Risks of Occupational Vibration Exposures

VIBRISKS

FP5 Project No. QLK4-2002-02650
January 2003 to December 2006

Annex 17 to Final Technical Report

Title: Whole-body vibration case-control study of low back pain and intervertebral disc pathology

Authors: Keith Palmer, Clare Harris, Michael J Griffin*, David Coggon, Isabel Reading, Nigel Arden, Madeleine Sampson**

Organisation: Medical Research Council Epidemiology Resource Centre
(Institute of Sound and Vibration Research*)
(Southampton University Hospitals NHS Trust**)
University of Southampton, U.K.

European Commission

Quality of Life and Management of Living Resources Programme
Key Action 4 - Environment and Health

13th December 2006

Annex 17: UK Case-control study of low back pain and intervertebral disc pathology: Associations with whole-body vibration

Keith Palmer, Consultant Occupational Physician and MRC Clinical Scientist

Clare Harris, Research Fellow

Michael J Griffin, Professor of Human Factors†

David Coggon, Professor of Occupational and Environmental Medicine

Isabel Reading, Statistician

Nigel Arden, Consultant Rheumatologist

Madeleine Sampson, Consultant Radiologist*

Medical Research Council Epidemiology Resource Centre, University of Southampton

† Institute of Sound and Vibration Research, University of Southampton

* Southampton University Hospitals NHS Trust, University of Southampton

Background

Low back pain (LBP) is common and an important cause of disability, but its aetiology is not fully understood. Epidemiological studies have implicated whole-body vibration in the occurrence of LBP,[1-7] alongside a list of many other physical, constitutional, and psychosocial risk factors (e.g. manual work, occupational lifting and twisting, forward bending and stooping, prolonged sitting, age, sex, taller stature, smoking, low mood, low job satisfaction and limited control over job demands);[1,8-11] but these do not explain all of its descriptive epidemiology. In particular, the striking time trends in disability attributed to LBP in some countries[11] do not seem to have been accompanied by a corresponding change in the prevalence of known risk factors.[12]

One of the difficulties in epidemiological investigation of LBP is the uncertain pathogenesis of most cases and the lack of objective diagnostic criteria.[13] As a consequence, case definition in most studies has been based on subjective report of symptoms. Inevitably this opens up the possibility for bias in the assessment of risk factors. For example, people whose work is physically demanding may be more aware of back symptoms and report them more readily.

An exception to the generally poor understanding of pathogenesis is prolapsed intervertebral disc (PID). PID can occur in the absence of back symptoms, and the coincidence of PID and back pain does not necessarily imply that the PID gave rise to the pain. Nevertheless PID, and related pathology such as tears of the posterior annulus, appear to account for a substantial minority of back pain cases.[14]

In the past, opportunities to study PID epidemiologically were limited because the disorder could only be diagnosed confidently by invasive investigations or at surgery. A classical case-control study, conducted by Kelsey and colleagues in the US during the 1970s,[3] provides an example of the limitations. This focussed mainly on the risks arising from whole-body vibration (WBV). Cases of PID, ascertained from radiology lists and confirmed by clinical interview, were compared with matched controls attending the same services. Subjects were asked about the time spent sitting during the job in which symptoms developed, and whether or not they drove motor vehicles for more than half the time or had a job title of truck driver. Elevated relative risks were found for prolonged driving (2.75, $P = 0.02$), and for being a truck driver 4.67 ($P < 0.02$), although the risk for being more sedentary than the paired control was also increased (1.58 overall, and 2.40 in those aged over 35). However, these investigations were conducted before the benefits of modern imaging technology were available to define the disc pathology, and before the importance of psychosocial factors to presentation was fully appreciated. Case definition was based on clinical impression (sometimes supplemented by observations at surgery). Also, the characterisation of certain physical exposures and confounders was limited. The role of potential confounders such as material handling (in loading and unloading vehicles)

was not considered in detail; the characterisation of exposure to WBV was more limited than modern methods of assessment; no allowance was made for the role of psychosocial factors in reporting and presentation; and the cases analysed included some drivers whose symptoms had followed on acutely from road traffic accidents.

More recently the advent of magnetic resonance imaging (MRI) has meant that PID and related disc pathology can now be diagnosed more easily, and, as indicated, there is greater understanding of the other risk factors that need to be considered. Given the paucity of epidemiological data on risk factors for objectively diagnosed PID and related disc pathology, a case-control study based on cases identified through MRI was undertaken.

Objectives

To investigate the risk factors (physical, psychosocial and constitutional) that underlie LBP presenting to radiology services, and to compare them among subjects with and without MRI evidence of PID or related disc pathology.

One particular focus was to assess, taking into account various potentially confounding exposures, how strongly the occurrence of LBP severe enough to merit MRI investigation is associated with exposure to WBV, and to which metrics of dose.

Methods

A case-control approach was adopted.

Study population

The study population comprised all men and women aged 20-64 years who were normally resident in the catchment area served by the radiology services at Southampton General Hospital - specifically, those living in Southampton, including Hedge End and West End, The Waterside area, Eastleigh, and Totton (UK postcodes SO1$ (wild-card), SO30, SO31, SO40, SO45 and SO50).

Cases and controls

Cases were a consecutive series of patients from the study population who had been referred to the radiology department at Southampton General Hospital (SGH) or to the private BUPA Southampton or Wessex Nuffield Hospitals for MRI because of LBP between 1st November 2003 to 1st September 2006 (approximately 30 months of recruitment in each hospital).

Subjects who had imaging of the spine because of external trauma or had non-mechanical pathology as the cause of LBP (e.g. cancer, metabolic bone disease, infections, previous back surgery, congenital disorders, ankylosing spondylitis) were excluded.

On a monthly basis, cases from SGH were recruited by:

- obtaining an Excel file from the SGH radiology information manager listing the name, address, sex, date of birth (dob), name of referring doctor, hospital number, episode number and date seen of each participant aged 20 – 64 years who had had an MRI scan of the lumbar spine in the previous month;
- selecting those subjects with qualifying address and referral from a doctor in rheumatology or trauma and orthopaedics who had provided written consent to the study (no subject was excluded for this reason);
- checking X-ray film envelopes in the library for referral card and report, making exclusions on the basis of this information and marking film envelopes as of interest to this study to prevent destruction in the future. Cases with missing film envelopes were checked on the computerised Radiology Management System (RMS) for the whereabouts of the envelope and the library was periodically checked for their return. Cases with possible reasons for exclusion/inclusion were checked by collaborating consultant rheumatologist, Dr Nigel Arden;
- checking all selected cases on the RMS for vital status and for any information missing from the film envelopes;
- providing a list of all selected cases to the radiology office staff for printing copies of reports;
- entering details of all selected cases to the MRC database from the Excel file originally provided;
- preparing batches of covering letters on joint MRC/SGH headed paper to the selected cases, countersigned by the collaborating consultant radiologist;
- mailing these to the cases with an accompanying questionnaire about personal characteristics, symptoms, disability and risk factors.

On a monthly basis, cases from BUPA Southampton and Wessex Nuffield Hospitals were recruited by:

- obtaining a list of names of subjects having an MRI of the lumbar spine in the previous month, from the MRI manager at the Wessex Nuffield Hospital and from the Diagnostic imaging manager at BUPA Southampton. Details of names, addresses, sex, dates of birth (dob), name of referring doctor, cause of referral and date seen of all subjects were then located by one of us (Clare Harris);
- selecting from among them subjects aged 20–64 years with qualifying places of residence and referral from a doctor in rheumatology or trauma and orthopaedics who had provided written consent to the study;

- checking X-ray referral cards and reports, making exclusions on the basis of this information and, for the cases selected, obtaining a copy of the report and obtaining copies of films or ensuring their availability for later evaluation (films not kept by the hospitals as they are the property of the patients);
- entering details of all selected cases to the MRC database;
- preparing batches of covering letters on joint private hospital/MRC headed paper to the selected cases, countersigned by the collaborating consultant radiologist;
- mailing these to the cases with an accompanying questionnaire about personal characteristics, symptoms, disability and risk factors.

Controls were subjects from the Accident and Emergency Department at SGH who had undergone radiological examination during the recruitment period. Those who had had X-rays of the lumbar spine or serious incapacitating illness were excluded. Controls were also excluded if they fulfilled the exclusion criteria applied to the cases. Eligible controls fulfilled the same residency requirement as cases and were group matched to them by sex and 5 year age bands. This strategy identified patients with a mix of diagnoses.

On a monthly basis, a table of male and female cases by 5-year age bands was produced and group matched controls (in the ratio 3:1) were recruited for these from the radiology casualty referral/report cards. The process entailed:

- selecting from among them the first three subjects who matched each case by age (5-year bands), sex, and qualifying place of residence;
- checking the referral card and report and making exclusions on the basis of this information;
- for the controls selected, recording name, sex, area of residence, postcode, date of birth, hospital number, date seen, cause of referral, X-ray procedure and report information;
- checking all selected controls on the records management system for vital status;
- producing an Excel file with details of all the controls selected for entering to the MRC database;
- preparing batches of covering letters to all selected controls, countersigned by the consultant radiologist;
- mailing these to the controls with an accompanying questionnaire about personal characteristics and risk factors.

Sample letters and information sheets have been published previously: (http://www.vibrisks.soton.ac.uk/members/documents/D8a%20WBV%20case-control%20year%203%20(Jan06).pdf).

Postal questionnaire

The questionnaire was based on and contained the key model elements of the VIBRISKS questionnaire, developed within the EU consortium. However, certain modifications were made necessary because of differences in study design (case-control vs. cohort) and in the source of sampling.

Cases were asked about their history of LBP/sciatica, and their current disability. The questionnaire ascertained the 1-year, 4-week, and 1-week period prevalence of LBP and sciatica, and assessed recent back pain using the Roland-Morris disability questionnaire[15] and a Visual Analogue Scale of pain intensity. Information was also collected on time off work and health care received because of LBP, duration of symptoms, and any acute precipitating events thought to underlie their onset.

Controls were also asked about their history of LBP/sciatica, including whether they had ever had a scan or surgery to their back because of LBP (subjects who reported these events were excluded).

Both **cases and controls** were also asked about:

- all jobs held for more than a year;
- potential risk factors which loaded the back (e.g. lifting, digging, posture while lifting, twisting, bending and stooping, sitting);
- professional driving (vehicle types and duration);
- personal characteristics (e.g. height, weight, age, sex, smoking habits);
- mental health (low mood, somatising tendency), health beliefs and illness behaviour.

Measuring instruments and their coding

The questionnaires for cases and controls can be found in full as follows:

Link to case questionnaire (working document No. WP4-N7):
http://www.vibrisks.soton.ac.uk/members/documents/WP4-N7%20CaseControlQuestionnaireCases%20(03Mar04).pdf

Link to control questionnaire (working document No. WP4-N8):
http://www.vibrisks.soton.ac.uk/members/documents/WP4-N8%20CaseControlQuestionnaireCntrls%20(03Mar04).pdf

Low mood was assessed using the Mental Health Section of the SF-36 (SF-36 MH),[16] with subjects categorised into bands (best, intermediate, worst) according to approximate thirds of

the distribution of scores across all subjects. Somatising tendency was assessed using elements of the Brief Symptom Inventory,[17] a validated self-reported measure of distress comprising items on bothersome nausea, faintness, dizziness, weakness, numbness in the body, chest pain and breathing difficulties during the past 7 days. The number of symptoms reported as 'extremely', 'quite a lot' or 'moderately' distressing were summed and the data analysed in three bands (0, 1, ≥2 distressing symptoms).

Questions on health beliefs focussed on three separate areas: fear-avoidance, beliefs about work as a cause of LBP, and propensity to consult over LBP. Fear-avoidance beliefs were assessed according to a series of statements about what to do in the event of LBP (e.g. physical activities should be avoided as they might make the pain worse, normal work should be avoided until the pain is treated, rest is needed for LBP to get better). These were based on the validated Fear-Avoidance Beliefs scale of Waddell *et al*.[18] A sum was made of the number of statements with which the respondent agreed. Three questions were also asked about work as a cause or aggravation of LBP and two questions on attitudes to consulting (whether it was important to see the doctor straightaway at the first sign of trouble, whether neglecting problems of this kind could lead to permanent health problems). In each case a sum was made of the number of items of agreement.

Questions on psychosocial risk factors at work were based on the Karasek demand-control-support model,[19] with subjects being subdivided according to decision latitude (three bands) and support (three bands), as well as according to self-reported job satisfaction (two bands).

In other coding decisions, subjects were classified by age in three bands, by height (approximate thirds for the distribution for all subjects), and body mass index (according to the National Heart Lung and Blood Institute BMI categories for underweight, normal weight, overweight and obesity). A count was also made of the number of other sites (knees, hips, shoulders, neck, wrist/hand, elbows) with pain lasting a day or more in the past four weeks, with subjects classified in three bands (0, 1 - 2, 3-6).

Physical activity in the current or most recently held job was assessed by a series of questions about digging (yes vs no), work with the arms above shoulder height (hours/day), lifting ≥20 lbs (times/day, times/day with back twisted), bending the trunk (hours/day, times/day), twisting (hours/day, times/day), standing (hours/day) and sitting while not driving (hours/day).

Exposure to WBV was assessed according to six metrics: (1) professional driving for ≥1 hour/day; (2) professional driving ≥3 hours at a time; (3) average weekly hours driven for the

commonest exposure source (in three bands: none, <16, >16); (4) average weekly hours driven for all exposure sources (in five bands); (5) maximum r.m.s. of any machine (three bands: 0, -6, $\geq$6 ms^{-2}) and (6) current r.m.s. A(8) (0, -0.5, -1.15, >1.15 ms^{-2}). To establish these last three metrics, questions were asked among professional drivers about the number of hours and minutes driven in a typical week for each of a list of vehicles as well as for an open category. Externally acquired estimates of vibration magnitude for the various commonly reported sources of exposure were applied as necessary to calculate exposures that included a component of vibration magnitude. The approach to exposure assessment builds on previously developed methodology,[20] with dose measures estimated according to standard methods proposed by the EU consortium (see Appendix 1 and WP4-N14),[*] with the exception of lifetime cumulative dose.

All questionnaire responses were checked for completeness, double-entered onto computer and cross-compared to detect errors of input; then subjected to range and consistency checks to detect improbable values. A note was made of any data cleaning and recoding decisions.

Imaging

For **cases**, images of the lumbosacral spine have been obtained according to routine departmental practice. Briefly, patients were placed on the spinal coil and scanned from the thoraco-lumbar junction to the mid sacrum; sagittal T1 weighted images were obtained using a coronal localiser, followed by sagittal and axial high resolution T2 images angled through the lower 3 discs using a fast spin echo; 9 sagittal sections with 5 mm width and 1 mm interspacing were acquired, covering both exit foramina and facet joints.

Two hundred and thirty-three scans from SGH have been located in the X-ray library and the envelopes marked to identify those to be retained for study. Copies of a further 148 scans performed at the private hospitals have been stored digitally and copied to CD-ROM for the MRC. These scans are currently being read by trained observers (specialists in radiology) who will be unaware of the patient's employment and exposure history, and other aspects of their questionnaire responses.

The aim will be to sub-classify cases according to the presence or absence of pathology that may give rise to LBP – specifically: disc herniation (disc impairment, disc bulge, disc protrusion and disc extrusion); disc degeneration (e.g. loss of disc height, endplate changes); nerve root

[*] http://www.vibrisks.soton.ac.uk/members/documents/WP4-N14%20(Calculation%20of%20dose%20for%20WBV%20-%2021%20Sept%202005).pdf

impingement and compression; high intensity zones and posterior annular tears; facet joint arthropathy; Schmorl's nodes; spondylolisthesis; and spinal canal stenosis. In part this will involve comparing a sagittal image of the spine with a previously validated and standardised picture atlas[21] to score for each of the L3 to L5 discs. Pilot work has been undertaken to define a suitable framework of operational definitions[22-28] and to train observers in a common methodology (see Appendix 2). The intra- and inter-observer repeatability of the atlas-based method is considered high (e.g. Kappa statistics of 0.70 to 0.72 for disc protrusion[29]); there is also evidence more generally that herniated/bulging discs,[30] nerve root compression,[27,30] disc degeneration,[27] and presence of a high intensity zone (early annular tear)[31] can be ascertained in a reproducible way. However, we plan that a random sample of 100 images (in random order) are read a second time by a trained observer, and also by a second blinded observer to confirm the within- and between-observer repeatability in our hands.

Outcomes

Eventually we will explore associations with WBV according to the presence or absence of specific findings on MRI. These results will not be available by February 2007.

Thus, for this report, two outcomes are used as the focus of analysis: (1) being a case – i.e. being referred for MRI imaging of the lumbar spine because of LBP; (2) being in the most disabled half of cases as assessed by Roland Morris Score ($\geq$ the median value of 11).

Analysis

Analysis was restricted to cases whose current episode of LBP came on in their current or most recent job and to controls who gave a current or most recent job history.

Associations of each potential risk factor with the two outcomes were examined initially in an analysis that adjusted for age and sex (which were factors of group matching and recruitment).

A process of stepwise logistic regression was then used to select a set of non-driving risk factors to be considered in multivariable analyses. This process was performed separately for the two outcomes with models explored using both forward and backward selection methods. Age and sex were selected *a priori* and forced to enter all of the models. In backward selection, variables were then dropped sequentially from a fully saturated model, the criterion being $P<0.05$ (with the least significant variable being deselected in each round until all were significant or *a priori* choices); in forward selection, variables were added sequentially to the model, the criterion for inclusion being $P<0.06$, and the choice at each stage representing the variable with the most significant association. (Both procedures generated the same list of potential confounders in relation to each outcome, but slightly different lists between outcomes.)

In the final stage, risk factors other than driving were identified by stepwise regression and added as factors of adjustment to a multivariable model that included a driving variable as a forced choice. Separate multivariable models were constructed for each of the six metrics of exposure to WBV.

Additionally, sensitivity analyses were conducted. Cases were recruited from private hospitals, but not controls; in case this selection strategy over-represented white-collar jobs among cases, analyses were re-run after exclusion of private cases.

Associations were expressed as odds ratios (OR) with 95% confidence intervals (95% CI). All analyses were performed using Stata 9.2 software.

Results

Altogether, 743 cases and 2,268 controls were approached. Usable replies were received from 385 (52%) of the cases and 965 (43%) of the controls. Reasons for non-response included moving away (45 subjects), malignancy (one subject), and mental handicap (seven subjects). Among the remainder, four cases were excluded because they did not report LBP on questionnaire, seven cases were ineligible because of previous surgery to the back, a further 67 cases were excluded because their LBP began before their current/most recent job and 36 cases did not have a job. Among the controls, 96 were excluded because they had previously had either a scan or surgery to the back and 59 did not report a current or recent job. Thus, a total of 271 cases and 809 controls were included in the analysis.

Among the cases, the median duration of LBP was 10 years (IQR 2 - 19), 67% reported taking time off work in the last year because of symptoms, and 84% reported sciatica (pain spreading down the leg to below the knee or causing distal neurological symptoms). The median Roland Morris score (RMS score) for the past four weeks was 11 (IQR 5 - 17).

Table 1 describes the distribution of demographic and personal characteristics in cases and controls and the associations with outcome overall, and among the more severe cases (Roland Morris Score $\geq$ 11).

Strong associations were seen with somatising tendency, SF-36 MH score and belief in work as a cause or aggravation of LBP. Thus, for cases overall, the OR was raised 3.8 fold in those who reported $\geq$2 somatic symptoms distressing vs. none, and raised 1.8 - 1.9 fold in those with low mood or attributing symptoms to work; and among the severe cases, associations were much

stronger again (12.5 and 3.5 - 5.4 respectively). Associations were also seen with tall stature (OR 1.6) and propensity to consult over LBP (OR 1.8 - 2.0), but of similar magnitude for the two outcome definitions. BMI, smoking status and fear avoidance beliefs were also associated with low back pain, but only among the more severe cases.

In comparison with personal risk factors, occupational risk factors (Table 2) were only weakly associated with low back pain, and in many cases risks were non-significantly elevated or close to the null value. For back pain overall, however, there was a non-significant association with frequent twisting of the back (OR 1.4) and a significant association with sitting for ≥3 hours while not driving (OR 2.0), and similar associations were also seen among the more severe cases. Finally, there were significant associations with low decision latitude (OR 1.3 - 2.1) and low support (OR 1.5 - 1.7).

The study included 200 professional drivers (54 cases and 146 controls), and of these 175 reported driving a single vehicle occupationally: the predominant exposure was to cars (124 reports), there being also 24 lorry drivers, seven bus drivers, nine drivers of forklift trucks, and seven ambulance drivers. The median weekly exposure time for drivers was 16 hours (IQR 10-30 hours), and the median A(8) was 0.79 (0.31-3.0) m/s^{-2}, but the upper interquartile limit when considered across the whole sample was zero for both parameters.

Few positive associations were seen between the six metrics of whole-body vibration and the two case outcomes (Table 3). Professional driving for ≥3 hours at a time was non-significantly associated with a higher odds for LBP overall (1.3) and for severe LBP (OR 1.5); and a non-significant increase in relative risks was found in the band with A(8) ≥0.5–1.15 vs. 0 ms^{-2} (OR 1.2 and 1.4), but no finding was significant at the 5% level and no exposure metric showed an exposure-response pattern.

These figures were adjusted for age and sex. Stepwise regression identified several other non-driving factors that were candidates for a multivariate model as indicated in Table 4. Associations with somatising tendency, beliefs about work as a cause of LBP, number of other sites with pain, and sitting while not driving, all tended to be stronger in the finally selected models.

Table 5 records adjusted risk estimates for driving and WBV after allowing for the factors in Table 4. Associations with professional driving for ≥3 hours at a time were weakened (OR 1.1 vs 1.3–1.5); other associations were not much changed, the only non-significant positive associations being with A(8) $\geq$ 0.5–1.15 ms^{-2} and driving 3-10 vs. 0 hours per week

professionally Again, no association was significant at the 5% level and no evidence was found of an exposure-response relation.

When we repeated the analysis after excluding cases from the private hospitals (n=117) then a broadly similar pattern of results was obtained. Significant univariate associations for both outcomes were seen with lifting, bending, twisting, beliefs about work as a cause of LBP, somatisation, SF-36 MH and propensity to consult about LBP; and also significant associations with sitting and fear-avoidance beliefs for LBP overall, and with smoking, decision latitude and number of other sites with pain for RMS $\geq$11. No metric of WBV showed a significant univariate or multivariate association and in only one comparison by A(8) ($\geq$ 0.5-1.15 vs. 0 m/s^{-2}) was an OR elevated (OR 1.5 for severe cases vs. controls). There was no evidence of an exposure-response relationship.

Discussion

As judged by these findings there are strong positive associations between severe LBP referred for imaging of the lumbar spine and somatising tendency, low mood, certain beliefs about LBP and consulting propensity, as well as moderate positive associations with being tall, smoking, and work involving: frequent or prolonged twisting, sitting while not driving, low decision latitude and poor support from colleagues or managers. Beyond this there was very little evidence of a risk from exposure to professional driving or WBV.

In weighing the findings a number of limitations need to be considered. Response was incomplete. This would be a source of bias in relation to questions about WBV only if non-responders had different associations with professional driving from responders; we have no reason to expect this.

A more important limitation is that, among cases, the exposure history came after the occurrence of LBP. The relevant exposures are those that precede onset of symptoms, but the most reliable and complete information came from the most recent or currently held job. Bias could arise if workers with LBP developed symptoms in driving jobs but then moved to work with lesser exposure because of symptoms ('healthy worker selection bias'). Assessing this bias is challenging in practice, as LBP often begins early on in adulthood,[32] sometimes before employment begins,[33] and then runs a relapsing and recurrent course.[11] Defining an exposure that predates symptoms is potentially arbitrary, while the distinction between WBV as an initiating factor as compared with a factor of aggravation is also less than straight forward; a censoring of recent exposure experience for cases would need to be mirrored by a censoring for controls. In practice, we focussed on the *current* episode of LBP and asked when this began,

and limited analysis to cases whose symptoms began in the current or most recent job, comparing their exposure to controls reporting a current/recent job. While it remains possible that some drivers reduced their exposure but remained within the same job, we consider the scope for this to be more limited than for a change of occupation; and no such selection was evident in relation to frequency and duration of occupational twisting.

Assessing exposures after the event has the potential also to inflate some risk estimates through reverse causation. Thus, low mood could arise as a consequence of severe LBP rather than causing it. However, it seems implausible that some exposures with positive associations could be influenced in this way – e.g. twisting, height, tendency to somatise.

As mentioned previously, although care was taken to ensure that cases and controls came from the same catchment areas, some cases were recruited from private hospitals whereas all controls came from public sector hospitals. To ensure that selection bias did not arise in relation to occupational activities (e.g. systematic over-representation of white-collar jobs among cases relative to controls), we conducted a sensitivity analysis investigating the impact of excluding private cases, and found this has no major impact on the findings**.** Selection bias could also arise if professional drivers had less ready access than their non-driving peers to MRI scan facilities; we have no reason to expect this however.

Another challenge lies in the assessment of exposures to WBV and other potential confounders. Estimates of dose rely on self-assessed exposure times and imputed values of vibration dose from other field observations. There is evidence, however, that professional drivers make a reasonably accurate assessment of their exposure times.[34] Moreover, it seems unlikely that there would be much misclassification of an exposure metric such as professional driving for $\geq$ 3 hours at a time. In our data set, sitting while not driving appeared to be a potentially relevant confounder, but adjustment did not have a big impact on the associations observed, which were close to the null value even in crude analyses.

Our failure to observe clear relations between LBP and WBV is at variance with several other research reports and reviews.[1-10] One explanation, given the relatively low prevalence of professional driving in our study population (18.5% overall), is that an effect was missed by chance. The upper confidence intervals for risk estimates did not exclude a doubling of risk from professional driving for $\geq$ 3hours at a time at the 5% level, although this is not a likely possibility, and the absence of any exposure-response effect tends also to argue against this explanation. A second possibility is that the drivers in our study - representing a population-based sample - were less heavily exposed to WBV than in surveys of occupational cohorts. Most were drivers of cars, with relatively few other sources of exposure reported. Associations with car driving have

been reported in several earlier surveys,[1-3,35] but there are also some contrary observations in general population-based samples.[36,37] In only 1 in 5 to 6 of our study subjects was the A(8) $\geq$ 0.5 m/s^{-2} and in only 1 in 20 $\geq$ 1.0 m/s^{-2}; in comparison, in certain positive studies from occupational settings average exposure levels were around 0.5 m/s^{-2} in crane drivers[4,5] and helicopter pilots,[6] 0.8 m/s^{-2} in lift truck drivers,[38] and 0.7-1.0 m/s^{-2} in tractor drivers,[39] and drivers of wheel loaders and freight containers.[40] Our findings on sitting while not driving raise a third possibility – that previously reported associations with WBV were confounded by constrained sitting, a characteristic ingredient of professional driving. A number of positive associations with sitting while not driving have been reported also in the wider literature,[8,9,41,42] but findings have been modest and not wholly consistent.[9,38] A fourth possibility is that WBV is generally associated with mild to moderate LBP, but not the severe kind studied here. (The studies by Kelsey et al[1,3] focussed on surgically-treated PID: a particular focus, when our findings on MRI imaging are available, will be on whether associations with WBV are any stronger in the subset of cases with PID or other structural abnormalities).

Whichever the explanation, our findings suggest that at the population level WBV is not an important cause of LBP severe enough to be referred for MRI imaging of the lumbar spine. Certain aspects of mental health and health beliefs (psychological factors) make a more important contribution.

References

1. Kelsey JL. An epidemiological study of acute herniated lumbar intervertebral discs. Rheumatol Rehab 1975;14:144-158.

2. Backman AL. Health survey of professional drivers. Scand J Work Environ Health 1983;9:30-35.

3. Kelsey JL, Githens PB, O'Conner T et al. Acute prolapsed lumbar intervertebral discs: an epidemiological study with special reference to driving automobiles and cigarette smoking. Spine 1984; 6: 608-613.

4. Bongers PM, Boshuizen HC, Hulshof CTJ, Koemeester AP. Back disorders in crane operators exposed to whole-body vibration. Int Arch Occup Environ Health 1988;60:129-137.

5. Bongers PM, Boshuizen HC, Hulshof CTJ, Koemeester AP. Long-term sickness absence due to back disorders in crane operators exposed to whole-body vibration. Int Arch Occup Environ Health 1988;61:59-64.

6. Bongers PM, Hulshof CTJ, Dijkstra L et al. Back pain and exposure to whole body vibration in helicopter pilots. Ergonomics 1990; 8: 1007-1026.

7. Bovenzi M, Zadini A. Self-reported low back symptoms in urban bus drivers exposed to whole-body vibration. Spine 1992;17:1048-1059.

8. Garg A, Moore JS. Epidemiology of low-back pain in industry. Occup Med State of the Art Reviews 1992;7: 593-608.

9. Riihimaki H. Back and limb disorders. In: McDonald C ed. Epidemiology of Work-related Diseases. BMJ Publishing Group: London 1995.

10. Burdorf A, Sorock G. Positive and negative risk factors for back disorders. Scand J Work Environ Health 1997;23: 243-256.

11. Clinical Standards Advisory Group. Back Pain: Report of a CSAG Committee on Back Pain. HMSO: London, 1995.

12. Coggon D. Occupational medicine at a turning point. Occup Environ Med 2005; 62: 281 - 283.

13. Lutz GK, Butzlaff M, Schultz-Venrath U. Looking back on back pain: trial and error of diagnoses in the 20th century. Spine 2003;28:1899-1905.

14. Hurri H, Karppinen J. Discogenic pain. Pain 2004; 112:225-8.

15. Roland M, Morris R. A study of the natural history of low-back pain. Part II: development of guidelines for trials of treatment in primary care. Spine 1983;8:145-50.

16. Jenkinson C, Coulter A, Wright L. Short form 36 (SF 36) health survey questionnaire: Normative data for adults of working age. BMJ 1993;306:1437-1440.

17. Derogatis LR, Melisaratos N. The brief symptom inventory: an introductory report. Psychol Med 1983;13:595-605.

18. Waddell G, Newton M, Henderson I, et al. A Fear-Avoidance Beliefs Questionnaire and the role of fear-avoidance beliefs in chronic low back pain and disability. Pain 1993;52:157-168.

19. Karasek RA. Job demands, job decision latitude, and mental strain: implications for job redesign. Adm Sci Q 1979; 24: 285-307.

20. Palmer KT, Griffin MJ, Bendall H, Pannett B, Coggon D. The prevalence and pattern of occupational exposure to whole-body vibration in Great Britain: findings from a national survey. Occup Environ Med 2000; 57: 229-236.

21. Jarosz J, Bingham JB, Pemberton J, Sambrook PN, Spector TD. An atlas for scoring cervical and lumbar disc degeneration. London: Springer Verlag, 1997.

22. Weishaupt D, Zanetti M, Hodler J, Boos N. MR imaging of the lumbar spine: Prevalence of intervertebral disk extrusion and sequestration, nerve root compression, end plate abnormalities and osteoarthritis of the facet joints in asymptomatic voulunteers. Radiology 1998;209:661-666.

23. Aprill C, Bogduk N. High-intensity zone: a diagnostic sign of painful lumbar disk on magnetic reonance imaging. British Journal of Radiology 1992;65:361-369.

24. Modic M, Steinberg P, Ross J, Masaryk T, Carter J. Degenerative disk disease: Assessment of changes in vertebral body marrow with MR imaging. Radiology 1988;166:193-199.

25. Boos N, Rieder R, Schade V, Spratt KF, Semmer N, Abei M. The diagnostic accuracy of magnetic responance imaging, work perception, and psychosocial factors in identifying symptomatic disc herniations. Spine 1995; 20: 2613-25.

26. Brant-Zawadzki M, Jensen M, Obuchowski N, Ross J, Modic M. Interobserver and intraobserver variability in interpretation of lumbar disk abnormalities: A comparison of two nomenclatures. Spine 1995;20:1257-1264.

27. Pfirrmann CW, Dora C, Schmid MR Zanetti M, Hodler J, Boos N. MR image-based grading of lumbar nerve root compromise due to disk herniation: reliability study with surgical correlation. Radiol 2004; 230: 583-8.

28. Ross J, Brant-Zawadski M, Moore K, Crim J, Chen M, Katzman G. Diagnostic imaging: Spine. Amirsys 2004.

29. Sambrook PN, MacGregor AJ, Spector TD. Genetic influences on cervical and lumbar disc degeneration. A Magnetic Resonance Imaging study of twins. Arth Rheum 1999; 42:366-372.

30. Van Rijn JC, Klemetso N, Reitsma JB et al. Observer variation in the evaluation of lumbar herniated discs and root compression: spinal CT compared with MRI. B J Radiol 2006; 79: 372-7.

31. Smith BM, Hurwitz EL, Solsberg D et al. Interobserver reliability of detecting lumbar intervertebral disc high-intensity zone on magnetic resonance imaging and association of high-intensity zone with pain and annular disruption. Spine 1998; 23: 2074-80.

32. Harkness EF, Macfarlane GJ, Nahit ES, Silman AJ, McBeth J. Risk factors for new-onset low back pain amongst cohorts of newly employed workers. Rheumatol 2003; 42:959-68.

33. Jones GT, Macfarlane GJ. Epidemiology of low back pain in children and adolescents. Arch Dis Child 2005; 90:312-6.

34. Palmer KT, Haward B, Griffin MJ, Bendall H, Coggon D. Validity of self-reported occupational exposures to hand-transmitted and whole-body vibration. Occup Environ Med 2000;57:237-24.

35. Pietri F, Leclerc A, Boitel L, Chastang J, Morcet J, Blondet M. Low-back pain in commercial drivers. Scand J Work Environ Health 1992;18:52-58.

36. Palmer KT, Griffin MJ, Syddall HE, Pannett B, Cooper C, Coggon D. The relative importance of whole-body vibration and occupational lifting as risk factors for low-back pain. Occup Environ Med 2003;60:715-721.

37. Xu Y, Bach E, Orhede E. Work environment and low back pain: the influence of occupational activities. Occup Environ Med 1997;54:741-745.

38. Boshuizen HC, Bongers PM, Hulshof CTJ. Self-reported back pain in forklift truck and freight-container drivers exposed to whole-body vibration. Spine 1992; 17: 59-65.

39. Bovenzi M, Betta A. Low-back disorders in agricultural tractor drivers exposed to whole-body vibration and postural stress. Applied Ergonomics 1994: 25: 231-241.

40. Bongers P, Boshuizen H. Back disorders and whole-body vibration at work. CIP-Gegevens Koninklijke Bibliotheek, Den Haag, 1990, ISBN 90-9003668-7.

41. Andersson GBJ. Epidemiologic aspects on low-back pain in industry. Spine 1981; 53-60.

42. Skovron ML. Epidemiology of low back pain. Balliere's Clinical Rheumatology 1992; 6: 559-573.

Table 1: Associations with demographic and personal characteristics in the study group

	Cases (n, %)				Controls (n, %) (n=809)		OR (95% CI)*			
	All (n=271)		RMS ≥11 (n=138)				All cases vs controls		RMS ≥11 vs controls	
Gender										
Male	139	(51.3)	69	(50.0)	374	(46.2)				
Female	132	(48.7)	69	(50.0)	435	(53.8)				
Age (years)										
20 - 34	58	(21.4)	32	(23.2)	170	(21.0)	All factors adjusted for age and sex			
35 - 49	131	(48.3)	71	(51.5)	344	(42.5)				
50 - 64	82	(30.3)	35	(25.4)	295	(36.5)				
Height										
Shortest third	82	(30.3)	43	(31.2)	308	(38.1)	1.0		1.0	
Middle third	75	(27.7)	34	(24.6)	221	(27.3)	1.3	(0.9 - 1.8)	1.1	(0.7 - 1.8)
Tallest third	107	(39.5)	58	(42.0)	260	(32.1)	1.6	(1.1 - 2.2)	1.6	(1.0 - 2.5)
Body Mass Index										
≤18.5	99	(36.5)	43	(31.2)	310	(38.3)	1.0		1.0	
18.5 - 24.9	100	(36.9)	51	(37.0)	294	(36.3)	1.0	(0.7 - 1.4)	1.2	(0.8 – 1.9)
≥30	53	(19.6)	33	(23.9)	166	(20.5)	1.0	(0.7 - 1.5)	1.5	(0.9 - 2.5)
Smoking status										
Never	123	(45.4)	45	(32.6)	355	(43.9)	1.0		1.0	
Ex	78	(28.8)	44	(31.9)	242	(29.9)	0.9	(0.7 - 1.3)	1.5	(0.9 - 2.3)
Current	69	(25.5)	49	(35.5)	204	(25.2)	1.0	(0.7 - 1.3)	1.8	(1.2 - 2.8)
Somatising tendency *(no of distressing symptoms)*										
0	63	(23.3)	13	(9.4)	371	(45.9)	1.0		1.0	
1	48	(17.7)	20	(14.5)	182	(22.5)	1.6	(1.1 - 2.4)	3.3	(1.6 - 6.9)
≥2	153	(56.5)	101	(73.2)	251	(31.0)	3.8	(2.7 - 5.4)	12.5	(6.8 - 23.0)
SF-36 MH score										
Best	68	(25.1)	16	(11.6)	268	(33.1)	1.0		1.0	
Intermediate	83	(30.6)	39	(28.3)	275	(34.0)	1.2	(0.8 - 1.8)	2.5	(1.4 - 4.6)
Worst	112	(41.3)	79	(57.3)	251	(31.0)	1.8	(1.3 - 2.5)	5.4	(3.1 - 9.6)

	Cases (n, %)				Controls (n, %) (n=809)		OR (95% CI)*			
	All (n=271)		RMS ≥11 (n=138)				All cases vs controls		RMS ≥11 vs controls	
Fear-avoidance beliefs *(no. of statements agreed)*										
0	72	(26.6)	27	(19.6)	256	(31.6)	1.0		1.0	
1 - 2	140	(51.7)	71	(51.5)	377	(46.6)	1.3	(0.9 - 1.8)	1.7	(1.1 - 2.8)
3 - 4	53	(19.6)	36	(26.1)	167	(20.6)	1.1	(0.7 - 1.7)	2.0	(1.2 - 3.5)
Beliefs about work as a cause of LBP *(no. of statements agreed)*										
0	46	(17.0)	14	(10.1)	219	(27.1)	1.0		1.0	
1-2	106	(39.1)	56	(40.6)	264	(32.6)	1.9	(1.3 - 2.8)	3.4	(1.8 - 6.2)
3	109	(40.2)	64	(46.4)	290	(35.9)	1.8	(1.2 - 2.6)	3.5	(1.9 - 6.4)
No. of other sites with pain *(past 4 weeks)*										
0	77	(28.4)	31	(22.5)	212	(26.2)	1.0		1.0	
1 - 2	132	(48.7)	70	(50.7)	350	(43.3)	1.0	(0.8 - 1.5)	1.4	(0.9 - 2.2)
3 - 6	58	(21.4)	35	(25.4)	226	(27.9)	0.7	(0.5 - 1.1)	1.2	(0.7 - 2.0)
Propensity to consult over LBP *(no. of statements agreed)*										
0	66	(24.4)	32	(23.2)	306	(37.8)	1.0		1.0	
1	90	(33.2)	45	(32.6)	222	(27.4)	1.8	(1.3 - 2.6)	1.9	(1.1 - 3.0)
2	109	(40.2)	57	(41.3)	269	(33.3)	1.8	(1.3 - 2.6)	2.0	(1.2 - 3.1)

* OR adjusted for age (in three bands) and sex.
RMS = Roland Morris score
Figures may not sum to 100% due to missing values.

Table 2: Occupational risk factors (other than whole-body vibration) in the study group

	Cases (n, %)				Controls (n, %)		OR (95% CI)*			
	All (n=271)		RMS ≥11 (n=138)		(n=809)		All cases vs controls		RMS ≥11 vs controls	
PHYSICAL										
Digging										
No	263	(97.1)	132	(95.6)	776	(95.9)	1.0		1.0	
Yes	8	(3.0)	6	(4.4)	33	(4.1)	0.6	(0.3 - 1.4)	1.0	(0.4 - 2.4)
Work with arms above shoulder height (hrs/day)										
0	134	(49.5)	63	(45.7)	360	(44.5)	1.0		1.0	
<1	99	(36.5)	51	(37.0)	277	(34.2)	1.0	(0.7 - 1.3)	1.1	(0.7 - 1.6)
≥1	38	(14.0)	24	(17.4)	172	(21.3)	0.6	(0.4 - 0.9)	0.8	(0.5 - 1.3)
Lifting ≥20 lbs (times/day)										
0	102	(37.6)	48	(34.8)	302	(37.3)	1.0		1.0	
1 - 10	117	(43.2)	56	(40.6)	340	(42.0)	1.0	(0.7 - 1.3)	1.0	(0.7 - 1.5)
>10	52	(19.2)	34	(24.6)	167	(20.6)	0.8	(0.6 - 1.2)	1.2	(0.7 - 1.9)
Lifting ≥20 lbs with back twisted (times /day)										
0	130	(48.0)	60	(43.5)	408	(50.4)	1.0		1.0	
1 - 10	107	(39.5)	57	(41.3)	288	(35.6)	1.1	(0.9 - 1.5)	1.3	(0.9 - 2.0)
>10	32	(11.8)	20	(14.5)	111	(13.7)	0.8	(0.5 - 1.3)	1.2	(0.7 - 2.0)
Bending trunk (times /day)										
0	107	(39.5)	48	(34.8)	302	(37.3)	1.0		1.0	
<20	98	(36.2)	52	(37.7)	302	(37.3)	0.9	(0.7 - 1.3)	1.1	(0.7 - 1.7)
≥20	65	(24.0)	37	(26.8)	202	(25.0)	0.9	(0.6 - 1.3)	1.1	(0.7 - 1.8)
Bending trunk (hrs/day)										
0	107	(39.5)	48	(34.8)	302	(37.3)	1.0		1.0	
<3	121	(44.7)	67	(48.6)	361	(44.6)	1.0	(0.7 - 1.3)	1.2	(0.8 - 1.8)
≥3	39	(14.4)	21	(15.2)	140	(17.3)	0.8	(0.5 - 1.2)	0.9	(0.5 - 1.6)
Twisting (times/day)										
0	158	(58.3)	70	(50.7)	511	(63.2)	1.0		1.0	
<20	74	(27.3)	45	(32.6)	211	(26.1)	1.1	(0.8 - 1.6)	1.6	(1.0 - 2.4)
≥20	39	(14.4)	23	(16.7)	86	(10.6)	1.4	(0.9 - 2.2)	1.9	(1.1 - 3.3)

	Cases (n, %)				Controls (n, %) (n=809)		OR (95% CI)*			
	All (n=271)		RMS ≥11 (n=138)				All cases vs controls		RMS ≥11 vs controls	
Twisting (hrs/day)										
0	158	(58.3)	70	(50.7)	511	(63.2)	1.0		1.0	
<3	86	(31.7)	48	(34.8)	224	(27.7)	1.2	(0.9 - 1.7)	1.6	(1.0 - 2.3)
≥3	26	(9.6)	19	(13.8)	72	(8.9)	1.2	(0.7 - 1.9)	2.0	(1.1 - 3.5)
Standing (hrs/day)										
<1	61	(22.5)	28	(20.3)	144	(17.8)	1.0		1.0	
1 - 3	88	(32.5)	42	(30.4)	221	(27.3)	0.9	(0.6 - 1.4)	1.0	(0.6 - 1.7)
≥3	122	(45.0)	68	(49.3)	444	(54.9)	0.6	(0.4 - 0.9)	0.8	(0.5 - 1.3)
Sitting, but not driving (hrs/day)										
<1	80	(29.5)	44	(31.9)	320	(39.6)	1.0		1.0	
1 - 3	60	(22.1)	35	(25.4)	219	(27.1)	1.1	(0.8 - 1.6)	1.2	(0.7 - 1.9)
≥3	131	(48.3)	59	(42.8)	270	(33.4)	2.0	(1.4 - 2.7)	1.6	(1.0 - 2.4)
Unloading a vehicle by hand (in professional drivers)										
No	241	(88.9)	121	(87.7)	725	(89.6)	1.0		1.0	
Yes	24	(8.9)	13	(9.4)	64	(7.9)	1.0	(0.6 - 1.7)	1.1	(0.6 - 2.2)
PSYCHOSOCIAL										
Decision latitude										
Often	117	(43.2)	49	(35.5)	384	(47.5)	1.0		1.0	
Sometimes	73	(26.9)	37	(26.8)	215	(26.6)	1.1	(0.8 - 1.6)	1.4	(0.9 - 2.2)
Seldom/Never	77	(28.4)	50	(36.2)	193	(23.9)	1.3	(1.0 - 1.9)	2.1	(1.3 - 3.2)
Support										
Often	107	(39.5)	50	(36.2)	366	(45.2)	1.0		1.0	
Sometimes	83	(30.6)	49	(35.5)	237	(29.3)	1.2	(0.9 - 1.7)	1.5	(1.0 - 2.3)
Seldom/Never	48	(17.7)	24	(17.4)	110	(13.6)	1.5	(1.0 - 2.3)	1.7	(1.0 - 2.9)
Not applicable	30	(11.1)	14	(10.1)	90	(11.1)	1.2	(0.7 - 1.9)	1.2	(0.6 - 2.3)
Job satisfaction										
No	230	(84.9)	116	(84.1)	698	(86.3)	1.0		1.0	
Yes	40	(14.8)	21	(15.2)	103	(12.7)	1.1	(0.8 - 1.7)	1.2	(0.7 - 2.0)

* OR adjusted for age (in three bands) and sex. RMS = Roland Morris score. Figures may not sum to 100% due to missing values.

Table 3: Exposures to whole-body vibration in the study group

	Cases (n, %)				Controls (n, %) (n = 809)		OR (95% CI)*			
	All (n = 271)		RMS ≥11 (n = 138)				All cases vs controls		RMS ≥11 vs controls	
Professional driving (≥1 hr/day)										
No	217	(80.1)	109	(79.0)	663	(82.0)	1.0		1.0	
Yes	54	(19.9)	29	(21.0)	146	(18.1)	1.1	(0.7 - 1.5)	1.2	(0.7 - 1.9)
Professional driving (≥3 hrs at a time)										
No	246	(90.8)	124	(89.9)	752	(93.0)	1.0		1.0	
Yes	24	(8.9)	14	(10.1)	55	(6.8)	1.3	(0.7 - 2.1)	1.5	(0.8 - 2.9)
Average hours driven/week for the commonest exposure source										
None	217	(80.1)	109	(79.0)	663	(82.0)	1.0		1.0	
<16	26	(9.6)	16	(11.6)	67	(8.3)	1.1	(0.7 - 1.8)	1.4	(0.8 - 2.6)
≥16	22	(8.1)	12	(8.7)	73	(9.0)	0.8	(0.5 - 1.4)	0.9	(0.5 - 1.9)
Total hours driven/week, all sources										
Not a regular driver	217	(80.1)	109	(79.0)	663	(82.0)	1.0		1.0	
3 -10	18	(6.6)	11	(8.0)	43	(5.3)	1.2	(0.7 - 2.1)	1.5	(0.7 - 3.0)
>10 - 20	14	(5.2)	8	(5.8)	39	(4.8)	1.0	(0.5 - 1.9)	1.2	(0.5 - 2.7)
>20 - 40	12	(4.4)	6	(4.4)	40	(4.9)	0.8	(0.4 - 1.6)	0.9	(0.3 - 2.1)
>40 - 81	4	(1.5)	3	(2.2)	18	(2.2)	0.6	(0.2 - 1.8)	1.0	(0.3 - 3.4)
Max r.m.s. of any machine (ms^{-2})										
0	217	(80.1)	109	(79.0)	663	(82.0)	1.0		1.0	
>0 - 0.6	35	(12.9)	18	(13.0)	89	(11.0)	1.1	(0.7 - 1.7)	1.2	(0.7 - 2.1)
≥0.6	17	(6.3)	11	(8.0)	56	(6.9)	0.8	(0.5 - 1.5)	1.2	(0.6 - 2.4)
Current r.m.s. A(8) (ms^{-2})										
0	217	(80.1)	109	(79.0)	663	(82.0)	1.0		1.0	
>0 -< 0.5	4	(1.5)	2	(1.5)	20	(4.8)	0.6	(0.2 - 1.8)	0.6	(0.1 - 2.7)
0.5 - 1.15	32	(11.8)	19	(13.8)	77	(9.5)	1.2	(0.7 - 1.8)	1.4	(0.8 - 2.5)
>1.15	12	(4.4)	7	(5.1)	41	(5.1)	0.8	(0.4 - 1.6)	1.0	(0.4 - 2.4)

* OR adjusted for age (in three bands) and sex.
RMS = Roland Morris score. Figures may not sum to 100% due to missing values.

Table 4: Non-driving factors selected by stepwise regression to be multivariate factors of adjustment

	OR (95% CI)			
	Cases vs controls*		RMS ≥11 vs controls†	
Gender				
Female	1.0		1.0	
Male	0.7	(0.5 - 1.0)	0.7	(0.5 - 1.1)
Age band (years)				
20 - 34	1.0		1.0	
35 - 49	1.4	(0.9 - 2.1)	1.7	(1.0 - 3.1)
50 - 64	1.1	(0.7 - 1.7)	1.2	(0.6 - 2.3)
Height				
Shortest third			1.0	
Middle third	-		1.1	(0.6 - 1.9)
Tallest third			2.1	(1.2 - 3.5)
Somatising tendency *(no. of distressing symptoms)*				
0	1.0		1.0	
1	1.9	(1.2 - 2.9)	3.8	(1.7 - 8.3)
≥2	4.6	(3.1 - 6.9)	12.9	(6.3 - 26.5)
SF-36 MH Score				
Best	-		1.0	
Intermediate			1.9	(0.9 - 3.7)
Worst			2.9	(1.5 - 5.6)
Beliefs about work as a cause of back pain *(no. of items agreed)*				
0	1.0		1.0	
1 - 2	2.1	(1.4 - 3.3)	3.3	(1.6 - 6.8)
3	2.6	(1.6 - 4.3)	4.3	(2.0 - 9.5)
Propensity to consult over low-back pain *(no. of items agreed)*				
0	1.0		1.0	
1	1.8	(1.2 - 2.6)	1.7	(1.0 - 3.1)
2	1.9	(1.3 - 2.8)	2.1	(1.2 - 3.7)
No. of other sites with pain				
0	1.0		1.0	
1 - 2	0.7	(0.5 - 1.1)	0.6	(0.3 - 1.1)
3 - 6	0.4	(0.2 - 0.6)	0.2	(0.1 - 0.5)
Work with arms above shoulder height (hrs/day)				
0	1.0			
<1	1.0	(0.7 - 1.5)	-	
≥1	0.6	(0.4 - 1.0)		
Sitting, but not while driving (hrs/day)				
<1	1.0		1.0	
1.3	1.3	(0.8 - 1.9)	1.6	(0.9 - 2.9)
≥3	2.8	(1.8 - 4.3)	3.0	(1.7 - 5.4)

Separate models were constructed for each of the two outcomes. RMS = Roland Morris score

Table 5: Adjusted risk estimates for exposure to whole-body vibration

	OR (95% CI)			
	Cases vs controls*		RMS ≥11 vs controls†	
Professional driving ≥1 hr/day				
No	1.0		1.0	
Yes	1.0	(0.6 - 1.5)	1.0	(0.6 - 1.8)
Professional driving ≥3 hrs at a time				
No	1.0		1.0	
Yes	1.1	(0.6 - 2.0)	1.1	(0.5 - 2.4)
Average hours driven/week for the commonest exposure source				
None	1.0		1.0	
<16	1.2	(0.7 - 2.1)	1.4	(0.7 - 2.8)
≥16	0.7	(0.4 - 1.3)	0.6	(0.3 - 1.5)
Total hours driven/week, all sources				
Not a regular driver	1.0		1.0	
3 -10	1.3	(0.7 - 2.6)	1.6	(0.7 - 3.7)
>10 - 20	0.8	(0.4 - 1.7)	0.9	(0.3 - 2.3)
>20 - 40	0.8	(0.4 - 1.6)	0.7	(0.2 - 2.2)
>40 - 81	0.5	(0.1 - 1.8)	0.6	(0.1 - 2.4)
Max r.m.s. of any machine (ms^{-2})				
0	1.0		1.0	
>0 - 0.6	1.2	(0.7 - 2.0)	1.2	(0.6 - 2.4)
≥0.6	0.7	(0.4 - 1.4)	0.8	(0.3 - 1.8)
Current r.m.s. A(8) (ms^{-2})				
0	1.0		1.0	
>0 -< 0.5	0.8	(0.2 - 2.5)	0.5	(0.1 - 2.7)
0.5 - 1.15	1.1	(0.7 - 1.9)	1.4	(0.7 - 2.8)
>1.15	0.6	(0.3 - 1.4)	0.6	(0.2 - 1.7)

Each row variable was analysed in a separate regression model.
RMS = Roland Morris score
* Adjustment was made for the variables in the first data column of Table 4
† Adjustment was made for variables in the last data column of Table 4.

Annex 17: Appendix 1 - Vibration magnitudes assumed in estimating exposures to whole-body vibration

The questionnaire provided the following information: (i) a list of vehicles driven in the current (or last) job, hours driven per week (Q23), and job duration. (ii) vehicles driven in past jobs - driven for >1 hour/day (Q31).

We assumed vibration magnitudes for the sources in these questions as set out in a previous study for the UK HSE:[20]

Vehicle	Estimated frequency-weighted acceleration (ms^{-2} r.m.s.)
Car or Van	0.5
Bus or Coach	0.6
Lorry or heavy goods vehicle	0.7
Motorcycle	1.0
Forklift truck	0.9
Tractor	0.75
Loader	1.2
Dumper or excavator	1.2
Other large off road vehicle (eg harvester, armoured tank)	1.2
Other large on road vehicle (e.g. ambulance, fire engine)	0.7

The consortium method allows the estimation of exposure severity using both r.m.s. and r.m.q. measures and also for assessments based on separate axes of vibration. However, the values suggested above are overall values and do not discriminate between r.m.s. and r.m.q. Hence, the suggested values are. r.s.s. (a_{wsi}).

Annex 17: Appendix 2 - Scoring System for MR images of the Lumbar Spine

James Shambrook
Phillip Mcnee
Madelene Sampson

Sagittal T1, T2 and axial T2 weighted images will be scored via the following classification system for all MR scans to be included the Southampton survey of lifestyle, work and health in radiology patients.

Disk Degeneration

Disk height

To be measured in millimetres, on midline sagittal images, perpendicular to the inferior end plate of the vertebral body above the disk; small endplate infractions and Schmorl's nodes to be ignored for assessment of disk height. On hard copy images the corresponding 10cm scale will also be measured in millimetres.

Disk signal (8)

Using a semi-quantative scale, comparing disk signal, at the mid-sagittal level to a standardized atlas for scoring lumbar disc degeneration:

Grade 0	Normal white signal
Grade 1	Diminished signal on at least one slice darker than the atlas
Grade 2	Diminished signal on at least one slice darker than the atlas
Grade 3	Signal indistinguishable from adjacent end plates

HIZ (High Intensity Zone)(2)

High intensity signal located in the substance of the posterior annulus fibrosus, clearly dissociated from the signal of the nucleus pulposus in that it is surrounded superiorly, inferiorly, posteriorly and anteriorly by the low intensity signal of the annulus fibrosus and is appreciably brighter than that of the nucleus pulposus.

End plate change (3)

End plates of adjacent vertebrae to be graded according to the system of Modic et al (3). When two different grades are present on both sides of an intervertebral space, only one diagnosis will be applied (first priority, type1; second priority, type 2; last priority, type 3).

Type	T1 weighted spin echo changes	T2 weighted spin echo changes
1	Decreased signal	Increased signal
2	Increased signal	Isointense or increased signal
3	Decreased signal	Decreased signal

Schmorl's nodes

A focal depression in the vertebral end plate continuous with the disk. The number present on superior and inferior end plates to be recorded.

Disk herniation (4,5)

The following classification will be used with regard to the type of disk herniation, as well as recording the site of herniation, as central, paracentral or lateral:

Score	Type	Description
0	Normal	No disc extension beyond interspace
1	Bulge	Circumferential, symmetric disc extension beyond interspace (around the end plates)
2	Protrusion	Focal or asymmetric disc extension beyond interspace into the canal; the base against the parent disc broader than any other diameter of the protrusion
3	Extrusion	Focal obvious disc extension beyond interspace; the base against the parent disc narrower than the diameter of the extruding material itself on axial images
4	Sequestration	Disc extension beyond interspace with no connection to the parent disk

Nerve root compression (1,6)

Both the nerve roots in the neural exit foramina and in the lateral recess of the canal will be scored according to the following classification:

Score	Type	Description
0	Normal	No compromise of the nerve root
1	Contact	Visible contact of disk material with the nerve root, and the normal epidural fat layer between the two is not evident. The nerve root has a normal position, and there is no dorsal deviation
2	Deviation	The nerve root is displaced dorsally by disk material
3	Compression	The nerve root is compressed between disk material and the wall of the spinal canal/exit foramen; it may appear flattened or be indistinguishable from disk material

Spondylolisthesis (7)

Vertebral spondylolisthesis will be classified as follows.

Grade I	<25% displacement vertebral body
Grade II	25-49% displacement vertebral body
Grade III	50-75% displacement vertebral body
Grade IV	76-100% displacement vertebral body
Grade V	Spondyloloptosis

Facet joint arthropathy

Will be scored as present or not present, and if present on which side.

Spinal canal stenosis

Spinal canal stenosis will be deemed to be present if there is no high cerebrospinal fluid signal at the disk level.

Other findings

Other positive findings will be recorded in a free text box, to include common variants and pathologies e.g. haemangioma, osteoporotic vertebral body collapse, pars defects.

References

1. Weishaupt D, Zanetti M, Hodler J, Boos N. MR imaging of the lumbar spine: Prevalence of intervertebral disk extrusion and sequestration, nerve root compression, end plate abnormalities and osteoarthritis of the facet joints in asymptomatic voulunteers. Radiology 1998;209:661-666.
2. Aprill C, Bogduk N. High-intensity zone: a diagnostic sign of painful lumbar disk on magnetic resonance imaging. British Journal of Radiology 1992;65:361-369.
3. Modic M, Steinberg P, Ross J, Masaryk T, Carter J. Degenerative disk disease: Assessment of changes in vertebral body marrow with MR imaging. Radiology 1988;166:193-199.
4. Masaryk TJ, Ross JS, Modic MT, Boumphrey F, Bohlman H, Wilber G. High resolution MR imaging of a sequestered lumbar intervertebral disks. Am J Neuroradiol 1988;9:351-358.
5. Brant-Zawadzki M, Jensen M, Obuchowski N, Ross J, Modic M. Interobserver and intraobserver variability in interpretation of lumbar disk abnormalities: A comparison of two nomenclatures. Spine 1995;20:1257-1264.

6. Pfirrmann CW, Dora C, Schmid MR, Zanetti M, Hodler J, Boos N. MR image-based grading of lumbar nerve root compromise due to disk herniation: Reliability study with surgical correlation. Radiol 2004; 230: 583-8.
7. Ross J, Brant-Zawadski M, Moore K, Crim J, Chen M, Katzman G. Diagnostic imaging: Spine. Amirsys 2004.
8. Jarosz J, Bingham J, Pemberton JB, Sambrook PN, Spector TD. An atlas for scoring cervical and lumbar disc degeneration. London: Springer Verlag, 1997.