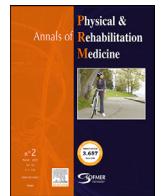




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Review

Modifiable lifestyle-related prognostic factors for the onset of chronic spinal pain: A systematic review of longitudinal studies



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ABSTRACT

Background: Stratified approaches to spinal pain that address psychosocial risk factors reduce long-term disability to a moderate extent. Identifying and managing other risk factors might help improve outcomes.

Objective: This systematic review of longitudinal studies aimed to evaluate possible associations between the onset of chronic spinal pain (including low back, back and neck pain) and putative modifiable lifestyle-related risk or protective factors.

Methods: This systematic review of longitudinal studies published during the last 2 decades followed PRISMA guidelines. Two reviewers screened Medline, Scopus, Pedro, Cochrane Library, Psycinfo, Science Direct, PTSDpubs and Google Scholar for relevant studies. The QUIPS tool was used to assess the risk of bias. A qualitative meta-synthesis of relevant factors was performed.

Results: Of 3716 unique records, 14 studies met the inclusion criteria (10 with low risk of bias and 4 moderate risk of bias). The highest bias observed was attrition. For chronic low back pain, we found moderate evidence for the involvement of high body weight, waist circumference, and hip circumference and conflicting evidence for high body mass index (BMI), smoking, and physical activity. For chronic neck pain, we found strong evidence for high BMI in women, moderate evidence for sleep disorders in women and conflicting evidence for high BMI in men and physical activity. For chronic back pain, we found limited evidence for gardening/yard work in men and more than one adult at home. Effect sizes were small.

Conclusions: Several modifiable lifestyle-related factors were identified. Evidence is still sparse and there is a need for more studies. PROSPERO database registration: Ref 172,112 CRD42020172112.

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Introduction

Spinal pain is the leading cause of pain and disability worldwide [1]. Therefore, we need preventive approaches that rely on the identification and management of modifiable risk factors. During the last 2 or 3 decades, the search for risk factors for long-term disability and work loss has mainly focused on psychosocial risk factors, called “yellow flags” [2–4]: attitudes and beliefs, behaviours, compensation issues, diagnosis and treatment, emotions, family and work-related factors. Screening tools such as the Orebro Musculoskeletal Pain Screening Questionnaire and the StarT Back tool were developed [5–7]. Stratified approaches gave interesting results, but the proportion of

individuals with significant disability remains high even after adequate management, leaving room for improvement [8]. These approaches focus mainly on the person's attitudes and beliefs as well as physical activity. Other modifiable risk factors might be involved in the development of chronic spinal pain (CSP) and could be the target of preventive approaches.

Lifestyle is defined as the “typical way of life or manner of living characteristic of an individual” (PubMed MeSH Database). It encompasses aspects such as leisure time physical activity, toxic consumption, social support and integration, nutrition behavior, spirituality and sexuality. These modifiable health behaviours influence physical condition, metabolism and well-being, all being theoretical protectors for chronic pain [9–11]. Although several behavioural factors have been included in the yellow flag list, few robust data are available concerning their involvement in the onset of CSP. Cross-sectional studies suggest an association between physical activity, obesity, smoking, and CSP [12–14]. Cross-sectional studies are useful to highlight potential areas of interest and provide prevalence

Abbreviations: BMI, body mass index; CBP, chronic back pain; CLBP, chronic low back pain; CNP, chronic neck pain; CSP, chronic spinal pain; LBP, low back pain; LRFs, lifestyle-related risk factors; MeSH, Medical Subject Headings; OR, odds ratio; QUIPS, Quality in Prognosis Studies; RoB, risk of bias; RR, risk ratio

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estimation, but they do not account for chronology and do not give the direction of the association [15].

In the present systematic review of longitudinal studies, we review the association between potential modifiable lifestyle-related risk or protective factors and the onset of CSP, including low back, back and neck pain.

Methods

Protocol and registration

This systematic review of longitudinal studies followed the PRISMA recommendations for systematic reviews [16]. The study protocol was registered on March 3, 2020 in the international prospective register of systematic reviews (PROSPERO, Ref 172,112). At that time, we found no other systematic review protocol registered on this topic.

Ethical considerations

This study was conducted in accordance with the ethical principles of the Helsinki statement (1964). Because the systematic review uses publicly accessible documents as evidence, neither institutional ethics approval nor informed consent were necessary.

Eligibility criteria

The inclusion criteria were prospective cohorts, case–control or randomised control studies assessing the correlation between lifestyle-related risk factors (LRFs) and the onset of CSP in adults (≥ 18 years old, representative of the general population of the given country). Individuals had to be free of chronic pain at inclusion (i.e., presenting a first acute pain episode or experiencing recurrent pain separated by pain-free intervals with < 3 months of continuous pain). Sufficient data had to be available concerning the influence of LRFs. Full-text articles had to be available in English or French and published in a peer-reviewed journal. The outcome had to be the onset of CSP with a minimal duration of 3 months of continuous pain.

The exclusion criteria were presence of CSP at baseline, insufficient statistical analysis, special groups not representative of the general population (e.g., high-risk occupational class workers or persons with specific diseases), studies focusing on psychosocial factors only and insufficient data concerning the pain status at baseline or follow-up.

Information source

Potentially relevant studies were searched in the electronic databases Medline (PubMed), Scopus, Pedro, Cochrane Library, Psycinfo, Science Direct, PTSDpubs, and Google Scholar. In addition, reference lists and citations were manually screened to include relevant studies not revealed by the search equation. For this study, no gray literature search was conducted. The timeframe for publication date was from January 1, 2000 to June 30, 2021.

Search

During the initial phase, our clinical question was framed by using the PICO process (Appendix A) [17]. To extend data collection and increase specificity, we broadened our initial term “spinal pain” to neck, thoracic, low back and back pain. We defined neck pain as “pain perceived anywhere in the posterior region of the cervical spine, from the superior nuchal line to the first thoracic spinous process” also including potential head, trunk or shoulder irradiation [18,19]. Thoracic spinal pain was defined as “pain experienced in the region of the thoracic spine, between the boundaries of T1–T12 and

across the posterior aspect of the trunk” [20]. Low back pain (LBP) was defined as “pain and discomfort, localised below the costal margin and above the inferior gluteal folds, with or without leg pain” following the definition from the European Guidelines for the prevention of LBP [21]. Back pain, defined as “pain in the posterior portion of the body that extends from the shoulder to the hips” [22]. Spinal pain is a broader definition including cervical, thoracic, and/or LBP. The onset of chronicity was defined as “continuous or intermittent pain or discomfort lasting 3 months or longer” [23].

For the search equation, selected terms were mapped by using the Medical Subject Headings index (MeSH) to increase pertinent results. No exclusion term was incorporated in the final search equation. Search domains were title and abstract. The search strategy and equations are summarised using the STARLITE framework [24] (Appendices B and C).

Study selection

Following the PRISMA recommendation, data extraction was performed in 3 phases. First, 2 independent reviewers (AM and MdF) screened the entire database and selected potentially relevant articles based on the title and abstract. Disagreements were resolved by discussion. Then, one author (AM) selected articles based on full-text analysis.

Data collection process

The Zotero software [25] and its Web navigator extension, v5.0.87, was used for reference management.

Data items

For each included study, one author (AM) obtained the following summary data: authors, date of publication, population at baseline and follow-up, total number of women and men at follow-up if provided, mean age and standard deviation if provided, LRFs at inclusion, outcome as presence or absence of chronic pain at follow-up, odds ratios (ORs, for dichotomous data) and risk ratios (RRs, for continuous data) with standard deviation for the association between LRFs and onset of CSP, confounding factors analysed by the authors, follow-up periods, and statistical methodology.

Risk of bias in individual studies

Risk of bias (RoB) was assessed by using the Quality in Prognosis Studies (QUIPS) tool. This tool considers several domains, including study participation, study attrition, prognostic factor measurement, outcome measurement, study confounding, statistical analysis and reporting. Each domain is assessed as “yes,” “partial,” “no,” or “unsure,” leading to a final judgment. The process leads to an overall subjective appreciation of the RoB (high, moderate or low). Originally designed for LBP, use of the QUIPS tool has been broadened to prognostic studies addressing different conditions such as rheumatologic or cardiovascular diseases. The tool’s validity, ease of use, assessment time and inter-observer agreement are good [26].

Synthesis of results

A statistically significant association was defined as $p < 0.05$ for univariate or multivariate associations and $a \geq 95\%$ confidence interval not including 1 for ORs and RRs. Additional analyses, such as p-trend [27], were also performed when relevant. Study results are summarised for each outcome (neck, low back or back chronic pain).

Given the study heterogeneity, no meta-analysis was performed. A qualitative meta-synthesis followed the method described by Verwoerd et al. [28]. According to these authors, a prognostic factor can

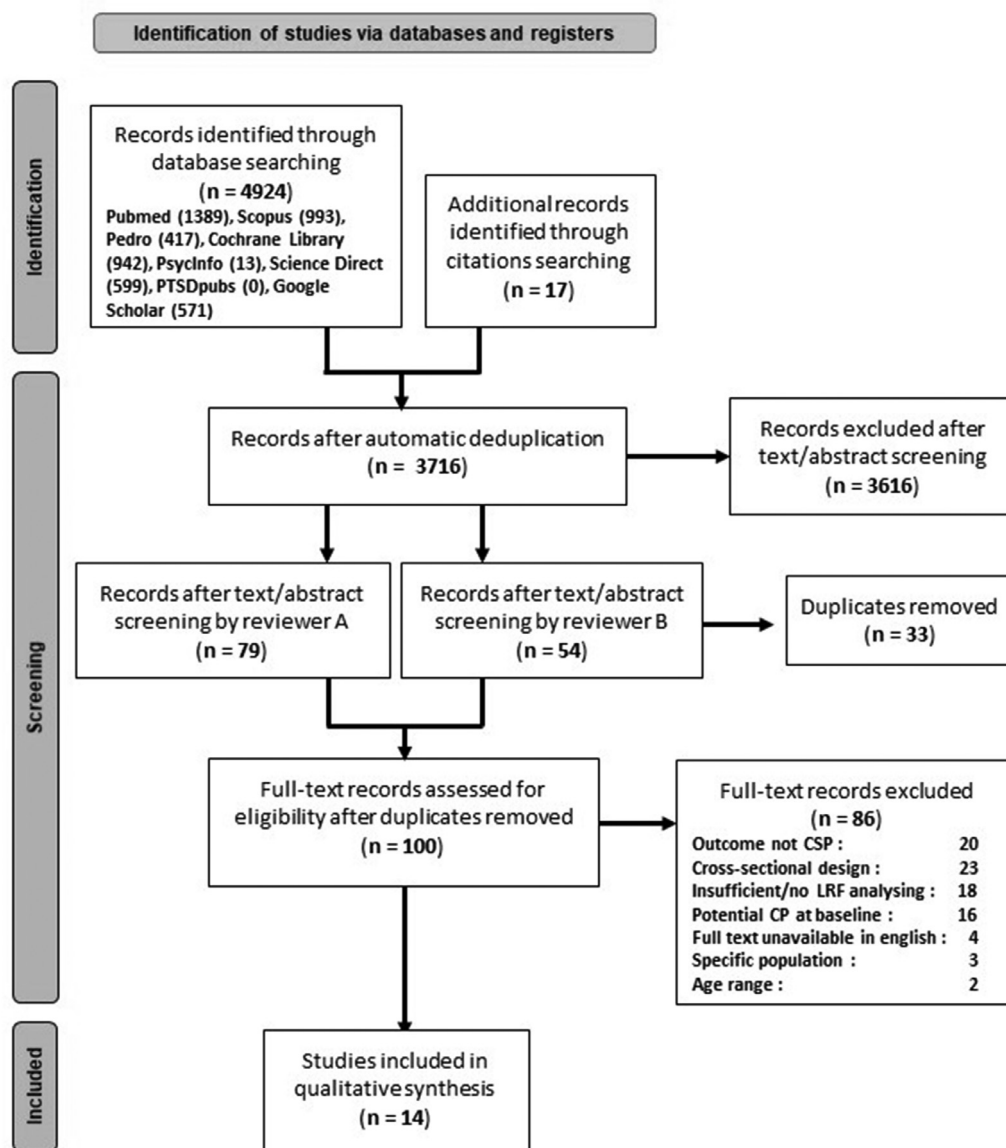


Fig. 1. PRISMA flow diagram. CP, chronic pain; CSP, chronic spine pain; LRF, lifestyle-related risk factor.

be qualified as having “limited evidence” if found in only one study, “moderate evidence” if found in one study at low RoB or more than one study at high RoB with >75% positive association with the outcome, and “strong evidence” if found in more than one study at low RoB with a positive association. “Conflicting evidence” was assigned if the evidence was inconsistent, with <75% studies showing the same direction of effect, and “no evidence” with no association found between variables.

Results

Study selection

The initial search identified 3716 unique records; 14 were included in the final analysis (Fig. 1).

Study characteristics (Table 1)

The 14 records selected for qualitative assessment included 13 inception cohort studies and one nested case–control study within a previously described cohort [29]. Cohorts originated from France,

Norway, Finland, Canada, New Zealand, the United States, Sweden and Scotland, for a total of 9 unique cohorts. Five studies included the Trøndelag Health Study cohort (HUNT studies, [29–33]). Nine studies followed large general population cohorts, one focused on municipal employees from Helsinki [34], 2 followed adult workers [35,36] and 2 recruited persons with acute/subacute back pain attending primary care centres [37,38]. Women prevailed in all cohorts but one [36]. The follow-up time varied greatly, from 6 months [37,38] to 13 years (HUNT studies). Eleven studies focused on chronic LBP (CLBP) occurrence, 3 chronic neck pain (CNP) and 2 chronic back pain (CBP).

All studies performed univariate and multivariate analyses for prognostic factors and confounding factors. The methodology varied among studies: 5 presenting results using RRs and the rest ORs.

Risk of bias within studies

Using the QUIPS tool, 10 studies were rated as low RoB and 4 as moderate RoB (Table 2). For the participation domain of the QUIPS tool, cohort samples were usually large, except for one study [36], which included a smaller sample of 315 adults. The highest RoB domain observed in these studies was attrition. Indeed, as frequently

Table 1
Characteristics of the included studies.

Authors	Population	N Baseline WomenN Men	Follow-upN WomenN Men	Lifestyle-related prognostic factor studied	Outcome	Follow-up-period	Statistical methodology
Esquirol et al., 2017 [36]	South France workers 32–52 years old No CLBP at inclusion	3237 1560 756 W 804 M		BMI (<25 or >25 kg/cm ²) Smoking (never/current or former smoker) Leisure time physical activity/sport (none or little/a lot) Leisure time physical activity/Gardening and DIY (none or little/a lot)	CLBP > 6 m prior to the follow-up period (yes/no)	5 y (1996 to 2001)	Binary logistic regression Backward multivariate logistic regression (OR) Akaike information criterion model selection ROC of selected model Multicollinearity analysis
Herin et al., 2014 [35]	France workers, 7 regions, 37–52 years No pain at inclusion	7808 6793 2447 W 4346 M		BMI (<25 or ≥25 kg/cm ²) Smoking (never, current or former) Sporting activities (yes or no)	CNP or CLBP ≥ 6 m (duration of current episode or intermittent complaints)	5 y (1990 to 1995)	Logistic regression analysis for univariate and multivariate models (HR)
Heuch et al., 2013 [32]	Nord-Trøndelag, Norway, 30–69 years No CLBP during previous year at inclusion	44,873 25,450 14,048 W 11,402 M		BMI (<25, 25–29.9 or ≥30 kg/cm ² and per 5 kg/m ²)	CLBP > 3 m continuously during the last year (yes/no)	11–13 y (1995–1997 to 2006–2008)	Logistic regression analysis for univariate and multivariate models (OR)
Heuch et al., 2014 [33]	Nord-Trøndelag, Norway, 30–69 years No CLBP during previous year at inclusion	44,923 25,450 14,048 W 11,402 M		Total cholesterol (≤4.8, 4.9–5.5, 5.6–6.1, 6.2–6.9 or ≥7 mmol/L) HDL cholesterol (≤1.0, 1.1–1.2, 1.3–1.4, 1.5–1.7 or ≥1.8 mmol/L) Triglycerides (≤0.94, 0.95–1.29, 1.30–1.70, 1.71–2.38 or ≥2.38 mmol/L) Or as continuous variables	CLBP > 3 m continuously during the last year (yes/no)	11–13 y (1995–1997 to 2006–2008)	Generalised linear modeling for binomially distributed data with a log link (RR) Linear analysis of continuous variable Likelihood test
Heuch et al., 2015 [30]	Nord-Trøndelag, Norway, 30–69 years No CLBP during year preceding inclusion	58,928 25,329 13,942 W 11,387 M		Body weight (kg) BMI (kg/cm ²) Waist circumference (cm) Hip circumference (cm) Waist-to-hip ratio	CLBP > 3 m continuously during the last year (yes/no)	11–13 y (1995–1997 to 2006–2008)	Generalised linear modeling for binomially distributed data with a log link (RR) Linear analysis of continuous variable
Heuch et al., 2016 [31]	Nord-Trøndelag, Norway, 30–69 years No CLBP during previous year at inclusion	31,145 18,068 9616 W 8452 M		Leisure time physical activity (light LTPA <1 h/week, light LTPA >1 h/w, hard LTPA <1 h/w, hard LTPA 1–2 h/w or LTPA ≥3 h/w)	CLBP > 3 m continuously during the last year (yes/no)	11–13 y (1995–1997 to 2006–2008)	Generalised linear modeling for binomially distributed data with a log link (RR) Quadratic analysis for U-shape relationship
Heuch et al., 2017 [29]	Nord-Trøndelag, Norway, 19–55 years No CLBP during previous year at inclusion	4822 (3137 controls 1685 cases*) 2720 W 2102 M		Serum 25-hydroxyvitamin D (<50.0, 50.0–74.9 or ≥75.0 nmol/L) Or as continuous variable Summer/autumn vs winter/spring season	CLBP > 3 m continuously during the last year (yes/no)	11–13 y (1995–1997 to 2006–2008)	Logistic regression analysis for univariate and multivariate models (OR) Quadratic analysis for linearity for continuous variable
Kääriä et al., 2012 [34]	Helsinki, Finland, municipal employees 40–60 years No CNP at inclusion	8960 5277 4220 W 1057 M		Sleep disorders (no, rare to occasional or frequent) BMI (<25, 25–29.9 or ≥30 kg/cm ²) Smoking (never, ex-smoker or current smoker) Leisure time physical activity (sport) (conditioning exercise, vigorous, moderate or inactive)	CNP > 3 m Continuously during the last 5–7 y	5–7 y (2000–2002 to 2007)	Logistic regression analysis for univariate and multivariate models (OR)

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Table 1 (Continued)

Authors	Population	N Baseline WomenN Men	N Follow-up N	Lifestyle-related prognostic factor studied	Outcome	Follow-up period	Statistical methodology
Kopec et al., 2004 [39]	Canadian households, adults > 18 years No back problems	11,063 10,007 5531 W 4476 M		Weight (kg) BMI (kg/m ²) Smoking (regular indoor cigarette smoke exposure, yes or no and number of years smoking and never smoked, former, occasional, or daily) Physical activity (monthly frequency ≥ 15 min and energy expenditure and sitting, stand/walk a lot, lift/carry light loads or heavy work and exercise regularly, sometimes or rarely and index active, moderate or inactive and LTPA type) Gardening/yard work (none, <3 h/m, ≥ 3 h/m) Drinking (not in pas 12 m, less than once per m, 1–3 times per m, 1+ time per w) Immigration status (yes or no) Marital status (married/partner, single or separated) Place of living (urban or rural)	CBP lasting or expecting to last > 6 m Excluding arthritis	2–4 y (1994–1995 to 1996–1997)	Logistic regression analysis for univariate model (OR) Logistic backward elimination for multivariate models (OR) ROC of selected models Bootstrap adjustment
Melloh et al., 2013 [37]	New Zealand adults 18–65 years Acute/recurrent BP at inclusion	315 168 104 W 64 M		Physical activity (IPAQ low, moderate or high) Smoking (pack/y) Drinking (Audit-C >4) Marital status (never, currently married, separated, divorced, widowed or cohabiting) BMI (kg/m ²)	Persistent LBP at 6 m, excluding specific LBP	6 m (3, 6, 12, 24 w)	Logistic regression analysis for univariate and multivariate models (OR)
Nilsen et al., 2011 [40]	Nord-Trøndelag, Norway, adults > 20 years Pain-free	45,925 32,417 (CNP) 16,952 W 15,465 M 30,575 (CLBP) 15,763 W 14,812 M		BMI (<18.5, 18.5–24.9, 25–29.9 or ≥ 30 kg/cm ²) Leisure time physical activity (sport) (<1, 1–1.9 or ≥ 3 times per w and average duration <15, 15–30, 31–60 or ≥ 60 min and average intensity without sweating/heavy breathing, sweating/heavy breathing or nearly exhausted and average number of hours)	CLBP > 3 m continuously during the last year (yes/no) CNP > 3 m continuously during the last year (yes/no)	9–13 y (1984–1986 to 1995–1997)	Generalised linear modeling for binomially distributed data with a log link (RR)
Skillgate 2017 [42]	Stockholm, Sweden, adults 18–84 years Pain-free	14,985 (CLBP) 9360 6225 W 3135 M 12,736 (CNP) 7760 4566 W 3194 M		Physical activity (< or ≥ 150 min moderate intensity or 75 min high intensity) Smoking (yes or no) Drinking ($\leq$ or > 168 g 100% alcohol/w for M and 108 g for F) Nutrition (healthy) (< or ≥ 4 portions of fruits and vegetables par day) Counted as healthy lifestyle behavior (0, 1, 2, ≥ 3 /4)	CLBP > 3 m, > a couple of days per week during the last 6 m (yes/no) CNP > 3 m, > a couple of days per week during the last 6 m (yes/no)	4 y (2006–2010)	Generalised linear modeling for binomially distributed data with a log link (RR)

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Table 1 (Continued)

Authors	Population	N Baseline WomenN Men	Follow-up N	Lifestyle-related prognostic factor studied	Outcome	Follow-up period	Statistical methodology
Smith 2004 [59]	Grampian, Scotland, adults > 25 years No CBP at inclusion	2422 1431 ? W ? M		Housing tenure (owned, council rented or private rented) Marital status (never, currently or no longer) Number of adults at home (1 or more)	CBP > 3 m	4 y (1996–2000)	Logistic regression analysis for univariate model (OR) Backward stepwise multiple logistic regression of multivariate model (OR)
Stevans et al., 2021 [38]	USA, 4 states, adults ≥ 18 years ALBP at inclusion	9547 5233 3029 W 2204 M		Smoking (no/former or current) BMI (< 25, ≥25 to <30, ≥30 kg/cm ²)	CLBP > 3 m and experiencing pain at least half the days in the past 6 m	6 m	Generalised linear mixed model with a logit link Backward stepwise multiple logistic regression of multivariate model and inverse probability weighting for missing data (adjusted OR)

BMI, body mass index; CLBP, chronic low back pain; CNP, chronic neck pain; CSP, chronic spinal pain; HDL, high-density lipoprotein; HR, hazard ratio LPTA, leisure time physical activity; M, men, m, month, OR, odds ratio, RR, risk ratio, w, week; y, year, W, women.

* Nested case control study of a prospective cohort.

Table 2

Assessment of risk of bias by using the Quality in Prognosis Studies tool.

	Esquirol et al., 2017 [36]	Herin et al., 2014 [35]	Heuch et al., 2013 [32]	Heuch et al., 2014 [33]	Heuch et al., 2015 [30]	Heuch et al., 2016 [31]	Heuch et al., 2017 [29]	Kääriä et al., 2012 [34]	Kopec et al., 2004 [39]	Melloh et al., 2013 [37]	Nilsen et al., 2011 [40]	Skillgate et al., 2017 [42]	Smith et al., 2004 [59]	Stevans et al., 2021 [38]
1. Study participation														
a) Adequate participation in the study by eligible persons	+	+	+	+	+	+	+	+	+	–	+	+	+	+
b) Description of the source population or population of interest	+	(+)	+	+	+	+	+	+	+	+	+	+	+	+
c) Description of the baseline study sample	+	+	+	+	+	+	+	+	+	+	+	+	+	+
d) Adequate description of the sampling frame and recruitment	+	+	+	+	+	+	+	+	+	+	+	+	+	+
e) Adequate description of the period and place of recruitment	+	+	+	+	+	+	+	+	+	+	+	+	+	+
f) Adequate description of inclusion and exclusion criteria	+	+	+	+	+	+	+	+	+	+	+	+	+	+
Risk of bias	Low	Low	Low	Low	Low	Low	Low	Low	Low	High	Low	Low	Low	Low
2. Study attrition														
a) Adequate response rate for study participants	(+)	+	+	+	+	+	/	+	+	+	+	+	+	+
b) Description of attempts to collect information on participants who dropped out	–	–	–	–	–	–	/	–	+	+	+	+	+	+
	+	–	+	+	+	+	/	–	+	+	+	–	–	–

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Table 2 (Continued)

	Esquirol et al., 2017 [36]	Herin et al., 2014 [35]	Heuch et al., 2013 [32]	Heuch et al., 2014 [33]	Heuch et al., 2015 [30]	Heuch et al., 2016 [31]	Heuch et al., 2017 [29]	Kääriä et al., 2012 [34]	Kopec et al., 2004 [39]	Melloh et al., 2013 [37]	Nilsen et al., 2011 [40]	Skillgate et al., 2017 [42]	Smith et al., 2004 [59]	Stevans et al., 2021 [38]
c) Reasons for loss to follow-up are provided														
d) Adequate description of participants lost to follow-up	—	—	—	—	—	—	/	—	+	+	+	+	+	+
e) There are no important differences between participants who completed the study and those who did not	?	?	?	?	?	?	/	?	—	(+)	+		(+)	(+)
Risk of bias	Moderate	High	Moderate	Moderate	Moderate	Moderate	/	High	Moderate	Moderate	Low	Moderate	Moderate	Low
3. Prognostic factor measurement														
a) A clear definition or description of the prognostic factor is provided	+	+	+	+	+	+	+	+	—	+	+	+	+	+
b) Method of prognostic factor measurement is adequately valid and reliable	+	(+)	+	+	+	+	+	+	(+)	+	+	+	+	+
c) Continuous variables are reported or appropriate cut points are used	+	+	+	+	+	+	+	+	+	+	+	+	+	+
d) The method and setting of measurement of prognostic factor is the same for all study participants	+	+	+	+	+	+	+	+	+	+	+	+	+	+
e) Adequate proportion of the study sample has complete data for the prognostic factor	+	+	+	+	+	+	/	+	+	+	+	+	—	+
f) Appropriate methods of imputation are used for missing prognostic factor data	—	—	—	—	—	—	/	—	—	—	—	—	—	+
Risk of bias	Low	Low	Low	Low	Low	Low	Low	Low	Moderate	Low	Low	Low	Moderate	Low
4. Outcome measurement														
a) A clear definition of the outcome is provided	+	+	+	+	+	+	+	+	+	+	+	+	+	+
b) Method of outcome measurement used is adequately valid and reliable	+	+	+	+	+	+	+	+	+	+	+	+	+	+
c) The method and setting of outcome measurement are the same for all study participants	+	+	+	+	+	+	—	+	+	+	+	+	+	+
Risk of bias	Low	Low	Low	Low	Low	Low	Moderate	Low	Low	Low	Low	Low	Low	Low
5. Study confounding														
a) All important confounders are measured	+	(+)	+	+	+	+	+	+	+	—	(+)	+	+	(+)
b) Clear definitions of the important confounder measures are provided	—	+	+	+	+	+	+	+	—	+	+	+	+	+
c) Measurement of all important confounders is adequately valid and reliable	—	(+)	+	+	+	+	+	+	?	+	+	+	+	+
d) The method and setting of confounding measurement are the same for all study participants	+	+	+	+	+	+	+	+	—	+	+	(+)	+	+
	—	—	—	—	—	—	/	—	—	—	—	—	—	+

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Table 2 (Continued)

	Esquirol et al., 2017 [36]	Herin et al., 2014 [35]	Heuch et al., 2013 [32]	Heuch et al., 2014 [33]	Heuch et al., 2015 [30]	Heuch et al., 2016 [31]	Heuch et al., 2017 [29]	Kääriä et al., 2012 [34]	Kopeck et al., 2004 [39]	Melloh et al., 2013 [37]	Nilsen et al., 2011 [40]	Skillgate et al., 2017 [42]	Smith et al., 2004 [59]	Stevens et al., 2021 [38]
e) Appropriate methods are used if imputation is used for missing confounder data														
f) Important potential confounders are accounted for in the study design	+	+	+	+	+	+	+	+	+	+	—	+	+	+
g) Important potential confounders are accounted for in the analysis	+	+	+	+	+	+	+	+	+	—	—	+	+	+
Risk of bias	Moderate	Low	Low	Low	Low	Low	Low	Low	Moderate	High	High	Low	Low	Moderate
6. Statistical analysis and reporting														
a) Sufficient presentation of data to assess the adequacy of the analytic strategy	+	+	+	+	+	+	+	+	+	+	+	+	+	+
b) Strategy for model building is appropriate and is based on a conceptual framework or model	+	+	+	+	+	+	+	+	+	+	+	+	+	+
c) The selected statistical model is adequate for the design of the study	+	+	+	+	+	+	+	+	+	+	+	+	+	+
e) There is no selective reporting of results	+	+	+	+	+	+	+	+	+	+	+	+	—	+
Risk of bias	Low	Low	Low	Low	Low	Low	Low	Low	Low	Low	Low	Low	Moderate	Low
Global risk of bias	Low	Low	Low	Low	Low	Low	Low	Low	Moderate	Moderate	Moderate	Low	Moderate	Low

(+) yes, (−) no, (+) partial, (?) unsure, and (/) irrelevant.

observed in large prognostic cohort studies, the proportion of participants with complete follow-up was often low, ranging from 43% [31] to 90% [39]. Individual studies assessed partly different sets of prognostic factors and included partly overlapping sets of potential confounding factors. This situation increases heterogeneity in the findings. In particular, one study assessed only a few confounding factors, without statistical justification [40]. Each study justified the choices for univariate and multivariate analyses. Data reporting was sufficient, although sometimes low for the excluded confounding factors in univariate analyses. Except for anthropomorphic parameters, the included studies relied on questionnaires to gather relevant information, thus inducing some risk of self-reporting bias (social desirability, recall period, selective recall and sampling approach) [41].

Results of individual studies

Table 3 presents relevant LRFs retrieved from each study, with details on statistical analysis. Results were often not statistically significant. When significant, ORs or RRs were generally small to moderate. Included studies reviewed the association between LRFs and onset of CNP, CLBP and CBP.

Esquirol et al. (low RoB) [36] and Herin et al. (low RoB) [35] did not find any significant association between the occurrence of CLBP and high body mass index (BMI), smoking or leisure-time physical activity. In the HUNT cohort (low RoB) [29–33], CLBP was significantly associated with high BMI (ORs 1.10–1.34, RRs 1.08–1.09) and related measures such as body weight, waist circumference or hip circumference (RRs 1.06–1.07). Cholesterol, triglycerides and 25-hydroxyvitamin D levels were not significantly associated with CLBP.

Kääriä et al. (low RoB) [34] found significant associations between the occurrence of CNP and sleep disorders or high BMI in women only (ORs 1.22–1.54). Data for smoking and leisure-time physical activity were not significant.

Kopec et al. (moderate RoB) [39] did not find any significant association between the occurrence of CBP and BMI, physical activity, drinking, smoking, immigration status, and marital status. The authors found a protective effect of gardening or yard work (OR 0.55–0.62) in men only.

Melloh et al. (moderate RoB) [37] found no association between CLBP occurrence and smoking, drinking, marital status, or BMI.

In the study of Nilsen et al. (moderate RoB) [40], CLBP was significantly associated with leisure-time physical activity (RRs 0.75–0.92) and BMI in some subgroups of participants (RRs 1.18–1.50). These authors included few confounding factors in their statistical analysis. Similar results were observed for CNP.

Of note, Skillgate et al. (low RoB) [42] found several healthy lifestyle behaviours (physical activity, absence of daily smoking, alcohol consumption ≤ 168 g 100% alcohol/week for men and ≤ 108 g 100% alcohol/week for women, consumption of fruits and vegetables) significantly associated with reduced CLBP occurrence only with the presence of at least 3 of these behaviours (RRs 0.72–0.86). These authors observed similar data for CNP, but in women only.

Smith et al. (moderate RoB) [38] found no association between CBP and marital status or housing tenure. The presence of more than one adult at home was protective.

Finally, Stevans et al. (low RoB) [38] found a significant association between high BMI and smoking for the transition from acute to CLBP.

Meta-synthesis of the results

Results of the meta-synthesis are summarised in Table 4 (details in Appendix D). For LBP, we found moderate evidence for an association with high body weight, high waist circumference and high hip circumference but no evidence for high waist-to-hip ratio after adjustment. The evidence for high BMI was conflicting: we found a positive association in 3 studies with low RoB and one with moderate

RoB and no associations in 2 studies with moderate and one study with low RoB. Leisure-time physical activity also showed conflicting evidence: 2 studies with low RoB showed no association, one with moderate RoB found a negative association and one study with low RoB found a negative association that was limited to the 50–59 age group. Gardening and/or yard work during leisure time showed no evidence as a protective factor [36]. We also found conflicting evidence for the association between smoking and CLBP (one low-risk and one moderate-risk study found no association, whereas one low-risk study found a small association). We found no association between LBP and 25-hydroxyvitamin D levels (one low-risk study), high cholesterol/triglyceride serum concentration (one low-risk study), marital status (one moderate-risk study) and alcohol consumption (one moderate-risk study). The cumulative association of several healthy lifestyle factors showed a trend for a negative association with CLBP.

Neck pain was addressed by 2 low-risk studies and one moderate-risk study. We found strong evidence for a positive association between high BMI and CNP for women only, with conflicting evidence observed for men (no association in one low-risk study, a positive association in another moderate-risk study). Similarly, sleep disorders showed moderate evidence for women only (one low-risk study). Evidence for leisure-time physical activity was conflicting, with no association in one low-risk study and a clear protective effect for men and women in a much larger study, although the latter analysed few confounding factors. We found no evidence for smoking (one low-risk study) and sleep disorders in men (one low-risk study). As with CLBP, we found some evidence for a negative association between CNP and several healthy lifestyle factors in women.

Two studies with moderate RoB focused on CBP. We found limited evidence for a negative association with gardening/yard work for men but a positive association with the presence of more than one adult at home. We found no association for marital status, housing tenure, place of living, immigration status, alcohol and tobacco consumption, high BMI and high body weight.

Discussion

Summary of evidence

This systematic review identified 14 longitudinal studies analysing the influence of LRFs on the occurrence of chronic spinal pain. The RoB was low to moderate. One study [36] included only 315 participants, but the others included large to very large cohorts (3137 to 77,216 participants). Despite these large cohorts, the evidence is still scarce and often conflicting. Although high body weight (or another related variable) could reasonably be considered a risk factor for CSP, the evidence was surprisingly conflicting for physical activity, and smoking was not associated with CSP. CIs for ORs and RRs were only slightly different from 1, showing that the association between LRFs and CSP was weak: if the prevalence of CSP in the general population is assumed to be 10%, then the effect size derived from OR data is 0.21 at best [43], corresponding to a small effect size.

How could LRFs affect the occurrence of CSP?

Two meta-analyses including data published before 2000 [44,45] observed a possible weak association between high body weight and CLBP. A first hypothesis accounting for this association is the chronic inflammation induced by the metabolic syndrome [46]. Although the lack of evidence for the high-cholesterol/triglyceride risk factor observed by Heuch et al. [33] gives lower credit to this theory, the observation that central adiposity is better related to CLBP than is BMI [30] supports it. A second hypothesis might be that the increased mechanical load induces chronic injury in the low spine, such as disk degeneration [47]. However, despite the frequent proposal of

Table 3

Odds ratios and risk ratios for lifestyle-related prognostic factors for chronic low back, neck, and back pain with multivariate analysis for confounding factors.

Authors	Odds ratios and risk ratios (95% CI)	Confounding factors	
	Chronic Low Back Pain		
Esquirol et al., 2017 [36]	BMI >25 kg/m² OR 1.25 (0.92; 1.70)** Smoking (current or former) OR 1.15 (0.86; 1.56)*	Leisure time physical activity (sport, a lot) OR 0.97 (0.63; 1.51)* Leisure time physical activity (gardening and DIY) OR 0.59 (0.28; 1.23)*	*No further adjustment **Age, sex, history of rheumatological and depression events, leisure time physical activity with sport and with gardening and DIY, job changes between both instances of data collection, age at first employment, shift work, job recognition and communication difficulties with colleagues
Herin et al., 2014 [35]	BMI (≥25 kg/cm²) HR 0.94 (0.82; 1.07) M* HR 1.08 (0.90; 1.30) W* Smoking (former) 1.03 (0.88; 1.21) M* 0.96 (0.76; 1.23) W*	Smoking (current) 1.06 (0.90; 1.24) M* 0.94 (0.74; 1.21) W* Sporting activities (yes) 0.88 (0.77; 1.01) M* 0.99 (0.82; 1.19) W*	*Age, social class, BMI, smoking status, participation in sporting activities
Heuch et al., 2013 [32]	BMI (25–29.9 kg/m²) OR 1.13 (0.97; 1.32) M* OR 1.19 (1.06; 1.34) W* ⊕ BMI (≥30 kg/cm²) OR 1.34 (1.08; 1.67) M* ⊕ OR 1.22 (1.03; 1.46) W* ⊕	BMI (per 5 kg/m²) OR 1.16 (1.05; 1.29) M* ⊕ OR 1.10 (1.03; 1.18) W* ⊕ for 50–69 yo	*Age, sex, education, work status, physical activity at work and in leisure time, smoking, blood pressure, lipid levels, and time between last meal and blood sampling
Heuch et al., 2014 [33]	Total cholesterol (per mmol/L) RR 0.98 (0.94; 1.02) W* RR 0.97 (0.92; 1.02) M* HDL cholesterol (per mmol/L)* RR 0.96 (0.85; 1.07) W* RR 0.91 (0.77; 1.08) M*	Triglycerides (per mmol/L) RR 1.16 (0.94; 1.42) W* RR 1.24 (0.96; 1.58) M*	*Age, sex, education, work status, physical activity, smoking, BMI, blood pressure and time between last meal and blood sampling
Heuch et al., 2015 [30]	Body weight (kg) RR 1.087 (1.039; 1.138) W* ⊕ RR 1.130 (0.995; 1.284) W** RR 1.091 (1.030; 1.157) M* ⊕ RR 1.124 (0.976; 1.294) M** BMI (kg/cm²) RR 1.075 (1.023; 1.128) W* ⊕ RR 1.091 (1.027; 1.158) M* ⊕	Waist circumference (cm) RR 1.078 (1.025; 1.134) W* ⊕ RR 1.064 (1.001; 1.131) M* ⊕ Hip circumference (cm) RR 1.073 (1.024; 1.123) W* ⊕ RR 1.060 (1.000; 1.123) M* ⊕ Waist-to-hip ratio RR 1.033 (0.985; 1.084) W* RR 1.037 (0.975; 1.102) M*	*Age, sex, education, work status, physical activity, smoking, HDL-cholesterol, triglycerides, and blood pressure **Age, education, work status, physical activity, smoking, HDL-cholesterol, triglycerides, blood pressure and mutual adjustment for body weight, BMI, waist circumference and hip circumference
Heuch et al., 2016 [31]	Leisure time physical activity (light >1 h/w) RR 0.97 (0.86; 1.09) W** RR 1.03 (0.87; 1.21) M** (hard <1 h/w) RR 0.99 (0.87; 1.13) W** RR 0.77 (0.60; 0.99) M* ⊕ RR 0.96 (0.82; 1.14) M**	(hard 1–2 h/w) RR 0.73 (0.55; 0.95) W* ⊕ RR 0.89 (0.77; 1.03) W** RR 0.65 (0.50; 0.85) M* ⊕ RR 0.81 (0.68; 0.97) M** (hard ≥3 h/w) RR 1.05 (0.86; 1.27) W** RR 0.66 (0.48; 0.90) M* ⊕ RR 0.96 (0.79; 1.17) M**	*Age 50–69 years old only **Age, sex, education, work status, physical activity at work, BMI, smoking
Heuch et al., 2017 [29]	Serum 25(OH)D (50.0–74.9 nmol/L) OR 1.01 (0.85; 1.20)* Serum 25(OH)D (≥75.0 per nmol/L) OR 0.98 (0.73; 1.31)*	Serum 25(OH)D (per 10 nmol/L) OR 1.01 (0.97; 1.06)*	*Age, sex, work status, physical activity at work and in leisure time, education, smoking, body mass index, subsample, and season of blood sample collection
Melloh et al., 2013 [37]	Smoking (pack/y) OR NS* Drinking (Audit-C >4) OR NS*	Marital status OR NS* BMI (kg/m²) OR 1.01 (0.90; 1.12)*	*Age, sex (and BMI)
Nilsen et al., 2011 [40]	Leisure time physical activity (sport) (inactive, <1 time/w) RR 0.90 (0.81; 1.01) W** RR 0.91 (0.80; 1.03) M** Leisure time physical activity (1–1.9 time/w) RR 0.84 (0.74; 0.95) W** ⊕ RR 0.88 (0.77; 1.00) M** ⊕ Leisure time physical activity (≥2 time/w) RR 0.92 (0.79; 1.07) W** RR 0.75 (0.64; 0.88) M** ⊕ p-trend 0.02 W p-trend < 0.001 M	BMI (<18.5 kg/m²) RR 0.94 (0.67; 1.32) W** RR 1.08 (0.47; 2.47) M** BMI (25–29.9 kg/m²) RR 1.18 (1.06; 1.30) W** ⊕ RR 1.03 (0.93; 1.14) M** BMI (≥30 kg/m²) RR 1.21 (1.04; 1.41) W** ⊕ RR 1.21 (0.99; 1.46) M** BMI ≥30 kg/m² and inactive RR 1.44 (1.13; 1.77) W* ⊕ RR 1.50 (1.16; 1.95) M* ⊕	*No further adjustment **Age, sex, BMI, smoking and occupation class
Skillgate et al., 2017 [42]	Number of healthy lifestyle behavior (HLB) (Physical activity, low smoking or drinking, Fruits and vegetables consumption) HLB (2) RR 0.87 (0.65; 1.16)* RR 1.07 (0.70; 1.64) W** RR 0.74 (0.49; 1.12) M*** HLB (3)	HLB (4) RR 0.84 (0.52; 1.34)* RR 1.04 (0.60; 1.82) W** RR 0.54 (0.17; 1.73) M*** HLB (3 or 4) RR 0.72 (0.53; 0.98)* ⊕ RR 0.86 (0.56; 1.32) W**	*Socioeconomic status, age, and sex **Socioeconomic status ***Socioeconomic status, economic stress, and neck pain

(continued)

Table 3 (Continued)

Authors	Odds ratios and risk ratios (95% CI)	Confounding factors
Stevens et al., 2021 [38]	RR 0.69 (0.49; 0.96)* [Ⓟ] RR 0.79 (0.50; 1.26) W** RR 0.65 (0.39; 1.06) M*** Smoking (current) 1.63 (1.35; 1.97)* [Ⓟ] BMI (≥ 25 to < 30) 1.57 (1.31; 1.87)* [Ⓟ]	RR 0.63 (0.39; 1.02) M*** As protective factors BMI (≥ 30)* 1.53 (1.28; 1.83)* [Ⓟ] *Age, race, ethnicity, BMI, smoking status, STarT Back risk level, Oswestry score, depression/anxiety status, geographic location, area deprivation index status (Neighborhood Atlas Tool), pharmacologic therapies, process of care status (concordant or not with guidelines)
Kääriä et al., 2012 [34]	Chronic Neck Pain Sleep disorders (frequent) OR 1.54 (1.22; 1.95) W** [Ⓟ] OR 1.14 (0.61; 2.13) M** BMI (25–29.9 kg/cm²) OR 1.22 (1.00; 1.48) W** [Ⓟ] OR 0.60 (0.38; 0.96) M* BMI (>30 kg/cm²) OR 1.39 (1.08; 1.80) W** [Ⓟ] OR 1.11 (0.61; 2.02) M*	Smoking (current smoker) OR 1.11 (0.89; 1.37) W* OR 0.85 (0.50; 1.44) M* Leisure time physical activity (inactive) OR 0.98 (0.71; 1.36) W* OR 1.05 (0.55; 2.00) M* (vigorous) OR 1.05 (0.76; 1.46) W* OR 0.84 (0.44; 1.60) M* *Age only **Age, sex, physical workload, emotional exhaustion, bullying, common mental disorders (GHQ), sleep disorders, acute NP, LBP and BMI
Nilsen et al., 2011 [40]	Leisure time physical activity (sport) (< 1 time/w) RR 0.95 (0.88; 1.02) W* RR 0.93 (0.85; 1.02) M* Leisure time physical activity (1–1.9 time/w) RR 0.87 (0.80; 0.95) W* [Ⓟ] RR 0.88 (0.80; 0.97) M* [Ⓟ] Leisure time physical activity (≥ 2 times/w) RR 0.91 (0.81; 1.01) W* RR 0.81 (0.72; 0.91) M* [Ⓟ] p-trend 0.002 W p-trend < 0.001 M	BMI (<18.5 kg/m²) RR 1.00 (0.81; 1.25) W* RR 0.75 (0.35; 1.58) M* BMI (25–29.9 kg/m²) RR 1.12 (1.04; 1.21) W* [Ⓟ] RR 1.12 (1.04; 1.21) M* [Ⓟ] BMI (≥ 30 kg/m²) RR 1.19 (1.07; 1.33) W* [Ⓟ] RR 1.22 (1.06; 1.41) M* [Ⓟ] *Age, sex, BMI, smoking and occupation class
Skillgate et al., 2017 [42]	Number of healthy lifestyle behavior (HLB) (physical activity, smoking, drinking, fruit and vegetable consumption) HLB (2) RR 1.05 (0.81; 1.37)* RR 0.75 (0.52; 1.09) W** RR 1.35 (0.93; 1.97) M*** HLB (3) RR 0.77 (0.57; 1.05)* RR 0.52 (0.34; 0.80) W** [Ⓟ] RR 1.03 (0.67; 1.60) M***	HLB (4) RR 0.88 (0.56; 1.40)* RR 0.53 (0.29; 0.95) W** [Ⓟ] RR 1.93 (0.95; 3.91) M*** HLB (3 or 4) RR 0.79 (0.59; 1.06)* RR 0.52 (0.35; 0.77) W** [Ⓟ] RR 1.13 (0.74; 1.71) M** *Sex, socioeconomic status, and personal support **Socioeconomic status ***Age, socioeconomic status, and economic stress
Kopec et al., 2004 [39]	Chronic Back Pain Weight (kg) OR NS* and ** BMI (kg/m²) OR NS* and ** Smoking OR NS* and ** Physical activity OR NS* and ** Drinking OR NS* and **	Immigration status OR NS* and ** Marital status OR NS* and ** Place of living OR NS* and ** Gardening/Yard work (<3 h/m) OR NS W* OR 0.62 (0.40; 0.94) M** [Ⓟ] Gardening/Yard work (≥ 3 h/m) OR NS W* OR 0.55 (0.38; 0.80) M** [Ⓟ] * (F) Income adequacy, weight, activity restriction, self-rated health, type of smoker, monthly frequency of physical activity, walking for exercise or errands, exercise or aerobics class, fishing, arthritis or rheumatism, high blood pressure, cancer, glaucoma, heart disease, migraine headaches, other chronic conditions, financial stress index, child-related stress index, personal stress index, psychological childhood trauma, mental distress scale, average frequency of contacts index, working status ** (M) Age, current main activity, urban or rural residence, height, body mass index, self-rated health, number of smoking years, usual daily activity, gardening or yard work, allergies other than food allergies, arthritis or rheumatism, heart disease, stomach or intestinal ulcers, other chronic conditions, specific chronic stress index, chronic stress index, child-related stress index, psychological childhood trauma, sense of coherence, social involvement score, recent life events score
Smith et al., 2004 [59]	Housing tenure (council rented) OR 1.09 (0.78; 1.54)* Marital status (married) OR 0.92 (0.58; 1.47)* Marital status (separated/divorced) OR 1.28 (0.72; 2.26)*	Number of adults at home (more than 1) SS** [Ⓟ] as protective factor In adult with no chronic pain at baseline *No further adjustment **Age, sex, number of adults at home, marital status, arthritis at baseline, chronic pain at baseline, level of expressed need at baseline, SF-36 dimensions

Ⓟstatistically significant at $p < 0.05$; BMI, body mass index; DIY, do-it-yourself leisure time physical activity; HDL, high-density lipoprotein; HR, hazard ratio; M, men; m, month; NS, non-significant; OR, odds ratio; RR, risk ratio; SS, statistically significant; w, week; W, women.

Table 4
Qualitative meta-synthesis.

Grade	CLBP	CNP	CBP
Strong evidence (consistent findings in multiple low RoB studies, with >75% of studies showing the same direction of effect)		High BMI in women	
Moderate evidence (consistent findings in multiple high RoB studies and/or one study with low RoB)	High body weight High waist circumference High Hip circumference	Sleep disorders in women	
Limited evidence (one study available)			More than one adult at home (protective) Gardening/Yard work in men (protective)
Conflicting evidence (inconsistent findings across studies)	High BMI Smoking Physical activity (protective)	High BMI in men Physical activity (protective)	
No evidence (No association between variables)	High cholesterol/triglycerides Serum 25(OH)D Marital status Waist-to-hip ratio Drinking Gardening/yard work	Sleep disorder in men Smoking	Marital status Housing tenure Gardening/Yard work in women Place of living Immigration status Drinking Physical activity Smoking High BMI High body weight

BMI, body mass index; CBP, chronic back pain; CLBP, chronic low back pain; CNP, chronic neck pain.

hypotheses attributing LBP to mechanical constraints, they are contradicted by several meta-analyses showing no association between LBP and occupational physical activity such as bending/twisting, awkward postures, sitting, standing/walking, carrying, pushing/pulling, lifting and manually handling/assisting disabled or sick persons [48].

Physical activity is classically considered a protective factor for LBP [49] and is recommended for preventing and managing LBP [50,51]. Exercise may decrease inflammatory factor levels [52]. Therefore, somewhat surprisingly, our study found only conflicting evidence for the association of physical activity and CLBP/CNP. Our results are in line with 2 recent meta-analyses, also reporting conflicting evidence [53,54]. Another meta-analysis [55] concluded strong evidence for physical activity as a protective factor for the severity and impact of CLBP. However, evidence was conflicting in support of the use of exercise to prevent LBP episodes. Methodological quality was low for 9 of 10 studies and heterogeneity was high.

In the present review, 2 of 4 studies showed a negative association between physical activity and CLBP. Our conclusion of “conflicting evidence” might be attributed to the meta-synthesis method, which is sensitive to grading individual studies. Moreover, prognostic studies often measure total physical activity (work-related physical activity as well as leisure). In the hypothesis of physical activity as a protective factor, we restricted our data extraction to studies clearly considering leisure-time physical activity only, hence reducing data volume. Another explanation for the discrepancy between studies might be the observation that the association between physical activity and health might follow a U-shaped curve, both low and very high activity levels being potentially detrimental [31]. Finally, most of these studies evaluated self-reported physical activity, which is probably over-evaluated. Considering the available literature and our present results, the benefits of leisure-time physical activity as a protective factor for CSP might not be as clear as previously thought.

The association between sleep disorders and chronic pain prevalence have been well documented. However, the direction of this association is uncertain, each outcome likely aggravating the other in a vicious circle [56]. In our study, sleep disorders were associated with CNP for women only, in a single study with low RoB [34]. Evidence for sleep disorders as a risk factor for CNP is scarce. A large cross-sectional study found an OR of 3.5 (3.0–4.0) for the association of insomnia/trouble falling asleep and neck pain in the last 3 months

[57]. A prospective study that did not meet our inclusion criteria [58] highlighted a poorer recovery rate among individuals with neck pain and poor sleep quality.

Social support was investigated in only one longitudinal study [59] and was reported as a protective factor. However, this association was not properly documented and only mentioned in the discussion.

Finally, we found conflicting evidence between smoking and outcome. Smoking and chronic pain share a complex relationship. Although smoking shows some evidence as a risk factor for chronic widespread pain [60], this association is blurred for spinal pain, as shown by another systematic review [61].

As stated above, the effect sizes measured in the studies included in this review are small, which suggests that their role in the development of CSP is weak when they are considered alone. However, the observation that the accumulation of healthy lifestyle habits may play a protective role [42] is encouraging and deserves further research. Another field of research should explore the interrelations between these variables (e.g., physical activity, body weight, smoking, and sleep quality). Finally, the association between lifestyle behaviours and cognitive–emotional factors merits further exploration. For example, the level of physical activity may highly depend on representations and beliefs such as kinesophobia.

Limitations

Limitations of this study can be ascribed to limitations in both our study protocol and the individual studies included. Concerning our study protocol, several limitations need to be considered. First, the only outcome variable considered was the presence of CSP at follow-up. However, the presence or absence of pain is a weak indicator. Functional outcomes relating to the International Classification of Function [62] domains “activity” and “participation” could have been included, but this would have greatly changed our methodology and further increased heterogeneity. Second, we limited our selection to prospective cohorts, case–control and RCT studies, which led to the elimination of 23 cross-sectional records. As mentioned above, cross-sectional studies cannot establish a link of causality between associated factors [63]. Third, our choice to assess RoB might be questioned. An assessment of the RoB is often missing in epidemiologic systematic reviews [64]. Numerous tools exist, but none is optimal [65]. The

PRISMA guidelines [66] and the Cochrane handbook [67] advise against the use of scales because item weighting is contextual and trying to reduce a multidimensional concept to a single numerical value can lead to further bias. We decided to use the QUIPS tool because it is specific to back pain epidemiologic systematic reviews and has been comprehensively evaluated [26]. Furthermore, it is compatible with meta-synthesis [28]. The meta-synthesis protocol has the advantage of providing a rating for the strength of evidence of prognostic factors. However, it does not account for the size of the studies (as would have been the case for a meta-analysis) and might be quite sensitive to small changes when rating the quality of the studies. Because of data heterogeneity, no meta-analysis could be performed.

Limitations of the included studies mainly relate to their high attrition rate, which is common in longitudinal epidemiological studies [68]. However, the RoB estimated by the QUIPS tool was low for 10 studies and moderate for the other 4. The use of a prevalence ratio for dichotomous variables and their integration as ORs for common long duration diseases such as CSP is discouraged because it tends to overestimate the association [63]. As discussed above, the absence of functional indicators is also a limitation. One third of the studies relied on the same cohort of participants [29–33], which might bias our conclusions.

Conclusions

This systematic review of longitudinal studies showed little and conflicting evidence for the association between several modifiable lifestyle factors and the onset of CSP. Clearly, more studies are needed. For CLBP, we found moderate evidence that being overweight is a risk factor but only conflicting evidence for being physically active. For CNP, being overweight and sleep disorders in women showed strong and moderate evidence as risk factors. For CBP, we found limited evidence for social support and gardening as a protective factor and only conflicting evidence for the role of smoking in the development of CSP. Effect sizes were small, which suggests that interventions specifically addressing one single factor might be of little clinical value. The association of several LRFs might be more significant than isolated factors, but this also deserves further exploration, as for the question of the interactions between factors. The medical literature often focuses on risk factors, fostering negative messages addressed to the individual (e.g., “being overweight is dangerous”). However, lifestyle-related behaviours as protective factors might also be considered much more positively. Gifford [69] called for the identification of “pink flags” to stop the “focus on bad bias” while positively encouraging people to strengthen their capacities.

Conflict of interest

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Supplementary materials

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