

Using heterogeneity in disease to understand the relationship between health and personality

Keywords: Pain; personality; arthritis; stratified medicine; ageing

Abstract

The aim of this study was to compare the relationship between two health outcomes (pain and self-reported health) and personality while accounting for heterogeneity in arthritic disease. Traditionally health psychology [and other health research](#) has ~~traded~~ [treated](#) patients' disease experiences as homogeneous_ but stratified medicine suggests that treating a disease as homogenous might over-generalise findings and miss important effects. We present a longitudinal analysis over 14 years, on a subsample of 443 arthritic respondents from the English Longitudinal Study of Ageing (ELSA). [Using linear regressions, w](#)We modelled how [the Big Five domains of](#) personality (wave 5) moderated the relationship between [past health \(at waves 1\) and present health](#) ~~predicting~~(at wave 7). [Then, t](#)o model ~~the~~ heterogeneity in arthritis experience we included assignment to 4 different sub-groups based on their experience of pain progression. The results showed that modelling heterogeneity led to the identification of specific stratified effects_ for personality (neuroticism, agreeableness_ and extraversion) not observed when these data are model treating the sample as homogenous. For example_ higher agreeableness was associated with worse pain for those in a sub-group reporting the greatest pain, and higher extraversion was protective against pain among those whose pain improved. The results highlight the importance of modelling ~~of~~ heterogeneity [in-of](#) disease-state.

Introduction

Stratified medicine ~~—~~grew out of the observation of wide variation in patients' reporting of symptoms and responsiveness to treatment (Kravitz et al., 2004). Stratified medicine identifies illness markers to classify sub-groups of people who are suffering from the same overall condition (Brown, 2011; Chin et al., 2011; Christaki & Giamarellos-Bourboulis, 2014; Kirchhof et al., 2014; Willis & Lord, 2015). We propose that this approach should be extended to health psychology by exploring how ~~standard commonly observed~~ effects ~~seen on health psychology~~ (e.g., personality – health associations) vary as a function of patient sub-groups stratified in terms of different disease experiences ~~of their disease~~ (Ferguson, 2013). In this paper, we set out a proof of principle by examining how the personality-health link varies as a function of different experiences of arthritis.

Disease Heterogeneity: Stratified Behavioural Medicine

Patients have heterogeneous experiences of disease in terms of their display of symptoms and disease progression (Ferguson, 2013). This heterogeneity offers the opportunity for behavioural medicine to model disease stratification. We contend that the effect of psychological factors, such as personality on self-reported health, will predict symptom experiences differentially depending on how patients are stratified. Specifically we examine this with respect to heterogeneity in sub-groups of arthritis patients with different patterns of pain progression.

Arthritis is a common condition ~~worldwide affecting older adults, particularly prevalent in older adults but with variants found in younger adults and children worldwide~~ (Johnson & Hunter, 2014), characterised by pain, swelling, and

discomfort. Arthritis has ~~a number of~~several aetiologies and the experience of pain varies considerably. Indeed, studies have shown there is heterogeneity in pain progress among arthritis patients, with 4 distinct sub-groups ~~with~~ experiencing very different ~~experiences~~patterns of pain progression (James et al. (2018). However, it is not known how stratifying by these sub-groups affects the link between personality and health. Indeed, studies that look at the relationship between personality and health have tended to treat diseases as homogeneous which may diminish the potential for ~~patient~~patient-tailored interventions (Mols et al., 2012; Smith & Zautra, 2002). Specifically within the context of arthritis, samples vary considerable in disease state (Meeus et al., 2012), as well as coverage of personality domains (Matcham et al., 2015).

Pain and personality in arthritis

Personality is important to understanding disease progression (Ferguson, 2013), as it affects disease pathogenesis (Weston et al., 2014), treatment choices (Israel et al., 2014), and compliance (Ironson et al., 2008). But how the effects of personality are moderated by disease heterogeneity is not known. Specifically, there is a core psychological component to pain (Gatchel et al., 2007; Quartana et al., 2009) and personality may influence this. Personality traits such as conscientiousness and neuroticism ~~have been shown to be~~ associated, in general, with better and poorer health respectively (Ferguson, 2013; Weston et al., 2014). Conscientiousness is thought to limit disease progression by limiting engagement in risky behaviours that might lead to the onset of disease (Weston et al., 2014), and being more likely to adhere to treatment regimens when disease develops (Molloy et al., 2014). Neuroticism is associated with a greater incidence of psychological dysfunction,

which may directly affect physical health, as well as through increased somatic symptomology and health service use (Lahey, 2009).

However, the relationship between personality and pain is less consistent. ~~High levels of neuroticism are~~ Neuroticism is linked with greater pain sensitivity (Ong et al., 2010) consistent with its effect on health, but, there is ~~some~~ e evidence that conscientiousness is also associated with pain sensitivity ~~in certain~~ in certain groups (Goubert et al., 2004; Martínez et al., 2011). While conscientiousness is associated with better health outcomes (Sutin et al., 2013), it may become less beneficial when chronic ~~poor health~~ disease emerges, and show a ‘dark side’ (Boyce et al., 2010; Ferguson et al., 2014). Not modelling for disease heterogeneity means this variation is likely to be missed. Therefore, in this paper, we examine, for the first time, the link between personality and pain in arthritis, when accounting for sub-groups of arthritis patients.

We also model the relationship between self-reported health, as a secondary outcome to strengthen the case for modelling heterogeneity. Self-reported health is a strong predictor of overall functioning (Hoffman et al., 2015), mortality in adults (DeSalvo et al., 2006), and health (Hampson et al., 2006). It is also associated with personality, with neuroticism and extraversion negatively and positively associated with evaluations of health respectively (Wasylikiw & Fekken, 2002). Like pain, we expect that disease trajectories moderate the personality-health association.

The present study

This paper, presents the findings of two analyses looking at the predictive relationship (over 14 years) between the Big 5 personality traits, and pain and self-reported health, when stratified by different patterns of disease progression in arthritis. The first set of

analyses treats arthritis as homogenous and examines how current pain, predicted by baseline pain recorded 14 years earlier, is moderated by personality. The second set of analyses includes reliably defined sub-groupings of arthritis patients with different patterns of pain progression and examines the moderating effects of personality are further moderated by the patient sub-group.

In the ELSA cohort, four trajectories of- arthritic pain have previously been identified (James et al., 2018), and are employed in this study. These four groups comprised: (1) a group of respondents reporting little to no pain at outset, which did not change over time ('low or no chronic pain'), (2) a group whose pain became considerably worse ('increasing chronic pain'), (3) a groups whose pain substantially receded ('decreasing chronic pain') and, (4) a group of respondents that reported moderate to severe pain at outset, which receded a little but varied considerably over time ('severe, fluctuating chronic pain'). These groups have previously been shown to predict engagement in social and civic activities (James et al., 2018), and impairments in activities of daily living (James et al., 2019). We will use these sub-groups to model disease heterogeneity in this paper.

Method

Participants

This study uses data from a subsample of arthritic respondents in the ELSA cohort. The ELSA consists of an interview and self-completion module, completed every two years. The data in this study comes from the first seven waves of the ELSA (2002 – 2015). The analysed subsample comprised respondents' self-reporting a doctor' s diagnosis of arthritis between 1998 and 2002, and participated in the ELSA at wave 1. Respondents were excluded if: (1) They reported a diagnosis of arthritis at

wave 2 as beginning in 2002, as their diagnosis was assumed to occur after their wave 1 interview or (2) They reported comorbid cancer at wave 1, as they were used as a comparison sample. This left a total of 889 patients ([Age \$M = 64.63\$, \$S.D. = 10.44\$, 61.98% Female; see James et al. \(2018\) \[for cross-trajectory descriptive statistics\]\(#\)\). Of these, 502 had pain data at wave 5, and 380 at wave 7. Pain data at wave 7 was imputed for respondents who completed the personality part of the self-completion module at wave 5 \(Agreeableness – 438, Conscientiousness – 443, Extraversion – 441, Neuroticism – 443, Openness – 443\). \[The details of this are reported in Figure 1\]\(#\)](#)

Cases were not subdivided based on kind of arthritis - prior analysis validating the trajectories showed that membership did not vary arthritis kind (Supplementary Table 1) (James et al., 2018). The sample is sufficiently powered to detect the effects being investigated in this study. Post-hoc power analysis using G-Power, ~~for the weakest R^2 reported in this analysis, using listwise deletion (rather than imputation), and using the largest number of predictors entered into a model, is 1.~~ [for all models was 1.](#)

Data availability

The data, [alongside documentation regarding the sampling and interviewing of the participants](#), is publicly available from the UK Data Archive (Marmot et al., 2017).

Measurements

Pain. Respondents rated if they were troubled by pain, and if so, would they rate their pain as mild, moderate or severe, at each wave of the study. These items were combined to create a scale from 0 (not troubled by pain) to 3 (severely troubled by pain). The responses to these pain questions at waves 1 and 7 were analysed in this study.

Poor Health. Across the ELSA, respondents were asked to subjectively rate their health (“Would you say your health is...”) At wave one, participants were randomly assigned to receive one of two five-point scoring schemes, (a) “Excellent”, “Very Good”, “Good”, “Fair” and “Poor”, or (b) “Very Good”, “Good”, “Fair”, “Bad” and “Very Bad”. In subsequent waves the first scheme (Excellent-Poor) was retained and given to all participants. In the [whole-sample ELSA cohort](#), the Very Good-Very Bad scoring scheme was associated with slightly more positive evaluations of health ($M(a) = 2.833$, $M(b) = 2.734$, $t(11787) = 4.779$, $p < .001$, $d = -0.088$), but this difference was not significant in the arthritis subsample [studied here](#) ($M(a) = 3.077$, $M(b) = 2.978$, $t(868.79) = 1.339$, $p = .18$, $d = -.09$). Therefore, values from these two measures were combined to give a single measure of poor health.

Big 5. At wave 5, respondents were asked to complete a revised version of the Midlife Development Inventory (MIDI) (Lachman & Weaver, 1997), previously administered in the Midlife in the United States (MIDUS) II study (Lachman & Weaver, 2005). The MIDI consists of 26 adjectives measuring the Big Five traits (Openness, (O, 7 items) Conscientiousness (C, 5 items), Extraversion (E, 5 items), Agreeableness (A, 5 items) and Neuroticism (N, 4 items)). Respondents were asked to “indicate how well each of the following [words] describes you”. Each adjective had four Likert response options (a lot, some, a little, not at all, scored 1-4). All except two items (calm and careless) are reverse-scored. The responses are then averaged to produce a trait score. Higher scores represent greater levels of endorsement of each trait (e.g. higher extraversion, higher neuroticism). Scores were not computed when more than half of the items (3 or more A, C & E; 2 N; 4 O) were missing.

Patient Sub-Group Assignment

Patients were assigned to one of the 4 patterns of pain progression defined in a previous latent class growth analysis of the ELSA sample, based on their self-reports of pain (James et al., 2018). The first group (“Low or No chronic pain”) reported minimal pain at wave 1 and did not show significant change over time. The second group (“Increasing chronic pain”) began at a similar pain level to the previous group, but showed increasing levels of pain over time. The third group (“Decreasing chronic pain”) started off in significant pain but improved over time. The fourth group (“Severe fluctuating chronic pain”) began in considerable pain, but fluctuated over time, getting better and worse at junctures but improving slightly overall. Self-reported pain at waves 1 and 7 by respondents in each trajectory are reported in Supplementary Tables S2 and S3. The model had high classification accuracy (entropy = 0.85).

Missing data

Sensitivity analyses using multivariate imputation by chained equations (MICE) were conducted. [The missing data ~~was~~were imputed because both pain and self-reported health are potential predictors of dropout, and thus may be missing at random, and so using an imputation approach like MICE is appropriate.](#) MICE assumes that the data are missing at random. Pain or self-reported health at wave 7 was imputed using respondents’ previous reports of pain and health respectively, and three auxiliary variables (depression, age and sex). For the cases with data at wave 7, these variables were strongly associated with pain ($R^2 = 0.383$) and health ($R^2 = 0.534$). Multiple imputation was conducted using the Multivariate Imputation by Chained Equations (‘mice’) package in *R* (Buuren & Groothuis-Oudshoorn, 2010). Because the level of missing data was considerable, we generated 50 imputations

(Bodner, 2008). The tables report the pooled estimates from ordinal least squares regressions of these imputations.

Modelling

Multiple linear regression was used to regress self-reported health or pain at wave 7, on health or pain at wave 1 and personality, with moderation between wave 1 health or pain and personality. Arthritis was first modelled homogeneously, before including dummies for the 4 patterns of pain progression. Likelihood ratio tests were used to examine whether adding pain progressions improved model fit. The personality traits were grand mean-centred. Patterns of pain progression were dummy coded, with the “low or no chronic pain” group used as the reference class. The modelling was conducted using *R* (R Core Team, 2017).

For listwise models, interactions at the homogeneous level were decomposed by plotting the simple intercepts and simple slopes at the mean and +/- 1 standard deviations (SDs) from the mean (Figure S1). The observed interactions in the heterogeneous analyses were decomposed by re-estimating the models using only the reference group and the trajectory where the interaction was located. These models are reported in Supplementary Tables S4 and S5. The simple slopes were calculated using the methods proposed by Preacher et al. (2003) which is integrated with *R*. The figures were developed using the *ggplot2* package (Wickham, 2016).

Results

The descriptive statistics of the personality items (Supplementary Table S6-S7) correspond closely to those reported in [the](#) user manual of the MIDI (Lachman & Weaver, 1997) and showed good internal consistency ($M\alpha = 0.74$).

Arthritis as a homogenous condition

The moderated regression models showed that pain reported at wave 1 is predictive of pain at wave 7 (Table 1). Personality traits were not directly associated with pain at wave 7. There was evidence of an interaction between conscientiousness and initial pain; the association between wave 1 and wave 7 pain was greatest when conscientiousness was low, and got weaker as conscientiousness increased (Figure S1). For the self-reported health measures, it was similarly found that worse health at baseline was predictive of worse health at wave 7 (Table 2), and there was no evidence of an association for personality, nor for ~~interaction~~interaction.

Arthritis as a heterogeneous condition

Each of the models (Table 3, Supplementary Table S8) shows that membership of two of the pain progression sub-groups (“Increasing chronic pain” and “severe, fluctuating chronic pain”) were predictive of pain at wave 7. There was a three-way interaction between pain, membership of the increasing chronic pain trajectory, and neuroticism. (Table 3). Respondents in the “increasing chronic pain trajectory” who were low in neuroticism (Figure 42), and had no pain at wave 1, reported greater pain at wave 7 relative to those troubled by pain at wave 1.

For the self-reported health, effects emerged for extraversion and agreeableness (Table 4, Supplementary Table S9), which were present in both the listwise and imputed data. There was a three-way interaction between extraversion, membership of the “decreasing pain trajectory” and worse health at wave 1: this is because higher extraversion and better health at wave 1 are associated with better health at wave 7 for those in the ‘no or low chronic pain’ trajectory group, and conversely lower extraversion and worse health at wave 1 is associated with worse health for those in the ‘decreasing pain trajectory’ group (Figure 23). There was a

two-way interaction between agreeableness and trajectory (Figure 34), with more agreeable respondents in the ‘severe chronic pain trajectory’ group reporting worse subsequent health. In the listwise deleted models, a fourth interaction between pain, the increasing pain trajectory, and conscientiousness (Tables S10-S15).

Arthritis type had no effect on these analyses (Tables S16-17).

Discussion

This study compared the relationship between markers of poor health and personality when modelling disease heterogeneity. When heterogeneity was modelled effects of personality on indicators of health were observed. Specifically, an interaction between neuroticism and pain, and extraversion and agreeableness on self-reported health were observed, that are not observed when these data are treated as homogeneous. The interaction between neuroticism and pain was observed in people following experiencing worsening pain over time. Respondents who were not troubled by pain at wave 1, and reported lower neuroticism, experienced greater pain at wave 7 compared to those who were in some pain at wave 1. A robust understanding of the effects of personality on arthritis or other diseases progression thus requires accounting for these distinct sub-groups experiencing different patterns of pain progression (Edwards et al., 2011).

The interaction between neuroticism may reflect the ‘bright’ side of neuroticism (Nettle, 2006). For those already troubled by pain at wave 1, the finding that greater neuroticism is associated with future pain concurs with the existing literature. However, the opposite appears to be the case for those not troubled by pain at wave 1; for this group the lowest pain at wave 7 was found among those with higher neuroticism. This may reflect a ‘healthy neuroticism’ phenotype (Ilieva, 2015;

Turiano et al., 2013; Weston & Jackson, 2015). Alternatively, people lower in neuroticism tend to report less pain but are also less likely to seek healthcare when pain gets worse (Costa & McCrae, 1987), and so when pain emerges they do not engage in behaviours that manage pain. This might be because low neuroticism is linked to reduced attention to danger signals (Lommen et al., 2010) and therefore may miss this early warning sign.

Effects of personality on self-reported health were only observed when heterogeneity in pain progression was accounted for. When pain progression was included, agreeableness and extraversion were moderators of initial self-reported health on later self-reported health. Higher extraversion was protective against worse health for respondents in the ‘low or no chronic pain’ trajectory who reported better health at wave 1. Additionally, for those in the ‘decreasing pain’ trajectory reporting worse health at wave 1, worse health at wave 7 was reported by patients with low extraversion. Our findings extend previous research showing that extraversion is associated with better subjective health (Williams et al., 2004).

There was an interaction between agreeableness and membership of the ‘severe pain’ trajectory, with more agreeable respondents in the ‘severe pain’ trajectory reporting worsening health. This may reflect a framing effect for agreeableness. People higher in agreeableness rate their subjective health more positively, especially among those with self-reported medical problems (Goodwin & Engstrom, 2002), and believe themselves to be less susceptible to poor health (Vollrath et al., 1999). Therefore, the emergence of poor health is felt more keenly amongst agreeable people. This has its basis in the heuristics people apply when making decisions, including health. It is well established that people use reference points to make judgements, using heuristics such as ‘anchoring and adjustment’ and

‘availability’, and that these reference points vary across individuals based on local contextual cues (Tversky & Kahneman, 1974). Agreeableness appears to create a biased reference point in judgements of health, and this may be anchoring subsequent judgements as chronic disease develops.

There are a number of limitations that ought to be considered with regard to these analyses. Personality was not measured at the same time as initial pain, but at wave 5. However, the heterogeneous analysis accounts for this somewhat, as it models changes in pain over time. However, using latent class membership to assign respondents can be problematic. Although the classification accuracy of the model was high, the analyses do not account for classification error nor the probabilistic nature of the assignment. Further, while the ELSA contains a wealth of data on many areas of functioning, it only provides limited information regarding respondents’ treatments; it is not possible to ascertain whether pain improvement is due to efficacious treatment. The pain measurement is of generalised pain, and ~~have~~has not been validated, although the content and structure are similar to measures of pain such as the EQ-5D-3L.

These analyses highlight the need for segmentation of diseases with a heterogeneous disease profile. Treating respondents with a recent arthritis diagnosis homogeneously, few effects of personality on health were identified. In contrast, by treating this sample as heterogeneous, relationships between personality and health indicators began to emerge. Capturing essentially variation in disease state is vital in understanding how psychological constructs affect health.

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RUNNING HEAD: HETEROGENEITY, HEALTH AND PERSONALITY

Table 1
Moderated linear regression models of the effects of each personality trait (and initial pain) on pain at wave 7 of the ELSA, with imputed data

Variable	<i>b</i>	<i>SE</i>	<i>p</i>	95% <i>CI</i>
Agreeableness				
Intercept	0.674	0.069	<.001 ***	0.538 – 0.810
Pain at W1	0.405	0.049	<.001 ***	0.309 – 0.502
Agreeableness	0.013	0.151	.929	-0.283 – 0.310
W1 Pain * Agree.	0.027	0.104	.797	-0.178 – 0.231
Conscientiousness				
Intercept	0.666	0.071	<.001 ***	0.526 – 0.805
Pain at W1	0.400	0.050	<.001 ***	0.301 – 0.495
Conscientiousness	0.237	0.156	.147	-0.080 – 0.534
W1 Pain * Conc.	-0.185	0.091	.042 *	-0.363 – -0.007
Extraversion				
Intercept	0.682	0.071	<.001 ***	0.544 – 0.821
Pain at W1	0.398	0.049	<.001 ***	0.301 – 0.495
Extraversion	-0.045	0.131	.734	-0.304 – 0.214
W1 Pain * Extra.	-0.057	0.082	.492	-0.221 – 0.106
Neuroticism				
Intercept	0.681	0.070	<.001 ***	0.544 – 0.819
Pain at W1	0.402	0.049	<.001 ***	0.305 – 0.498
Neuroticism	0.140	0.133	.294	-0.122 – 0.402
W1 Pain * Neur.	-0.001	0.087	.995	-0.171 – 0.170
Openness to Experience				
Intercept	0.677	0.071	<.001 ***	0.538 – 0.815
Pain at W1	0.403	0.049	<.001 ***	0.306 – 0.500
Openness	0.029	0.131	.828	-0.230 – 0.287

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W1 Pain * Open.	-0.084	0.081	.305	-0.255 – 0.077
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Note: Agree = Agreeableness, Conc. = Conscientiousness, Extra. = Extraversion,

Neur. = Neuroticism, Open. = Openness to Experience. W1 = wave one. *** = $p <$

.001.

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Table 2

Moderated linear regression models of the effects of each personality trait (and initial SRH) on SRH at wave 7 of the ELSA, with imputed data.

Variable	<i>b</i>	<i>SE</i>	<i>p</i>	95% <i>CI</i>
Agreeableness				
Intercept	1.871	0.129	<.001 ***	1.617 – 2.125
SRH at W1	0.447	0.042	<.001 ***	0.364 – 0.531
Agreeableness	-0.019	0.263	.943	-0.536 – 0.498
W1 SRH * Agree.	-0.053	0.082	.517	-0.215 – 0.109
Conscientiousness				
Intercept	1.927	0.131	<.001 ***	1.670 – 2.184
SRH at W1	0.432	0.043	<.001 ***	0.347 – 0.517
Conscientiousness	-0.431	0.259	.096	-0.940 – 0.077
W1 SRH * Conc.	0.033	0.075	.662	-0.114 – 0.180
Extraversion				
Intercept	1.977	0.129	<.001 ***	1.723 – 2.231
SRH at W1	0.413	0.042	<.001 ***	0.329 – 0.496
Extraversion	-0.357	0.217	.102	-0.784 – 0.071
W1 SRH * Extra.	-0.025	0.067	.706	-0.156 – 0.106
Neuroticism				
Intercept	1.845	0.129	<.001 ***	1.592 – 2.099
SRH at W1	0.458	0.042	<.001 ***	0.374 – 0.541
Neuroticism	-0.147	0.211	0.487	-0.563 – 0.269
W1 SRH * Neur.	0.130	0.069	0.062	-0.007 – 0.266
Openness to Experience				
Intercept	1.908	0.129	<.001 ***	1.654 – 2.162
SRH at W1	0.435	0.043	<.001 ***	0.352 – 0.519
Openness	-0.187	0.216	.386	-0.612 – 0.237
W1 SRH * Open.	-0.046	0.068	.501	-0.181 – 0.088

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Note: SRH = Self reported (worse) health, Agree = Agreeableness, Conc. = Conscientiousness, Extra. = Extraversion, Neur. = Neuroticism, Open. = Openness to Experience. W1 = wave one. *** = $p < .001$.

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Table 3
Moderated linear regressions of the relationship between pain, trajectory and personality in Neuroticism, imputed data

Effect	<i>b</i>	<i>S.E.</i>	<i>p</i>	95% <i>C. I.</i>
Neuroticism				
Intercept	0.376	0.077	<.001***	0.224 – 0.527
W1	0.058	0.160	.716	-0.257 – 0.374
Class B	1.253	0.158	<.001***	0.941 – 1.565
Class C	0.234	0.629	.711	-1.005 – 1.473
Class D	1.212	0.471	.011*	0.285 – 2.139
Neuroticism	0.012	0.140	.932	-0.264 – 0.288
W1*Class B	-0.282	0.254	.269	-0.783 – 0.220
W1*Class C	0.125	0.312	.689	-0.490 – 0.740
W1*Class D	0.173	0.255	.498	-0.329 – 0.675
W1*Neur.	0.036	0.274	.895	-0.503 – 0.575
Class B*Neur.	-0.348	0.288	.228	-0.916 – 0.220
Class C*Neur.	1.524	1.181	.198	-0.803 – 3.851
Class D*Neur.	-0.545	0.778	.484	-2.076 – 0.985
W1*Class B*Neur.	0.985	0.438	.025*	0.122 – 1.848
W1*Class C*Neur.	-0.626	0.606	.303	-1.821 – 0.570
W1*Class D*Neur.	0.169	0.425	.692	-0.668 – 1.006

Note on abbreviations: Reference = low or no chronic pain, Class B = increasing chronic pain, Class C = decreasing chronic pain, Class D = severe fluctuating chronic pain. W1 = wave 1 pain, Neur = Neuroticism. W1 = wave one. * = $p < .05$, *** = $p < .001$.

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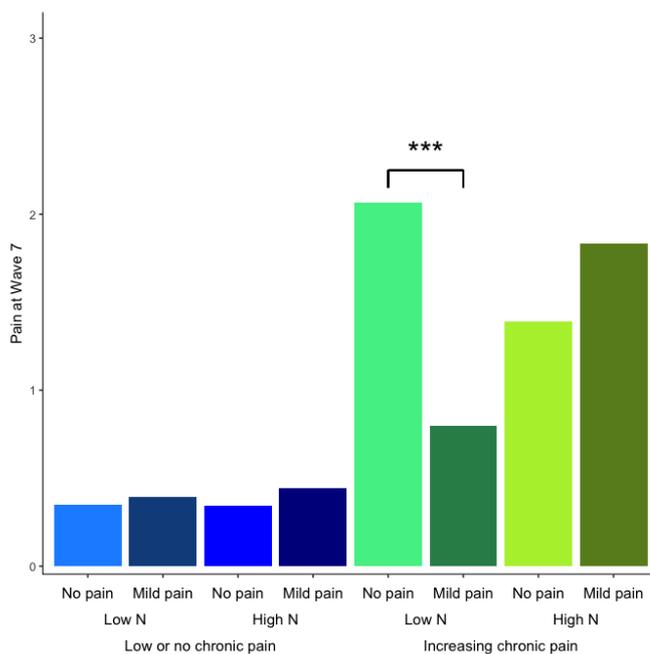
Table 4
Moderated linear regressions of the relationship between SRH, trajectory and personality in Agreeableness and Extraversion, imputed data

Effect	<i>b</i>	<i>S.E.</i>	<i>p</i>	95% <i>C. I.</i>
Agreeableness				
Intercept	1.776	0.178	<.001***	1.425 – 2.126
W1	0.381	0.069	<.001***	0.246 – 0.516
Class B	1.079	0.348	.002**	0.395 – 1.763
Class C	-0.029	0.455	.949	-0.924 – 0.865
Class D	0.634	0.419	.131	-0.190 – 1.458
Agreeableness	-0.336	0.339	.321	-1.002 – 0.330
W1*Class B	-0.194	0.118	.102	-0.426 – 0.039
W1*Class C	0.033	0.142	.818	-0.246 – 0.311
W1*Class D	0.028	0.120	.819	-0.209 – 0.264
W1*Agre.	0.045	0.130	.728	-0.211 – 0.301
Class B*Agre.	-0.496	0.816	.543	-2.102 – 1.109
Class C*Agre.	0.973	1.001	.332	-1.000 – 2.946
Class D*Agre.	2.136	0.877	.015*	0.412 – 3.860
W1*Class B*Agre.	0.091	0.269	.735	-0.439 – 0.621
W1*Class C*Agre.	-0.378	0.285	.185	-0.940 – 0.183
W1*Class D*Agre.	-0.420	0.241	.083	-0.894 – 0.055
Extraversion				
Intercept	1.907	0.183	<.001***	1.547 – 2.266
W1	0.340	0.069	<.001***	0.204 – 0.475
Class B	0.920	0.332	.006**	0.266 – 1.573
Class C	-0.268	0.416	.520	-1.086 – 0.550
Class D	0.832	0.438	.058	-0.031 – 1.695
Extraversion	-0.480	0.301	.112	-1.072 – 0.112
W1*Class B	-0.129	0.114	.262	-0.353 – 0.096
W1*Class C	0.108	0.134	.421	-0.155 – 0.370
W1*Class D	-0.027	0.130	.838	-0.283 – 0.230
W1*Extr.	0.068	0.104	.516	-0.137 – 0.273

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Class B*Extr.	-0.551	0.617	.372	-1.765 – 0.662
Class C*Extr.	1.144	0.742	.124	-0.316 – 2.605
Class D*Extr.	0.251	0.657	.703	-1.041 – 1.543
W1*Class B*Extr.	0.072	0.207	.729	-0.335 – 0.479
W1*Class C*Extr.	-0.451	0.226	.047*	-0.896 – -0.006
W1*Class D*Extr.	-0.069	0.187	.713	-0.438 – 0.300

Note on abbreviations: Reference = low or no chronic pain, Class B = increasing chronic pain, Class C = decreasing chronic pain, Class D = severe fluctuating chronic pain. W1 = wave 1 self-reported worse health, Agree = Agreeableness, Extr = Extraversion. W1 = wave one. * = $p < .05$, ** = $p < .01$, *** = $p < .001$.



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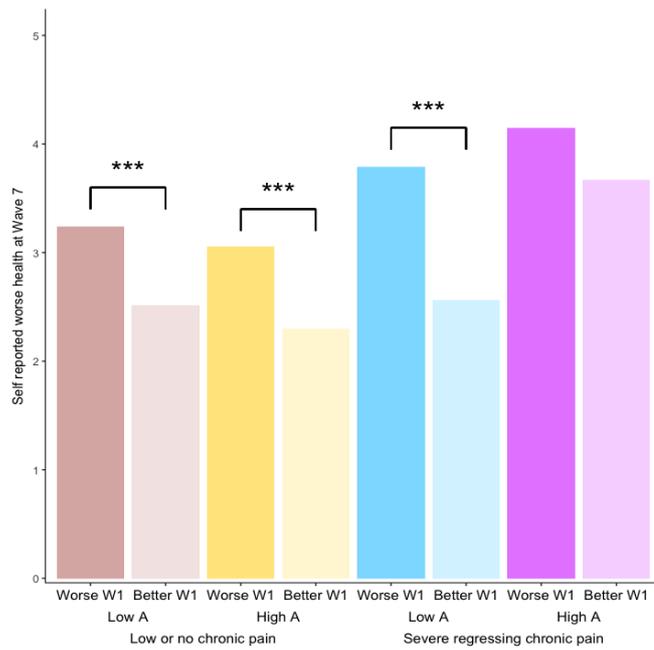
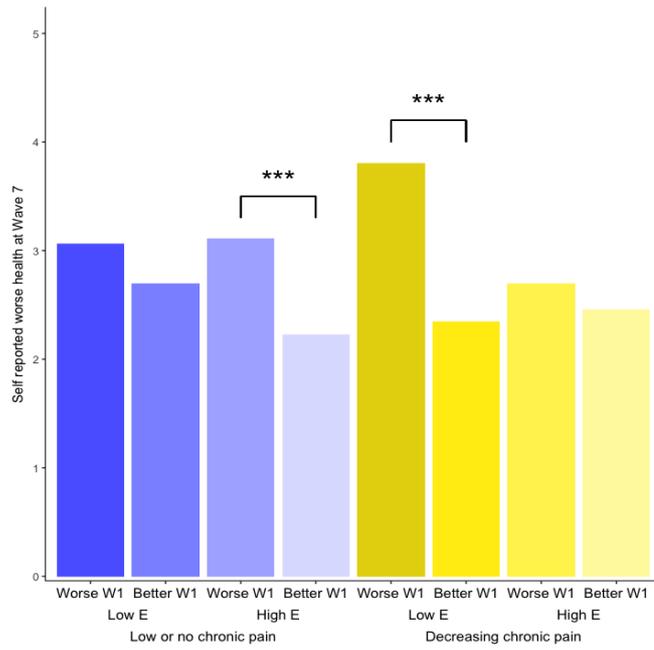


FIGURE LEGENDS

[Figure 1](#)

[Flowchart of the sample analysed in this study.](#)

Figure ~~12~~

Decomposition of the three way interaction between membership of the increasing pain group, pain at wave 1 (respondent reported 'not being troubled by pain' vs. troubled by pain with mild severity) and Neuroticism (+/- 1 SD from the mean) on wave 7 pain. [Note: * = \$p < .05\$, ** = \$p < .01\$, *** = \$p < .001\$.](#)

Figure ~~23~~

Decomposition of the three way interaction between membership of the decreasing pain group, self-reported worse health at wave 1 (+/- 1 SD from the mean) and Extraversion (+/- 1 SD from the mean) on wave 7 pain. [Note: * = \$p < .05\$, ** = \$p < .01\$, *** = \$p < .001\$.](#)

Figure ~~43~~

Decomposition of the interaction between membership of the severe regressing chronic pain group and Agreeableness (+/- 1 SD from the mean) on wave 7 pain. [Note: * = \$p < .05\$, ** = \$p < .01\$, *** = \$p < .001\$.](#)