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The effect of executive function on the development of chronic pain: A prospective longitudinal study

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ABSTRACT

Increasing evidence suggests a close association between chronic pain and executive function, a set of cognitive processes necessary for goal-directed behaviors. However, there is a dearth of longitudinal studies examining the predictive effect of executive function on the development of chronic pain. Drawing on the cyclical model of executive function and health, we sought to examine how executive function, measured at baseline, may predict chronic pain etiology approximately 9 years later. Using a large-scale dataset of midlife adults ($N = 1553$) from the MIDUS 2 and 3 (Midlife Development in the United States) studies, we employed multivariate logistic regression to examine the etiology of new chronic pain for individuals who did not have chronic pain at baseline. Further, we also tested whether executive function predicted the degree of pain interference, among individuals with chronic pain. Our results revealed that lower baseline executive function was associated with a significant likelihood of developing chronic pain 9 years later (OR = 0.812, $p = .001$), even after adjusting for demographics, health, and psychosocial confounds (OR = 0.827, $p = .014$). However, executive function failed to robustly predict the etiology and degree of chronic pain interference. Our findings underscore the critical role of executive function on the development of chronic pain.

Chronic pain, persistent pain that endures beyond normal healing time, is a common health concern among older adults, affecting an estimated 20% of the population in the United States (Dahlhamer et al., 2018). Chronic pain affects individuals' personal relationships and quality of life (Reid et al., 2011), and their work productivity (Azevedo et al., 2016). Given its toll on the individual and on society, it is therefore important to identify factors associated with the development of chronic pain. Beyond biomedical factors such as post-surgery acute pain (Fassoulaki et al., 2008) and changes in health status (Bergman et al., 2004), emerging research suggests that cognitive factors, specifically executive function (EF), could contribute to the development of chronic pain and pain-related outcomes (e.g., pain intensity; Attal et al., 2014). Despite this possibility, scant research has examined the predictive value of executive function on the development of chronic pain. In view of this, the current paper sought to elucidate how executive function, measured at baseline, may relate to the development of chronic pain approximately 9 years later, using a large national sample of midlife adults. Further, we explored if EF would predict the degree of pain interference, among individuals who developed chronic pain.

Chronic pain is a complex experience with wide-ranging implications. Recognized as a major health problem with higher prevalence among older adults (Dahlhamer et al., 2018), chronic pain may bring about deleterious effects on the individual including decreased quality of life (Ataoğlu et al., 2013), depression (Magni et al., 1994), anxiety (McWilliams et al., 2003), and impaired cognitive functioning (Baker et al., 2016; Rouch et al., 2021). Further, chronic pain has been associated with dependence on opioids (Institute of Medicine, 2011) and is considered to be an underlying cause contributing to the opioid crisis (Voon et al., 2017). From an economic viewpoint, chronic pain can affect individuals' work productivity, resulting in days of work missed (Azevedo et al., 2016). It is estimated that chronic pain may cost up to $635 billion annually, inclusive of healthcare and labour costs (Institute of Medicine, 2011). The prevalence of chronic pain, together with the societal and economic costs it incurs, has fueled increasing concerns about the prevention of pain (Institute of Medicine, 2011), generating a concerted effort among researchers to identify modifiable risk factors that may influence individuals' vulnerability to the development of chronic pain.

An array of risk factors associated with pain have been identified, including old age (Elliott et al., 1999), health conditions such as obesity

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(Hitt et al., 2007), nerve injury (Kehlet et al., 2006), as well as psychosocial factors including anxiety, depression, discrimination, socioeconomic status, trauma exposure, and family strain (Bonathan et al., 2013; Brown et al., 2018; Hinrichs-Rocker et al., 2009; Theunissen et al., 2012; Woods et al., 2019). Beyond these risk factors, a promising factor that has received less attention is cognitive function, despite existing research literature suggesting a close association between cognitive function and chronic pain.

Growing evidence suggests that the experience of pain may compromise cognitive functions such as executive function (EF), a set of cognitive processes crucial for goal-directed behaviors which may include health-promoting behaviors (Hall et al., 2006; Miyake and Friedman, 2012). Several studies have demonstrated that individuals with chronic pain reported overall EF impairments (Baker et al., 2016), had poorer working memory performance (Berryman et al., 2013), and demonstrated EF impairment (see Berryman et al., 2014 for a review), compared to their pain-free counterparts. However, while the experience of chronic pain may compromise EF, it is also plausible that impaired EF may contribute to the development of chronic pain (Attal et al., 2014). Despite this possibility, there is a dearth of longitudinal studies that have examined this relation.

Several mechanisms may shed light on the causal influence of EF on pain. First, neural systems involved in EF and pain may reciprocally modulate one another given that they both engage an overlapping set of mental resources (Buhle and Wager, 2010). Affirming the idea of shared mental resources, functional imaging studies have established that the brain regions involved in EF and pain processing share a significant overlap. Specifically, the prefrontal cortex (Miller and Cohen, 2001) and anterior cingulate cortex (Moriarty et al., 2011) are both involved in pain modulation and aspects of executive function, including working memory and inhibitory control (Klein et al., 2007; Rubia et al., 2003). This implies that the areas involved in EF and pain may have reciprocal modulatory effects. Indeed, Attal et al. (2014) found that performance on the Trail-Making Test B and Rey-Osterrieth Complex Figure Test predicted the presence and severity of pain 6 months and 12 months later in a small surgical cohort, providing preliminary evidence that poorer EF may be related to the mechanisms of pain chronicity.

Second, drawing on the cyclical model of executive function and health (Allom and Mullan, 2014), EF are regarded to be essential in carrying out favorable health behaviors (Reimann et al., 2020) and have been consistently linked to more favorable physical health outcomes that may buffer against the development of chronic pain. For example, research suggests a significant relationship between higher EF and behaviors such as healthier dietary behaviors (Allom and Mullan, 2014), greater levels of physical activity (Daly et al., 2015), and lower levels of tobacco dependence (Flaudias et al., 2016). These behaviors, in turn, are associated with lower risk of chronic pain (Jakobsson and Larsson, 2014; Landmark et al., 2013; Van Hecke et al., 2013). Given the importance of EF for health-related outcomes, it is plausible that individuals with enhanced EF may engage in activities that lower the risk of developing chronic pain. Taken together, the findings above suggest that EF can possibly influence the development of chronic pain.

1. Present study

In view of the theoretical and empirical evidence discussed above, the present research has two goals. First, we sought to examine the predictive role of baseline EF on the development of chronic pain approximately 9 years later. To achieve our goals, we utilized a longitudinal approach to analyze a large nationally representative sample of middle-aged and older adults from the MIDUS (Midlife Development in the United States) study. Second, we also aimed to examine relation between cognition and pain interference. While the impairment in EF may incline individuals to allocate attention toward pain-relevant information (Godfrey et al., 2020) instead of inhibiting the pain experience (Oosterman et al., 2010), the relation between cognition and pain interference is mostly inconsistent in the literature (van der Leeuw et al., 2016; Oosterman et al., 2012). Given the mixed results, we also sought to explore if baseline EF would be related to the degree of pain interference approximately 9 years later, among middle-aged and older adults with chronic pain.

2. Method

2.1. Participants

The present study comprises midlife adults who completed the second (II) and third (III) waves of MIDUS and participated in the Cognitive Project component of MIDUS II. The MIDUS study is a national longitudinal study of non-institutionalized English-speaking American adults, aged 25 to 74, from across 48 states who were recruited through random digit sampling (Brim et al., 2004). MIDUS I, the first wave of data collection, was conducted in 1995–1996. MIDUS II was conducted in 2004–2006 and MIDUS III was conducted in 2013–2014 as a longitudinal follow-up to MIDUS II. Participants completed phone interviews and self-administered questionnaires returned by mail. Participants who took part in the Cognitive Project were a subset of participants from MIDUS II and had to undergo a series of cognitive tasks that were verbally administered over a 30-min phone interview.

In accordance with Woods et al. (2019), to assess the etiology of chronic pain, we only included participants who responded "No" to a pain-screener item ("Do you have chronic pain, that is do you have pain that persists beyond the time of normal healing and has lasted anywhere from a few months to many years?") at MIDUS II and responded to this same item at MIDUS III. We further excluded 40 participants who reported chronic pain and were undergoing cancer treatment at MIDUS III. This resulted in a final sample of 1553 participants. The average age of the sample was 54.43 years old ($SD = 11.30$), with majority being male (53.70%), married (74.61%), and had completed 2 years of college education (52.3%). Of the 1553 participants who denied chronic pain at baseline (MIDUS II), 406 participants (26.1%) later reported experiencing chronic pain at MIDUS III (see Table 1). These participants mostly experienced pain on their back (44.3%; see Table S1). All participants provided informed consent and data collection was approved by the Health Sciences IRBs at the University of Wisconsin-Madison. Data can be freely accessed via the Inter-University Consortium for Political and Social Research (http://www.icpsr.umich.edu). Our data files and analysis scripts are openly available at OSF via https://osf.io/24qmh/.

2.2. Measures

All measures, except the parental abuse and dependent variables (i.e., chronic pain and chronic pain interference), were obtained via the MIDUS II self-administered questionnaire. We used the abuse data from MIDUS I, and chronic pain data from MIDUS II and MIDUS III self-administered questionnaire to assess the etiology of chronic pain.

2.2.1. Chronic pain

Chronic pain was assessed with the question "Do you have chronic pain, that is do you have pain that persists beyond the time of normal healing and has lasted anywhere from a few months to many years?". Participants who responded "No" at MIDUS II and "Yes" at MIDUS III received an etiology score of 1 ($N = 406$). Participants who responded "No" at MIDUS II and MIDUS III received a value of 0 ($N = 1147$).

2.2.2. Chronic pain interference

Participants who indicated experiencing chronic pain at MIDUS III completed a short 5-item version of the Brief Pain Inventory (BPI) scale (Cleeland, 1991), a reliable and valid instrument used to assess pain-related interference (Raichle et al., 2006). Participants reported the extent to which chronic pain interfered with their general activity,

Table 1
Descriptive statistics.

	N	*M*	(*SD*)	Range
Demographics				
Age (years)	1553	54.43	(11.30)	33–83
Sex (% male)	1553	53.70%		
Marital status (% married)	1552	74.61%		
Income	1529	78522.30	(62056.56)	0–300,000
Income group (quartile)	1529	2.50	(1.12)	1–4
Education	1550	7.75	(2.50)	1–12
Health				
Smoking (% current smokers)	1553	11.65%		
Number of surgeries	1486	1.11 (2.52)		0–54
Body Mass Index	1499	27.25 (4.95)		16.99–49.92
Sleep problems	1553	7.02% (.26)		
Physical health	1553	2.12 (.86)		1–5
Personality				
Depressive symptoms	1553	.38	(1.41)	0–7
Anxiety symptoms	1553	.07	(.60)	0–8
Agreeableness	1542	3.44	(.49)	1.80–4.00
Extraversion	1542	3.12	(.56)	1.20–4.00
Neuroticism	1541	1.99	(.59)	1.00–4.00
Conscientiousness	1542	3.51	(.42)	1.75–4.00
Openness	1528	2.94	(.52)	1.43–4.00
Religious coping	1541	5.65	(2.13)	2–8
Parental abuse	1505	3.18	(0.80)	1–4
Not in workforce	1548	26.3%		
Predictor				
Executive function	1553	0.27	(.90)	−4.80–3.40
Dependent variable				
Etiology (% chronic pain)	1553	26.1%		
% Chronic pain interference	1553	23.1%		
Severity of pain interference	399	2.82	(2.50)	0–10

mood, relationships with others, sleep and enjoyment in the past week (0 = *not at all*; 10 = *completely*). Scores were averaged across the five items to generate a BPI interference rating used to index the severity of pain interference. Of the 406 participants, 399 participants completed the BPI scale, whereas data was missing for 7 participants.

Regarding the etiology of chronic pain interference, participants were categorized as having chronic pain interference, receiving a score of 1, when they reported chronic pain with a positive BPI rating (indicating chronic pain sufficient to interfere with their life; $N = 359$). All other participants who reported no chronic pain ($N = 1147$), or chronic pain with a 'zero' BPI rating ($N = 40$) were classified as having no pain interference, receiving a score of 0.

2.3. Executive function

Executive function was assessed using the Brief Test of Adult Cognition by Telephone (BTACT; Tun and Lachman, 2006), which comprised six subtests of cognitive function, and the Stop and Go Switch Task (SGST). Following exploratory and confirmatory factor analyses of the six BTACT subtests scores and SGST, an episodic memory factor and an executive function factor were obtained. The executive function score was computed using the mean z-scores for five tasks that loaded on the executive function factor. The five tasks include backward counting, digit span backward, categorical fluency, number series, and SGST, and assessed several domains of EF, including working memory, inhibitory control, and task-switching. We provide a description of the tasks in the following paragraphs.

In the backward counting task, participants were instructed to count backward from 100. The number of items correctly reported served as an index of speed of processing. The digits backward span task measured working memory. Participants were given an increasing sequence of digits (maximum 8 digits) and tasked to repeat them backward. The highest number of digits correctly recalled was recorded. For the category fluency task (Drachman and Leavitt, 1972), participants were given 60s to produce as many words as possible from a given category (e.g. animals). The total number of unique responses served as an index of verbal ability and speed. In the number series task (Salthouse and Prill, 1987), participants were presented strings of numbers (e.g., 35, 30, 25, 20, 15) and asked to deduce the next number in the series (e.g., 10). The total number of correct responses was recorded and used as an index of fluid intelligence and reasoning.

Lastly, the SGST, which measured task-switching and inhibitory control, required participants to verbally respond to the words "RED" and "GREEN". The test comprised 2 single-task blocks and a mixed-task block. In the first single-task block which consisted of congruent trials cued by the word "NORMAL", participants were instructed to reply "STOP" and "GO" when they were presented the words "RED" and "GREEN", respectively. The second single-task block comprised incongruent trials wherein participants were given the "REVERSE" cue and had to reverse their responses, replying "GO" and "STOP" to "RED" and "GREEN", respectively. In the mixed-task block, participants received the "NORMAL" and "REVERSE" cues at random, and were required to switch between congruent and incongruent trials. Inhibitory control was calculated using the difference in reaction time between the congruent and incongruent trials. Task-switching ability was calculated using the difference in reaction time between the switch (i.e., trials that require participants to switch taskset) and non-switch (i.e., trials of the same taskset as a preceding trial) trials. Together, the z-scores for these five subtests were averaged and served as an index of EF.

2.4. Covariates

Anxiety. Anxiety symptoms were assessed using the 10-item generalized anxiety disorder (GAD) subscale drawn from the broader Composite International Diagnostic Interview (Costa and McCrae, 1992; Kessler et al., 1998). The GAD subscale was developed based on the revised third edition of the Diagnostic and Statistical Manual of Mental Disorders (Drachman and Leavitt, 1972). Participants reported the frequency of these anxiety symptoms (e.g., "trouble falling asleep") experienced over the past 12 months using a 4-point scale (1 = *most days*, 4 = *never*). Following established procedures (Marcus et al., 2014), a continuous GAD score was constructed by summing the total number of "most days" responses.

Consistent with previous research (Kessler et al., 1998), participants received this interview only if they responded that they 1. Worry "A lot more" than most people, and 2. Worried "Every day, Just about every day, or Most days" in the past 12 months, and 3. Worry about "More than one thing" or have different worries "At the same time" in the pre-condition GAD questionnaire. Participants who did not qualify to do the GAD subscale received a score of 0.

Depressed affect. Depressed affect was assessed using the short form of the World Health Organization's CIDI (WHOs CIDI-SF; 'Kessler et al., 1998). The total number of depressive symptoms was computed by summing up the number of "Yes" responses to 7 items (e.g., "lose interest in most things") in the past 12 months.

Personality traits. The Big Five personality traits – agreeableness, extraversion, neuroticism, conscientiousness, and openness to experiences – were assessed by asking participants to rate the extent to which 26 adjectives described them on a scale of 1 (*a lot*) to 4 (*not at all)*. The scale was developed based on existing adjective measures of the Big Five (Goldberg, 1992; Trapnell and Wiggins, 1990). Agreeableness was measured using 5 adjectives: Helpful, warm, caring, softhearted, sympathetic. Extraversion was assessed using 5 adjectives: Outgoing, friendly, lively, active, talkative. Conscientiousness was measured using 5 adjectives: Organized, responsible, hardworking, careless (R), thorough. Neuroticism was measured using 4 adjectives: Moody, worrying, nervous, calm (R). Openness to experience was assessed using 7

adjectives: Creative, imaginative, intelligent, curious, broad-minded, sophisticated, adventurous.

All items, excepted the ones marked with (R), were reverse-coded so that high scores reflected higher standings in each dimension. Thereafter, each trait scale was calculated using the mean across each set of items. The scale has been validated in a study comprising 1000 respondents (Lachman and Weaver, 1997).

Religious coping. Religious coping was measured using two items (Bradshaw and Ellison, 2008; Brown and Lee, 2020). Participants indicated on a 4-point scale (1 = *never*; 4 = *often*) the extent to which they sought spiritual or religious support when they faced problems or had to make decisions.

Parental abuse. Parental abuse was assessed in MIDUS I using questions taken from the Conflict Tactics Scale (CTS; Straus and Gelles, 1990). Participants were presented with a list of emotionally abusive behaviors, such as "Insulted you or swore at you" and were asked "During your childhood, how often did your mother, or the woman who raised you, do any of the things" on the list "to you?". The same question was asked with reference to "your father, or the man who raised you". Participants responded to these two questions using a 4-point scale (1 = *often*; 4 = *never*). An index of parental abuse was calculated using the average of both items; lower scores reflect more frequent abuse.

Employment status. Participants reported on their current employment situation. Following Brown and Lee (2020), individuals who identified as working now, self-employed, looking for work and temporarily laid off were classified as being in the workforce. Individuals who identified as retired, homemaker, full-or part-time student, maternity or sick leave, permanently disabled, and others, were classified as not in the workforce.

Health variables. We included health variables such as smoking, number of surgeries, Body Mass Index (BMI), sleep problems (1 = *sleep problems*; 0 = *no sleep problems*), and self-rated physical health (1 = *excellent*; 2 = *very good*; 3 = *good*; 4 = *fair*; 5 = *poor*) that have been shown to predict future chronic pain (Brown and Lee, 2020). Participants who indicated that they smoke regularly now were considered to be current smokers. Participants who never had a cigarette or did not smoke regularly now were considered to be non-current smokers (1 = *smoker*; 0 = *non-smoker*).

Demographic variables. We included demographic variables such as participants' age, gender (recoded as 1 = *male*, 0 = *female*), marital status, and socioeconomic status (SES) as covariates. SES was operationalized using education attainment and household income. Participants reported their education attainment on a scale of 1 (*No school*) to 12 (*Ph.D, ED. D, MD, LLB, LLD, JD, or other professional degree*). Household income was calculated based on participants' household total income obtained through wages, pension, social security, government assistance, and other sources. Consistent with previous studies (Radler and Ryff, 2010), we created quartiles of income and included them as a continuous variable (Q1: less than $38,250; Q2: $38,250-$65,250; Q3: $65,550-$61,250; Q4: more than $104,687.50).

2.5. Data analysis

The present study aimed to examine the relationship between EF at baseline and one's onset of 1. Chronic pain and 2. Chronic pain interference, approximately 9 years later. To achieve our aims, we conducted two separate multivariate logistic regression analyses using SPSS, with chronic pain and chronic pain interference as the criterion variable in each analysis. Further, using ordinary least squares (OLS) regression, we examined if one's EF at baseline would be related to the severity of pain interference. To take into account confounding variables, we controlled for numerous covariates that have previously been linked to chronic pain. Prior research suggests that health factors such as smoking (Andersson et al., 1998), number of surgeries, Body Mass Index, sleep problems, and physical health (Brown and Lee, 2020), psychosocial factors including depressive symptoms (Magni et al., 1994), anxiety (McWilliams et al., 2003), personality (Wade et al., 1992), religious coping (Brown and Lee, 2020; Wachholtz and Pearce, 2009), parental abuse (Dennis et al., 2019), employment status (Brown and Lee, 2020), and demographic variables such as age (Rustøen et al., 2005), gender (Tsang et al., 2008), marital status (Wade et al., 2013), and socioeconomic status (Bonathan et al., 2013) are related to chronic pain.

In the first set of logistic regression analyses, we examined if EF would predict the etiology of chronic pain. In the second set of logistic regression analyses, we examined if EF would be associated with chronic pain that interferes with one's daily life activities. Lastly, we used OLS regression to examine if EF would predict the degree or severity of chronic pain interference for those who report chronic pain. In all analyses, we estimated three models to ensure the robustness of our results. In the first model, we presented an unadjusted model without controlling for any covariates. In the second model, we controlled for demographics and health factors, which have been shown to be associated with chronic pain (e.g., McWilliams et al., 2003; Tsang et al., 2008). In the third model, we controlled for psychosocial factors such as the big five personality to ensure that our results were not an artifact of personality factors (Wade et al., 1992). Missing data was imputed using a Markov chain Monte Carlo algorithm with a fully conditional specification procedure, resulting in five imputed datasets (Rubin, 1987). The percentage of missing values for the non-imputed dataset is reported in Table S2.

3. Results

3.1. Chronic pain

We first conducted multivariate logistic regression to examine the predictability of EF on the development of chronic pain. Our results in Model 1 revealed that lower baseline EF was associated with a significant likelihood of developing chronic pain approximately 9 years later (OR = 0.812, 95% CI [0.715, 0.922], p = .001; see Table 2). This longitudinal association remained even after controlling for demographics and health factors in Model 2 (OR = 0.810, 95% CI [0.697, 0.941], p = .006) and psychosocial factors in Model 3 (OR = 0.827, 95% CI [0.710, 0.962], p = .014; see Tables S3 and S4 for zero-order correlation for all main variables).

Additional analyses were conducted on the non-imputed dataset using listwise deletion. We found similar results, suggesting that imputation did not affect our results. Specifically, EF significantly predicted the etiology of chronic pain in Models 1, 2, and 3 for both the imputed (Table 2) and non-imputed dataset (Table S5).

We also calculated the corresponding effect size of EF in Model 3. We converted the OR (i.e., 0.827) to 1/OR, since OR is less than 1 (Chen et al., 2010), and utilized the formula: effect size (d) = ln (OR)/1.81 (Chinn, 2000). We calculated an effect size of 0.105, which aligns with Chen et al.'s (2010) calculation and corresponds to a small effect size (Cleeland, 1991).

A sensitivity power analysis using G*Power (Faul et al., 2009) demonstrated that with a sample size of 1,553, our study had 80% power to detect an effect size as small as 0.098. Therefore, our study is appropriately powered to detect a small effect size of 0.105 as discovered in the present study. Further, a post-hoc power analysis indicated that our study had 84% power to detect an effect size of 0.105. Taken together, our sample demonstrated that EF is a meaningful clinical variable for predicting the development of chronic pain, after controlling for demographics, health, and psychosocial factors.

3.2. Chronic pain interference and severity

We performed a logistic regression analysis to examine the effect of EF on chronic pain interference for all participants. We found that lower baseline EF was significantly associated with an increased risk of having chronic pain interference approximately 9 years later in Model 1 (OR =

Table 2
Model summaries with chronic pain as the outcome variable.

	Model 1		Model 2				Model 3			
Predictor	*B* (*SE*)	*OR (95% CI)*	*B (SE)*		*OR (95% CI)*		*B* (*SE*)		*OR (95% CI)*	
Executive function	−.208 (.065)**	.812 (.715, .922)	−.211	(.077)**	.810	(.697, .941)	−.190	(.078)*	.827	(.710, .962)
Demographics										
Age			−.005	(.006)	.995	(.984, 1.007)	−.005	(.007)	.995	(.982, 1.008)
Gender			−.075	(.122)	.928	(.731, 1.177)	−.084	(.134)	.919	(.707, 1.194)
Marital status			−.031	(.048)	.970	(.883, 1.065)	−.039	(.049)	.962	(.874, 1.059)
Income quartile			.001	(.064)	1.001	(.883, 1.135)	.010	(.066)	1.010	(.887, 1.149)
Education			.021	(.027)	1.021	(.968, 1.077)	.017	(.028)	1.017	(.962, 1.075)
Health										
Smoking			.230	(.181)	1.258	(.882, 1.795)	.200	(.185)	1.221	(.850, 1.755)
Number of surgeries			.043	(.023)	1.043	(.998, 1.091)	.041	(.023)	1.042	(.996, 1.090)
Body Mass Index			.009	(.012)	1.010	(.985, 1.034)	.010	(.013)	1.010	(.985, 1.035)
Sleep problems			.147	(.223)	1.158	(.748, 1.794)	.072	(.232)	1.075	(.682, 1.693)
Physical health			.173	(0.072)*	1.189	(1.033, 1.370)	.113	(.076)	1.119	(.964, 1.300)
Psychosocial factors										
Depressed affect							.011	(.043)	1.011	(.930, 1.100)
Anxiety							−.078	(.103)	.925	(.755, 1.132)
Agreeableness							−.191	(.146)	.826	(.621, 1.100)
Extraversion							.140	(.135)	1.150	(.883, 1.497)
Neuroticism							.312	(.110)**	1.366	(1.102, 1.695)
Conscientiousness							−.309	(.148)*	.734	(.549, .982)
Openness							.125	(.141)	1.133	(.858, 1.495)
Religious coping							.001	(.029)	1.001	(.945, 1.060)
Parental abuse							−.133	(.077)	.875	(.753, 1.018)
Not in workforce[1]							.316	(.156)*	1.372	(1.011, 1.862)

Note. [1]Not in workforce was scored as 1 = *not in workforce;* 0 = *in workforce.* *$p < .05$; **$p < .01$; ***$p < .001$.

0.873, 95% CI [0.766, 0.996], $p = .044$) in Model 1. However, this longitudinal association became non-significant in Model 2 (OR = 0.864, 95% CI [0.740, 1.010], $p = .066$) and Model 3 (OR = 0.887, 95% CI [0.757, 1.039], $p = .136$; see Table 3), suggesting that the link between EF and chronic pain interference can be accounted for by other factors such as personality and employment status.

Next, we performed an OLS regression to examine the associations between EF and the severity of chronic pain interference for participants who reported chronic pain and completed the BPI scale ($N = 399$). Similarly, although our OLS regression showed that EF was a significant longitudinal predictor of the severity of chronic pain interference in Model 1 ($b = −0.262$, $SE = 0.132$, $p = .047$), this association became non-significant in Model 2 ($b = −0.019$, $SE = 0.149$, $p = .899$) and Model 3 ($b = 0.071$, $SE = 0.147$, $p = .629$; see Table 4). This suggests that EF is unlikely to significantly influence the degree or amount of chronic pain interference among individuals with chronic pain.

We also conducted additional analyses on the non-imputed dataset and obtained similar results. Specifically, EF significantly predicted the etiology of chronic pain interference in Model 1 only, for both the imputed (Table 3) and non-imputed dataset (Table S6). Similarly, EF predicted the severity of chronic pain interference in Model 1 only, for both the imputed (Table 4) and non-imputed dataset (Table S7).

Table 3
Model summaries with chronic pain interference as the outcome variable.

	Model 1		Model 2				Model 3			
Predictor	*B* (*SE*)	*OR (95% CI)*	*B (SE)*		*OR (95% CI)*		*B* (*SE*)		*OR (95% CI)*	
Executive function	−.135 (.067)*	.873 (.766, .996)	−.146	(.079)	.864	(.740, 1.010)	−.120	(.081)	0.887	(.757, 1.039)
Demographics										
Age			−.007	(.006)	.993	(.981, 1.005)	−.008	(.007)	0.992	(.979, 1.006)
Gender			−.093	(.127)	.911	(.711, 1.168)	−.095	(.140)	0.909	(.691, 1.196)
Marital status			−.038	(.050)	.963	(.873, 1.062)	−.048	(.051)	0.953	(.862, 1.053)
Income quartile			−.011	(.066)	.989	(.868, 1.126)	−.002	(.068)	0.998	(.873, 1.142)
Education			.027	(.028)	1.028	(.972, 1.086)	.025	(.030)	1.025	(.968, 1.086)
Health										
Smoking			.307	(.186)	1.359	(.945, 1.956)	.269	(.190)	1.309	(.902, 1.901)
Number of surgeries			.043	(.023)	1.044	(.999, 1.091)	.043	(.023)	1.044	(.998, 1.091)
Body Mass Index			.012	(.013)	1.012	(.987, 1.037)	.011	(.013)	1.011	(.986, 1.038)
Sleep problems			.221	(.228)	1.247	(.797, 1.950)	.109	(.239)	1.115	(.698, 1.779)
Physical health			.186	(.075)*	1.204	(1.040, 1.395)	.095	(.080)	1.100	(.941, 1.286)
Psychosocial factors										
Depressed affect							.014	(.044)	1.014	(.931, 1.105)
Anxiety							−.069	(.104)	0.934	(.761, 1.146)
Agreeableness							−.163	(.152)	0.850	(.631, 1.146)
Extraversion							.109	(.140)	1.116	(.848, 1.468)
Neuroticism							.390	(.114)**	1.476	(1.180, 1.848)
Conscientiousness							−.413	(.153)**	0.661	(.490, .894)
Openness							.081	(.147)	1.085	(.812, 1.448)
Religious coping							−.010	(.031)	0.990	(.932, 1.052)
Parental abuse							−.157	(.079)*	0.855	(.732, .999)
Not in workforce[1]							.403	(.163)*	1.496	(1.087, 2.058)

Note. [1]Not in workforce was scored as 1 = *not in workforce;* 0 = *in workforce.* *$p < .05$; **$p < .01$; ***$p < .001$.
Severity of chronic pain interference.

Table 4
Ordinary least squares (OLS) regression with severity of chronic pain interference as the outcome variable.

	Model 1		Model 2			Model 3		
Predictor	*β*	*B (SE)*	*β*	*B (SE)*		*β*	*B (SE)*	
Executive function	−.099	−.262 (.132)*	−.007	−.019	(.149)	.027	.071	(.147)
Demographics								
Age			−.002	.000	(.012)	.036	.008	(.014)
Gender			−.109	−.548	(.251)*	−.078	−.396	(.269)
Marital status			−.131	−.257	(.101)*	−.143	−.280	(.100)**
Income quartile			−.061	−.139	(.135)	−.056	−.128	(.133)
Education			−.082	−.082	(.057)	−.042	−.042	(.059)
Health								
Smoking			.142	1.024	(.366)**	.137	.988	(.358)**
Number of surgeries			−.023	−.018	(.039)	−.020	−.015	(.038)
Body Mass Index			.096	.049	(.028)	.089	.045	(.027)
Sleep problems			.034	.316	(.453)	−.002	−.017	(.451)
Physical health			.137	.377	(.155)*	.068	.187	(.164)
Psychosocial factors								
Depressed affect						−.001	−.001	(.079)
Anxiety						−.033	−.126	(.187)
Agreeableness						.014	.073	(.303)
Extraversion						.036	.159	(.252)
Neuroticism						.175	.722	(.217)**
Conscientiousness						−.080	−.452	(.293)
Openness						−.114	−.552	(.284)
Religious coping						.021	.025	(.058)
Parental abuse						−.139	−.421	(.152)**
Not in work force[1]						.079	.431	(.302)

Note. [1]Not in workforce was scored as 1 = *not in workforce*; 0 = *in workforce*. *$p < .05$; **$p < .01$; ***$p < .001$.

4. Discussion

The present research investigated whether baseline EF would predict the development of chronic pain in middle-aged and older adults approximately 9 years later. Using multivariate logistic regression analyses, we demonstrated that EF predicted the development of chronic pain in middle-aged and older adults approximately 9 years later. Specifically, individuals with enhanced EF at baseline have a lower risk of developing chronic pain later, even after adjusting for potential confounding variables such as depressive symptoms, personality, age, and gender. We failed to find, however, any significant relation between baseline EF and pain interference among individuals with chronic pain.

Our results support and extend previous findings of the close relationship between EF and chronic pain. Critically, our study provides the first evidence in demonstrating the prospective effect of EF on the development of chronic pain. This is noteworthy, given that past research has mainly examined EF deficits as an outcome of chronic pain, rather than a potential predictor, despite their close relations. Our findings are, in part, in line with the Cyclical model Of Pain, Executive function, emotion regulation and Self-management (COPES; Caes et al., 2020), which proposes that individuals with EF deficits have a greater risk of developing chronic pain plausibly because they are less likely to successfully engage in and sustain patterns of health-promoting behavior, compared to those with enhanced EF. In turn, this may increase the risk of health conditions (Allan et al., 2016) that are associated with chronic pain.

We note that the effect size found was small based on Cleeland (1991) classification, and our results should be interpreted with caution. However, we believe that our findings are non-trivial, given the well-established detrimental physical and psychological implications of chronic pain (Azevedo et al., 2016; Fowler-Brown et al., 2013; Reid et al., 2011). Moreover, considering that the outcome of chronic pain is difficult to change (Ashburn and Staats, 1999), the significant, albeit small, effect size has clinical significance (Dennis et al., 2019). It is also likely that the small effect size in our study is underestimated, since effect sizes in longitudinal outcomes that control for stability effects tend to be underestimated and considerably smaller than effect sizes in cross-sectional studies (Adachi and Willoughby, 2015). The small effect size found in this study is consistent with the small effect sizes of other well-established psychosocial predictors of chronic pain such as depression and anxiety (Magni et al., 1994; McWilliams et al., 2003). Therefore, notwithstanding the small effect size, our results demonstrate the importance of EF for chronic pain outcomes.

Next, our findings add to an existing body of evidence that EF and pain likely engage an overlapping set of mental resources (Buhle and Wager, 2010). Accordingly, impairment in EF could weaken one's ability to manage pain and also increases hypervigilance to pain (Legrain et al., 2011; Giusti et al., 2020). Since the perception of pain is dependent on the attention that is allocated to a nociceptive stimulus, individuals with EF deficits may have more difficulties directing attention away from the stimulus. As a result, these individuals may be over-attentive to pain-related signals in the long run.

Notably, our results hint at a potential bidirectional relationship between EF and chronic pain. Previous research has mainly examined how chronic pain may adversely influence EF, rather than investigate if EF may influence the development of chronic pain. For instance, researchers suggest that chronic pain can manifest in structural, functional, and chemical changes in neural networks that are common to both pain and EF (Berryman et al., 2014; Seminowicz and Davis, 2007). Further, pain is noted to consume resources otherwise directed to EF (Legrain et al., 2009; Oosterman et al., 2012). However, preliminary findings from our, as well as Attal et al.'s (2014), study, indicate that there could be a bidirectional relation, whereby EF deficits could have long term consequences for the development of chronic pain and chronic pain can impair EF (Caes et al., 2020). Taken together, the evidence points to a potential circularity between EF and chronic pain, whereby less efficient EF is a predisposing factor to developing chronic pain, and that the persistent nature of chronic pain may compromise EF by draining cognitive resources (Caes et al., 2020).

Interestingly, we found that baseline EF was not significantly associated with pain interference and the degree of pain interference among individuals with chronic pain. This suggests that among individuals with chronic pain, baseline EF does not predict whether chronic pain interferes with their daily life, including their mood and sleep. One possible explanation is that for patients who already have chronic pain, chronic pain interference may negatively influence one's EF (Baker et al., 2016), rather than baseline EF predicting chronic pain interference. For these patients, the presence of pain may compete for limited

attentional resources (Eccleston and Crombez, 1999), thereby reducing EF performance. This could explain cross-sectional associations between EF abilities and chronic pain interference (van der Leeuw et al., 2016), but a negligible relation between baseline EF and chronic pain interference and severity as found in the present study.

Consistent with previous studies (Jorm et al., 1993; Judge et al., 2021), high neuroticism and low conscientiousness were associated with an increased likelihood of developing pain interference. Individuals high in neuroticism are prone to experience negative affect such as distress, worry, and anxiety (Costa and McCrae, 1992). These individuals have heightened vigilance to pain and are more likely to report higher levels of pain severity (Goubert et al., 2004). Therefore, they may report greater pain interference as they are likely to perceive ways in which pain interrupts their daily activities. In contrast, conscientiousness is characterized by perseverance, and goal-directedness, and is associated with enabling various positive health behaviors (Lodi-Smith et al., 2010). Individuals high in conscientiousness may persevere in proactive and adaptive health behaviors that reduce the severity of pain interference (Judge et al., 2021).

Our findings add to extant research that has demonstrated the detrimental effects of adverse childhood experiences such as parental abuse on poor health outcomes in adulthood. Consistent with previous studies (e.g., Dennis et al., 2019), we found that individuals who reported increased parental abuse had greater pain interference. The tumultuous early family environment could contribute to individuals' vulnerability and negative attitude towards overcoming pain (Goldberg et al., 1999), and their psychological response to abuse may also exacerbate the pain experience (Marin et al., 2021).

Our results also dovetail previous findings demonstrating the negative association between unemployment and pain intensity among chronic pain patients (Voon et al., 2017). Not being in the workforce may deprive individuals of opportunities for control over their health and is associated with spending less time being engaged in activity and spending more time lying down and resting (Jackson et al., 1997; Tait et al., 1990). Such health behaviors may exacerbate the experience of pain leading individuals who are not in the workforce to report more pain intensity than those in the workforce (Voon et al., 2017).

Our study is not without limitations. First, our use of a composite EF does not allow us to delineate which aspects of EF may better predict the development of chronic pain. Given that EF comprises a cluster of high-level cognitive processes, including inhibition, updating, and shifting (Miyake et al., 2000), the relation between EF and pain may be largely driven by specific processes such as inhibition (Bjekić et al., 2018) or updating (Legrain et al., 2011). Second, our findings are based on an American sample and may have limited generalizability. For instance, Asians, in comparison to Caucasians, may report higher pain scores and worse pain-related outcomes due to unhelpful cognitive appraisal processes (Day et al., 2020). Therefore, future research should seek to examine these findings using diverse samples.

These limitations notwithstanding, our findings hold important clinical implications. Given that a lowered EF is a predictive factor for the development of chronic pain, it may be useful for healthcare professionals to incorporate EF tests in screening for individuals who are at higher risk of developing chronic pain. Identifying these individuals may facilitate targeted interventions at delaying the development of chronic pain. Further, our findings have practical implications for the development of intervention programs to target older adults at risk of developing chronic pain. Specifically, our results provide support for the use of preventive EF-training interventions, such as EF computerized cognitive training (CCT), to enhance EF among older adults (Nguyen et al., 2019). Given that EF and pain engage overlapping neural networks (Buhle and Wager, 2010), improvements in EF may strengthen one's ability to direct attention away from the pain stimulus and effectively manage pain. Future studies should seek to investigate if there may be transfer of EF training effects to chronic pain outcomes.

Credit author statement

Wee Qin Ng: Conceptualization, Methodology, Formal analysis, Writing – original draft. Andree Hartanto: Conceptualization, Writing – review & editing, Supervision.

Data availability

I have shared the link to my data/code at the Attached File step.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.socscimed.2022.115478.

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