

Original Investigation

Effects of Smoking Cessation on Pain in Older Adults

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Abstract

Introduction: Smokers are at increased risk of developing chronic pain and suffering higher pain intensity. However, nicotine has analgesic properties, and smokers may view smoking as a means to cope with pain. Smoking cessation is clearly beneficial to the long-term health of smokers. However, it is not known how abstinence from smoking affects pain. The aim of this study was to determine the association between smoking cessation and changes in pain symptoms by secondary analysis of a large longitudinal dataset of older adults.

Methods: Secondary analyses were performed of longitudinal biennial survey data (1992 through 2006) from the nationally representative Health and Retirement Study of United States adults older than 50 years. Multivariate logistic regressions were utilized to determine the relationship between the changes in smoking status and changes in pain symptoms, controlling for demographics, depression, self-rated health, history of arthritis, and body mass index.

Results: In multivariate analyses, among the 4,695 smokers who reported no pain or mild pain at enrollment, smoking status was not independently associated with exacerbation of pain (odds ratio [OR]: 0.95, 95% CI: 0.84, 1.08). Among the 1,118 smokers who reported moderate to severe pain at enrollment, smoking status was not independently associated with improvement of pain (OR: 0.87, 95% CI: 0.70, 1.08).

Conclusions: Smoking cessation was not independently associated with changes in pain symptoms in older adults. These results suggest that concerns regarding the effects of abstinence from smoking on pain should not pose a barrier to offering tobacco use interventions to smokers with chronic pain.

Introduction

Cigarette smoking is associated with an increased risk of developing painful disorders (Andersson, Ejlertsson, & Leden, 1998; Eriksen, Natvig, Rutle, & Bruusgaard, 1999; Hestbaek, Leboeuf-Yde, & Kyvik, 2006; Kaila-Kangas, Leino-Arjas, Riihimaki, Luukkonen, & Kirjonen, 2003; Mattila, Saarni, Parkkari,

Koivusilta, & Rimpela, 2008; Mikkonen et al., 2008; Miranda, Viikari-Juntura, Martikainen, Takala, & Riihimaki, 2002; Miranda, Viikari-Juntura, Punnett, & Riihimaki, 2008; Mustard, Kalcevic, Frank, & Boyle, 2005; Palmer, Syddall, Cooper, & Coggon, 2003; Power, Frank, Hertzman, Schierhout, & Li, 2001; Scott, Goldberg, Mayo, Stock, & Poitras, 1999). Compared with nonsmokers, smokers report higher levels of pain, and their pain has a greater impact on physical, psychosocial, and occupational functioning (Hooten et al., 2009; John, Hanke, et al., 2006; Vogt, Hanscom, Lauerman, & Kang, 2002; Weingarten et al., 2008, 2009). Although these associations are well established, it is not known how abstinence from smoking affects pain. Nicotine has acute analgesic properties (Jamner, Girdler, Shapiro, & Jarvik, 1998; Perkins et al., 1994), but nicotine deprivation may acutely worsen painful symptoms and nicotine withdrawal may also contribute to a worsening perception of pain (Anderson et al., 2004; Biala, Budzynska, & Kruk, 2005; Schmidt, Tambeli, Gear, & Levine, 2001; Yang, Wu, & Zbuzek, 1992). In addition, many smokers view tobacco use as a means to cope with stress and anxiety (Parrott, 1995) such that abstinence may have an untoward influence on an individual's capacity to cope with pain. Alternatively, due to the complex effects of chronic nicotine exposure on nicotinic receptor pharmacology, long-term abstinence from nicotine may lead to improvements in pain symptoms. Understanding the potentially beneficial and deleterious effects of smoking cessation on pain is particularly relevant to the development of tobacco use interventions targeted to smokers with chronic pain.

The aim of this study was to determine the association between smoking cessation and changes in pain symptoms in older adults by secondary analysis of a large longitudinal dataset of older United States residents.

Methods

Study Population

The Health and Retirement Study (HRS) surveys a nationally representative sample of older adults in the United States (National Institutes on Aging, 2009). The HRS collects data on older adults' physical and mental health, insurance coverage, financial situations, family support systems, work status, and

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Smoking cessation and pain

retirement planning. In 1992, the study enrolled the first cohort of U.S. residents between the ages of 50 and 60 years, and follow-up interviews were conducted every two years. A sample of U.S. residents age 70 and older were enrolled in 1993, and these individuals were reinterviewed in 1995 and 1998. A group of adults in the age group that falls between the original two cohorts and a cohort consisting of people in their early 50s was enrolled in 1998. Since 1998, all living individuals continue to be interviewed biennially. Survey response rates (70%–81%) and retention rates (86%–90%) have been excellent. The HRS is approved by the University of Michigan Health Sciences Human Subjects Committee. Data collected from 1992 to 2006 were used in the current analysis. Because this study used publicly available de-identified data, it was exempted from review by the Mayo Clinic Institutional Review Board.

Data Analyzed

Demographic information analyzed included sex, age, race and ethnicity, education, marital status, annual household income, and health insurance coverage (government or private plan). Pain was defined by a positive response to the question, “Are you often troubled with pain?” Participants who reported pain were asked in an additional question to rate pain intensity as mild, moderate, or severe. Smoking-related data collected by the HRS included current smoking status, current number of cigarettes smoked, age of smoking initiation, maximum number of cigarettes smoked in the past, and year of quitting for former smokers. During each cycle of interviews, participants were asked if they were a current cigarette smoker. For purposes of this analysis, current smokers at enrollment who reported not smoking at a follow-up interview were defined as having quit smoking.

Depression was assessed by the 8-item Center for Epidemiological Studies Depression Scale, with scores ranging from 0 to 8. Participants with a score ≥ 4 were considered to be depressed. The presence of arthritis was determined by self-report. Body mass index (BMI) was calculated from self-reported height and weight and categorized a priori into four groups: <25 , 25–29.9, 30–34.9, and >35 . Participants were asked to rate their health as excellent, very good, good, fair, and poor.

Statistical Analysis

The study population for this analysis included subjects who reported current smoking at the time of enrollment. Baseline demographics and the frequency of pain were tabulated to characterize this population.

Two sets of analyses were performed using two pairs of outcome measures: pain compared with no pain and moderate to

severe pain compared with no pain or mild pain. Table 1 summarizes the analyses performed in this study.

The first set of analyses sought to identify the occurrence or worsening of pain. The first analysis in this set examined smokers who reported having no pain at enrollment (Group A). The occurrence of pain in this group of smokers was defined as reporting pain at any subsequent interview. The second analysis in this set included smokers who reported experiencing no pain or mild pain at enrollment (Group B). Worsening of pain in this group of smokers was defined as reporting moderate to severe pain at any subsequent interview.

The second set of analyses sought to identify the resolution or improvement of pain. The first analysis in this set examined smokers who reported having any level of pain at enrollment (Group C). The resolution of pain in this group of smokers was defined as reporting no pain at any subsequent interview. The second analysis in this set included smokers who reported moderate to severe pain at enrollment (Group D). The improvement of pain in this group of smokers was defined as reporting either no pain or mild pain at a subsequent interview.

Generalized estimating equations (GEE) logistic regressions were used to evaluate the relationship between quitting smoking and the four different pain outcomes mentioned above at follow-up assessments, adjusting for other factors that we and others have shown in prior studies to be associated with pain (Mantyselka, Turunen, Ahonen, & Kumpusalo, 2003; Shi, Hooten, Roberts, & Warner, 2010). The analyses included data from all follow-up assessments, exploring the relationship between smoking status and pain outcomes in each subject at each assessment. Because each subject could have multiple assessments, intra-individual correlations were taken into account by the GEE analysis. Age at enrollment (in 10-year intervals), sex, race, and education were not modeled as time dependent. Marital status, annual household income (in 10,000 U.S. dollars as a continuous variable), insurance coverage (any government or private plans), arthritis, quitting smoking, depressive symptoms, and BMI in four categories were entered into the models as time-dependent variables (i.e., were allowed to change at each assessment). In the multiple regression models, the independent contribution of covariates of interest was determined after adjustment for the effects of other covariates. Adjusted odds ratios (ORs) were reported.

Analyses was performed using Stata, version 10.0 (College Station, TX), and $p < .05$ was considered to be statistically significant.

Table 1. Summary of Analysis Groups

| Group | A | B | C | D |
|------------------|--|--|---|---|
| Study population | Smokers with no pain at enrollment | Smokers with no pain or mild pain at enrollment | Smokers with any pain at enrollment | Smokers with moderate or severe pain at enrollment |
| <i>n</i> | 4,285 | 4,695 | 1,948 | 1,118 |
| Outcome: | Pain occurrence: report of any pain in follow-up assessments | Pain worsening: report of moderate or severe pain in follow-up assessments | Pain resolution: report of no pain in follow-up assessments | Pain improvement: report of no pain or mild pain in follow-up assessments |
| Predictor | Smoking status | Smoking status | Smoking status | Smoking status |

Results

Table 2 shows the baseline characteristics of the study population. At the time of enrollment, 6,258 subjects were current smokers. The majority were aged 50–60 years and were self-identified as non-Hispanic Whites. Approximately one third reported having pain, and among those who reported pain, approximately three quarters reported experiencing moderate to severe pain. Of subjects who reported pain at enrollment, 420 did not provide information on pain intensity because this question was not administered at the 1993 assessment. During follow-up, 2,760 subjects (44%) reported not currently smoking at a subsequent interview (i.e., quitting smoking).

In univariate analysis, quitting smoking was associated with an *OR* of 1.22 (1.09, 1.35, $p < .001$) for the occurrence of any pain in smokers who did not report pain at enrollment (Group A), demonstrating that smokers who quit smoking were more likely to develop pain. The univariate *OR* relating quitting smoking and the transition to moderate or severe pain among those who initially reported no pain or mild pain (Group B) was 1.09 (0.98, 1.22, $p = .108$). However, in multivariate analyses, quitting smoking was not independently related to either the occurrence or the worsening of pain (Table 3). The adjusted *ORs* for the other factors included in the model as covariates were similar in the analyses of Groups A and B. In the two multivariate regressions (Table 3), factors consistently associated with a lower likelihood of reporting the occurrence or worsening of pain included being non-Hispanic Black, older age, not being depressed, better self-rated health, not having arthritis, and a BMI lower than 30.

In univariate analysis, quitting smoking was related to an *OR* of 0.98 (0.85, 1.14, $p = .829$) for the resolution of pain in

those who reported any pain at enrollment (Group C), demonstrating that smokers who quit were not more likely to experience resolution of pain. The univariate *OR* relating quitting smoking and the transition to no pain or mild pain among those who initially reported moderate or severe pain (Group D) was 0.89 (0.73, 1.08, $p = .226$). In multivariate analyses, quitting smoking was not independently associated with either the resolution or the improvement of pain (Table 4). Factors independently associated with higher likelihood of reporting the resolution or improvement of pain included not being depressed, better self-rated health, and not having arthritis.

Discussion

The main finding of this longitudinal study of older smokers was that smoking abstinence was not independently associated with either the improvement or the exacerbation of pain.

The mechanisms responsible for the association between smoking and pain have not been fully elucidated but may involve several factors (Shi, Weingarten, Mantilla, Hooten, & Warner, 2010). First, chronic exposure to nicotine may change the central nervous system (Mukhin et al., 2008; Sallette et al., 2005) such that pain perception is altered (Perkins et al., 1994; Silverstein, 1982). Second, smoking may worsen disease processes, such as osteoporosis, which may cause pain (An et al., 1994; Glassman et al., 2000; Law & Hackshaw, 1997). Third, smoking may be a coping strategy utilized to manage anxiety and stress induced by pain such that pain may contribute to the maintenance of smoking behavior (Ditre, Heckman, Butts, & Brandon, 2010). Finally, smoking is associated with other factors that can affect pain such as depression. Symptoms of depression are more prevalent in smokers compared with non smokers (Breslau, Peterson, Schultz, Chilcoat, & Andreski, 1998; Brown, Madden, Palenchar, & Cooper-Patrick, 2000; Murphy et al., 2003), and smoking predicts the onset of depression as well as exacerbates existing symptoms of depression (Klungsoyr, Nygard, Sorensen, & Sandanger, 2006; Pasco et al., 2008).

Given that the mechanisms underlying the association between smoking and pain are not known, it is difficult to predict how abstinence from smoking might affect pain. Several considerations may apply. In experimental humans and animal studies, smoking can have acute analgesic effects in subjects without chronic pain (Jamner et al., 1998; Perkins et al., 1994), which could favor pain exacerbation when exposure to smoke is eliminated. However, it is not known whether smoking a cigarette changes pain perception in smokers with chronic pain. Animal models suggest that nicotine withdrawal is associated with increased sensitivity to pain stimuli (Anderson et al., 2004; Biala et al., 2005; Schmidt et al., 2001; Yang et al., 1992). If smoking represents a coping strategy for pain, abstinence may result in the loss of a means of dealing with pain, which may increase the reporting of pain. To the extent that the body may recover from altered pain perception and structural damage caused by smoking, long-term abstinence may improve pain symptoms. If smoking is just an indicator of other causal factors for pain such as depression, abstinence may have little effect. We did not observe a consistent effect of smoking abstinence on pain in this analysis of data of older adults, suggesting that (a) any changes in pain perception or structural damage caused by smoking are not reversible,

Table 2. Selected Characteristics of Smokers at Time of Enrollment ($n = 6,258$)

| | <i>n</i> (%) |
|--|-----------------|
| Age | |
| Years ($M \pm SD$) | 58.1 \pm 9.0 |
| <50 | 618 (9.9) |
| 50–60 | 3,684 (58.9) |
| 60–70 | 949 (15.2) |
| ≥ 70 | 1,007 (16.1) |
| Sex (female) | 3,177 (50.8) |
| Race/ethnicity | |
| White, non-Hispanic | 4,445 (71.2) |
| Black, non-Hispanic | 1,145 (18.3) |
| Hispanic | 493 (7.9) |
| Other | 159 (2.6) |
| Number of cigarettes smoked per day ($M \pm SD$) | 19.0 \pm 12.8 |
| Any pain | 1,948 (31.3) |
| Pain intensity ^a | |
| Mild | 410 (26.8) |
| Moderate | 788 (51.6) |
| Severe | 330 (21.6) |

Note. ^aPain intensity was provided by 1,528 of the subjects who reported pain. This question was not asked of those subjects enrolled in 1993.

Table 3. The Association Between Selected Factors and the Occurrence (Group A) Or Worsening (Group B) of Pain (OR and 95% CI)

| | Pain occurrence ^a (Group A) | <i>p</i> value | Pain worsening ^b (Group B) | <i>p</i> value |
|-----------------------------------|--|----------------|---------------------------------------|----------------|
| Female | 1.06 (0.92, 1.21) | .437 | 1.27 (1.10, 1.47) | .001 |
| Race/ethnicity | | | | |
| White, non-Hispanic | 1.00 | | 1.00 | |
| Black, non-Hispanic | 0.63 (0.63, 0.75) | <.001 | 0.57 (0.47, 0.70) | <.001 |
| Hispanic | 0.79 (0.61, 1.02) | .072 | 0.77 (0.59, 1.00) | .049 |
| Other | 0.78 (0.50, 1.21) | .261 | 0.70 (0.44, 1.11) | .126 |
| Age at enrollment | | | | |
| <50 | 1.00 | | 1.00 | |
| 50–60 | 0.70 (0.58, 0.85) | <.001 | 0.67 (0.55, 0.82) | <.001 |
| 60–70 | 0.46 (0.36, 0.58) | <.001 | 0.46 (0.35, 0.59) | <.001 |
| ≥70 | 0.41 (0.31, 0.54) | <.001 | 0.38 (0.28, 0.52) | <.001 |
| Married | 0.91 (0.79, 1.03) | .141 | 0.90 (0.78, 1.04) | .152 |
| Education >12 years | 1.13 (0.98, 1.31) | .097 | 1.07 (0.92, 1.24) | .407 |
| Household income (per 10,000 USD) | 0.98 (0.97, 0.99) | .003 | 0.99 (0.97, 1.00) | .118 |
| Insurance plan | 1.24 (1.06, 1.46) | .006 | 1.15 (0.98, 1.35) | .084 |
| Depression | 1.94 (1.70, 2.21) | <.001 | 1.96 (1.72, 2.24) | <.001 |
| Self-rated health | | | | |
| Excellent | 1.00 | | 1.00 | |
| Very good | 1.67 (1.29, 2.15) | <.001 | 1.57 (1.17, 2.11) | .003 |
| Good | 2.95 (2.30, 3.79) | <.001 | 3.34 (2.50, 4.45) | <.001 |
| Fair | 6.03 (4.65, 7.83) | <.001 | 7.30 (5.40, 9.89) | <.001 |
| Poor | 13.26 (9.92, 17.73) | <.001 | 15.06 (10.90, 20.80) | <.001 |
| Arthritis | 3.11 (2.75, 3.52) | <.001 | 3.16 (2.77, 3.61) | <.001 |
| Body mass index | | | | |
| <25 | 1.00 | | 1.00 | |
| 25–30 | 1.12 (0.98, 1.28) | .085 | 1.08 (0.94, 1.24) | .296 |
| 30–35 | 1.51 (1.27, 1.79) | <.001 | 1.51 (1.27, 1.80) | <.001 |
| ≥35 | 1.38 (1.09, 1.75) | .007 | 1.56 (1.22, 1.98) | <.001 |
| Quitting smoking | 1.04 (0.92, 1.17) | .557 | 0.95 (0.84, 1.08) | .472 |

Note. ^aPopulation analyzed included smokers who reported no pain at enrollment ($n = 4,285$). Outcome: reporting any pain at a subsequent assessment. Analysis adjusted for all other factors in the table. OR = odds ratio.

^bPopulation analyzed included smokers who reported either no or mild pain at enrollment ($n = 4,695$). Outcome: reporting either moderate or severe pain severity at a subsequent assessment. Analysis adjusted for all other factors in the table.

(b) smoking per se does not affect chronic pain but rather is associated with other factors such as depression that are causal for pain exacerbation, or (c) that there is no net effect of changes in multiple factors that contribute to a relationship between smoking and pain.

To our knowledge, no prior study has directly investigated the relationship between smoking cessation and changes in pain. However, other studies that have included former smokers suggest that chronic pain is also more common in this subgroup of adults (Jakobsson, 2008; John, Alte, et al., 2006; Palmer et al., 2003). In a recent meta-analysis of the relationship between smoking status and low back pain, both former and current smokers had increased odds of having prevalent pain and incident pain compared with never-smokers (Shiri, Karppinen, Leino-Arjas, Solovieva, & Viikari-Juntura, 2010). Although the OR for former smokers tended to be lower compared with current smokers, the difference was not significant (Shiri et al., 2010). This observation may be due, in part, to changes in the central nervous system caused by chronic nicotine exposure, which may be slowly or only partly reversible (Perkins et al., 2001).

In our prior work using the HRS data, we examined multiple factors associated with incident pain in the general population (which included nonsmokers; Shi, Hooten, et al., 2010). This analysis confirmed that current smoking is a risk factor for incident pain in older adults. The current analysis (which includes only smokers) examining the occurrence of pain in those patients who did not report pain at enrollment is consistent with this prior analysis in terms of which factors were associated with pain. Depression and increased BMI were important factors associated with changes in pain symptoms. Among smokers in this study, higher depression scores were related to a higher likelihood of reporting worsened pain symptoms and a lower likelihood of reporting improvement in pain, regardless of the change in smoking behavior. For the most part, factors associated with the occurrence or worsening of pain had the expected association with the resolution or improvement of pain (e.g., depression, self-reported health status), further supporting the validity of our method and analysis.

This study has several limitations. First, all the information in the HRS was self-reported by survey participants such that smoking status could not be biochemically confirmed. Second,

Table 4. The Association Between Selected Factors and Resolution (Group C) Or Improvement (Group D) of Pain (OR and 95% CI)

| | Pain resolution ^a (Group C) | <i>p</i> value | Pain improvement ^b (Group D) | <i>p</i> value |
|-----------------------------------|--|----------------|---|----------------|
| Female | 0.72 (0.60, 0.87) | .001 | 0.64 (0.50, 0.81) | <.001 |
| Race/ethnicity | | | | |
| White, non-Hispanic | 1.00 | | 1.00 | |
| Black, non-Hispanic | 1.40 (1.08, 1.80) | .010 | 1.34 (0.99, 1.83) | .060 |
| Hispanic | 1.51 (1.16, 1.97) | .003 | 1.31 (0.93, 1.85) | .128 |
| Other | 0.72 (0.37, 1.38) | .320 | 0.69 (0.39, 1.23) | .204 |
| Age at enrollment | | | | |
| <50 | 1.00 | | 1.00 | |
| 50–60 | 0.94 (0.72, 1.23) | .656 | 0.98 (0.70, 1.38) | .929 |
| 60–70 | 1.35 (0.96, 1.89) | .087 | 1.45 (0.94, 2.23) | .096 |
| ≥70 | 1.87 (1.30, 2.70) | .001 | 0.98 (0.49, 1.98) | .964 |
| Married | 0.94 (0.79, 1.31) | .533 | 0.89 (0.71, 1.12) | .306 |
| Education >12 years | 1.00 (0.83, 1.21) | .987 | 1.02 (0.80, 1.29) | .897 |
| Household income (per 10,000 USD) | 1.00 (0.99, 1.02) | .682 | 1.00 (0.99, 1.02) | .640 |
| Insurance plan | 0.96 (0.77, 1.19) | .681 | 0.97 (0.74, 1.28) | .839 |
| Depression | 0.53 (0.45, 0.62) | <.001 | 0.56 (0.46, 0.69) | <.001 |
| Self-rated health | | | | |
| Excellent | 1.00 | | 1.00 | |
| Very good | 1.00 (0.71, 1.42) | .978 | 0.84 (0.51, 1.39) | .493 |
| Good | 0.55 (0.39, 0.76) | <.001 | 0.42 (0.25, 0.68) | <.001 |
| Fair | 0.30 (0.22, 0.42) | <.001 | 0.24 (0.15, 0.40) | <.001 |
| Poor | 0.14 (0.10, 0.21) | <.001 | 0.11 (0.07, 0.18) | <.001 |
| Arthritis | 0.44 (0.36, 0.53) | <.001 | 0.43 (0.33, 0.55) | <.001 |
| Body mass index | | | | |
| <25 | 1.00 | | 1.00 | |
| 25–30 | 1.06 (0.89, 1.26) | .532 | 1.00 (0.80, 1.27) | .955 |
| 30–35 | 0.93 (0.74, 1.17) | .516 | 1.09 (0.83, 1.45) | .536 |
| ≥35 | 0.58 (0.42, 0.81) | .001 | 0.73 (0.51, 1.06) | .102 |
| Quit smoking | 0.97 (0.82, 1.15) | .719 | 0.87 (0.70, 1.08) | .202 |

Note. OR = odds ratio.

^aPopulation analyzed included smokers who reported pain at enrollment ($n = 1,948$). Outcome: reporting no pain at a subsequent assessment. Analysis adjusted for all other factors in the table.

^bPopulation analyzed included smokers who reported moderate to severe pain at enrollment ($n = 1,118$). Outcome: reporting either no or mild pain at a subsequent assessment. Analysis adjusted for all other factors in the table.

the HRS did not collect detailed information on other types of tobacco use such as smokeless tobacco, which might also affect pain symptoms. Third, it could only be known that the change in smoking behavior and the change in pain happened in the same 2-year interval prior to the time of interview, so that the exact temporal relationship between events could not be ascertained. Thus, the analysis could only provide evidence of association, and causal inferences cannot be established. Fourth, details such as the type of pain were not available from the HRS, and our analysis treated “pain” as a single outcome. Because the pathophysiology leading to the development of pain is diverse, it is possible that changes in smoking status may affect some types of pain but not others. Fifth, pain intensity rating at enrollment was missing for more than 20 percent of subjects who reported pain because this question was not asked in the initial assessment for some subjects. Finally, the HRS included older adults only, and the results from this study may not be generalizable to other age groups.

These results have potential clinical relevance. Smoking is the most preventable cause of death (Mokdad, Marks, Stroup, & Gerberding, 2004), and every patient who smokes, including

those with chronic pain, should be assisted by health care professionals in quitting. However, limited clinical evidence suggests that it is difficult for smokers with chronic pain to achieve abstinence from tobacco (Fishbain et al., 2008; Hooten et al., 2009). This study does not provide evidence that the potential for improvement in pain should be used as a motivator for cessation in smokers with pain or that integrating tobacco use treatment with pain treatment will significantly benefit improvement of pain symptoms with treatment, although this has not been directly tested. However, smoking abstinence does not appear to worsen pain, which should be reassuring to patients and health care providers involved in the delivery of smoking cessation services. Nonetheless, as smoking may represent a coping strategy to manage pain and be associated with other comorbid conditions including depression, tobacco use interventions targeted to smokers with chronic pain may need to specifically address these important clinical factors.

In conclusion, we did not find evidence that abstinence from smoking consistently affects pain symptoms in older adults. These results suggest that concerns regarding the effects

of abstinence from smoking on pain should not pose a barrier to offering tobacco use interventions to smokers with chronic pain.

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Declaration of Interests

None declared.

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