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Predictors of Low Back Pain Onset in a Prospective British Study

[Chris Power](#), PhD, [John Frank](#), MD, [Clyde Hertzman](#), MD, [Gill Schierhout](#), PhD, and [Leah Li](#)

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Abstract

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Objectives. This study examined predictors of low back pain onset in a British birth cohort.

Methods. Univariate and multivariate analyses focused on individuals who experienced onset of low back pain at 32 to 33 years of age ($n = 571$) and individuals who were pain free ($n = 5210$). Participants were members of the 1958 British birth cohort.

Results. Incident pain was elevated among those with psychological distress at 23 years of age (adjusted odds ratio [OR] = 2.52, 95% confidence interval [CI] = 1.65, 3.86) and among persistent moderate or heavy smokers (adjusted OR = 1.63, 95% CI = 1.23, 2.17). Significant univariate associations involving other factors (e.g., social class, childhood emotional status, body mass index, job satisfaction) did not persist in multivariate analyses.

Conclusions. This prospectively studied cohort provides evidence that psychological distress more than doubles later risk of low back pain, with smoking having a modest independent effect. Other prospective studies are needed to confirm these findings before implications for low back pain prevention can be assessed.

Low back pain, a leading cause of disability in the industrialized world, is well recognized as having a multifactorial etiology. The main predictors of back pain include physical stress (e.g., prolonged lifting, driving, forceful or repetitive movements involving the back),^{1–7} psychosocial stress (e.g., high perceived workload and time pressure, low control and lack of social support at work),^{8,9} personal characteristics (e.g., psychological status^{10,11} and tobacco use^{12–14}), and physical characteristics (e.g., obesity and height).^{15,16} Although these factors represent conditions existing during working life as well as conditions accumulated during the prework period, most studies focus exclusively on ergonomic and psychosocial working conditions. With a few exceptions,^{15,17} there exists, to our knowledge, almost no research on the contribution to low back pain of prework experiences in combination with working life factors.

Relevant longitudinal data on early life risk factors and adult low back pain are available from the 1958 British birth cohort study. First, with regard to back pain, data obtained in the 23- and 33-year follow-ups allow differentiation of incident and chronic pain. The aim of this article is to identify independent risk factors for incident pain in the year before the 33-year follow-up.

Second, with regard to risk factors, the 1958 study contains extensive information on prework experiences as well as working life factors. Prework experiences include early life factors (e.g., social class at birth, growth patterns, and socioemotional status), physical characteristics (e.g., obesity and height), and early adult factors (e.g., educational attainment, psychological status, and smoking behavior). Information relevant to working

life includes job title (from which ergonomic exposure can be inferred), psychosocial work characteristics, family circumstances, and adult emotional and practical support. The study therefore provides an opportunity for simultaneous investigation of both earlier and working life risks from birth to 33 years of age.

METHODS

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The 1958 birth cohort¹⁸ includes all children born in England, Wales, and Scotland between March 3 and March 9, 1958. The cohort was originally part of the Perinatal Mortality Study, which included information on 17 414 births. Five subsequent follow-up studies have been conducted (when the cohort members were aged 7, 11, 16, 23, and 33 years), with 11 407 subjects included in the most recent sweep (1991).¹⁹ Despite sample attrition, those remaining in the study have been found to be generally representative of the original sample.^{19,20}

There are 2 sources of information on back pain in the cohort. When cohort members were aged 23 and 33 years, information on backache was recorded as part of a self-completion checklist of psychological and somatic symptoms (the Malaise Inventory).^{21,22} The specific question asked was “Do you often have backache?” When subjects were aged 33 years, they responded to questions on low back pain that had been shown to be appropriate for epidemiologic studies.²³ Subjects were asked, “Have you ever had back pain in the area shaded in this picture, that is, pain which lasted for more than one day, but not counting the kind of pain you can get with flu or [if female respondent] with periods and pregnancy?” (The shaded area, between the 12th rib and the gluteal folds, was illustrated on a body diagram.) They were then asked, “How old were you when you first had back pain lasting more than one day?”

We identified subjects with incident cases who reported a first episode of low back pain in the year preceding the 33-year interview and who, in addition, did not report backache at the age of 23 years. Excluded from the analyses were those with persistent pain at 23 and 33 years ($n = 1157$), those with incident pain between 23 and 32 years ($n = 1638$), those with pain at 23 years who recovered ($n = 1628$), and those with missing data ($n = 1203$). The total sample comprised 5781 subjects, 5210 who were pain free throughout the study and 571 with recent incident low back pain. Because recall is more repeatable over the preceding 12 months than over longer periods, we selected incident pain at 32 to 33 years of age as the study outcome.^{23,24}

Potential explanatory variables were initially categorized as those occurring during working life and those usually developing before an individual joins the labor market. The variables were then grouped according to nominal characteristics (referred to here as factors).

Working Life

Ergonomic factors. A proxy measure of physical ergonomic stress was constructed from subjects' current (33 years of age) or most recent job title, based on a modification of the 9 major Standard Occupational Classification categories used by Liira et al.²⁵ in the Ontario Health Survey. Occupational categories were modified by separating out transport and construction workers from the British “unskilled manual” (class V) occupational classification and collapsing administrative and professional occupations (classes I, II, and III). This yielded 9 groups that were comparable to the categories used in the Ontario Health Survey, for which data on ergonomic stress have been previously published.²⁵ A “driving in the past year” variable was based on respondents' estimates of the total annual mileage that they had driven (in specified mileage categories) during the 12 months before the 33-year interview.

Psychosocial factors. Psychosocial work characteristics were obtained from questions asked at the 33-year interview regarding respondents' current work situation. Most subjects recorded information

relating to a paid job, but characteristics of unpaid work (e.g., housework or child care) were also included. Lack of learning opportunities, monotony, inability to vary one's work pace, and not having control over breaks were characterized as negative psychosocial work characteristics. The "learning" and "monotony" variables were similar to Karasek and Theorell's items addressing the skill discretion component of control at work.²⁶ The "breaks" and "pace" variables related more directly to the decision authority component of control at work. A sum of negative work characteristics across the 4 variables was calculated (range: 0–4); a high level was defined as the presence of 2 or more negative work characteristics. Subjects also reported on job satisfaction at 23 years (Table 1).

**TABLE 1—
Unadjusted Odds Ratios for Adult Life Factors Related
to Incident Low Back Pain at 32–33 Years of Age: 1958
British Birth Cohort, 1990–1991 (n=5781)**

Early adult factors. Psychological distress at 23 years of age was assessed through a self-report 24-item checklist (the Malaise Inventory)²¹ focusing on psychological and somatic symptoms. Because one Malaise Inventory item relates to backache and overlaps with the definition of back pain used in our analyses, we calculated malaise scores (range: 0 to 23) excluding this item. A score of 7 or higher defined high distress.

On the basis of reports at 16, 23, and 33 years of age, subjects were classified into 5 lifetime smoking groups: (1) never smoked, (2) never smoked moderately or heavily, (3) early moderate or heavy smoking (at or before 23 years) and cessation by 33 years, (4) early moderate or heavy smoking continuing at 33 years, and (5) late initiation (started heavy or moderate smoking between 23 and 33 years). Subjects who smoked 10 or more cigarettes per day were defined as moderate/heavy smokers.

Educational level obtained by 33 years of age was classified as shown in Table 1. Classifications were comparable to those used in the United States: high school or above ("A level"), completion of grade 10 ("O level"), completion of less than grade 10, and no educational qualifications. The 1980 British registrar general's classification of occupations was used to determine social class at 23 years of age. Women were classified according to their own occupation and not that of their partner. The following classes were included: professional and intermediate (classes I and II), skilled nonmanual and skilled manual (class III), and semiskilled and unskilled manual (classes IV and V).

Other factors. Social support was assessed via 6 questions on social relationships derived from the British Social Attitudes Survey²⁷; these questions were asked at the 33-year interview. Separate scales were constructed for emotional support (3 items) and practical support (3 items). For each scale, 1 or no positive responses indicated low support. Respondents reported the number of children they had had by 33 years of age. Life control was constructed as the sum of responses to 3 questions that provided alternative choices, one in the direction of control over life and the other in the direction of lack of control. Scores ranged from 0 (high control) to 3 (low control). Inactivity at 23 years was assessed from reports on frequency of sports activity and television viewing per week. Subjects with more than 5 episodes of television viewing and no sports activity were categorized as inactive.

Prework

Early life factors. Social class at birth was based on father's occupation (according to the 1950 registrar general's classification): class I or II, class III nonmanual, class III manual, class IV or V, and no male head of household. Somatization at 16 years of age was defined as a long-duration sickness absence (more than 1 month in the preceding year) from school without a medical condition identified as serious. Psychological status in childhood was indexed by measures of socioemotional functioning assessed by teachers at the ages of 7 years (via the Bristol Social Adjustment Score²⁸) and 16 years (via the Rutter Behaviour Scale²⁹). Transformed total scores (square root transformations) were grouped in 3 categories—well adjusted, intermediate, and poorly adjusted—and a combined (7- and 16-year) socioemotional status score was computed.

Physical factors. Obesity at 7 and 23 years of age was defined according to body mass index (BMI), calculated as weight in kilograms divided by height in meters squared. BMI at 7 and 23 years and height at 7 and 33 years were categorized according to percentile, as shown in Table 2.

The image is a small thumbnail of a table. The title of the table is "TABLE 2—Unadjusted Odds Ratios for Prework and Physical Factors Related to Incident Low Back Pain at 32–33 Years of Age: 1958 British Birth Cohort, 1990–1991 (n=5781)". The table has columns for "Factor", "No.", "Odds Ratio", and "95% CI". The rows include "Sex", "Social class at birth", "Somatization", "Psychological status", "Obesity", and "Height".

TABLE 2—

Unadjusted Odds Ratios for Prework and Physical Factors Related to Incident Low Back Pain at 32–33 Years of Age: 1958 British Birth Cohort, 1990–1991 (n=5781)

Data Analysis

We calculated unadjusted odds ratios for back pain in relation to the potential explanatory variables by comparing, using logistic regression, subjects with incident cases and subjects with no pain. Variables for which at least one category was associated (at $P < .05$) with an increased risk of back pain were retained for the multivariate analysis, which was conducted in 2 stages. In the first stage, variables were analyzed in subgroups (e.g., all early life factors were examined simultaneously but separately from all adult life factors). Nonsignificant variables at this stage were excluded from a second stage of analysis representing the full multivariate model. Sex, ergonomic stress, and psychosocial work characteristics were retained in this full model on a priori grounds.

Each variable was inspected (at the univariate stage) in order to collapse adjacent categories for which there were roughly equivalent risks and thereby maximize the statistical power of the multivariate analysis. To test whether some factors operated differently in men and women, we conducted univariate analyses separately by sex. In general, the relationships were similar, and hence results are presented for men and women combined (with adjustment for sex in the multivariate analysis).

In addition to sex, 9 variables were selected for the multivariate analysis. For the most part, correlations between these variables were rather low, ranging from 0.02 to 0.40; a majority (33 of 36) were below 0.2. The full multivariate model provided adjusted odds ratios for each of the selected variables and also indicated the adequacy of fit of the final model from which we calculated percentages of variance explained.

In all analyses described thus far, psychological distress was used as a dichotomous variable; however, we also tested whether the relationship between incident pain and number of psychological symptoms (continuous variable) was linear. Number of symptoms

was truncated at 10 because too few subjects reported symptom levels above this point. We used a quadratic term to assess linearity.

RESULTS

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Of subjects who were previously pain free, 9.9% reported incident pain at 32 to 33 years of age (11% of men and 8.8% of women). Tables 1 and 2 present, for each predictor variable, unadjusted odds ratios for recent incident back pain. Working life factors associated with an increased risk of low back pain (Table 1) included high level of exposure to ergonomic stress, high number of negative psychosocial work characteristics, job dissatisfaction and psychological distress at 23 years of age, early and continuing smoking, manual social class, less than an “A level” education, and moderate to low life control.

Prework predictors (Table 2) included manual social class at birth, poorer emotional status during ages 7 to 16 years, and high BMI (above the 70th percentile) at 23 years. No effect of child care factors emerged, and few sex differences were identified. Ergonomic stress and poor emotional status at 16 years appeared to have a stronger effect for men than for women; the reverse pattern was found for social class at birth and somatization.

Table 3 presents unadjusted odds ratios for the restricted sample with complete data on all predictor variables included in the multivariate analyses. Although Table 3 includes fewer variable categories than Tables 1 and 2, comparison of the unadjusted risk estimates suggests that few sample biases occurred despite the reduced sample available. Table 3 also presents adjusted risks from models of 3 groups of factors. Most risks attenuated slightly after adjustment, except for psychological distress at 23 years, which was largely unaffected by adjustment at this first multivariate stage.

**TABLE 3—
Adjusted Odds Ratios for Incident Low Back
Pain at 32–33 Years of Age: 1958 British Birth
Cohort, 1990–1991 (n=2773)**

Finally, Table 3 shows the full model in which all variables were included. In this model, elevated risks of incident low back pain remained for only 3 variables: sex, psychological distress at 23 years, and smoking (early and continuing). The increased risks for high ergonomic occupational exposure and low BMI (below the 15th percentile) were of borderline significance. Social class at birth and 23 years and socioemotional status between the ages of 7 and 16 years had weak or nonexistent effects in the full model. With a percentage of variance explained of 7%, the explanatory power of the model was poor; this was not entirely unexpected, however, for a model in which most variables were dichotomized.

In further analyses, we examined the relationship between number of psychological and somatic (malaise) symptoms reported at 23 years and incidence of low back pain at 32 to 33 years of age. There was a significant ($P < .05$) nonlinear relationship between pain incidence and psychological symptoms. Figure 1 shows that pain incidence increased with number of symptoms reported at 23 years, but the rate of

increase was greater for those reporting more than 7 symptoms.

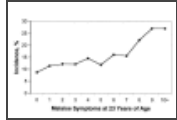


FIGURE 1—
Incidence of low back pain onset: 1958 British birth cohort.

DISCUSSION

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In this sample of individuals aged 33 years, we found strong predictive effects on low back pain onset of psychological status 10 years earlier and continued smoking. These risk factors were established for the 11% of men and 8.8% of women with incident low back pain at 32 to 33 years. These incidence rates are difficult to compare with rates from other studies because they apply to a specific age; however, another national survey reported that 9% of respondents aged 25 to 34 years had back pain in the previous year,³⁰ as compared with 5.6% of all subjects in the 1958 cohort at 33 years.

Our study was unique in its inclusion of risk factors from early life in relation to pain onset in early adulthood. Also, a major strength of the study was that many factors were recorded before pain onset, thus providing firmer evidence regarding the temporal sequence between putative risk factors and low back pain.³¹ This long-term perspective provides support for the emphasis in the literature on adult life risk factors.^{8,16}

It is generally accepted, however, that ergonomic factors, specifically biomechanical load on the spine, have an important influence on the development of low back pain,^{5,32,33} and our study failed to show a strong effect. We do not interpret our results as showing no effect of workload on the genesis of back pain, because we had no direct measure of load. Our measure did not even represent a direct self-report of physical load; rather, values were imputed from published estimates of general job exposures for broad occupational categories or entire industrial sectors.²⁵ Consequently, substantial misclassification of workplace physical load is to be expected, leading to underestimations of effects.

When examined separately, several early life characteristics were associated with elevated risks; however, their effects were eliminated when later life characteristics were included in the analyses (even though, in general, the correlations between predictors were low). This pattern directed our attention to continuities over time in individual characteristics. In particular, the present study provides evidence that certain factors carry risks from around the time of labor market entry. Presence of psychological distress at 23 years of age was associated with a more than 2-fold greater risk of low back pain onset, and this risk was largely unaffected by adjustment for other putative risk factors.

Moreover, we demonstrated that risk of pain onset was influenced by number of psychosomatic symptoms recorded, although risks were most pronounced among those at higher symptom levels. Psychological distress in early adulthood is in part determined by earlier life characteristics, such as poorer socioeconomic circumstances in childhood (as indicated by rented housing and

manual social class) and poorer school achievement and behavior,²⁰ as well as later experiences, such as early childbearing and single parenthood.³⁴ Thus, although early life factors did not independently predict back pain onset in this study, they contribute to psychological distress in early adulthood, which in turn affects risk of pain onset.

Alternative interpretations need to be considered in relation to psychological distress and back pain. It could be argued that our findings reflect negative affectivity; that is, individuals' emotional states influence their propensity to report back pain. Although we measured psychological distress prospectively, this characteristic exhibited a degree of stability, even over a 9-year period; thus, we are unable to discount the possibility that our findings in regard to psychological distress reflect negative affectivity. Confirmation of the relationship is therefore needed in studies involving a disability instrument specifically focusing on back pain.

Recent literature supports the biological plausibility of this relationship, however, even though there is little information on the specific pathophysiologic connections between emotional states and perception of pain in the lower back. According to the recent National Institute for Occupational Safety and Health review of work-related musculoskeletal problems, including low back pain, there is accumulating scientific evidence of connections between the brain and the locomotor system.⁵ In particular, it is suspected that perception of lack of well-being operating through the hypothalamic–pituitary–adrenal axis and the sympatho-adrenal medullary axis alters muscle tone and function, leading to a predisposition to injury.

Associations between psychosocial factors and back pain have been reported,^{8,10,13,16} but only rarely with prospectively obtained information.¹¹ One group of reviewers remarked on the limited research addressing the influence of psychological problems on the development of back pain, in contrast to the better evidence that psychological disorders exacerbate chronic back problems.⁸

The direction of association between psychological status and back pain is therefore not well established. It is pertinent to the issue of direction of association that our measure of psychological distress preceded onset of low back pain by several years, and all cases in which the direction of association may have been in question were removed from the analysis. Yet it might be argued that individuals' psychological states would not remain stable over this long an interval. If this were an issue, however, it would lead to random misclassification, which in turn would lead to underestimates of effects.

When other factors were taken into account, the risk associated with perceived life control was reduced and (although remaining elevated) no longer significant. No statistically significant effects were observed, even in univariate analyses, in regard to negative psychosocial work characteristics, as assessed by lack of learning opportunities, monotony,

inability to vary work pace, and lack of control over breaks. These work characteristics, conceptualized as indicating job strain, have been proposed as having adverse effects on health, particularly cardiovascular disease outcomes.²⁶

Research has been extended to musculoskeletal disorders, in that it has been suggested that high job demands increase muscle tension, which in turn could have an effect on musculoskeletal symptoms.³⁵ Some studies have implicated monotonous work and other aspects of control, but there is less convincing evidence for psychological work demands.^{5,8,9,33} Our results may have been affected by the fact that we assessed perceived life control and control at work only contemporaneously.

Smoking also had a predictive effect on low back pain incidence. This finding is consistent with findings of previous studies.^{12–14,17,25} Uncertainty exists as to whether smoking plays a causal role in the development of low back pain or whether it is an indicator of other factors affecting pain onset, such as psychosocial problems.^{12,16} A causal role would result from the direct physiologic effects of decreased arterial oxygen on muscles or from increases in carboxyhemoglobin or nicotine effects on receptors in the neuromuscular system.

On the other hand, smoking may represent “self-medication” for psychological distress. Subjects in the 1958 cohort who commenced smoking by 16 years and continued to smoke moderately or heavily to 33 years had an excess risk of back pain onset of 90% or more relative to those who had never smoked. An excess risk of 63% persisted after psychosocial factors (childhood socioemotional status and adult psychological distress, perceived life control, and psychosocial work characteristics) and other relevant risk factors had been taken into account. This would argue against the self-medication hypothesis.

Although further residual confounding cannot be discounted, the findings from the present cohort argue for a causal role of smoking in view of the prospective measurement of confounders such as social class on more than one occasion. Further research is needed to elucidate the probable mechanism through which smoking affects back pain risk.

Unlike most other studies to date, ours attempted to examine the effect of physical stress related to child care responsibilities. However, this variable was simply imputed from the number of children 16 years or younger living in the same household as the respondent at the 33-year follow-up. Therefore, it is perhaps not surprising that there was no increased risk of back pain associated with child care.

Because physical load would arise especially in relation to young

children, we repeated the analysis including only the number of children 5 years or younger, but there were no differences in the results. Additional research should attempt to better quantify physical stress due to child care and domestic work. With regard to physical inactivity, we found no effect of leisure-time inactivity at 23 years; however, given that our measure did not include activity at work, we may have underestimated the effect of this factor.

Consistent with other work on socioeconomic health differences in this cohort,³⁶ low back pain onset was more likely among subjects in lower social classes than among subjects in higher classes (the odds ratio at 23 years of age was 1.78 for classes IV and V relative to classes I and II) before adjustment for other factors. This finding is consistent with findings of previous studies specifically focusing on back pain.⁸ One previous investigation that involved smoking, BMI, and psychological characteristics as variables was unable to account for social differences in back pain.¹³

In the present analysis, adjustment for psychological distress and smoking reduced the odds ratio associated with social class from 1.61 to 1.32. Further adjustment for other factors eliminated the social class difference in incident pain (Table 3 ►). These findings are similar to results from a study of self-rated health conducted with this cohort in which class differences were largely explained.³⁷

A primary strength of the present study was the availability of prospectively collected data on a wide range of putative risk factors from birth through the age of 33 years. The restriction of the analysis to incident cases occurring in the year preceding the 33-year follow-up further strengthened the study. Indeed, very few studies¹⁷ have focused on risk factors for the onset of low back pain in early adult life, even though low back pain episodes occurring in later life frequently represent recurrences.^{33,38}

A limitation of our study design was the exclusion of approximately 43% of the 33-year sample who had suffered back pain before the age of 32 years. This raises the possibility that our risk factors do not apply to individuals with the most “frail” backs who contribute to the population burden. A second limitation was the 9-year interval between ascertainment of some of the risk factors at 23 years and onset of pain. However, exposure misclassification would be expected to lead to false-negative results or underestimated effect sizes. Hence, the estimates described here are likely to be conservative.

In conclusion, this prospectively studied cohort provides evidence that psychological distress at 23 years of age more than doubles the risk of incident low back pain several years later, with smoking having almost as great an effect. Until other prospective studies of similar scope provide independent confirmation, however, caution should be applied in drawing conclusions for preventive interventions. Future research on the genesis of low back pain will need to incorporate frequent, repeated measures of both outcomes and risk factors to advance the knowledge base.

Acknowledgments

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