

# Occupational driving and lumbar disc degeneration: a case-control study

Michele C Battié, Tapio Videman, Laura E Gibbons, Hannu Manninen, Kevin Gill, Malcolm Pope, Jaakko Kaprio

## Summary

**Background** Back problems are reported more by occupational drivers than by any other occupational group. One explanation is that whole-body vibration caused by the vehicle leads to accelerated disc degeneration, herniation, and associated symptoms. We aimed to investigate the effects of lifetime driving exposure on lumbar disc degeneration in monozygotic twins with very different histories of occupational driving during their life.

**Methods** We assessed 45 male monozygotic twin pairs from the population-based Finnish Twin Cohort who had greatly different patterns of occupational driving during their life. Data were obtained for driving exposures and potential confounding factors through an extensive, structured interview. We assessed disc degeneration with lumbar MRI.

**Findings** Disc degeneration did not differ between occupational drivers and their twin brothers. We also did not identify any overall tendency for greater degeneration or pathology in occupational drivers than their twin brothers.

**Interpretations** Although driving may exacerbate symptoms of back problems, it does not damage the disc. Our inability to identify structural damage should be encouraging to those employed in occupations involving motorised vehicles and operation of heavy equipment.

*Lancet* 2002; **360**: 1369–74. Published online Oct 15, 2002  
<http://image.thelancet.com/extras/01art9329web.pdf>

**Faculty of Rehabilitation Medicine, University of Alberta, Edmonton, Canada** (Prof M C Battié PhD, Prof T Videman MD); **Department of Environmental Health, University of Washington, Seattle, USA** (L E Gibbons PhD); **Department of Clinical Radiology, Kuopio University Hospital, Kuopio, Finland** (H Manninen MD); **Department of Orthopedic Surgery, Southwest Orthopedic Institute, Dallas, USA** (K Gill MD); **Department of Environmental and Occupational Medicine, University of Aberdeen, Aberdeen, UK** (Prof M Pope PhD); **Department of Public Health, University of Helsinki, Helsinki** (Prof J Kaprio MD)

**Correspondence to:** Dr Michele C Battié  
 (e-mail: mc.battie@ualberta.ca)

## Introduction

Work-related low back pain can be one of the most challenging disorders seen in primary and specialty care. Clear, verifiable diagnoses of underlying causes are rare, as are treatments with proven effectiveness, and work-related factors sometimes complicate recovery. The main causes of back problems in the work place include heavy lifting, repeated loads to the spine from manual handling of materials, work postures incurring postural stress, and whole-body vibration associated with driving.<sup>1</sup> A leading hypothesis for such problems is that the negative effects of driving on back problems are the result of disc degeneration and herniation caused by whole-body vibration, implying irreparable damage. In fact, in 1993, the Federal Ministry of Labour in Germany added a new occupational disease to its official list of occupational diseases, “diseases of the lumbar spine from disc degeneration caused by long-term (mainly vertical) whole-body vibration exposure”.<sup>2</sup>

Results of animal and in-vitro studies<sup>3–5</sup> suggest that vibration could adversely affect the disc through several mechanisms, especially with vibration at the disc’s natural frequency of about 5 Hz. Findings from epidemiological studies<sup>6–8</sup> have also provided some support for the hypothesis that driving adversely affects the intervertebral disc, with higher rates of disc herniation in occupational drivers than in other occupations. However, investigators have had difficulties isolating the effects of driving and associated whole-body vibration from those of other potentially confounding factors, such as twisted or forwardly inclined work postures, extended sitting times, postural stresses in confined work places, and lifting and lifestyle factors that differ between people in driving and non-driving occupations. In an extensive review, Kjellberg and colleagues<sup>9</sup> reported that most investigators<sup>10–12</sup> have recorded significantly higher frequencies of back symptoms and degenerative changes of vertebrae and intervertebral discs in drivers than referents. However, Kjellberg and colleagues also note that uncontrolled confounding factors could have affected the results in all studies, and the conclusions about the causal role of whole-body vibration for injuries or disorders therefore become uncertain. Investigators of earlier studies<sup>9,10,12</sup> were also unable to clarify a dose-response relation, and a basis for setting specific limits has not been established. We aimed to investigate the effects of lifetime driving exposure on lumbar disc degeneration in monozygotic twins with very different histories of lifetime occupational driving.

## Methods

### Participants

We selected male monozygotic twin pairs from the population-based Finnish Twin Cohort<sup>13</sup> on the basis of their present and previous job titles and the probability

of substantial differences in occupational driving or other exposures of interest suspected of affecting disc degeneration. The Finnish Twin Cohort database includes job titles from 1975 and 1981 when extensive health-related questionnaires were mailed to all twins in the cohort, with response rates of 89% and 84%, for the 2 years, respectively. We contacted pairs who seemed to have very different exposures to occupational driving or one of the other exposures of interest (occupational materials handling, sedentary work, or regular exercise) and asked them if they would be interested in participating in the study. Since we were investigating differences within pairs, we included a participant only if both co-twins agreed to participate. Of 141 pairs approached, the co-twins of 116 pairs (82%) agreed to participate. Of those, 45 pairs had appropriate co-twin differences in occupational driving, as verified in a study interview in 1991 or 1992. Pairs were included only if they had differences in occupational driving of at least 5 years of full-time driving.

Mean age of participants was 50.7 years (SD 7.5). At the time of the interview and imaging, 14 (16%) of 90 participants were on pension—six from the driver group and eight controls. Every participant was asked whether they had ever changed jobs for health reasons; two drivers and two non-drivers had changed jobs because of health and all were because of back problems. One driver changed from driving trucks to driving buses, a non-driver changed from a management position to mink breeding, and the remaining two were siblings who both moved from forest work to other occupations.

Study protocols were reviewed and approved by the ethical committee of the Department of Public Health at the University of Helsinki and the human subjects committee at the University of Washington. All participants received written information about the study procedures and provided written informed consent before participation.

#### Driving exposure

Data for exposure were obtained from an extensive, structured interview that reviewed the participant's work history and common leisure activities. The participant was asked to review every job held and its associated tasks from the time he began working. For every job noted in the occupational history, the participant was asked to describe the tasks done and to estimate exposure to specific conditions. Participants noted whether they spent time during the day in motorised vehicles and, if so, the approximate mean number of hours per day of driving or riding. The type of vehicle was also noted.

We calculated driving exposure for every job by multiplying the mean number of driving hours per day by 220 work days per year, which was then multiplied by the number of years in the job, with the totals summed for every job held. To investigate whether co-twins had

substantial differences in exposure to whole-body vibration, we investigated the types of vehicles driven. As a quantitative indicator of discordance, occupational driving time was adjusted by the mean vibration values reported by Dupuis and Zertlett<sup>14</sup> and Oortman and colleagues<sup>15</sup> for comparable vehicle types from the 1980s, an intermediate period in the participants' driving histories.

We also obtained data for possible confounding factors, such as handling of occupational materials, work in bent postures, and smoking. Participants were asked to describe their job activities and estimate the most common weights lifted, the frequency of lifting, the number of hours spent working in specified bent or twisted postures, and the number of hours spent sitting. The product of the most common weight lifted, multiplied by the frequency, formed a summary measure of lifting per day. Every job was placed in one of four categories on the basis of material handling activities and associated positional loading (ie, twisted and bent postures): 1=sedentary work, 2–3=progressive degrees of materials handling and positional loading, and 4=very heavy physical loading. These categories did not include driving exposure.

We also did a limited assessment of the reliability of the interview. In a phone interview 12 months later, we asked participants how many hours they spent sitting and driving, the most common weight lifted, and the frequency of lifting in their current jobs. For participants who reported that they were still working at the same job as 12 months earlier, responses were then compared with those for the job held at the time of the initial interview. The assessment showed that the reliability of the interview was moderate to good (intraclass correlation coefficient 0.74 for sitting time, 0.83 for driving time, and 0.60 for mean total lifting per day).<sup>16</sup>

After each job position was described, we asked the participant to recall whether he had had any back injuries (defined as sudden onset, associated with a work activity or accident) during their work. During the interview, participants were also asked how frequently they had had back pain during the previous 12 months, with responses ranging from daily to none at all on a seven-point scale.

#### Assessment of disc degeneration

We assessed disc degeneration with MRI. Patients were imaged consecutively, and to control for diurnal and activity effects on the disc, every participant spent at least 45 min lying supine immediately before the procedure.<sup>17</sup> Images were obtained with a 1.5 Tesla imager (Magnetom, Siemens AG Erlangen, Germany) with a surface coil, using a protocol and sequences described previously.<sup>18</sup>

The images were assessed qualitatively by two independent readers—a radiologist and an orthopaedic surgeon—who were unaware of the twinship and exposure. Every spinal level was assessed for reduction in disc height, disc bulging, osteophytes, and irregularities in the endplate (Schmorl's nodes) with a 0–3 rating scale; in which 0 was

Variable	Drivers (n=45)	Co-twins (n=45)	Paired difference	p*
Lifetime occupational driving hours†	34 000 (20 500)	6300 (7000)	27 700 (18 500)	<0.0001
Vibration-weighted driving hours‡	23 900 (16 400)	4200 (5000)	19 700 (14 900)	<0.0001
Job code (1–4 scale)§	2.7 (0.8)	2.4 (0.9)	0.2 (1.2)	0.2471
Occupational lifting/day (kg lifted×frequency/day)§	1700 (3900)	1000 (2200)	700 (4600)	0.1010
Time working twisted/bent (h/day)§	1.8 (1.8)	1.6 (1.5)	0.2 (2.2)	0.7306
Commute time (min)	26.7 (24.2)	34.2 (25.6)	7 (29)	0.0507
Cigarette smoking (pack-years)	17.8 (20.8)	15.5 (19.4)	2.3 (19.5)	0.2667

Values are mean (SD). \*Calculated with Wilcoxon rank test. †A work year of 220 days of 8 h/day of occupational driving would equal 1760 driving hours. ‡This measure is the number of hours spent in driving jobs multiplied by an estimate of the vibration frequency for the corresponding vehicle type, as measured in the 1980s by ISO 2631 vibration measurement standards. §Means are weighted by number of months spent in each job over the participant's lifetime work history.

Table 1: Exposures to occupational driving and possible confounding

	Scale*	Driver	Co-twin	Difference (95% CI)	p
<b>Disc outcome</b>					
Upper lumbar					
Signal intensity (digital)	CSF-adjusted	-0.26 (0.05)	-0.27 (0.05)	0.01 (-0.01 to 0.03)	0.1840
Annular tears	Disks with tears	0.30 (0.59)	0.33 (0.64)	-0.05 (-0.27 to 0.17)	1.0000
Bulging	0-3 score	0.93 (0.53)	1.00 (0.59)	-0.07 (-0.21 to 0.07)	0.6079
Herniations	Disks with herniations	0.07 (0.25)	0.07 (0.25)	0.00 (-0.09 to 0.09)	1.0000
Disc height narrowing	0-3 score	0.22 (0.26)	0.26 (0.33)	-0.04 (-0.12 to 0.04)	0.3053
Endplate changes					
Upper	Multiple or large	0.12 (0.19)	0.12 (0.12)	0.00 (-0.06 to 0.06)	0.8801
Lower	Multiple or large	0.05 (0.09)	0.06 (0.12)	-0.01 (-0.04 to 0.01)	0.2970
Osteophytes	Any large	0.18 (0.25)	0.19 (0.27)	-0.01 (-0.07 to 0.05)	0.9416
Lower lumbar					
Signal intensity (digital)	CSF-adjusted	-0.20 (0.06)	-0.21 (0.05)	0.00 (-0.02 to 0.02)	0.9817
Annular Tears	Disks with tears	0.57 (0.62)	0.67 (0.71)	-0.11 (-0.35 to 0.13)	0.4151
Bulging	0-3 score	1.09 (0.51)	1.22 (0.46)	-0.12 (-0.28 to 0.03)	0.1921
Herniations	Disks with herniations	0.30 (0.51)	0.56 (0.78)	-0.27 (-0.55 to 0.01)	0.0414
Disc height narrowing	0-3 score	0.97 (0.65)	1.20 (0.63)	-0.05 (-0.23 to 0.13)	0.6302
Endplate changes					
Upper	Multiple or large	0.14 (0.24)	0.09 (0.17)	0.06 (-0.02 to 0.13)	0.1612
Lower	Multiple or large	0.09 (0.22)	0.06 (0.15)	0.04 (-0.03 to 0.11)	0.3711
Osteophytes	Any large	0.18 (0.32)	0.17 (0.25)	0.02 (-0.07 to 0.10)	0.6715

Values are mean (SD). CSF=cerebrospinal fluid. \*All variables are scaled so that a higher score represents more degeneration.

Table 2: Differences in lumbar degenerative findings in upper and lower lumbar regions between drivers and their co-twins

normal and 1-3 were progressive degrees of abnormality. A set of standard images and written instructions for assessment accompanied the images. We assessed inter-rater agreement by calculating intraclass correlation coefficients (ordinal variables) or  $\kappa$  statistics (dichotomous variables). Inter-rater agreement coefficients were 0.62 for disc height reduction, 0.63 for disc bulging, 0.85 for osteophytes, and 0.91 for endplate irregularities or Schmorl's nodes. Annular tears and disc herniations were assessed by another radiologist. Intrareader reliability, as determined from a sample of 20 participants, gave a  $\kappa$  of 0.69 for absence or presence of annular tears contiguous with the outer disc margin and 0.71 for disc herniations.  $\kappa$  values greater than 0.6 suggest substantial agreement, with those over 0.8 suggesting perfect agreement.<sup>19</sup>

We obtained quantitative measures of disc degeneration using digital data from MRI and a computerised spine image analysis program. Specifically, disc signal intensity adjusted for cerebrospinal fluid, disc height, and bulging were assessed as described previously.<sup>18</sup> The reproducibility of these quantitative assessments is better than that of their qualitative counterparts (signal intensity interclass correlation coefficient 0.95-0.98 for lumbar spinal level, 0.96-0.97 for disc bulging, and 0.75-0.79 for disc height). Such measures should provide good power for detecting effects.<sup>18,20</sup>

#### Statistical analysis

At an  $\alpha$  level of 0.05, 45 pairs would be expected to have 80% power to detect effect sizes of around 0.6 SDs in paired differences. Such effects are judged to be of moderate size.<sup>21</sup> We compared measures of MRI, driving exposures, potential confounders, and reports of back pain history between co-twins using the Wilcoxon signed rank test, a non-parametric test for paired data. Differences in work-related back injuries and in previous lumbar surgery were assessed with McNemar's test for paired comparisons. We also used paired *t* tests to assess differences in the quantitative MRI scores. Results were similar to the Wilcoxon signed rank test and are thus not reported. We investigated potential confounders by regressing the paired difference in an outcome measure on the paired difference in a potential confounder, and noting changes in the estimate of the intercept, as has been used in earlier twin studies.<sup>22</sup> Because of the high degree of control of extraneous and possible confounding factors

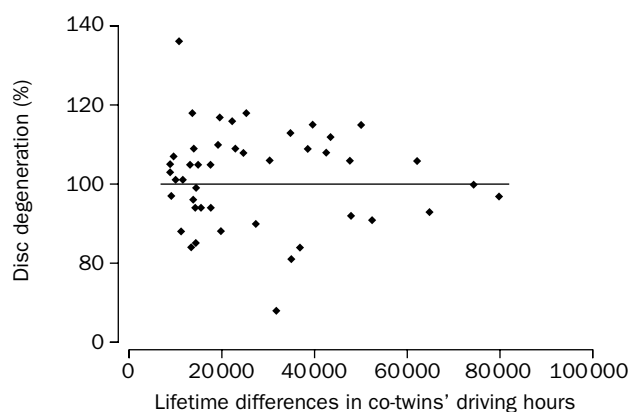
and co-twin similarities in degenerative disc findings,<sup>18,23</sup> this model is very efficient for detecting even small effects.<sup>24</sup> Disk degeneration may be largely determined by genetic factors,<sup>20,25,26</sup> further strengthening use of a case-control study design with monozygotic twin pairs with differing exposures to occupational driving.

#### Role of the funding source

The sponsors of the study had no role in study design, data collection, data analysis, data interpretation, or in the writing of the report.

#### Results

Participants exposed to driving had on average 5.4 (95% CI 4.2-6.6) times more driving exposure than their co-twins, with differences ranging from slightly more than 5 years to over 45 years of 8-h driving per workday (table 1). Co-twin differences were greater than that indicated by driving hours alone if whole-body vibration levels of different vehicle classes driven were accounted for. Adjustment for driving hours by the values reported in the 1980s<sup>14,15</sup> provided an estimate of a 5.7-fold (4.4-7.0) discordance. This result could have been predicted by



#### Difference in disc signal intensity in participants exposed to driving relative to their lesser-exposed co-twins, plotted by hours of driving discordance

Points are difference in degeneration, as measured through signal intensity, between siblings of one pair of monozygotic twin participants.

Variable	Drivers (n=45)	Twin siblings (n=45)	Paired difference	p*
Frequency of low back pain over past 12 months (1–7 scale)	4.7 (2.3)	4.7 (2.2)	0 (2.9)	0.9664
Severity of worst episode over past 12 months (0–100 scale)	31 (31)	42 (38)	10.4 (40.5)	0.0804
Back pain interfering with daily activities over past 12 months (days)	18 (61)	22 (77)	4.2 (96.6)	0.5547

Values are mean (SD). \*Wilcoxon signed rank test.

Table 3: Recent history of back problems in drivers and their twin siblings

looking at the vehicles driven; in 42 of 45 pairs, the twins with more driving drove the same class of vehicle for more years or a class of vehicle associated with higher whole-body vibration levels than did their siblings (webtable at <http://image.thelancet.com/extras/01art9329webtable.pdf>). In no case was the direction of driving discordance between co-twins reversed when vibration frequency-weighted exposure hours were calculated. Potential confounding factors did not differ between drivers and their co-twins, with the exception of higher estimates for occupational lifting in drivers than their twins (table 1).

Driving exposure was not associated with accelerated lumbar degeneration and structural abnormalities as measured through disc signal intensity, annular tears, disc bulging, disc herniations, disc height, endplate irregularities, and Schmorl's nodes or osteophytes (table 2). Despite substantial differences in driving exposure, disc degeneration did not differ between occupational drivers and their twins, with the exception of disc herniations, which were more frequent in the lower lumbar regions of non-drivers than drivers ( $p=0.044$ ). Controlling for smoking, occupational lifting, time spent in twisted or awkward work postures, and job code as possible confounding factors did not change the results.

Furthermore, disc degeneration did not differ between occupational drivers and their lesser-exposed co-twins by extent of exposure to motorised vehicles, and differences in driving did not show a threshold effect (figure). We also recorded no association or threshold effect between driving and degeneration in analyses adjusted for quantitative vibration scores.

Occupational drivers and their co-twins reported similar amounts of low back pain. 24 drivers and 21 of their co-twins reported sudden onset back injuries related to work ( $p=0.4910$ ). Of those reporting injuries, a mean number of 1.7 (SD 0.9 for drivers and 1.4 for non-drivers) injuries was reported in both groups. Two participants in each group, all from different twin pairs, reported previous lumbar surgery ( $p=1.00$ ). The frequency of low back pain over the previous 12 months was almost identical in the two groups. The severity of pain and the number of days that back pain interfered with daily activities did not differ between groups (table 3).

## Discussion

Almost everyone is exposed to motorised vehicles and associated whole-body vibration, the only difference is extent. Health concerns over vibration exposure (frequency, amplitude, and duration) were first raised in the 1950s and since that time, many studies have been done. Investigators have typically pointed to an association between occupational driving and back symptoms, admissions for disc disorders, and lumbar degeneration. We, however, did not identify any acceleration of lumbar degeneration related to occupational driving. This result persisted even when considering co-twins with the greatest differences in

driving exposure. Moreover, by contrast with the original hypothesis, twins with less driving had more herniated discs in the lower lumbar levels than did occupational drivers. However, this finding was marginally significant, and is probably due to chance.

In most studies,<sup>9</sup> occupational driving is associated with degeneration of the lumbar spine with degeneration assessed by radiographs, thereby focusing on measures of disc space narrowing and vertebral changes. Although results of most early studies show more lumbar degeneration in occupational drivers than in referents, investigation of specific types of findings and inclusion of more recent studies<sup>27–30</sup> paints a less consistent picture. The results of earlier studies probably conflict because the investigators did not adequately control for possible confounding factors.

Because we investigated monozygotic twins with greatly differing patterns of exposure to driving, in addition to controlling for obvious attributes such as sex, age, genotype, our analysis accounted for many other factors, including unidentified confounding factors, that are probably more similar in co-twins than in unrelated matched-controls. The participants' estimates of specific weights lifted and frequencies of occupational lifting and time spent in awkward work postures should not be viewed as precise measures, but are indicators of possible differences between co-twins. Co-twins had similar baseline characteristics, with the exception of occupational lifting, which drivers reported 1.7 times more than their co-twins. Such a difference would be expected to exaggerate, rather than mask, an apparent negative effect of driving exposure on disc degeneration. Thus, the difference between comparison groups cannot account for the absence of a harmful effect of driving on lumbar degeneration. Controlling for occupational lifting and small variations in smoking, time spent in twisted or awkward work postures, and job code did not change the results.

Because we assessed only 45 co-twin pairs, only effects of moderate size would be significant. However, our results provide no evidence of even small consistent differences between groups to suggest a deleterious effect of driving on the disc. Furthermore, we did not have precise measurements of whole-body vibration exposure. Such precision is not feasible when studying lifetime effects because of variations in vehicle models and the effects of maintenance and road conditions, among other factors, that typically vary over lifetime work histories. We considered obtaining measures of a sample of current vehicles driven, but did not think this would give a reasonable depiction of lifetime exposures and might give a false precision of measurement. There is also some question as to whether the routinely used international standard, ISO 2631 frequency-weighted root-mean-square acceleration value,<sup>31</sup> is the best method to quantify vibration exposure.<sup>9</sup> Although peak shocks might be more relevant for some possible injury mechanisms, such as endplate fracture, the effects of rally driving characterised by vibration with high impact loading were not associated with disc degeneration.<sup>32</sup>

While determination of dose-response relations may not be possible without vibration exposure measurements of greater precision, the case-control design should be appropriate for identification of driving effects.

Our results lend support to those of Brinckmann and coworkers,<sup>30</sup> who concluded that occupational driving has no detrimental effects on vertebral height, sagittal plane displacement, or disc height if the machine operators' seats are dampened—as is the case in equipment produced in the last couple of decades. They conclude that “primary overload damage to vertebrae or discs is not responsible for the high prevalence of back problems in labour forces with sustained exposure to whole-body vibration”.<sup>30</sup>

Driving includes several conditions, most notably long-term confined postures and whole-body vibration. Other possible explanations for the association of driving with back pain problems include effects on muscles and tendons through constrained work postures and vibration-induced muscle fatigue.<sup>33</sup> Another possible mechanism through which vibration may affect back pain problems is through neural changes at the cellular level.<sup>34</sup>

Although occupational driving might be associated with higher rates of back-related symptoms, the mechanism for this association is probably not the result of irreparable damage of lumbar discs and vertebrae. Our inability to identify damage to these structures, despite substantial exposures, should be encouraging to those employed in occupational driving jobs. Although driving might exacerbate symptoms from back problems, it might not be associated with permanent damage. We suggest that attention be shifted from degenerative and morphological changes of the disc and vertebrae to other possible explanations for the link between occupational driving and the frequently reported higher occurrence of back problems.

#### Contributors

M C Battié was responsible for the idea for the study and overall planning, oversaw data collection, analysis, and interpretation, and wrote the report. T Videman was responsible for study idea, overall planning, overseeing data collection, and contributed to data interpretation and writing of the report. L Gibbons was mainly responsible for data analysis and interpretation and contributed to the writing of the report. H Manninen and K Gill planned and did the MRI qualitative assessments and contributed to the data interpretation and writing of the report. M Pope assisted in the planning of the driving and whole-body vibration measures and contributed to data interpretation and writing of the report. J Kaprio contributed to the study planning, participant recruitment, data interpretation, and writing of the report.

#### Conflict of interest statement

M Battié, L Gibbons, H Manninen, K Gill, and J Kaprio have no financial or personal relationships with other people or organisations related to the effects of driving and whole-body vibration that could bias their work on this research project. T Videman and M Pope gave expert testimony in Occupational Safety and Health Administration hearings in 2000 on research findings on the effects of workplace exposures, including driving, on spine pathology, and disorders. They have no funding conflict. M Pope has funding from the Health and Safety Executive on a study of whole-body vibration.

#### Acknowledgments

Funding for this study was provided by the National Institutes of Health (RO1 AR40857), the Alberta Heritage Foundation for Medical Research, the Finnish Ministry of Education, and the Academy of Finland (51038).

#### References

- 1 Frank JW, Kerr MS, Brooker AS, et al. Disability resulting from occupational low back pain. Part I: What do we know about primary

prevention? A review of the scientific evidence on prevention before disability begins. *Spine* 1996; **21**: 2908–17.

- 2 Dupuis H. Medical and occupational preconditions for vibration-induced spinal disorders: occupational disease no 2110 in Germany. *Int Arch Occup Environ Health* 1994; **66**: 303–08.
- 3 Keller TS, Hansson TH, Holm S, Pope MH, Spengler DM. In vivo creep behavior of the normal and degenerated porcine intervertebral disc: a preliminary report. *J Spinal Disord* 1988; **1**: 267–78.
- 4 Holm S, Nachemson A. Nutrition of the intervertebral disc, effects induced by vibration. *Orthop Trans* 1985; **9**: 525.
- 5 Brinckmann P, Biggemann M, Hilweg D. Fatigue fracture of human lumbar vertebrae. *Clin Biomech* 1988; **3**: 1–23.
- 6 Kelsey JL, Hardy RJ. Driving of motor vehicles as a risk factor for acute herniated lumbar intervertebral disc. *Am J Epidemiol* 1975; **102**: 63–73.
- 7 Heliövaara M. Occupation and risk of herniated lumbar intervertebral disk and sciatica leading to hospitalization. *J Chron Dis* 1987; **40**: 259–64.
- 8 Jensen MV, Tüchsen F, Ørsted E. Prolapsed cervical intervertebral disc in male professional drivers in Denmark, 1981–90: a longitudinal study of hospitalizations. *Spine* 1996; **21**: 2352–55.
- 9 Kjellberg A, Wikström B-O, Landström U. Injuries and other adverse effects of occupational exposure to whole-body vibration: a review for criteria documentation. *Arbete Och Hälsa vetenskaplig skriftserie* 1994; **41**: 1–74.
- 10 Bovenzi M, Hulshof CT. An updated review of epidemiologic studies on the relationship between exposure to whole-body vibration and low back pain (1986–97). *Int Arch Occup Environ Health* 1999; **72**: 351–65.
- 11 Hoogendoorn WE, van Poppel MNM, Bongers PM, Koes BW, Boulter LM. Physical load during work and leisure time as risk factors for low back pain. *Scand J Work Environ Health* 1999; **25**: 387–403.
- 12 Lings S, Leboeuf-Yde C. Whole-body vibration and low back pain: a systematic, critical review of the epidemiological literature 1992–99. *Arch Occup Environ Health* 2000; **73**: 290–97.
- 13 Kaprio J, Koskenvuo M, Artimo M, Sarna S, Rantasalo I. The Finnish Twin Registry: baseline characteristics. Section 1: materials, methods, representativeness, and results for variables special to twin studies. Helsinki: Department of Public Health Science, University of Helsinki, M47, 1979.
- 14 Dupuis H, Zerlatt G. Vibration behavior in the spinal column. In Dupuis H, Zerlatt G, eds. The effects of whole body vibration. Berlin: Springer, 1986: 31–39.
- 15 Oortman Gertling P, van Drimmelen D, Musson Y. Trillen en schokken tijdens het werk. Resultanten van de inventarisatie-metingen. ICG LA-DR-10-04, 's Gravenhage, 1987.
- 16 Portney LG, Watkins MP. Foundations of clinical research: applications to practice. 2nd edn. Upper Saddle River, New Jersey: Prentice-Hall, 2000: 565.
- 17 Boos N, Wallin Å, Gbedegbegnon T, Aebi M, Boesch C. Quantitative MR imaging of lumbar intervertebral disks and vertebral bodies: influences of diurnal water content variations. *Radiology* 1993; **188**: 351–54.
- 18 Battié MC, Videman T, Gibbons LE, Fisher LD, Manninen H, Gill K. Determinants of lumbar disc degeneration: a study relating lifetime exposures and magnetic resonance imaging findings in identical twins. *Spine* 1995; **20**: 2601–12.
- 19 Landis JR, Koch GG. The measurement of observer agreement for categorical data. *Biometrics* 1977; **33**: 159–74.
- 20 Videman T, Leppävuori J, Kaprio J, et al. 1998 Volvo award in basic sciences: intragenic polymorphisms of the vitamin D receptor gene associated with intervertebral disc degeneration. *Spine* 1998; **23**: 2477–85.
- 21 Cohen J. Statistical power analysis for the behavioral sciences. New York: Academic Press, 1969.
- 22 Rönnemaa T, Karonen SL, Rissanen A, Koskenvuo VA. Relation between plasma leptin levels and measures of body fat in identical twins discordant for obesity. *Ann Intern Med* 1997; **126**: 26–31.
- 23 Bijkerk C, Houwing-Duistermaat JJ, Valkenburg HA, et al. Heritabilities of radiologic osteoarthritis in peripheral joints and of disc degeneration of the spine. *Arthritis Rheum* 1999; **42**: 1729–35.
- 24 Battié MC, Videman T, Gill K, et al. 1991 Volvo award in clinical sciences: smoking and lumbar intervertebral disc degeneration: an MRI study of identical twins. *Spine* 1991; **16**: 1015–21.
- 25 Jones G, White C, Sambrook P, Eisman J. Allelic variation in the vitamin D receptor gene, lifestyle factors, and lumbar spinal degenerative disease. *Ann Rheum Dis* 1998; **57**: 94–97.

- 26 Annunen S, Paassilta P, Lohiniva J, et al. An allele of *COL9A2* associated with intervertebral disc disease. *Science* 1999; **285**: 409–12.
- 27 Wukasch W. Zur Wirkung langzeitiger Ganzkörpervibrationsexposition auf die Wirbelsäule von Traktoristen (eine epidemiologische Studie). Dissertation aus dem Zentralinstitut für Arbeitsmedizin der DDR, 1979.
- 28 Kumar A, Varghese M, Mohan D, Mahajan P, Gulati P, Kale S. Effect of whole-body vibration on the low back: a study of tractor-driving farmers in North India. *Spine* 1999; **24**: 2506–15.
- 29 Luoma K, Riihimäki H, Raininko R, Luukkonen R, Lamminen A, Viikari-Juntura E. Lumbar disc degeneration in relation to occupation. *Scand J Work Environ Health* 1998; **24**: 358–66.
- 30 Brinckmann P, Frobin W, Biggemann M, Tillotson M, Burton K. Quantification of overload injuries to thoracolumbar vertebrae and discs in persons exposed to heavy physical exertions or vibration at the workplace. *Clin Biomech* 1998; **13** (suppl): S1–36.
- 31 International Organization for Standardization. ISO 2631/1 Evaluation of human exposure to whole-body vibration—Part I: general requirements. Geneva: ISO, 1985.
- 32 Videman T, Simonen R, Usenius JP, Österman K, Battie MC. The long-term effects of rally driving on spinal pathology. *Clin Biomech* 2000; **15**: 83–86.
- 33 Hansson T, Broman H, Magnusson M. Back muscle fatigue and seated whole body vibrations. *Clin Biomech* 1991; **6**: 173–78.
- 34 McLain RF, Weinstein JN. Effects of whole body vibration on dorsal root ganglion neurons: changes in neuronal nuclei. *Spine* 1994; **19**: 1455–61.

## Clinical picture

### Obstructive nephropathy and iliac aneurysm

Robbie Dedi, John H Turney

A 67-year-old smoker with intermittent claudication was referred with serum creatinine rising from 167  $\mu\text{mol/L}$  to 326  $\mu\text{mol/L}$  over a period of 3 months. His blood pressure was 169/106 mm Hg. We suspected that he had atherosclerotic renovascular disease. Contrast-enhanced magnetic resonance angiography confirmed bilateral renal artery stenosis and showed a large abdominal aortic aneurysm, extending to involve the common iliac arteries (figure). The left kidney was shrunk (arrows: upper and lower poles), probably due to chronic ischaemic damage, and the right was hydronephrotic. Contrast-enhanced computed tomography showed that the expanded right common iliac artery was compressing the ureter on that side causing renal tract dilatation, and obstructing the functioning right kidney. We placed a temporary right-sided percutaneous nephrostomy tube before right renal artery revascularisation and repair of the abdominal aortic and iliac aneurysms. The patient made a good post-operative recovery.



Department of Nephrology, Wellcome Wing, Leeds General Infirmary, Leeds, West Yorkshire LS1 3EX, UK (R Dedi MRCP, J H Turney FRCP)