 years) from the county of Västerbotten were randomly selected from the population registry, stratified for age and sex. Eighty persons were excluded since they could not be reached by post, resulting in a sample size of 8520 persons. Of these, 3406 (40%) responded to the questionnaires. The second data collection took place in 2013 (T2), where 3181 participants were invited 2336 participants responded (73.4%). Finally, the last collection took place in 2016 (T3) and comprised 2226 participants who were still alive and lived in Västerbotten remained, of which 1837 (82.5%) responded.

### 2.2. Study sample

The following analyses are based on 1837 subjects who participated both in T1 and T3. We chose to omit T2 and maximize the length between two time points to increase the probability to observe substantial change in any of the investigated constructs. A further discussion of optimal time frames and T2 are addressed in the discussion and supplemental analyses in the appendix. Table 1 describes the age, sex, and response rates of the analyzed sample. Background variables of the sample are reported in Table 2 (see Palmquist, 2017, for more details).

**Table 1**

Numbers of respondents across age and sex strata at the first (T1) and last (T3) wave of data collection.

| Age at T1 (years) | T1 (year 2010) |             | T3 (year 2016) |            |
|-------------------|----------------|-------------|----------------|------------|
|                   | women          | men         | women          | men        |
| 18–29             | 307 (32.1)     | 179 (17.3)  | 84 (70.0)      | 48 (72.7)  |
| 30–39             | 266 (40.3)     | 177 (24.7)  | 127 (78.4)     | 66 (73.3)  |
| 40–49             | 288 (40.5)     | 230 (31.0)  | 155 (80.3)     | 106 (76.8) |
| 50–59             | 367 (50.9)     | 295 (39.5)  | 239 (86.6)     | 191 (86.4) |
| 60–69             | 405 (58.4)     | 356 (50.7)  | 267 (84.8)     | 250 (89.0) |
| 70–79             | 265 (53.8)     | 271 (63.9)  | 146 (81.1)     | 158 (85.9) |
| Total sample      | 1898 (45.2)    | 1508 (34.9) | 1018 (81.7)    | 819 (83.6) |

Note. Percentage of those invited at each data collection wave in parentheses.

**Table 2**  
Descriptive statistics of background variables at T1 of the analyzed sample.

|  | T1              |
|--|-----------------|
| Age (years; means $\pm$ SD)                | 54.8 $\pm$ 14.8 |
| Female (%)                                 | 55.7            |
| Education (highest) (%)                    |                 |
| Primary school                             | 25.4            |
| High school                                | 32.4            |
| University                                 | 42.2            |
| Smoker (%)                                 | 8.2             |
| General health status (%)                  |                 |
| Very good or excellent                     | 41.5            |
| Good                                       | 33.5            |
| Somewhat good or poor                      | 25.0            |
| Self-reported diagnosis from physician (%) |                 |
| Hypertension                               | 27.5            |
| Diabetes                                   | 5.3             |
| Rheumatic disorder                         | 4.8             |
| Back, joint, or muscle disorder            | 14.4            |
| Migraine                                   | 4.4             |
| Tinnitus                                   | 7.9             |
| Generalized anxiety disorder               | .07             |
| Depression                                 | 3.9             |
| Burnout syndrome                           | 4.4             |
| Panic disorder                             | 1.0             |
| Posttraumatic stress disorder              | .08             |
| Chronic fatigue syndrome                   | .06             |
| Irritable bowel syndrome                   | 2.1             |
| Fibromyalgia                               | 2.3             |

Note. N = 1837.

## 2.3. Measures

### 2.3.1. Idiopathic environmental intolerance scales

The 11-item versions of the Chemical Sensitivity Scale (CSS-11; also referred to as the Chemical Sensitivity Scale for Sensory Hyperreactivity; Nordin et al., 2004; Nordin et al., 2013a), the Electromagnetic Field Sensitivity Scale (EMFSS-11; Nordin et al., 2013b), and the Noise Sensitivity Scale (NSS-11; Nordin et al., 2013b) were used to assess IEs. More specifically, the scales quantify affective reactions and behavioral disruptions attributed to odorous/pungent chemicals, electrical devices, and sounds. Thus, the scales focus on the cognitive-affective aspects rather than the perception of somatic symptoms but have been shown to correlate with self-reported symptoms (e.g., Nordin et al., 2004; Nordin et al., 2013b).

The 11 items are analogous across the three scales, such that a certain item statement about environmental odorous/pungent chemicals in the CSS-11 corresponds as closely as possible to a statement in the EMFSS-11 about the EMF environment the NSS-11 about the sound environment. For example, the CSS-11 item “At movies, other persons’ perfume, and aftershave disturb me” corresponds to the EMFSS-11 items “At music concerts or similar events, it is disturbing to know that there is a lot of electrical equipment around me”, and the NSS-11 item “At movies, whispering, and crinkling candy wrappers disturb me.” Furthermore, the response alternatives in the three scales are identical for each corresponding statement. The three scales have ten 6-level and one 5-level Likert scale, and the mean score can range from 0 to 4.9. All scales show good reliability and have been comprehensively validated (Nordin et al., 2004; Nordin et al., 2013b).

### 2.3.2. Modern Health Worries Scale

The 12-item version of the Modern Health Worries Scale (MHWS-12; Olaru et al., 2019) was used to assess worries about aspects of modernity and technology affecting personal health. It has a four-factor structure that includes Toxic Interventions (e.g., “toxic chemicals in household products”), Environmental Pollution (e.g., “traffic fumes”), Tainted Food (e.g., “hormones in food”), and Radiation (e.g., “cell phones”). The items are rated on a 5-level scale, and the mean score can range from 1 to

5. The items were written in Swedish and have shown good reliability and validity (Palmquist et al., 2017).

### 2.3.3. Perceived Stress Scale

The degree to which life situations are appraised as stressful was measured with the 10-item Perceived Stress Scale (PSS-10; Cohen and Williamson, 1988). It refers to how frequently certain events or emotions have emerged (e.g., “In the last month, how often have you felt that you could not cope with all the things that you had to do”). The items are rated on a 5-level scale, and the mean score can range from 0 to 4. The Swedish version was used, which has shown good reliability and validity (Nordin and Nordin, 2013).

### 2.3.4. Shirom-melamed burnout questionnaire

The Shirom-Melamed Burnout Questionnaire (SMBQ; Melamed et al., 1999) was used to assess burnout. It consists of 22 items and measures the dimensions Physical Fatigue (e.g., “I feel physically exhausted”), Cognitive weariness (e.g., “I am too tired to think clearly”), Tension (e.g., “I feel tense”), and Listlessness (e.g., “I feel sleepy”). The items are rated on a 7-level scale, and the mean score can range from 1 to 7. The Swedish version was used, which has shown good reliability and validity (Grossi et al., 2003).

### 2.3.5. Karolinska Sleep Questionnaire

Poor sleep was assessed with seven items of the two dimensions sleep quality (four items; e.g., “Difficulties falling asleep”) and non-restorative sleep (three items; e.g., “Not well-rested on awakening”) of the Karolinska Sleep Questionnaire (KSQ; Nordin et al., 2013d). The items are rated on a 6-level scale, and the mean score can range from 0 to 5. The Swedish version was used, which has shown good reliability and validity (Nordin et al., 2013d).

### 2.3.6. Hospital anxiety and Depression Scale

The Hospital Anxiety and Depression Scale (HADS; Zigmond and Snaith, 1983) was used to assess anxiety and depression. It has two subscales, each with seven items, to measure symptoms of anxiety (e.g., “Worrying thoughts go through my mind”) and depression (e.g., “I have lost interest in my appearance”). The extent of anxiety and depression are rated on a 4-level scale, and a mean score across the 14 items was used that can range from 0 to 3. Support for combining the two scales is provided by prior results from a Swedish population showing that this measure correlated more strongly than each individual score with established questionnaire instruments on anxiety and depression (Lisspers et al., 1997).

### 2.3.7. Somatic symptom severity scale

The Patient Health Questionnaire 15-Item Somatic Symptom Severity Scale (PHQ-15; Kroenke et al., 2002) was used to assess somatization. It includes 14 of the 15 most common somatic complaints characterizing somatoform disorders (Hinz et al., 2017; van Ravesteijn et al., 2009). The extent of symptoms (e.g., “Stomach pain”, “Shortness of breath”, and “Feeling tired or having low energy”) is rated on a 3-level scale, and the mean score can range from 0 to 2. The Swedish version was used, which has shown good reliability and validity (Nordin et al., 2013c).

## 2.4. Procedure

The procedure was the same at T1 and T3. A questionnaire was sent by mail, a reminder was sent to non-responders after three weeks, and an additional reminder and a new copy of the questionnaire after another three weeks. All data was collected between March and April, before the onset of the allergy season. The study was approved by the Umeå Regional Ethics Board (09-171M and 2016-83-32M) and conducted in accordance with the declaration of Helsinki.

### 2.5. Statistical analysis

Mean scores were computed for all scales and used as indicators for the subsequent analyses. To investigate the development of IEs and neighboring constructs, as well as their reciprocal associations, we analyzed their mean trajectories and covariation. Thus, we operationalized development in the investigated constructs as the individual or joint growth, stability or decline over the period of observation. More specifically, we estimated cross-lagged panel models with latent variables. Cross-lagged models can be applied to investigate the causal direction between two (or more) repeatedly measured variables. Importantly, cross-lagged models allow controlling for auto-regressive effects in longitudinal data. Using latent variables with multiple indicators instead of single observed variables resolves challenges commonly encountered in cross-lagged panel models. As a primary benefit, auto-regressive and cross-lagged effects are estimated more accurately as the measurement error of the constructs can be modeled. Further, unequal or changing reliabilities can be accommodated, and residual correlations between observed variables across time points can be estimated that would otherwise inflate estimates of stability. Clearly, we use observational data, and interpretation of the causal nature of effects hinges upon additional assumptions such as the exclusion of plausible alternative hypotheses. In the present context, these would primarily pertain to unmeasured moderators of any cross-lagged effects we might find.

Latent factor models were estimated with *Maximum Likelihood* (ML) estimation or *Robust Maximum Likelihood* (MLR) estimation in cases where variables deviated substantially from normality. Latent factors were scaled with the effects coding method (Little et al., 2006). Model fit was considered good if *Comparative Fit Index* (CFI) > 0.95 and *Root Mean Square Error of Approximation* (RMSEA) < 0.06 (Hu and Bentler, 1999). Missing data was handled using multiple imputation (see Palmquist, 2017). All analyses were performed on one imputed dataset and cross-validated in four other datasets. Data preparation, recoding, and analysis was performed in R (version 4.0.0; R Core Team, 2020). Latent models were estimated using the package *lavaan* (version 0.6–6; Rosseel, 2012). We provide annotated R code and outputs for the main analyses on an online repository of the *Open Science Framework*: <https://osf.io/we7sd/>.

## 3. Results

### 3.1. Descriptive results

Table 3 summarizes the descriptive statistics and reliability estimates of all scales across time points. Average scores were as would be expected in a general population, with no substantial mean shifts on any variable from T1 to T3, indicating stationarity on the population level. Reliabilities were generally good, with estimates between  $\alpha = 0.77$  (NSS-11 T1) and  $\alpha = 0.96$  (MHWS-12, T1 and T3).

**Table 3**  
Descriptive statistics, reliability estimates, and test-retest-correlations.

| Scale    | Items | T1   |      |          | T3   |      |          | $r_{T1,T3}$ |
|----------|-------|------|------|----------|------|------|----------|-------------|
|          |       | M    | SD   | $\alpha$ | M    | SD   | $\alpha$ |             |
| CSS-11   | 11    | 2.74 | .78  | .81      | 2.69 | .79  | .82      | .68         |
| EMFSS-11 | 11    | 1.65 | .75  | .84      | 1.57 | .77  | .85      | .66         |
| NSS-11   | 11    | 2.53 | .72  | .77      | 2.52 | .72  | .79      | .61         |
| MHWS-12  | 12    | 2.64 | .88  | .96      | 2.61 | .88  | .96      | .59         |
| PSS-10   | 10    | 1.30 | .60  | .83      | 1.33 | .62  | .82      | .52         |
| SMBQ     | 22    | 2.75 | 1.10 | .95      | 2.82 | 1.15 | .95      | .60         |
| KSQ      | 7     | 1.47 | .85  | .83      | 1.49 | .86  | .83      | .56         |
| HADS     | 14    | .49  | .42  | .89      | .51  | .45  | .90      | .58         |
| PHQ-15   | 15    | .41  | .31  | .82      | .43  | .32  | .83      | .51         |

Note.  $N = 1837$ .  $M$  = mean,  $SD$  = standard deviation,  $\alpha$  = Cronbach's alpha,  $r_{T1,T3}$  = Test-retest-correlation between scores at T1 and T3.

Zero-order correlations of all scales across time points are provided in Table 4. Correlations were positive across both time points and scales. Hence, there is a substantial amount of positive manifold in the variables reported here. Clusters of even higher correlations were apparent between the IEI scales (CSS-11, EMFSS-11, NSS-11) and MHWS-12, as well as between scales of negative affectivity (PSS-10, SMBQ, KSQ, PHQ-15). Test-retest correlations between T1 and T3 were generally high.

### 3.2. Latent factor models

Based on the positive scale correlations and conceptual overlaps between the CSS-11, EMFSS-11, and NSS-11, we used the respective mean scores as indicators of a general IEI factor. This latent general factor captures the common variance of the three IEI scales and abstracts from item and scale specificities. The same method was applied to extract a general psychopathology factor from indicators of PSS-10, SMBQ, KSQ, and HADS. Each scale's total score was regressed on a common factor, ajar to common factor models of mood and anxiety disorders discussed in the literature (Simms et al., 2012; Smith et al., 2020; see Nimnuan et al., 2001 and Wessely et al., 1999, for similar thoughts on functional somatic syndromes). For the MHWS-12, a general factor was estimated with four indicators reflecting the four postulated facets "Environmental Pollution", "Radiation", "Tainted food" and "Toxic Interventions". Somatic symptoms had a single indicator, the total score of the PHQ-15. All multiple-indicator models indicated good model fit and longitudinal invariance of factor loadings.

#### 3.2.1. General Psychopathology Hypothesis

The cross-lagged model estimated to test the *General Psychopathology (GP) Hypothesis* (Fig. 1) fits the data well ( $n = 1837$ ;  $\chi^2[69] = 267.7$ ,  $p < .01$ ; CFI = 0.99; RMSEA = 0.04; SRMR = 0.03). Correlations at T1 indicated a moderate association of IEs and GP ( $\rho = 0.36$ ). As expected, the auto-regressive parameters indicated a fair amount of stability in both IEs ( $B = 0.78$ ) and GP ( $B = 0.68$ ). The path from GP[1] on IEI[3] is central to the *General Psychopathology Hypothesis*: it reflects the effect of GP at T1 on IEs at T3 after controlling for initial (T1) levels of IEs. The observed regression coefficient of 0 indicated that GP did not predict IEs 6 years later. In order to develop a better evaluation of cross-lagged effects, we also estimate cross-lagged effects from IEs at T1 on covariates at T3. For the General Psychopathology model this effect was also 0, indicating no causal impact of IEs on GP. The correlation of the disturbance terms at T3 captures the remaining association of T3 measurement after accounting for T1 in both constructs. Interestingly, this correlation ( $\rho = 0.30$ ) was almost as high as the relation of both constructs at T1. This might be due to nuisance variance of scale usage or point towards common state effects on the measurement of the constructs (e.g., daily mood). In sum, the cross-lagged model of IEs and GP did not provide evidence for the hypothesis that GP cause IEs in the observed time frame.

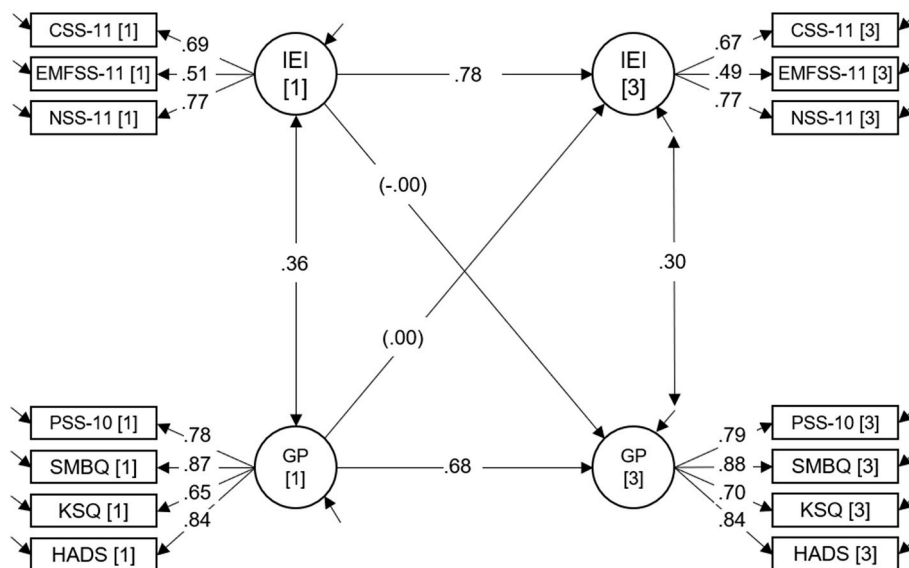
#### 3.2.2. Attribution Hypothesis

To test the *Attribution Hypothesis*, we estimated a cross-lagged model of IEs and somatic symptom distress, as indicated by the PHQ-15 (Fig. 2). Model fit was excellent ( $n = 1837$ ;  $\chi^2[15] = 29.7$ ,  $p = .01$ ; CFI = 1.00; RMSEA = 0.02; SRMR = 0.02). The number of somatic symptoms at T1 was positively correlated with higher levels of IEs at T1 ( $\rho = 0.31$ ). Somatic symptoms were rather stable across time ( $r = 0.47$ ). The seemingly lower stability coefficient is partly due to attenuation by measurement error, as the PHQ-15 was treated as a single-indicator variable. Contrary to the *Attribution Hypothesis*, somatic symptoms did not predict IEs at T3 ( $B = 0.00$ ). However, the cross-lagged effect of IEs on PHQ-15 was low but significant ( $B = 0.11$ ). This result might suggest that IEs predict levels of self-reported somatic symptoms, after controlling for initial levels of self-reported somatic symptoms. Given the small effect size, however, this relation should not be stressed too much. We ran an additional analysis where all subjects with a physician-based

**Table 4**  
Zero-order correlation matrix of all scales across time points.

|                  | 1.  | 2.  | 3.  | 4.  | 5.  | 6.  | 7.  | 8.  | 9.  | 10. | 11. | 12. | 13. | 14. | 15. | 16. | 17. | 18. |
|------------------|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|
| 1. CSS-11 [1]    | –   |     |     |     |     |     |     |     |     |     |     |     |     |     |     |     |     |     |
| 2. EMFSS-11 [1]  | .39 | –   |     |     |     |     |     |     |     |     |     |     |     |     |     |     |     |     |
| 3. NSS-11 [1]    | .53 | .36 | –   |     |     |     |     |     |     |     |     |     |     |     |     |     |     |     |
| 4. MHWS-12 [1]   | .39 | .43 | .32 | –   |     |     |     |     |     |     |     |     |     |     |     |     |     |     |
| 5. PSS-10 [1]    | .17 | .12 | .25 | .10 | –   |     |     |     |     |     |     |     |     |     |     |     |     |     |
| 6. SMBQ [1]      | .17 | .13 | .28 | .10 | .69 | –   |     |     |     |     |     |     |     |     |     |     |     |     |
| 7. KSQ [1]       | .16 | .08 | .28 | .09 | .43 | .58 | –   |     |     |     |     |     |     |     |     |     |     |     |
| 8. HADS [1]      | .14 | .11 | .25 | .13 | .68 | .72 | .56 | –   |     |     |     |     |     |     |     |     |     |     |
| 9. PHQ-15 [1]    | .20 | .18 | .25 | .16 | .43 | .54 | .49 | .44 | –   |     |     |     |     |     |     |     |     |     |
| 10. CSS-11 [3]   | .68 | .26 | .40 | .29 | .13 | .13 | .13 | .10 | .16 | –   |     |     |     |     |     |     |     |     |
| 11. EMFSS-11 [3] | .33 | .66 | .29 | .36 | .10 | .09 | .06 | .09 | .14 | .36 | –   |     |     |     |     |     |     |     |
| 12. NSS-11 [3]   | .42 | .25 | .61 | .22 | .22 | .21 | .21 | .17 | .18 | .51 | .36 | –   |     |     |     |     |     |     |
| 13. MHWS-12 [3]  | .32 | .36 | .27 | .59 | .09 | .08 | .08 | .10 | .12 | .35 | .41 | .31 | –   |     |     |     |     |     |
| 14. PSS-10 [3]   | .08 | .10 | .16 | .08 | .52 | .46 | .31 | .47 | .33 | .11 | .15 | .23 | .08 | –   |     |     |     |     |
| 15. SMBQ [3]     | .11 | .08 | .20 | .09 | .46 | .60 | .40 | .49 | .41 | .14 | .12 | .25 | .09 | .70 | –   |     |     |     |
| 16. KSQ [3]      | .11 | .08 | .22 | .09 | .34 | .43 | .56 | .41 | .40 | .15 | .11 | .27 | .11 | .50 | .63 | –   |     |     |
| 17. HADS [3]     | .08 | .06 | .18 | .10 | .46 | .50 | .35 | .58 | .34 | .11 | .12 | .23 | .13 | .67 | .73 | .61 | –   |     |
| 18. PHQ-15 [3]   | .15 | .17 | .20 | .11 | .29 | .39 | .35 | .33 | .51 | .18 | .20 | .23 | .13 | .43 | .56 | .51 | .45 | –   |

Note. N = 1837, all  $p < .05$ .



**Fig. 1.** Cross-lagged panel model of IEIs and General Psychopathology. N = 1837;  $\chi^2(69) = 267.7, p < .01$ ; CFI = 0.99; RMSEA = 0.04 [0.03-0.04]; SRMR = 0.03; residual correlations between the same indicators across time points were omitted for clarity. Coefficients not in parentheses were significantly different from zero ( $p < .05$ ).

diagnosis at T1 were excluded (remaining N = 745) to consolidate our results. This allowed investigating if the results differed for subjects which did not have a traditional narrative for their complaints at the onset of the study. However, none of the main model parameters (i.e., cross-lagged effects) changed substantially.

**3.2.3. Nocebo hypothesis**

To test the *Nocebo Hypothesis*, we fitted a cross-lagged model of MHWs and IEIs (Fig. 3). The initial model did not fit the data sufficiently well (n = 1837;  $\chi^2[69] = 1081.9, p < .01$ ; CFI = 0.93; RMSEA = 0.09; SRMR = 0.08). Inspection of modification indices revealed that the main source of misfit was attributable to residual correlations between the EMFSS-11 and the Radiation scale of the MHWS-12 across both time points. This points towards some commonality of emotional and behavioral reactions to and worries about electricity and radiation beyond the general commonality of the constructs, but this cannot be answered conclusively from the available data.

To accommodate the residual correlations, we let the four indicators (across 2 time points) load on an orthogonal electro/nuisance factor,

which led to a significantly improved model fit (n = 1837;  $\chi^2[67] = 441.2, p < .01$ ; CFI = 0.97; RMSEA = 0.06; SRMR = 0.06). IEIs and MHWs were substantially correlated at T1 ( $\rho = 0.57$ ), as were the disturbances at T3 ( $\rho = 0.37$ ). MHWs did not predict IEIs after controlling for initial levels of IEIs (B = -0.00), contradicting the *Nocebo Hypothesis*. The cross-lagged effect of IEIs of MHWs (B = 0.17), in turn, was low but significant, suggesting a causal precedence of IEIs in the development of MHWs. Care should be taken concerning the interpretation of this effect, given its small magnitude.

**4. Discussion**

In the current study, we investigated temporal dependencies between IEIs and neighboring constructs in a near-representative population-based large-scale longitudinal study spanning six years. In general, cross-lagged panel models did not support the hypotheses that initially high levels of general psychopathology, somatic symptoms, or modern health worries predict high levels of IEIs at a later time point after controlling for initial levels of IEIs. However, significant cross-lagged

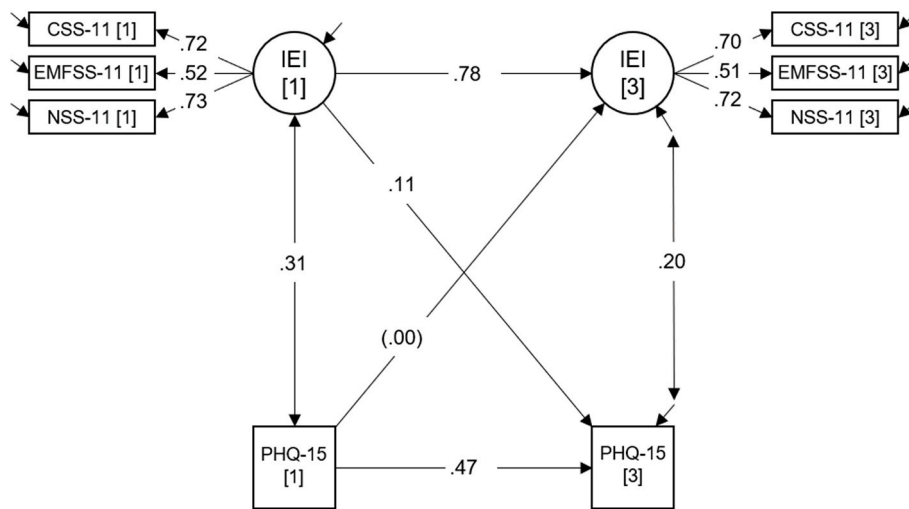


Fig. 2. Cross-lagged panel model of IEs and somatic symptoms (PHQ-15). N = 1837;  $\chi^2(15) = 29.7$ ,  $p = .01$ ; CFI = 1.00; RMSEA = 0.02 [0.01-0.03]; SRMR = 0.02; residual correlations between the same indicators across time points were omitted for clarity. Coefficients not in parentheses were significantly different from zero ( $p < .05$ ).

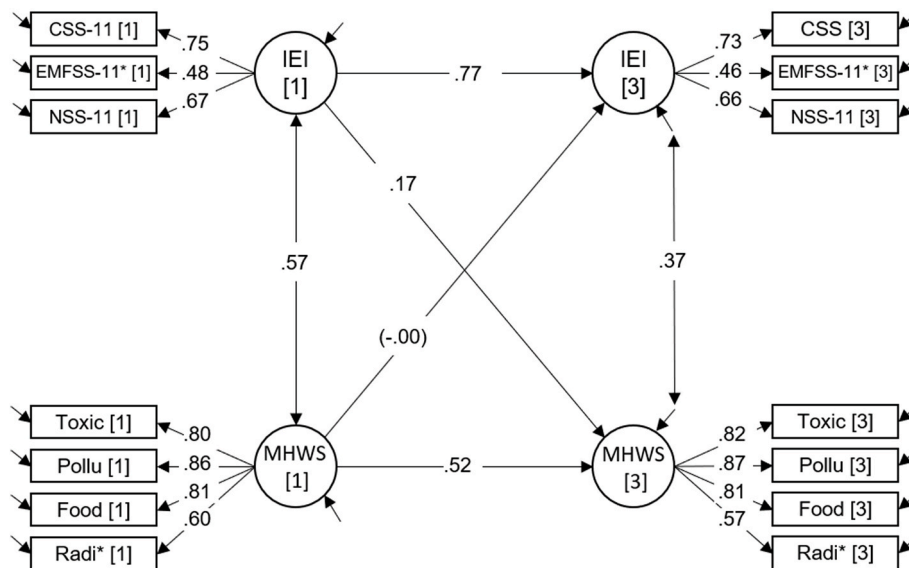


Fig. 3. Cross-lagged panel model of IEs and MHWs. N = 1837;  $\chi^2(67) = 441.2$ ,  $p < .01$ ; CFI = 0.97; RMSEA = 0.06 [0.05-0.06]; SRMR = 0.06; residual correlations between the same indicators across time points were omitted for clarity. Indicators with an asterisk additionally load on an orthogonal „electro“-factor. Coefficients not in parentheses were significantly different from zero ( $p < .05$ ).

effects, suggested weak positive associations between IEs and self-reported somatic symptoms and modern health worries six years later.

#### 4.1. Positive manifold of conditions and its possible interpretations

The lack of substantial cross-lagged effects should not be confused with a general lack of association between constructs. All conditions correlated substantially at T1, and correlations of disturbance terms at T3 were considerable as well. The investigated constructs thus shared large parts of common variance and temporal trajectories. Given the high correlations of disturbance terms at T3 (i.e., the association of constructs at T3 after controlling for initial levels at T1), parts of the covariance between constructs might be attributable to shared state-variance in addition to trait-variance. The positive manifold between conditions and their strong temporal stability make investigations of causal relations between them and other variables challenging.

Overall, our results support earlier findings that demonstrated a

comparatively high temporal stability of IEI (Bailer et al., 2008; Dieudonné, 2016, 2019; Gruber et al., 2018). The high levels of stability might be partly responsible for the current lack of effective treatments for IEI, both in terms of psychotherapeutic and pharmacological interventions (but see Van den Bergh et al., 2020, for a novel psychotherapeutic approach). Given the dimensional approach to IEs applied in the current study, however, high temporal stability also means that people with low levels or the complete absence of perceived environmental intolerances are not likely to acquire these conditions in the future. For example, it has been shown that mass media reports can increase MHWs and symptoms attributed to electromagnetic exposure (Bräscher et al., 2017; Köteles et al., 2016; Szemerszky et al., 2015a; Witthöft et al., 2018; Witthöft and Rubin, 2013), but this change does not appear to be stable over time (Dieudonné, 2020; Valkenburg et al., 2016).

The positive correlations between IEI scales and the large parts of shared variance captured by a common latent factor might indicate that

the constructs involved in this study simply tap different aspects of the same phenomenon. In this case, we actually model facets of an overarching condition with similar parallel temporal trajectories but no causal interactions. For example, it is often difficult to differentiate between affective and bodily aspects of perceived stress (Pennebaker, 1982). Several factors, such as the lack of appropriate words, personal styles favoring affective or somatic interpretation, or the actual overlapping of these aspects, are supposed to lie behind this issue (Ádám, 1998; Barsky et al., 1999; Kóteles, 2021; Pennebaker, 1982). This explanation highlights the necessity of a more holistic approach to IEIs. For example, symptoms attributed to a certain environmental factor by patients may simply indicate “uneasiness of mind” that manifests itself as a somatic interpretation (Petersen et al., 2015) but is not associated with peripheral pathophysiology. From a psychopathological point of view, the positive manifold observed in this study might be indicative of a hierarchical structural model involving a higher-order explanatory construct such as postulated in the Hierarchical Taxonomy of Psychopathology (HiTOP; Kotov et al., 2017). More specifically, the constructs involved in this study (i.e., IEI, somatic symptoms) might show substantial associations because they belong to a higher-order somatoform spectrum or an emotional dysfunction superspectrum (Watson et al., 2021). This explanation renders the assumption of causal associations among the involved constructs meaningless.

The construct overlap was especially pronounced between IEIs and MHWs. Although these conditions are often investigated separately based on their distinct theoretical definitions, it was difficult to distinguish them empirically in the current data. In some cases, correlations of MHWs with individual IEI scales were slightly larger than correlations within IEI scales, and MHWs could be subsumed as a fourth indicator under a general IEI factor without substantial loss in model fit. This raises questions about the discriminant validity of the constructs. It is important to note here that the scales used to assess IEIs in the current study focus on the cognitive-affective aspect (i.e., the awareness of and the unpleasantness caused by the respective environmental factors) of the condition and do not include the perception of somatic symptoms. This approach to IEIs is quite close to health-related worries as measured by the MHWs. Given the low discriminant validity of the scales and their strong general factor, substantially diverging associations between individual IEI scales with other scales or criteria are unlikely (e.g., that the nocebo hypothesis may hold strongly for EMF hypersensitivity, less strongly for chemical intolerance, and perhaps not at all for sound intolerance). Further, such discriminant associations would have caused misfit in the estimated factor models. Whereas an investigation of modification indices did not hint towards such effects in our data, and the current study instead opted for a global approach, discriminant associations of IEIs with external variables are interesting from both a theoretical and practical perspective and might be investigated further in the future.

Another possible explanation is that an unknown part of the common variance of all scales is due to a general propensity towards negativity or a tendency to use self-report response scales more or less enthusiastically. The artifact hypothesis we want to put forward here is that self-reports in questionnaires on somatic symptoms, modern health worries, anxiety, depression, and burnout might constitute a demand characteristic much like bobo-doll did for some participating children in Bandura's (1965) classical aggression experiments. An upward bias of correlations between such measures could be further investigated by pursuing a multi-trait multi-method approach, which might also be better suited to control for demand characteristics and to provide evidence of convergent and discriminant validity with other constructs. In summary, given the substantial positive manifold within the data, we were concerned that all three theories would gather some support, i.e., something akin to the dodo bird verdict (Carroll, 1865; “Everybody has won and all must have prizes.”). In reality, the cross-lagged effects were either absent or too small to derive major support for one theory relative to another.

#### 4.2. Methodological considerations

We want to highlight two important methodological aspects in the conception and analysis of our study, namely the choice of time lag and adequate statistical models. The choice of time lag between measurement points in panel studies can substantially influence the observed regression coefficients (Dormann and Griffin, 2015). To uncover within-person dynamics, the time lag should be chosen in accordance with the stability of the variables of interest. Hence, to detect highly fluctuating processes, shorter time intervals are necessary than would be to uncover rather stable processes and vice-versa. As the evidence suggests that the conditions in our study develop slowly and are rather stable (Bailer et al., 2008; Dieudonné, 2016, 2019; Gruber et al., 2018), we chose to maximize the time interval between the two measurements in order to maximize the probability of observing substantial change. However, no consensus about theoretically optimal time lags for the developmental investigation of IEIs exists to date. Future studies might therefore supplement the main measurement intervals with additional shorter interval measurements to investigate potential covariations on a shorter time scale.

Concerning the question of adequate statistical models, CLPMs have been termed “developmental workhorse” for their common application in the study of reciprocal influences in longitudinal studies (Berry and Willoughby, 2017). In recent years, however, they have been criticized for their lack of adequate decomposition of within- and between-subject variation, thus potentially leading to biased estimates (Hamaker et al., 2015; Mund and Nestler, 2019). The random-intercept cross-lagged panel model (RI-CLPM; Hamaker et al., 2015; Mulder and Hamaker, 2020) has been proposed as an extension to the conventional CLPM, which explicitly separates trait-like and state-like components of variance. To validate our results, we replicated the analysis with the newly developed RI-CLPM. As the RI-CLPM requires three or more measurement points, we included T2 from our study in the analysis (see Appendix). In general, the results of both analyses converged. Cross-lagged effects between the constructs of interest were not significant, and the low significant cross-lagged effects between IEIs and PHQ-15 and MHWs observed in the initial analysis, respectively, were ultimately non-significant. Thus, this validation of results reinforced our conclusion that no meaningful reciprocal associations between the constructs analyzed were apparent in the data.

#### 4.3. Conclusion

In the current study, cross-lagged panel models were applied to investigate reciprocal association between IEIs and general psychopathology, somatic symptoms, and modern health worries in longitudinal data of a large near-representative community sample. Contrary to theoretical predictions, higher initial levels in either of the three constructs were not associated with increases in IEIs over a period of 6 years. The proneness to idiopathic environmental intolerances (IEIs), such as chemical intolerance, noise sensitivity, and electromagnetic hypersensitivity, appeared very stable. Somatic symptoms, indicators of negative affectivity, and modern health worries showed reliable associations with IEIs. However, they did not predict IEIs. Therefore, popular theories of the development of IEIs could not be confirmed empirically.

#### Credit author statement

Luc Watrin: Conceptualization, Methodology, Formal analysis, Writing – original draft, Writing – review & editing, Visualization, Steven Nordin: Conceptualization, Methodology, Investigation, Resources, Writing – review & editing, Funding acquisition, Renáta Szemerszky: Conceptualization, Writing – review & editing, Oliver Wilhelm: Conceptualization, Methodology, Writing – review & editing, Michael Witthöft: Conceptualization, Writing – original draft, Writing – review & editing, Ferenc Kóteles: Conceptualization, Supervision, Writing –

original draft, Writing – review & editing

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**Ethics**

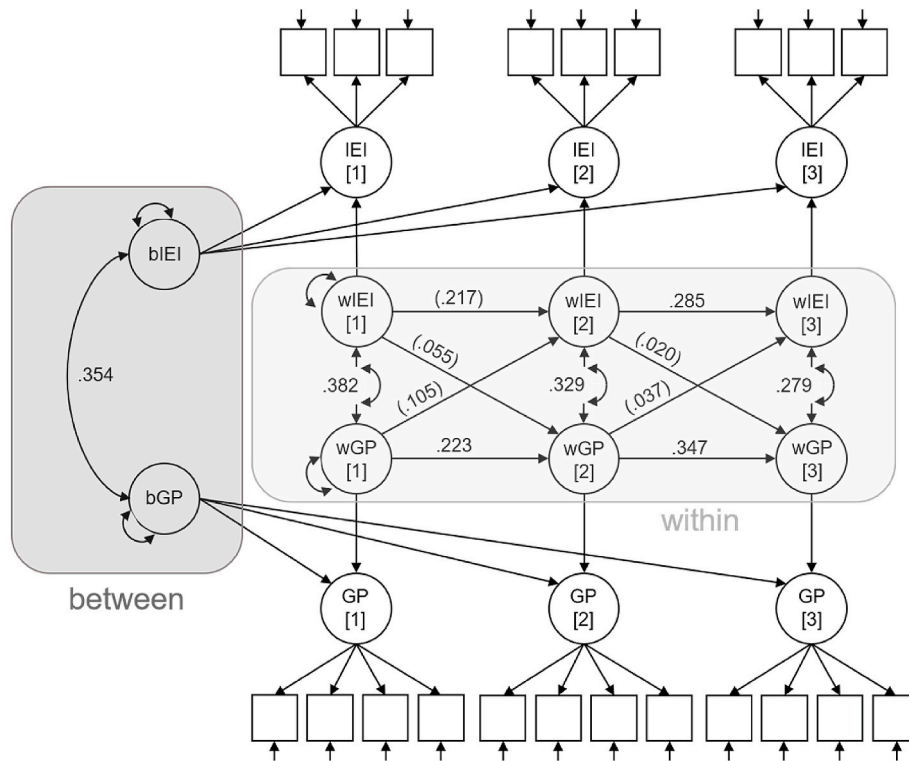
The work described in this article has been carried out in accordance

with *The Code of Ethics of the World Medical Association (Declaration of Helsinki)* for experiments involving humans. The study was approved by the Umeå Regional Ethics Board (Dnr 09–171M and 2016-83-32M).

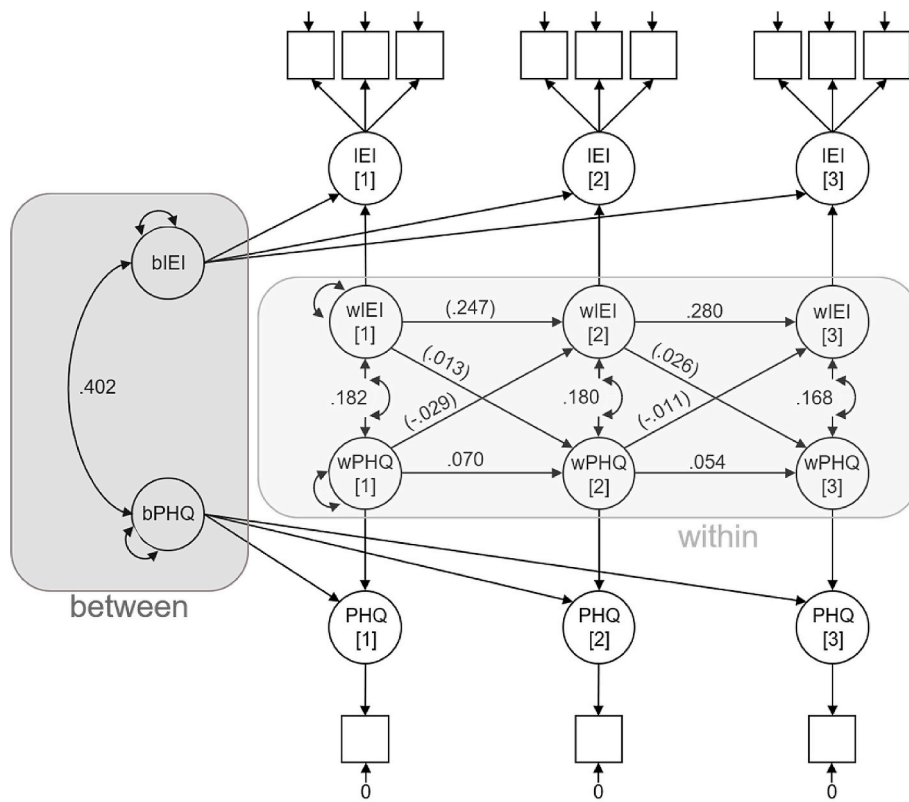
**Declaration of competing interest**

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

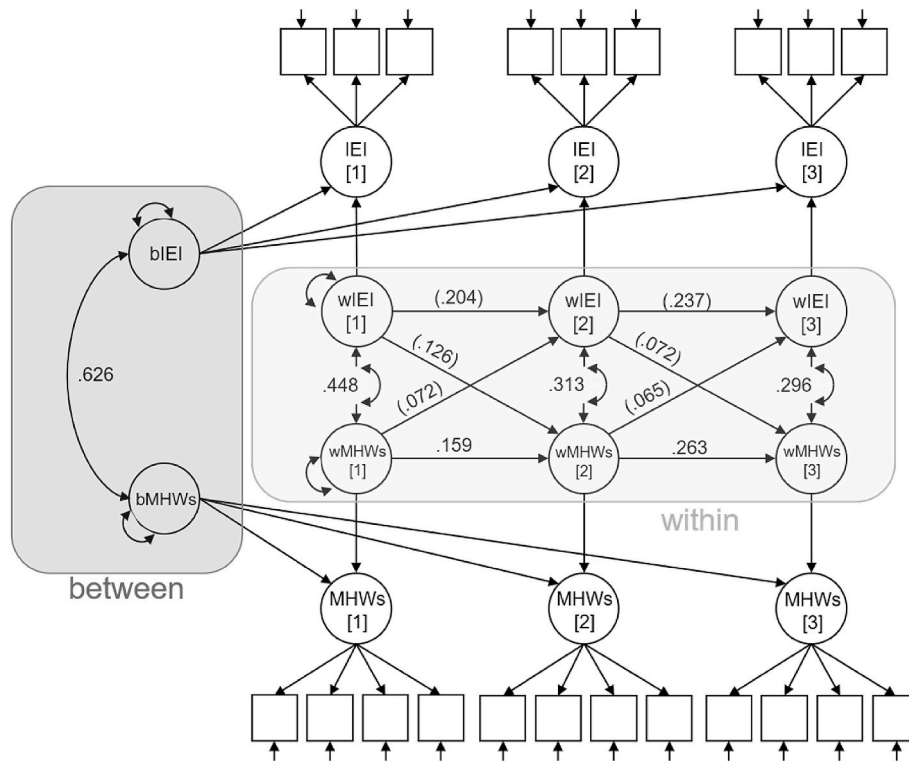
**Appendix A. Random-Intercept Cross-Lagged Panel Models**



**Fig. A.1.** Random-Intercept Cross-Lagged Panel Model for IELs and General Psychopathology.  $N = 1837$ ;  $\chi^2(174) = 630.0$ ,  $p < .01$ ; CFI = .983; RMSEA = .038 [.035-.041]; SRMR = .034; Estimates from the measurement models and residual correlations between the same indicators across time points were omitted for clarity. All coefficients not in parentheses were significantly different from zero ( $p < .05$ ). See the online supplement for a comprehensive model output.



**Fig. A.2.** Random-Intercept Cross-Lagged Panel Model for IELs and Somatic Symptoms (PHQ).  $N = 1837$ ;  $X^2(42) = 106.1$ ,  $p < .01$ ; CFI = .995; RMSEA = .029 [.022-.036]; SRMR = .021; Estimates from the measurement models and residual correlations between the same indicators across time points were omitted for clarity. All coefficients not in parentheses were significantly different from zero ( $p < .05$ ). See the online supplement for a comprehensive model output.



**Fig. A.3.** Random-Intercept Cross-Lagged Panel Model for IELs and MHWs.  $N = 1837$ ;  $X^2(174) = 1508.3$ ,  $p < .01$ ; CFI = .951; RMSEA = .065 [.062-.068]; SRMR = .082; Estimates from the measurement models and residual correlations between the same indicators across time points were omitted for clarity. All coefficients not in parentheses were significantly different from zero ( $p < .05$ ). See the online supplement for a comprehensive model output.

## Appendix B. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.envres.2021.111774>.

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