wrist bending or other stressful postures.

Epidemiological Evidence

NIOSH reviewed 30 epidemiological studies that addressed workplace risk factors and CTS. Exposed workers in theses studies were usually engaged in job activities involving forceful and repetitive hand/finger or wrist movements and therefore were concurrently subjected to a combination of physical factors. These studies are summarized in Table V–5. Thirteen studies used physical examination or electrophysical indicators to diagnose CTS as well as direct observation or measurement of exposure to risk factors during job activities. The remaining studies either relied on symptom questionnaires to determine health outcomes or self reports and job title descriptions to evaluate exposures. CTS was solely determined by the presence of numbness, pain or tingling in the fingers enervated by the median nerve, and a positive Tinel's or Phalen's test (symptoms triggered upon wrist flexion and palpation) in about half the studies. Nerve conduction (NC) tests were not used in defining cases in these studies. In the other half of the studies, abnormal median nerve conduction was required in addition to symptomatology to diagnose CTS. Since normal NC was often defined and measured differently in various laboratories, CTS case definition is unlikely to be uniform across studies. Several investigations quantitatively estimated force, either from EMG measurements or based on weights of tools or other handled parts, and recorded job task observations. Repetitive hand/wrist movements were sometimes quantitatively measured and categorized based on task frequency, quantity of work performed in a specified time, or ratio of work time to recovery time.

Of the 13 studies (eleven cross-sectional and two case control) that relied on both objective determination of exposure and medical diagnosis of CTS, 10 reported finding statistically significant associations between CTS and exposure to biomechanical risk factors. The reported ORs ranged from 1.1 to 21.3. Some cross-sectional studies provided evidence of an exposure-response relationship with respect to CTS and exposure to force and repetition. Silverstein *et al.*studied 652 workers in 39 jobs from 7 different plants (Silverstein *et al.*1987, Ex. 26–34). Jobs were grouped into high and low repetitiveness and force categories based on cycle time and EMG measurements, respectively. The OR for CTS (defined by physical tests/symptoms) in highly repetitive jobs compared to low repetitive jobs, irrespective of force, was 5.5 ($p<0.05$) in a multiple logistic model that included age, gender, plant site and years on the job. The corresponding OR for high-force jobs, irrespective of repetitiveness, was 2.9 ($p>0.05$) but the OR for combined exposures to high repetition and force was 15.5 ($p<0.05$).

TABLE V–5.—SUMMARY OF EPIDEMIOLOGY STUDIES EXAMINING CARPAL TUNNEL SYNDROME

Study	Job type studied	Physical factors	Exposure basis	Diagnosis	Risk measure (95% CI)[1]
Bovenzi (1991) Ex. 26–1433	Forestry	V	Measurement	Physical exam	OR=21* (NR)
Roquelaure (1997) Ex. 38–396	Manufacturing	F/R/P	Measurement	Physical exam+NC	OR=9.0* (2.4–33.4)
Silverstein (1987) Ex. 26–34	Manufacturing	V/F/R/P	Measurement	Physical exam	OR=1.8–15.5* (1.7–142)
Chatterjee (1992) Ex. 26–942	Rock drilling	V	Measurement	Physical exam+NC	OR=10.9* (1.0–524)
Osorio (1994) Ex. 26–807	Supermarket	F/R/P?	Observation	Physical exam+NC	OR=6.7–8.3* (2.6–26.4)
Barnhart (1991) Ex. 26–1216	Ski manufacture	F?/R/P	Measurement	Physical exam+NC	OR=1.9–40* (1.0–15.8)
Frost (1998) Ex. 38–198	Slaughter house	F/R/P	Measurements	Physical exam+NC	OR=4.2* (1.8–10.1)
Bovenzi (1994) Ex. 26–774	Stone drilling	V	Measurement	Physical exam	OR=e.4* (1.4–8.3)
Baron (1991) Ex. 26–697	Grocery checking	F/R/P	Measurement	Physical exam	OR=3.7 (0.7–16.7)
Moore (1994) Ex. 26–1364	Meat processing	F/R/P	Measurement	Physical exam+NC	OR=2.8 (0.2–36.7)
Chiang (1990) Ex. 26–1118	Frozen Food Packing.	F?/R/P?	Measurement	Physical exam+NC	OR=1.9–11.7* (2.9–46.6)
Chiang (1993) Ex. 26–1117	Fish processing	F/R/P?	Cycle time, EMG	Physical exam	OR=1.1–1.8* (1.1–2.9)
Stetson (1993) Ex. 26–1221	General industry	F/R/P	Checklist	NC only	NR*
Latko (1999) Ex. 38–123	Manufacturing	F/R/P?	Measurement	Physical exam+NC	OR=2.3–3.1 (0.9–10.9)
Armstrong (1979) Ex. 26–348	Sewing machine use	F/R/P	EMG, flexion	Physical exam	OR=1.1–2.0* (1.6–2.5)
Nathan (1988) Ex. 26–990	Multiple industries	F/R/P	Observation	NC only	PRR=1.0–2.0* (1.1–3.4)
Nathan (1992) Ex. 26–989	Multiple industries	F/R/P	Observation	NC, symptoms	PRR=1.0–1.5 (1.0–2.2)
Canon (1981) Ex. 26–1212	Aircraft plant	V/R	Hand tool measurement.	Workers' comp	OR=2.1–7.0* (3.0–17)
English (1995) Ex. 26–848	CTS case/control	F/R/P	Questionnaire	Physical exam	OR=0.4–1.8* (1.2–2.8)

TABLE V–5.—SUMMARY OF EPIDEMIOLOGY STUDIES EXAMINING CARPAL TUNNEL SYNDROME—Continued

Study	Job type studied	Physical factors	Exposure basis	Diagnosis	Risk measure (95% CI)[1]
Feldman (1987) Ex. 26–1210	Electronics plant	F?/R/P	Cycle time flexion	Questionnaire	OR=2.3* (1.4–4.5)
Koskimies (1990) Ex. 26–973	Forestry	V	Job title	Physical exam+NC	NR*
McCormack (1990) Ex. 26–1334	Textile	F/R/P?	Job title	Physical exam	OR=0.4–0.9 (0.3–2.9)
Morgenstern (1991) Ex. 26–1493	Grocery cashiers	F?/R/P	Job title	Questionnaire	OR=1.9 (0.9–3.8)
Punnett (1985) Ex. 26–995	Garment	F?/R/P?	Job title	Physical exam	OR=2.7* (1.2–7.6)
Schottland (1991) Ex. 26–1001	Poultry processing	F/R/P	Job title	NC only	OR=1.9–2.9* (1.1–7.9)
Weislander (1989) Ex. 26–1027	CTS case/control	V/F/R/P?	Questionnaire	Physical exam+NC	OR=1.8–3.3* (1.6–6.8)
Liss (1995) Ex. 26–55	Dental hygienist	F?/R/P	Questionnaire	Questionnaire	OR=3.7* (1.1–11.9)
DeKrom (1990) Ex. 500–41–28	CTS case/control	F/R?/P	Questionnaire	Physical exam+NC	OR=5.4–8.7* (3.1–24.1)
Tanaka (1997) Ex. 26–1185	Household survey	V/P	Questionnaire	Questionnaire	OR=1.8–5.9* (3.4–10.2)
Farkkila (1988) Ex. 26–947	Chair saw use	V	Questionnaire	Physical exam+NC	NR*
SHARP (1993) Ex. 500–41–116	Poultry processing	F/R	Measurement	Questionnaire	NR* (p< 0.0004)
Rosecrance (1994) Ex. 38–203	Newspaper work	F/P/R/ (pinch)	Questionnaire	Physical exam	NR*
Rossignol (1997) Ex. 500–205–24	Manual labor	F/R?/P?	Questionnaire	Surgery for CTS	OR=4.1* (1.5–3.2)
LeClerc (1998) Ex. 500–41–85	Assembly line	R/P?	Questionnaire	Physical exam+NC	OR=3.1–6.6*
Atroshi (1999) Ex. 38–181	General Population	F/P/R/V	Questionnaire	Physical exam+NC	OR=1.0–3.0* (1.4–6.8)
Gorsche (1999) Ex. 500–121–23	Meat packing	V	Questionnaire	Physical exam	NR
Katz (1998) Ex. 38–393	CTS case control	F/R/P	Questionnaire	Physical exam	NR
Kerns (2000) Ex. 500–71–34	Pork processing	F/R/P	Job title	NC only	NR

F=forceful exertions; R=repetitive motion; P=awkward posture; V=vibration; ?=presence of risk factor unclear.
NCV=nerve conduction; IR=incidence rate; OR=odds ratio; PRR=prevalence rate ratio; NR=not reported.
*=p<0.05.
[1] 95% confidence interval expressed for the upper end of the risk measure range.

Chiang *et al.*studied 207 workers from 8 fish processing factories in Taiwan (Chiang *et al.*1993, Ex. 26–1117). Jobs were divided into three groups based on level of repetitiveness and force using cycle times (upper arm movements, not just wrist) and EMG of the forearm flexor muscles. There was a statistically significant trend in prevalence of CTS (defined by physical tests/symptoms) with exposure from low force/repetition, high force *or* high repetition, and high force/repetition. Force alone, but not repetitiveness, significantly predicted CTS (OR=1.8; 95% CI 1.1–2.9).

Several other epidemiological investigations found physical risk factors to be significantly associated with prevalence of CTS. In another Chiang *et al.*study of 207 workers from 2 frozen food processing plants, job tasks were grouped by low and high repetitiveness based on wrist movement cycle time (Chiang *et al.*1990, Ex. 26–1118). Statistical modeling that included gender, age, and cold temperatures resulted in an OR of 1.9 (p<0.05) for CTS (defined by physical tests/symptoms/NC studies). This study stressed the association between CTS and repetitive movements, although some forceful hand/wrist exertion probably existed in the study group.

Stetson *et al.*studied median NC on 103 automotive workers with symptoms consistent with CTS compared with 137 asymptomatic automotive workers and an unexposed group of 105 administrative and professional workers (Stetson *et al.*1993, Ex. 26–1221). Repetitiveness was evaluated by cycle times, hand/wrist grip forces were estimated based on weights of handled tools and parts, and wrist deviation was judged from videotape analysis. Both symptomatic and asymptomatic workers had significantly lower median sensory amplitudes and significantly longer distal latency times than the referent group. The same NC trends were found between automotive workers in jobs requiring grip force greater than 6 pounds compared to those requiring less than 6 pounds. This grip force variable probably combines forceful exertion with wrist deviation. It was not possible to adequately compare repetitive and non-repetitive work since this risk factor was present in almost the entire study group.

Barnhardt *et al.*found ski manufacturing workers with highly repetitive job tasks had a statistically elevated OR of 4.0 (95% CI 1.0–15.8) for CTS (defined by physical tests/NC studies) compared to those workers engaged in non-repetitive tasks (Barnhardt *et al.*1991, Ex. 26–1216). Exposure was evaluated by observational analysis and included repetitive jobs with sustained flexion, extension, or ulnar deviation of the wrists by 45 degrees. The participation rate for this study was lower (less than 70 percent) than most of the other investigations. Armstrong and Chaffin reported that CTS (defined by physical tests/symptoms) was significantly associated (OR=2.0; 95% CI 1.6–2.5) with pinch force exertion (combination of force and deviated wrist posture) in female sewing machine operators (Armstrong and Chaffin 1979, Ex. 26–348). Because of the case-control study design, it is not clear whether deviated postures contributed to the development of CTS or whether the CTS symptoms

led to the use of abnormal postures during work.

Four of the studies addressed CTS and manual work involving vibrating power tools. A case control study by Chatterjee *et al.*found a significant difference (OR=10.9; 95% CI 1.0–524) in the prevalence of CTS cases (defined by NC studies/symptoms) in rock drillers compared with controls (Chatterjee *et al.*1982, Ex. 26–942). The rock drillers were exposed to vibration frequencies between 31.5 and 62 Hertz. The highest relative risks (OR=21.3; p<0.002) for CTS (defined by physical tests/symptoms) were found in forestry workers using chain saws compared to maintenance workers who did not use power tools (Bovenzi *et al.*1991, Ex. 26–1433). Differences in ambient temperatures (potential confounder) between outdoor (chain saw operators) and indoor work (maintenance workers) may have contributed to the results. Koskimies *et al.*reported significant correlations between reductions in NC velocities in the median and ulnar nerves and number of years of vibration exposure in forestry workers who used chain saws greater than 500 hours in the previous 3 years (Koskimies *et al.*1990, Ex. 26–973). The prevalence of CTS (defined by physical tests/symptoms) in these workers was 20 percent. In another study, Bovenzi *et al.*reported an OR of 3.4 (95% CI 1.4–8.3) for CTS (defined by NC studies/symptoms) among stone quarry drillers/carvers exposed to hand-transmitted vibration when compared to polishers who performed manual operations and were not exposed to vibration (Bovenzi et al., 1994, 26–774). In these four studies, flexor tendons and the median nerve of the hand were probably subjected to a considerable degree of forceful exertion as well as mechanical injury during use of these power tools. Vibration can also cause direct damage to the digital arteries leading to sensory loss and numbness.

There were three studies that did not find statistically significant association between CTS and exposure to physical risk factors, even though each reported substantially raised ORs. Moore and Garg found an OR of 2.8 (95% CI 0.2–36.7) for CTS (defined by NC studies/symptoms) among pork processing workers in hazardous jobs compared to safe jobs (Moore and Garg 1994, Ex. 26–1033). Jobs were categorized based on videotape analysis for estimates of force, repetitition and awkward postures. The possible presence of a healthy worker effect (most workers were laid off in the year prior to the study) and the short latency period (8–32 months) limits confidence in the relative risk estimate.

An OR of 6.7 (95% CI 0.8–52.9) for CTS (defined by NC studies/symptoms) was reported in a study of supermarket workers rated for high versus low exposure to repetitive and forceful wrist motions as judged by an ergonomist and industrial hygienist (Osorio *et al.*1994, Ex. 26–807). However, the entire study consisted of only 56 workers grouped into 3 categories for analysis limiting the power of the study to find a statistically significant association. Baron et. al. (Ex. 26–697) also studied CTS (defined by physical tests/symptoms) in 124 grocery store checkers and reported an OR of 3.7 (95% CI 0.7–16.7) compared to 157 non-checkers. Physical examinations were not done on all workers and the relative risk measure was based on responses to a standardized questionnaire. The exposure level for checkers was characterized as having low peak force and a medium level of repetition; therefore, the intensity of exposure to physical risk factors was less than that among workers examined in other studies.

Almost all studies controlled for the obvious confounders of age, gender, and predisposing medical conditions by selection of an appropriate referent population, stratification, or use of a multiple logistic regression model. Many of the cross-sectional studies either excluded workers with pre-existing CTS prior to employment or excluded recently hired workers from the study. Therefore, it is unlikely that the reported associations between CTS and exposure to biomechanical risk factors reflected preferential employment of those with CTS (*i.e.,* the requirements for entry into the cohort made it likely that exposure preceded the onset of CTS).

NIOSH (1997, Ex. 27–1) concluded that there was epidemiological evidence of a positive association between CTS and highly repetitive work, either alone or in combination with other risk factors. They also found evidence of positive associations between forceful work and work involving hand/wrist vibration and CTS. However, NIOSH concluded there was insufficient evidence of an association between CTS and exposure to extreme postures alone because of individual variability in work methods and difficulties in measuring postural characteristics. NIOSH did recognize that there was strong evidence that exposure to a combination of physical risk factors along with non-neutral wrist postures was related to the onset of CTS.

A large number of studies that addressed physical work factors and CTS were submitted into the OSHA docket following publication of the proposal; those that OSHA found to be of adequate study design are included in Table V–5 (Frost *et al.*1998, Ex. 38–198; Roquelaure *et al.*1997, Ex. 500–41–111; Latko *et al.*1999, Ex. 38–123; Rossignol *et al.*1997, Ex. 502–420; Leclerc *et al.*1998, Ex. 500–41–85; Atroshi *et al.*1999, Ex. 38–181; Gorsche *et al.*1999, Ex. 500-121–23; Kearns *et al.*2000, Ex. 500–71–34; Katz *et al.*1998, Ex. 38–393). All but three of these studies (Ex. 500–121–23; Ex. 500–71–34; Ex. 38–393) found significantly increased prevalence of CTS among workers exposed to physical risk factors. The three studies that did not find a statistically significant association did not rely on independent assessment or observation of exposure to physical work factors, but instead used job titles or self-reported survey information to infer exposure. One of these studies, Gorsche *et al.*(Ex. 500–121–23), found an increased prevalence and incidence of CTS in a cross-sectional and longitudinal study of meat packers but it was not statistically significant. Kearns *et al.*(Ex. 500–71–34), who ascertained cases only by nerve conduction studies and did not rely on symptoms or clinical evaluation to diagnose CTS, also failed to find a statistically significant association. Katz *et al.*(Ex. 38–393) studied factors associated with long-term disability rather than the development of CTS.

In contrast, three studies that did measure or observe exposures and used a combination of symptoms, physical tests, and nerve conduction velocity measurements to diagnose CTS found strong associations with exposure to repetition and/or force (Exs. 38–198, 500–41–111, 38–123). Another study, the SHARP study (Ex. 500–41–116) of poultry processing workers summarized in the hand/wrist tendinitis section above, found that the number of forceful exertions per hour was significantly predictive of CTS (p=0.004).

Many studies of CTS contained in the rulemaking docket are not included in Table V–5 either because it was questionable whether exposure to physical risk factors occurred or because the study did not address the relationship between physical risk factors and CTS (Nathan and Keniston 1993, Ex. 351–14; Stallings *et al.*1997, Ex 351–20; Franzblau *et al.*1994, Ex. 38–175; Nordstrom *et al.*1988, Ex. 500–25–9; Zetterberg and Ofverholm 1999, Ex. 500–121–78). Other studies were not included on OSHA's summary table because they used a flawed study design or a flawed statistical analysis to examine the relationship between exposure to biomechanical risk factors and CTS (Malchaive *et al.*1996, Ex. 500–

66–5; Homan *et al.*1999, Ex. 38–172; Olafsdotti *et al.* 2000, Ex 38–288.

One of the above studies is among several papers published by Dr. Peter Nathan and colleagues, which were based on two group of workers whom they have followed prospectively (Ex. 26–990; Ex. 26–988; Ex. 26–989; Ex. 26–1294; Ex. 26–517; Ex. 38–437; Ex. 38–13; Ex. 351–14). Because of the importance of these studies to the ergonomics rulemaking, they are addressed in detail here. In one of the earlier studies (Nathan *et al.*1988, Ex. 26–990), nerve conduction was assessed on 471 randomly chosen individuals from four industries (steel mill, meat/food packaging, electronics, and plastics manufacturing). The group was divided into the following exposure categories:

- Group 1, very low force, low repetition;
- Group 2, low force, very high repetition;
- Group 3, moderate force, moderate repetition;
- Group 4, high force, moderate repetition; and
- Group 5, high force, high repetition.

No significant difference in median nerve sensory latency values was found between Group 1 and Group 2, which differed primarily by the amount of repetition exposure. There was a statistically significantly higher number of subjects with median nerve slowing in Group 5 compared to Group 1, but not when compared to Groups 2, 3, or 4. When individual hands were used to base calculations rather than number of individuals, only Group 3 showed a significantly higher prevalence of median nerve conduction slowing. When prevalence ratios were calculated, Groups 3, 4, and 5 had significantly higher PRs compared to Group 1.

This same group of workers was followed up for five years in a 1992 study (Ex. 26–988) and eleven years in a 1998 study (Ex. 38–13). The study used hands, rather than individuals, as the basis for analysis. The authors stated that they found no significant difference in the prevalence of median nerve slowing among any of the exposure groups. The authors claimed to have confirmed this finding in a second combined cohort of Japanese and American industrial workers (Ex. 38–437) as well as validated their exposure categories (Ex. 26–1294). They went on to show that slowing of nerve conduction was significantly associated with obesity (Ex. 26–989), body mass index (Ex. 26–517), wrist depth/width and a number of other non-occupational risk factors (Ex. 351–14).

However, their research has a number of flaws in the study design, analysis and interpretation of the results, which call their conclusions into doubt. Chief among these is the failure to adequately justify and validate their grouping and rank order of occupational hand use. This provides multiple opportunities for exposure misclassification and will tend to underestimate the association of exposure with health outcome. This aspect of the study has been criticized by several experts (Ex. 26–1010; Ex. 26–952; Tr. 1000). Despite this potential for misclassification, there was a significant increase in prevalence between the lowest (Group I) and higher exposure groups combined (Groups III, IV, and V) in the cross-sectional study (Ex. 26–990). Others have also concluded that, methodological shortcomings aside, the articles by Nathan *et al.*demonstrate a positive exposure-response relationship between ''occupational hand activity'' and slowed conduction of the median nerve (Tr. 1519–1522; Tr. 9862). Others have testified that alternative exposure grouping of the data resulting in less exposure misclassification would result in a clear exposure-response relationship between job group and median nerve latency (Punnett testimony, Ex. 37–2; Gerr testimony, 27–2). Some who testified at OSHA's informal hearing have also stated that Dr. Nathan's articles use statistical presentation and analysis methods that obscure the evidence, and that not enough data are presented for the reader to independently evaluate whether the authors' conclusions are justified (Tr. 1521; Tr. 7850.). Low participation rates, unusual and inconsistent case definition, and inappropriate statistical analysis may also have limited the ability to detect increases in CTS prevalence over time in these studies with respect to work-related biomechanical factors. For example, the authors reported in the baseline study that they randomly selected the study participants (Ex. 26–990). However, they did not report the proportion of those who were selected and invited that actually participated. Since the 471 subjects represented only 26 percent of the total workforce of the participating companies, the representativeness of the sample is unknown, the ability to generalize from the data is highly limited, and the potential for selection bias is substantial. There is no comparative information on participants and non-participants with respect to demographics, occupational history or exposure, or medical history. The lack of clarifying information is particularly problematic because the direction of the selection bias could be either toward or away from the null value. This problem affects not only the 1988 baseline study but all future follow-up studies of the same cohort. Because of these criticisms, OSHA finds that the Nathan studies do not convincingly demonstrate a lack of association between workplace exposure to biomechanical risk factors and CTS.

In his written testimony, Dr. Peter Nathan calls into question the case definition for CTS relied upon by OSHA in their evaluation of the epidemiological studies (Ex. 32–241–3–13). He testifies that ''there is general agreement among experts that classic symptoms associated with positive electrodiagnostic findings for the median nerve are necessary for a diagnosis of CTS'' but that ''there is no general agreement that symptoms, in the presence of negative electrodiagnostic findings is equivalent to CTS.'' (Id., pg 4). Dr. Nathan then goes on to criticize OSHA and NIOSH, in their 1997 review, for accepting studies that use CTS case definitions without electrodiagnostic confirmation. He argues that longitudinal studies are the only study design of value for determining causation and concludes ''if one required electrodiagnostic studies for a valid case definition of CTS, and a longitudinal design for establishing temporal relationships, then only one [his own] of the 31 studies analyzed by NIOSH would have met standard criteria for establishing causation.'' (Id., pg 11).

OSHA accepts that specific symptoms determined during clinical exam in combination with objective evidence of median nerve dysfunction through electrodiagnostic tests is the most definitive case definition for CTS at the present time. This has been supported by expert testimony not only from Dr. Nathan but Dr. Frederick Gerr (Ex. 37–2) and Dr. Gary Franklin (Tr. 13363). OSHA also does not dispute lack of agreement among experts on CTS diagnosis when symptoms exist in the presence of normal median nerve conduction. However, the relevant issue is whether clinical symptoms and signs in the absence of electrodiagnostic testing are an invalid CTS case ascertainment for the purposes of evaluating epidemiological evidence to determine if work-related physical factors are associated with the disorder. NIOSH addressed the issue in its 1997 review and cited studies that found satisfactory correlations between CTS diagnosed by nerve conduction and the disorder diagnosed by symptom questionnaire and physical examination (Ex. 26–1501; Ex. 26–439). It was also reported that clinical examination for CTS diagnosis without the benefit of

nerve conduction studies has a sensitivity of 84 percent and a specificity of 76 percent (Ex. 26–1208). This indicates that without the aid of electrodiagnostics, one would make a CTS diagnosis when the disorder is not present (false positive) in about one in four subjects. On the other hand, clinical exam is expected to miss a diagnosis (false negative) when CTS is present about one in six subjects. While this degree of sensitivity and specificity may not be acceptable when making treatment decisions, such as surgery, OSHA does not believe it introduces substantial bias for purposes of evaluating epidemiological evidence.

OSHA does not agree with Dr. Nathan's assertion that only longitudinal studies are relevant in evaluating causation. Longitudinal prospective cohort studies are indeed the strongest epidemiological study design, especially for establishing temporal relationships. However, they often require extended periods of time, are more costly, and are not as numerous other study designs. Other types of epidemiology, such as cross-sectional and case-control studies, add evidence of causality in terms strength and consistency of association and exposure-response.

OSHA has examined the epidemiological data base and finds that even if one restricts the evidence to studies that used abnormal median nerve conduction to establish CTS case ascertainment, there is reasonable evidence of association between repeated, forceful exertions of the hand and CTS. There were eleven studies either reviewed by NIOSH in their 1997 review or submitted to the OSHA docket during the rulemaking process that found statistically significant associations between combinations of force, repetitive motion, awkward posture, and segmental vibration and CTS defined by electrodiagnostic criteria (Ex. 38–396; 26–942; 38–198; 26–1118; 26–1221; 23–1001; 26–1027; 500–41–28; 500–41–85; 38–181; 26–973). The entire body of epidemiological studies described in the preceding paragraphs is also supported by impressive biomechanical and psychophysical data, discussed in the following subsection, that shows sustained force on the flexor tendons along with flexion/extension of the wrist increases carpal tunnel pressure and reduces exertion and perceptions of discomfort. In his written testimony (Ex. 37–2), Dr. Fredric Gerr discussed his evaluation of the epidemiological studies that used abnormal nerve conduction to diagnose CTS and made the following statement in his oral testimony at the hearing:

> However, when significant positive associations between work and carpal tunnel syndrome are observed repeatedly, in study after study, by investigator after investigator, in country after country and at many different times, we must ask ourselves why. In my opinion, after reading these studies and considering all the possible reasons why so many studies show this relationship, the most reasonable, plausible, and likely explanation is that work really did cause the carpal tunnel syndrome observed in these studies. (Tr. 1525)

Biomechanical and Psychophysical Evidence

Several clinical and cadaver studies confirm that fingertip force, wrist flexion/extension, repetitive tasks and combinations of the above are able to raise carpal tunnel pressure (CTP) in a dose-dependant manner. Mean CTP was raised from 5 mm Hg in a neutral wrist position to approximately 100 mm Hg at 60 degree wrist extension and 80 mm Hg at 60 degree flexion in a population of CTS patients and controls (Weiss et. al. 1995, Ex. 26–236). CTP has been shown to significantly increase with increasing finger tip force (Rempel et. al. 1997, Ex. 26–889) and with clenching a fist or holding an object in a power grip (Seradge et. al. 1995, Ex. 26–325). There was a two- to three-fold increase in CTP when performing a repetitive task involving change in wrist posture 20 times per minute for 5 minutes (Rempel et. al. 1994, Ex. 26–1151). The elevated CTPs found in these human biomechanical studies are within the range of neuronal pressures shown to impair blood flow, axonal transport, and nerve conduction in experimental animals.

Psychophysical data support the biomechanical findings. They show that maximum acceptable weight (MAW) and torque (MAT) decrease and perceived exertion and discomfort increase with the frequency and duration of repetitive wrist motions. The psychophysical method was used to determine the preferred weights for one-handed horizontal transfer tasks (*e.g.* hand/wrist motion used to move an object across a supermarket scanner). Frequency and duration of the transfer movement significantly decreased MAW in an exposure-dependent manner and increased perceived exertion over an eight-hour session (Krawczyk et. al. 1992, Ex. 26–974). In another study, MAT was reduced over the course of a seven-hour trial of repeated flexion and extension of the wrist (Snook et. al. 1995, Ex. 26–212). The magnitude of MAT reduction correlated with the frequency of the task and perceived discomfort increased with increasing repetition.

Conclusion

The 1997 NIOSH report concluded the following with regard to the relationship between work-related physical risk factors and CTS:

> Based on the epidemiologic studies reviewed, especially those with a quantitative evaluation of the risk factors, the evidence is clear that exposure to a combination of the job factors studied (repetition, force, posture, etc.) increases the risk of CTS. This is consistent with the evidence in the biomedical, physiological, and psychosocial literature (Ex. 26–1).

OSHA also finds convincing evidence that jobs involving repetitive and forceful movements of the hand and wrist are linked to CTS. The epidemiological findings are supported by clinical, biomechanical, and psychophysical studies showing that repetitive tasks involving flexion/extension of the wrist and force to the flexor tendons result in substantial increases in CTP, reductions in measured exertion, and perceptions of discomfort. This evidence is clearly consistent with the pathophysiology of CTS in which elevated CTP can lead to compression of the median nerve resulting in the clinical signs and symptoms characteristic of this MSD. OSHA finds that the epidemiological and biomechanical literature convincingly demonstrates a causal relationship between forceful and repetitive exertions to the hand, especially in combination with a flexed wrist, and an increased risk of carpal tunnel syndrome. Forceful and repetitive exertion includes vibration from the use of hand-held power tools.

Hand-Arm Vibration Syndrome

Hand-arm vibration syndrome (HAVS) refers to a collection of signs and symptoms that occurs when vibration from a tool is transferred to a worker's hand or arm. The symptoms include numbness, blanching of the fingers, pain in response to cold exposure, and reduction in grip strength. These manifestations are similar to Raynaud's phenomenon triggered by cold temperatures. HAVS symptoms are believed to be the result of both neurological and circulatory disturbances, probably occurring independently and by unrelated mechanisms. Vibration may directly injure (as opposed to indirect damage from compression as in CTS) peripheral nerve endings and neuroreceptors causing numbness, tingling and pain in the fingers. Histopathology of persons suffering from HAVS indicate that

vibration may also directly damage endothelial cells of the digital arteries resulting in a lack of response to certain vasodilators and thickening of the vessel walls. These physiological changes can cause vascular constriction and ischemia of the surrounding musculoskeletal and neural tissue. The clinical outcome is blanching of the fingers (''white finger''), loss of feeling, muscle weakness, and weakened grip strength.

Epidemiological Evidence

NIOSH reviewed 20 post-1988 epidemiological studies that addressed workplace risk factors and HAVS. Table V–6 summarizes some key aspects of these investigations, such as the occupations examined, the biomechanical risk factors they were exposed to, whether exposures were directly observed or measured during the study, and whether the health outcomes were verified by trained medical personnel during physical examination. Previous investigations were reviewed as part of the 1989 NIOSH criteria document on exposure to HAV (Ex. 26–392). In its 1997 evaluation, NIOSH featured four cross-sectional studies (Bovenzi *et al.*1988, Ex. 26–1500; 1994, Ex. 26–1239; 1995, Ex. 26–354; Nilsson *et al.*1989, Ex. 26–1148) and one prospective study (Koskimies *et al.*1992, Ex. 26–1490), which met most of NIOSH's criteria for high quality. These investigations determined HAVS based on medical exam and did not strictly rely on self-reported questionnaires. Standard and relatively uniform diagnostic criteria were used in defining HAVS cases. This generally included episodes of cold-provoked, well-demarcated blanching of the fingers, occurrence of vibration white finger attacks after employment and following use of power tools, and abnormal digital artery response to cold provocation. All studies used the Stockholm Taylor-Palmear scale to grade and stage symptoms. The five investigations included vibration measurements of exposure on tools used by the study subjects combined with information on exposure time obtained by direct interview.

The four cross-sectional studies found statistically significant positive relationship between exposure to vibration and prevalence of HAVS. The strength of this association was high with reported ORs ranging from 6 to 85. The one prospective study showed significant decreases in HAVS prevalence with decreasing exposure to vibration over time. All five investigations contributed evidence of exposure-response relationships between HAVS and vibration acceleration or duration of exposure. One study also documented a relationship between exposure and symptom severity.

TABLE V–6.—SUMMARY OF EPIDEMIOLOGY STUDIES EXAMINING HAND-ARM VIBRATION

Study	Job type studied	Physical Factors	Exposure basis	Diagnosis	Risk measure (95% CI)[1]
Nilsson (1989) Ex. 26–1148	Pulp mill machining.	V	Tool acceleration	Physical exam	OR=14–85* (15–486)
Bovenzi (1995) Ex. 26–354	Forestry	V	Chain saw acceleration.	Cold provocation	OR=6.2–32* (11–93)
Bovenzi (1994) Ex. 26–1239	Stone drilling	V	Tool acceleration	Physical exam	OR=9.3* (4.9–17.8)
Bovenzi (1988) Ex. 26–1500	Stone cutting	V	Tool acceleration	Physical exam	OR=6.1* (2.0–19.6)
Brubaker (1987) Ex. 26–762	Forestry	V	Chain saw acceleration.	Symptoms ischemia.	NR
Koskimies (1992) Ex. 26–1490	Forestry	V	Chain saw acceleration.	Physical exam	NR
Brubaker (1983) Ex. 26–763	Forestry	V	Questionnaire	Symptoms ischemia.	NR
Dimberg (1991) Ex. 26–1395	Aircraft machining.	V	Questionnaire	Questionnaire	NR
Krivekas (1994) Cited in Ex. 26–1	Forestry	V	Questionnaire	Pyhsical exam	OR=3.4–6.5* (2.4–17.5)
Letz (1992) Ex. 26–384	Ship-yard	V	Tool acceleration	Questionnaire	OR=5.0–40.6* (11–176)
McKenna (1993) Ex. 26–745	Machine riveting	V	Questionnaire	Cold provocation	OR=24* (3.1–510)
Mirbod (1992) Ex. 26–1492	Forestry	V	Chain saw acceleration.	Physical exam	NR
Mirbod (1997) Ex. 500–121–49	Motorcyclists	V	Handlebar acceleration.	Questionnarie	NR*
Mirbod (1999) Ex. 500–121–48	Metal grinding	V	Job title	Physical tests	NR*
Mirbod (1994) Ex. 26–1491	Multiple industries.	V	Tool acceleration	Questionnarie	OR=3.8* (2.1–6.8)
Musson (1989) Ex. 26–743	Power tool use	V	Tool acceleration	Questionnaire	NR
Nagata (1993) Ex. 26–1494	Chain saw operation.	V	Job title	Physical exam	OR=7.1* (2.5–19.9)
Saito (1987) Ex. 26–1440	Chain saw operation.	V	job title	Cold provocation	NR
Palmer (1998) Ex. 500–121–56	Pavement breaking.	V	estimated tool acceleration.	Physical exam cold test.	OR=2.2–2.6* (1.4–4.8)
Palmer (2000) Ex. 500–121–57	Multiple industries.	V	Questionnaire	Questionnaire	PRR=1.5–2.2* (1.9–2.4)
Lindsell (1999) Ex. 500–205–13	Dockyard work	V	Job title	Cold provocations.	NR*
McGeoh (2000) Ex. 500–41–96	Welding	V	Questionnaire	Questionnaire	NR*
Shinev (1992) Ex. 26–836	Polishing	V	Tool acceleration	Physical exam	NR

TABLE V–6.—SUMMARY OF EPIDEMIOLOGY STUDIES EXAMINING HAND-ARM VIBRATION—Continued

Study	Job type studied	Physical Factors	Exposure basis	Diagnosis	Risk measure (95% CI)[1]
Starck (1990) Ex. 26–1510	Machining	V	Tool acceleration	Questionnaire	NR
Virokannas (1995) Ex. 26–891	Railway	V	Questionnaire	Questionnaire	NR
Miyashita (1992) Ex. 26–1223	Construction	V	Questionnaire	Questionnaire	OR=0.5 (0.1–11.8)

V=vibration; OR=odds ratio; NR=not reported.
* =p<0.05.
[1] 95% confidence interval expressed for the upper end of the risk measure range.

Bovenzi *et al.*found a significantly greater prevalence of HAVS in a group of 222 active forestry workers engaged in chain saw work as compared to randomly chosen shipyard workers unexposed to hand vibration (Bovenzi *et al.*1995, Ex. 26–354). The reported OR was 11.8 (95% CI 4.5–31.1) for all forestry workers and 6.3 (95% CI 2.3–17.1) for workers only using ''anti-vibration'' saws. The study found a nearly linear dose-response between HAVS prevalence and both vibration acceleration and years of exposure. Vibration exposure was determined from questionnaire reports on frequency of chain saw work and direct measurement of vibration produced by 30 different saws.

In two earlier studies, Bovenzi *et al.*examined HAVS in stone quarry drillers and carvers exposed to vibration from hand-held power tools along with an unexposed referent group. The first investigation found a statistically significant HAVS prevalence (OR=6.1; 95% CI 2.0–19.6) in 75 drillers/cutters compared to unexposed mill workers (Bovenzi *et al.*1988, Ex. 26–1500). There was a significant association between the level of vibration acceleration and severity of symptoms. In a much larger study of 570 quarry drillers/carvers and 258 polishers/machine operators not using power tools, an OR of 9.3 (95% CI 4.9–17.8) was reported (Bovenzi *et al.*1994, Ex. 26–1239). HAVS prevalence showed a significant increasing trend with estimates of lifetime vibration exposure.

In the Nilsson study, HAVS was examined in 89 platers and 61 office workers from a pulp mill machine manufacturing plant (Nilsson *et al.*1989, Ex. 26–1148). Prevalence of HAVS (OR=85; 95% CI 15–486) was much greater for platers with current exposure to vibration than unexposed office workers. There was a clear dose-response between HAVS and years of exposure.

Koskimies *et al.*investigated HAVS in a group of 124 forestry workers from 1972 to 1990 using a series of ten cross-sectional studies over time (Koskimies *et al.*1992, Ex. 26–1490). Their analysis showed a monotonic decrease in prevalence from 40 percent in 1972 to 6 percent in 1990. In a subcohort of 57 workers followed prospectively, HAVS cases decreased from 35 percent in 1975 to 6 percent in 1986. Over the same time period, modifications in chain saws used by the workers resulted in a reduction vibration acceleration from 14 m/s^2 to 2 m/s^2. While it is likely that the decline in HAVS is due to changes in the vibration acceleration, exposures and outcomes were never linked for individual workers.

The 1989 NIOSH criteria document (Ex. 26–392) provides some epidemiological evidence for an exposure-response relationship and temporal association between HAVS and vibration exposure. NIOSH analyzed HAV acceleration levels and prevalence of HAV-related vascular symptoms from 23 cross-sectional studies and found the two variables linearly correlated (R=0.67; p<0.01). Many of these earlier studies determined latency between vibration exposure and onset of HAVS symptoms providing some evidence of a temporal relationship. Unfortunately these data may be subject to recall bias since the mean latency was about six years and onset of symptomatology was often self-reported.

Most studies accounted for potential age-related effects by stratification of the analysis or through the use of multiple logistic regression. These studies also controlled for non-occupational disorders that involve symptoms similar to HAVS, such as idiopathic Raynaud's phenomena, peripheral neuropathy, alcohol-related illness, etc. According to NIOSH (1997, Ex. 26–1), it does not appear that these potential confounders account for the fairly strong and consistent association between HAVS and vibration.

Four studies that address vibration and HAVS were submitted into the OSHA docket following publication of the proposal (Mirbod *et al.*1999, Ex 500–121–48; Mirbod *et al.*1997; Ex 500–121–49; Ex 500–205–21; Palmer *et al.*1998, Ex 500–121–56; McGeoch and Gilmour 2000, Ex. 500–42–96; These are summarized in Table V–6. Studies that either measured tool acceleration or based HAVS on a combination of symptoms and medical tests found a significant association between segmental vibration exposure and this MSD (Ex. 500–121–49; Ex. 500–121–56 Ex. 500–121–48).

Conclusion

The 1997 NIOSH report concluded the following with regard to the relationship between work-related physical risk factors and HAVS:

> The 20 epidemiological studies show *strong evidence* of a positive association between high level exposure to hand-arm vibration and vascular symptoms of hand-arm vibration syndrome (HAVS). These studies are of workers with high levels of exposures such as forestry workers, stone cutters or carvers, shipyard workers, or platers. These workers were typically exposed to HAV acceleration levels of 5 to 36 m/s^2 * * * There is substantial evidence that as intensity and duration of exposure to vibrating tools increase, the risk of developing HAVS increases. [Ex. 27–1, Emphasis in original]

OSHA agrees with the NIOSH statements that intensity and duration of exposure to vibrating tools is linked to the risk of developing HAVS. Most of the epidemiological studies show a strong and consistent association between high-level exposure to HAV and HAVS symptomatology. The data indicate there are strong exposure-response relationships between the magnitude and duration of exposure and HAVS prevalence and severity. Some studies indicate temporal correlation between the chronic use of vibrating power tools and the onset of the disorder. A causal association between vibration and HAVS is consistent with clinical evidence showing that vibration damages nerve tissue and blood vessels in the fingers leading to symptoms characteristic of this MSD. Therefore, OSHA concludes that workers exposed to segmental vibration exposure, such as from long-term use of hand held power tools, are

at increased risk of developing hand-arm vibration syndrome.

Hypothenar Hammer Syndrome

Hypothenar hammer syndrome (HHS) is a collection of vascular and neurological signs and symptoms that have been related to repeated trauma to the hand. HHS is associated with striking or pushing hard objects with the hypothenar region of the hand using the hook of the hamate bone as an anvil. At this location, the palmar blood vessels of the ulnar artery and the sensory branch of the ulnar nerve lie virtually unprotected near the surface of the skin and become trapped between ''the hammer'' (i.e. the hard object) and ''the anvil'' (i.e. the hamate bone). As a result, the blood vessels and nerves are especially vulnerable to injury by external mechanical stress. The repeated blunt trauma can lead to ulnar artery spasm, aneurysm formation, and/or thrombosis. These lesions cause arterial occlusion, vascular insufficiency, and post-traumatic ischemia of the surrounding tissue. The damage to neural tissue and reduced blood flow to the fingers are responsible for the most frequently reported symptoms of pain, numbness, cold feeling, discoloration and stiffness of the affected digits. A diagnosis can be made based on symptoms and a physical examination test of the radial and ulnar arterial blood supply to the hand, termed the Allen test. This test measures reflow time through the arteries following compression. Reflow time is substantially delayed in patients that suffer ulnar artery occlusion. More recently, arteriography has been used to confirm diagnosis of HHS. If elimination of the contact stress fails to resolve symptoms, vascular reconstructive surgery is often performed (Ex. 500–41–29).

There are four case studies of hospital or surgery clinic patients with HHS in the OSHA docket that have consistently implicated occupational exposure to repeated palmar trauma as a critical risk factor (Conn *et al.*1970 Ex. 26–821; Vayssairet *et al.*1987 Ex. 500–41–47; DeMonoco *et al.*1999 Ex 500–41–39; Ferris *et al.*2000 Ex. 500–41–33). These studies report on 58 patients altogether. In almost every case, the individuals suffering from the disorder reported a history of repetitive blunt trauma to the hand related to their jobs. Occupations such as carpenter, metal worker, machinist, and mechanic were most often cited. More infrequently, the HHS patients were engaged in hobbies in which the hand was exposed to frequent impact, such as karate and wood working. It should be noted that use of the hand as a hammer or to repeatedly apply direct impact to a hard object is a specialized combination of repetitive motion and mechanical force applied directly to a specific anatomical region. Other studies have reported HHS in workers repeatedly exposed to high-frequency mechanical stress to the palm from occupational use of hand-held vibrating tools (Nilsson *et al.*1989 Ex. 26–1148; Kaji *et al.*1993 Ex. 500–41–70). Thus, HHS is clearly another example of a work-related injury that occurs as a result of combined exposure to biomechanical risk factors (*e.g.* repetition, force, vibration) associated with other MSDs of the upper extremities.

Epidemiological Evidence

Besides the case studies mentioned above, there were two cross-sectional studies in the rulemaking docket that investigated HHS among workers (Little and Ferguson 1972 Ex. 500–41–89; Kaji *et al.*1993 (Ex. 500–41–70). Little and Ferguson examined 79 male vehicle maintenance workers from Australia for HHS who self-reported daily use of the hand as a hammer and 48 employees in the same shops who did not report habitual hand hammering. HHS was identified by both a positive Allen and Doppler test. The Doppler test charted blood flow from the radial artery and had shown good correlation with ulnar artery occlusion as measured by arteriography. The prevalence of HHS was 14 percent (11 out of 79) in the exposed workers and 0 percent in the referent population. The mean duration of employment (29.9 years) was significantly greater ($p<0.02$) in subjects with HHS than in men exposed to repeated trauma without the disorder (mean duration of 18.7 years).

Kaji *et al.*used arteriography to examine the hands of 330 Japanese workers that used vibrating tools in mining, forestry, and several other industries. They found a 7.3 percent (24 cases) prevalence of HHS among the workers. The injured subjects were predominantly coal miners, rock drillers and forestry workers that reportedly used air and jack hammers or chain saws. All suffered from HAVS as well as HHS. The mean duration of vibration exposure was 19.4 years (range 5 to 30 years). There was no unexposed referent group and no direct observation or measurements of vibration exposure in the study.

Conclusion

There is clear evidence that repeated and forceful impact between the hypothenar region of the hand and hard objects, such as hand hammering while on the job, or frequent exposure to mechanical stress from use of hand-held vibrating tools increase the risk of developing HHS. The occluded blood vessels that develop in the palmar region of the hand as a result of the blunt trauma created by these occupational risk factors have been cited in numerous case studies. The pathophysiology that links the initial damage with tissue ischemia and the characteristic symptoms that define HHS are also well established in the medical literature. Although limited in terms of numbers and design, the epidemiological findings are consistent with the clinical evidence and provide support for a causal association between repeated and forceful contact stress to the hand and this disorder. OSHA concludes that workers exposed to repeated and forceful impact between the hypothenar region of the hand and hard objects, such as hand hammering while on the job, or frequent exposure to mechanical stress from use of hand-held power tools, are at increased risk of developing hypothenar hammer syndrome.

E. Disorders of the Low Back

Low-back pain has long been associated with the performance of heavy physical work (Hales and Bernard 1996, Ex. 26–896; Klein, Jensen, and Sanderson 1984, Ex. 26–972; Rowe 1969, Ex. 26–318; 1971, Ex. 26–319). Studies have demonstrated that back disorder rates vary substantially by industry, occupation and by job within given industries or facilities (see Bigos *et al.*1986a, Ex. 26–871; Riihimaki *et al.*1989a, Ex. 26–58; Schibye et al 1995, Ex. 26–1463; Skovron *et al.*1994, Ex. 26–795). Recently, a NIOSH review (Bernard and Fine 1997, Ex. 26–1) concluded that several work-related factors are associated with low-back disorders. The National Academy of Sciences (NAS 1999, Ex. 26–37) also concluded that there is an association between certain work factors and low-back disorders. This section summarizes and discusses the evidence that physical work-related risk factors contribute to the pathogenesis of specific disorders of the back. The risk factors are (1) heavy physical work, (2) lifting and forceful movement, (3) bending, twisting and awkward posture, (4) static work postures, and (5) whole body vibration. Exposure to several factors often occurs concurrently in occupational settings and the evidence indicates that the risk of injury is greatest when more than one factor is present, reinforcing the concept that these MSDs are both multi factorial in etiology and that the joint effects of these risk factors can be synergistic. The

terms ''back disorder'' or ''back MSDs'' are used to encompass all adverse health outcomes related to the back.

There are several types of evidence that interrelate to support the five risk factors stated above as causative factors for MSDs of the back. Information on pathophysiology provides evidence that links risk factors to the physiological, anatomical, and pathological alterations in soft tissues of the back. This speaks to the biologic plausibility that work-related risk factors contribute to these injuries. There is also a large volume of epidemiological data that provides evidence of an association between worker exposure to the identified risk factors and the occurrence of MSDs of the back. Finally, there is biomechanical and psychophysical laboratory research that provides much corroboration and adds to the plausibility and coherence arguments for a causal association determination.

Epidemiologic and laboratory-based research methods have both been used to evaluate the significance of various risk factors associated with work-related musculoskeletal disorders (MSDs). Epidemiologic studies are designed to look for significant associations between exposure to ergonomic risk factors and selected health outcomes (ranging from medically diagnosed disease entities to subjective reports of pain or discomfort) in selected populations of workers. NIOSH (Bernard and Fine, 1997, Ex. 26–1) performed a comprehensive review of the occupational epidemiology back MSD literature and after carefully selecting those highest quality studies, performed an assessment of the 42 studies by type of work-related risk factor. This evaluation draws from the NIOSH assessment and appends it with additional and more recent studies added to the record.

Although epidemiologic studies provide important insights into understanding the causes of MSDs, these studies are sometimes criticized due to their inability to precisely measure exposures to risk factors and the associated biomechanical and/or physiological responses to these exposures. Biomechanical models and laboratory studies do not replace epidemiological studies. However, these approaches provide important complementary information toward understanding the complex process of how exposures to ergonomic risk factors result in physiological responses that may ultimately lead to work-related injuries and illnesses. Presented here is a summary of laboratory studies and biomechanical models of work factors associated with increased risk of low-back injuries and disorders.

Laboratory studies are controlled scientific investigations of how humans respond when exposed to specific ergonomic risk factors (*e.g.,* forceful exertions, awkward work postures, high repetition, etc.) during simulated work activities. Responses include both objective biomechanical/physiological measurements, such as the electromyographic (EMG) activity of a working muscle, and subjective psychophysical measurements, such as ratings of perceived exertion. Most of the studies cited were performed in true laboratory settings. A few studies were performed in operational workplaces modified as necessary to collect data under carefully controlled conditions. Because of ethical issues related to the protection and safety of human subjects, laboratory studies are designed to keep exposures to risk factors at levels below the threshold of injury. As a result, these studies are generally incapable of ''proving'' a relationship between exposure and injury. Despite this limitation, laboratory studies provide important scientific insights as to how the body responds to ergonomic stresses. Combined with pathophysiological models of musculoskeletal injury mechanisms and epidemiological findings of positive relationships between exposure to ergonomic risk factors and musculoskeletal injury, laboratory studies are an essential element in understanding the causes and prevention of work-related overexertion injuries.

Biomechanical models simulate and/or predict how the musculoskeletal system responds to work factors such as external loads placed on the hands, work posture, and movement dynamics. These models can be used to estimate musculoskeletal stresses in the absence of a human experiment.

To understand the mechanisms by which work causes or contributes to the genesis or expression of low-back pain, it is first necessary to comprehend basic low-back anatomy and potential sources of pain. The majority of low-back disorders involve soft tissues (muscle and ligament) or the three-disc complex (the intervertebral disc and two facets). The latter may involve degenerative disc disease, disc herniation or osteoarthritic conditions. To understand how the performance of work causes lumbar disc disease, a review of lumbar anatomy, disc biochemistry, and disc biomechanics is presented here. With this foundation, pathogenic models are better appreciated. Several references are available for additional information (Bogduk and Twomey 1991, Ex. 26–720; Chaffin and Andersson 1991, Ex. 26–420; Williams, McCulloch, and Young 1990, Ex. 26–1563; Wiesel *et al.*1996, Ex. 26–1394). This discussion of the anatomy of the low back region is followed by a summary of the occupational epidemiology literature on the low back. This section is followed by a discussion of the biomechanical and laboratory research literature on stressors on the back.

The epidemiology literature is examined, to the extent possible, by grouping by specific work-related stress factor. The biomechanical and laboratory section discusses specific stressors separately for soft tissue disorders, disc disorders, and arthritis/spondylosis. In the conclusion section OSHA makes a determination of causality based on the consistency and strength of the epidemiology evidence and the coherence with the biomechanical and laboratory evidence. OSHA makes a determination of causality on each occupational risk factor examined, where possible; however, the final determination of work-related back MSDs is based on the totality of the evidence, not on each factor separately. OSHA believes that determining causal associations between individual work-related risk factors and MSDs is helpful, both in making a final determination of causality and in determining ways to abate risk. However, in discussing the epidemiology evidence it becomes clear that work often involves simultaneous exposure to multiple risk factors, even though in any particular situation exposure to one risk factor may predominate.

Anatomy of the Low Back

The lumbar spine is required to redistribute forces related to both intrinsic weight bearing and extrinsic load carrying. It is composed of five vertebral bodies separated by intervertebral discs acting as shock absorbers and stabilizers, as well as the posterior vertebral ring composed of pedicles, laminae, spinous and transverse processes, and facet joints that enclose and protect the spinal cord and spinal nerve roots. The lumbar vertebrae are numbered from the upper (cephalad) or first lumbar vertebra (L1) to the lower (caudad) or fifth lumbar vertebra (L5). Lumbar vertebrae are larger and wider than those in the dorsal and cervical spine, with the fifth vertebra generally the largest. This affords a larger surface area for the intervertebral disc and for load distribution. Disc anatomy and function will be discussed further in this section. At the lower end of the lumbar spine is the sacrum, a large, triangular bone

representing the fusion of five sacral vertebrae, and the small coccyx.

Consistent with the greater vertebral size, the lumbar pedicles are shorter and wider than in the dorsal spine. Lumbar facets are posterior articular processes where the adjacent vertebrae interface. These joints help permit motion and bear some of the compressive load in addition to helping maintain stability of the spine against torsion and shear. Facet joints are synovial, and they contain nerve innervations in the synovial lining.

Anterior and posterior longitudinal ligaments attach to the superior and inferior margins of the lumbar vertebrae, and are innervated by pain fibers. The ligamentum flavum is a non-innervated structure that runs down the vertebral ring, and may hypertrophy after injury. This may become significant when a hypertrophied ligament infolds during spinal extension in an individual with disc bulging and facet arthropathy, thereby creating relative spinal stenosis. The interspinous ligament, also non-innervated, runs down the posterior margins of the spinous processes, posterior projections from the vertebral ring.

In adults, the spinal cord terminates as the conus medullaris at about the level of the first lumbar vertebra in the upper lumbar spine. Branching off from the conus is a bundle of lumbosacral nerve roots that resemble a horse's tail, called the cauda equina. These nerve roots pass through the lumbar and sacral portions of the spinal canal surrounded by the vertebrae, intervertebral discs, pedicles, laminae, facet joints, and spinal ligaments and eventually emerge as individual nerve roots through the intervertebral foramina. The neural foramen is bordered by the transverse processes of adjacent vertebrae, and the spinal nerve root takes its name from the adjacent (cephalad) vertebrae. The spinal cord is covered by the thecal sac, composed of meningeal tissue and cerebrospinal fluid.

Nerve roots in the lumbosacral spine include ventral (motor) and dorsal (sensory) components. Ventral roots contain motor axons sending signals to distal areas and control various skeletal muscle motor functions. Dorsal roots comprise primarily sensory axons receiving signals from distal areas or dermatomes. Thus, symptoms and signs of nerve root compression will vary with the location of the compressive lesion. As the intrathecal nerve roots reach the intervertebral foramen, the root sleeve gradually encloses the nerve more tightly, and eventually become extrathecal. Cell bodies for sensory axons are located in an extrathecal area of swelling called the dorsal root ganglion. These ganglia are encountered in or close to the intervertebral foramina. Axons of the nerve roots consist of collagen tissue called the endoneurium. This is covered by a thin root sheath that separates the endoneurium from a small amount of cerebrospinal fluid, and the epineurium and perineurium covering. Blood flow derives from segmental arteries that divide into three branches when approaching the intervertebral foramen. Nociceptors are present in facet synovium and outer layers of annulus (or extension of the posterior longitudinal ligament).

There are several important muscles of the low back. The psoas muscles are major spinal flexors that originate at the anterior vertebral borders and combine with the iliacus from the crest of the pelvic ilium and insert on the pelvis and lesser trochanter of the hip. Posteriorly, the erector spinae muscles attach to the spinous processes and laminae down to the sacrum to act as major spinal extensors. The interspinales muscles run between the five spinous processes of the lumbar spine and contribute to extension. Several other coactivating muscles assist in spinal stabilization and rotation. The rectus abdominis extends from the lower border of the rib cage to the pelvis and assist in flexion and maintenance of lordosis. The obliques and transversus are coactivators, and contribute to the generation of increased intraabdominal pressure, which some feel helps decrease compressive loading on the spine. External moments imposed on the lumbar spine during lifting are proportional to the weight and distance of the load from the spine and the weight and location of the individual's body segments. This results in a state of equilibrium where the external moments are counteracted by internal moments, primarily created by muscle contractions of flexors balancing extensors with additional stabilization from co-activators. Ligaments provide passive resistance or restorative moment to muscles. It is not clear, however, under what lifting conditions the ligaments play a significant biomechanical role.

Epidemiology of Work-Related Low Back Disorders

When discussing causal factors for low-back disorders, outcome measures vary and include low-back pain, impairment, and disability. Outcome measures may be defined in terms of severity and also whether the information was based on self-reports (interview or questionnaire) or objective criteria, *e.g.,* lumbar disc pathology.

Because there are numerous conditions in the low back which may cause low back pain, regardless of their relationship to work factors, and, in most cases the cause(s) cannot be determined with any degree of clinical certainty, the most common form of back disorder is ''non-specific symptoms,'' which often cannot be diagnosed. Therefore, in its review of the epidemiologic evidence for work-relatedness of low-back musculoskeletal disorders NIOSH (Bernard 1997; Ex. 26–1) included subjectively-defined health outcomes (*e.g.,* ''back pain'') because they comprise such a large subset of the total. From a total of 42 studies, 24 investigations defined the health outcome only by report of symptoms on questionnaire or interview, 2 used sick leaves and medical disability retirements and 6 used injury/illness reports. The NIOSH review of epidemiologic studies of low-back disorders examined the following potential risk factors related to physical aspects of the workplace: (1) Heavy physical work (HPW, work that has high energy demands or requires some measure of physical strength, jobs that impose large compressive forces on the spine), (2) lifting and forceful movements (LFM), (3) bending and twisting (BT, awkward postures), (4) static work postures (SWP), and (5) whole-body vibration (WBV). These physical risk factors almost always appear in workplaces in combinations with other work-related risk factors, as well as a myriad of personal, psychosocial and other factors. However, to the extent possible the review seeks to examine the physical factors separately. Furthermore, since this ergonomics rule does not contain provisions relating to WBV, this last portion of the NIOSH review will be substantively omitted from this analysis. Table V–7 contains a listing of both the higher quality back studies used in the NIOSH 1997 (Ex. 26–1) review and additional back studies in the record.

TABLE V–7.—SUMMARY OF EPIDEMIOLOGY STUDIES EXAMINING MUSCULOSKELETAL DISORDERS OF THE BACK

Study/exhibit number	Job type studied	Physical factors	Exposure basis	Physical exam.	Risk measure (95% CI)[1]
Punnett, 1991 Ex. 26–39	Auto assembly	HPW/BT LFM	Observation measurement.	Yes	OR=2.2–8.1* (1.4–4.4)
Astrand, 1987 Ex. 26–527	Pulp mill	HPW	Questionnaire job title.	Yes	OR=2.3*
Bigos, 1991 Ex. 26–1242	Aircraft assembly	HPW	Observation questionnaire.	No	NR*
Burdorf, 1991 Ex. 26–454	Concrete fabrication.	HPW/BT LFM	Observation measurement.	No	OR=2.8* (1.3–6.0)
Clemmer, 1991 Ex. 26–1345	Offshore drilling	HPW	Questionnaire job title.	No	OR=2.2–4.3*
Hildebrandt, 1995 Ex. 26–1516	Population based	HPW	Questionnaire job title.	No	OR=1.2* (1.33–1.55)
Heliovaara, 1991 Ex. 26–959	Population based	HPW/LFM	Questionnaire job title.	Yes	OR=1.9–2.5* (1.4–4.7)
Hildebrandt, 1996 Ex. 26–770	Steel maintenance	HPW	Questionnaire job title.	No	NR
Johansson, 1994 Ex. 26–1132	Metal workers	HPW/BT LFM	Questionnaire job title.	No	PRR=1.76 (1.25–2.47)
Leigh, 1989 Ex. 26–750	Population based	HPW	Questionnaire job title.	No	OR=1.5* (1.1–2.2)
Masset, 1994 Ex. 26–1470	Steel workers	HPW/BT	Questionnaire job title.	No	NR
Partridge, 1968 Ex. 26–1, pg. 6–81	Dock workers	HPW	Questionnaire job title.	Yes	OR=1.2
Riihimaki, 1989 Ex. 26–998	Concrete workers	HPW/BT	Questionnaire job title.	No	OR/1.0–1.5*
Riihimaki, 1994 Ex. 26–1188	Heavy equipment operators.	BT	Questionnaire job title.	No	NR
Ryden, 1989 Ex. 26–809	Hospital employees.	HPW/BT	Questionnaire job title.	No	OR=2.2* (1.25–4.15)
Svensson, 1989 Ex. 26–732	Population based	HPW/BT LFM	Questionnaire job title.	No	OR=1.2*
Videman, 1990 Ex. 26–1023	Hospital patients	HPW/SWP LFM	Questionnaire job title.	autopsy	OR=2.8–24.6* (1.5–409)
Bergenudd, 1988 Ex. 26–1342	Population based	HPW	Questionnaire job title.	No.	OR=1.8* (1.2–2.7)
Burdorf, 1990 Ex. 26–1518	Crane operators	HPW/SWP LFM	Questionnaire job title.	No	OR=0.5–4.0 (0.8–21.2)
Chaffin, 1973 Ex. 26–876	Electronics manufact..	LFM	Job title	No	OR=5.0*
Holmstrom, 1992 Ex. 26–36	Manual handling	LFM/BT SWP	Questionnaire job title.	Yes	OR=1.3* for BT (1.1–1.5)
Huang, 1988 Ex. 26–1204	School lunch workers.	LFM	Observation measurement.	No	NR
Kelsey, 1975 Ex. 26–1134	Case/control herniated lumbar disc.	LFM/SWP	Questionnaire job title.	No	NR
Kelsey, 1984 Ex. 26–752	Case/control prolapsed lumbar disc.	LFM/BT	Questionnaire job title.	Yes	OR=3.1* (1.3–7.5)
Knibbe, 1996 Ex. 26–766	Nurses	LFM	Questionnaire job title.	No	OR=1.3
Magora, 1972, 1973 Ex. 26–1513	8 occupations	LFM/BT	Observation measurement.	No	OR=1.0–1.7* (1.3–2.1)
Liles, 1984 Ex. 26–33	Manual handling	LFM	Measurement	No	OR=4.5* (1.02–19.9)
Marras, 1995 Ex. 26–14–12	Manufacturing workers.	LFM/BT/ HPW	Observation measurement.	No	OR=10.7* (4.9–23.6)
Toroptsova, 1995 Ex. 26–1, pg. 6–92	Machine builders	LFM/BT/ SWP	Questionnaire job title.	Yes	OR=1.4*–1.7*
Undeutsch, 1982 Ex. 26–731	Airport baggage handlers.	LFM	Questionnaire job title.	Yes	NR
Walsh, 1989 Ex. 26–1437	Population based	LFM/SWP	Questionnaire job title.	No	OR=1.5–2.0* (1.1–3.7)
Skov, 1996 Ex. 26–674	Saleworkers	SWP	Questionnaire job title.	No	OR=2.5* (1.2–4.9)
Mandel, 1987 Ex. 500–41–92	Hospital nurses	LFM	Questionnaire	No	OR=1.4*
Thorbjornsson, 1998 Ex. 500–119–7	Random selection from 2500 medical exams.	HPW	Questionnaire	Yes	OR=1.4* (1.0–2.0)

TABLE V–7.—SUMMARY OF EPIDEMIOLOGY STUDIES EXAMINING MUSCULOSKELETAL DISORDERS OF THE BACK—Continued

Study/exhibit number	Job type studied	Physical factors	Exposure basis	Physical exam.	Risk measure (95% CI)[1]
Kuh, 1993 Ex. 500–41–80	Population based	LFM	Job title	No	RR=1.3* (1.0–1.7)
Smedley, 1995 Ex. 500–41–40	Hospital nurses	LFM	Questionnaire	No	OR=1.3–1.8* (1.3–2.5)
Venning, 1987 Ex. 500–41–49	Nurses	LFM	Questionnaire job title.	No	OR=1.7–4.3*
Xu, 1997 Ex. 500–119–9	Population based	BT/HPW	Questionnaire	No	OR=1.3–1.7* (1.51–1.93)
Stobbe, 1988 Ex. 500–41–45	Hospital nurses, LPNs, attendants.	LFM	Questionnaire	No	OR=1.0–2.7*
Park, 1997 Ex. 500–41–104	Population based	HPW/LFM/BT	Questionnaire	No	OR=1.88* (1.64–2.15) for HPW
Latza, 2000 Ex. 500–41–83	Population based	HPW/BT/SWP/LFM	Questionnaire	No	OR=1.77–1.89*
Latza, 2000 Ex. 500–119–6	Laying sandstone	HPW/LFm	Questionnaire	Yes	PR=1.8–2.6* (1.1–6.5) for hours/shift
Kerr, in press Ex. 500–39	Automotive workers.	LFM/BT	Measurement	No	OR=1.7–2.0* (1.22–3.59) for biomechanical factors
Krause, 1998 Ex. 500–87–2	Transit vehicle workers.	HPW	Questionnaire records.	Yes	OR=3.04* (1.85–5.00)
MacFarlane, 1997 Ex. 500–41–91	Population based	LFM	Questionnaire	Yes	OR=1.1–2.5* (1.5–4.1)
Waters, 1999 Ex. 500–41–54	Lifting case/control.	LFM	Questionnaire measurement.	No	OR=2.45* (1.29–4.85)
Wang, 1998 Ex. 500–41–52	Manual handling	LFM	Measurement	No	Significant correlation p<0.01
Van Poppel, 1998 Ex. 500–121–71	Airline baggage handlers.	HPW	Questionnaire	No	NR
Vingard, 2000 Ex. 500–41–51	Population based	HPW/LFM/BT	Questionnaire	No	RR=1.4–2.9* (1.2–6.8)
Luoma, 1998, (2000) Exs. 500–71–39, (38)	Not by identifiable risk factor but by title—office carpenter machine driver		Job title	Yes	OR=2.0–8.1* (2.4–21.1)
SHARP, 1993 Ex. 30–7	Data entry	SWP	Questionnaire	No	NR* (p<0.05)
Larese, 1994 Ex. 38–130	Hospital nurses	LFM	Measurement	Yes	OR=1.9–2.4*
Myers, 1999 Ex. 500–119–10	Case/control municipal workers.	HPW/BT/LFM	Questionnaire measurement job title.	No	OR=1.6–2.0* (1.13–3.67) for BT

HPW=heavy physical work; LFM=lifting or forceful movements;
BT=bending and twisting or other awkward postures; SWP=static work postures
IR=incidence rate; OR=odds ratio; RR=relative risk; NR=not reported; *=p<0.05
[1] 95% confidence limits expressed for the upper end of the risk measure range.

Heavy Physical Work

The NIOSH summary reviewed the eighteen higher quality studies which address the association between HPW and LBP (Ex. 26–1, pgs. 6–4 to 6–13). Of these eighteen, 14 were cross-sectional, 3 were prospective) and one was a case-control (Ryden *et al.* 1989, Ex. 26–801). Study populations included individuals working in health care, office work, manufacturing and construction, and all with different physical work requirements. Despite the fact that the studies assessed different groups of workers, defined disorders and assessed exposures in many ways, nine of these eighteen found statistically significant positive associations. The relative risk estimates for these significant associations generally ranged from 1.1 to 4.3, although one study of cadavers found a significant OR=12.1 (95% C.I. 1.4—107) for the risk of osteophytosis among those in the HPW category. OSHA notes that if there were no true associations only one of these eighteen studies should have shown a statistically significant result.

With regard to temporality, this is usually most easily studied with a cohort study design. Of these three studies, one had no association (Bigos *et al.* 1991, Ex. 26–1241), while two showed statistically significant increases (Clemmer *et al.* 1991, Ex. 26–1345; Bergenudd *et al.* 1988 Ex. 26–1342). Two cross sectional studies also considered temporal relationships by including in the analysis only those MSD-free when starting their current jobs, and both showed positive associations (Burdorf *et al.* 1991, Ex. 26–454; Burdorf and Zondervan 1990, Ex. 26–1518). Thus, these results are consistent with a positive finding for temporality.

OSHA also notes that the Bureau of Labor Statistics Annual Survey of

Injuries and Illnesses as well as other health interview surveys have found elevated LBP rates and MSDs in typical HPW associated occupations, (female) nursing aides, orderlies and attendants; personal care; and air transportation workers (see the risk assessment in section VI of this preamble). While survey statistics may not be definitive in themselves, they show a pattern of consistency with the results from the epidemiology studies discussed above. OSHA notes that these types of population-based studies can be less reliable than other epidemiology designs due to generally less knowledge about individual exposures.

Since OSHA's Ergonomics proposal was published, several other studies on HPW and LBP have been put into the record. Several are discussed below:

The Vingard *et al.*, 2000 (Ex. 500–41–114) population-based case-referent study suggests that prolonged exposure to many years of heavy work and forward bending (cumulative exposure) increases the risk of LBP. The Latza *et al.,* 2000 (Ex. 38–424) prospective study, after adjusting for trade, found exposure-response relationships for hours per shift laying sandstone (PR=1.8, 95% C.I. 0.7—4.7, for 0 to <2 hours; PR=2.6, 95% C.I. 1.1—6.5, for ≥ 2 hours; trend test p=0.03), and stone load (PR=1.8, 95% C.I. 0.4—9.5, for intermediate level; PR=4.0, 95% C.I. 0.8—19.8 for high level; trend test p=0.03). The Krause *et al.* 1998 study (Ex. 38–272) found that cable car crews performing the heaviest physical labor had a three-fold increased risk of spinal injury compared with bus driver (OR=3.04, 95% C.I. 1.85—5.00). This five year prospective study of 1,871 transit vehicle operation also found both physical workload and psychosocial job factors independently predict spinal injury in transit vehicle operators.

OSHA has also considered three other studies available since the proposal on HPW. Two of these three studies found at least one statistically significant association between LBP and HPW while the third suffered from methodological problems. Myers *et al.* (1999, Ex. 500–119–10) carried out a case-control study of 274 workers with symptoms and signs of low back pain from four municipal departments (a 73% participation rate). The stated purpose was to identify factors, both physical work characteristics and psychosocial factors, associated with acute low back injury. Two randomly selected controls were chosen, one matched according to work tasks, which the authors stated ''could be used to examine importance of non-ergonomic factors'' and one matched by department. Cases were defined from reports from the city Occupational Medicine Clinic, and were those who had been assigned restricted work or had lost work time due to back injury. Further information was gathered from questionnaire about work history, work characteristics, work injuries, back pain, psychosocial behaviors, and demographics. Exposure was assessed by questionnaire and measurement; the strenuousness of each worker's job classified as light, medium, or heavy according to weight capacity, frequency and duration of sitting-standing-walking. Analyses screened for 2-way, 3-way and 4-way interactions. The variables examined included a work movement index, which combined twisting, extended reaching, and stooping. Factor analysis was used to determine the important psychosocial factors from the Job Content Questionnaire. There was no difference in the prevalence among the cases and controls regarding physical work demands (light, medium, and heavy), nor twisting or extended reach. However, because the cases and controls were matched on job department and/or job title, the design provided little ability to examine those job factors. This would also preclude any conclusions pertaining to the relative strength between psychosocial and physical factors. Although the authors noted that their ''findings underscore the importance of adopting a model that does not focus entirely on physical factors in relation to the multifactorial problem of back injury,'' their study design did not allow them to focus adequately on the physical factors. This study focused on the psychosocial aspects of the acute back pain but did not adequately address work factors.

Park *et al.* (1997, Ex. 500–41–104) carried out a cross-sectional study using data from the National Health Interview Survey Occupational Health Supplement, 1988. In this survey, 30,074 randomly selected employed persons were asked about back pain occurring every day for a week or more in the previous 12 months. The response rate was 87%. Causes of back pain were classified into 3 groups: (1) Injury and/or repeated activities that occurred at work; (2) injury and/or repeated activities that occurred outside work; and (3) other reasons (illness, diseases, unknown). Self-reported work activities included repeated activities with lifting, pushing, pulling, bending, twisting, or reaching. Occupation was coded according to the 1980 classified Index of Industries and Occupations of the U.S. Bureau of Census. Confounders were controlled for in the regression models. Results found that 17.6% of workers reported back pain every day for one or more weeks during a 12 month period; 26.9% of these reports were attributed to repeated activities (RA) at work; 17% to RA and injury, and 8.2% to injury at work. The majority of back pain found in blue collar workers (OR=1.38, 95% C.I. 1.22—1.54), was attributed to work; whereas the majority of BP in white-collar workers was not attributed to work conditions. A higher proportion of workers with work-related back pain caused by injury or RA had pain in the lower back extending to lower body parts, had missed work more than 5 days, and had changed jobs than had workers with non-work related back pain. Other significant variables were ''strenuous physical activities at work more than 4 hours per day [HPW]'' (OR=1.88, 95% C.I. 1.64—2.15), ''repetitive movements more than 4 hours per day'' (OR=2.4, 95% C.I. 2.1—2.77) and current smoking (OR=1.57, 95% C.I. 1.39—1.76), BMI greater than 28 kg/m^2 (OR=1.35, 95% C.I. 1.2—1.52) and age 35–59 (OR=1.31, 95% C.I. 1.2—1.46). The strength of this study is the rigorousness used by the National Center for Health Statistics in their study design and analysis. A weakness is that it is based on questionnaire data.

Thorbjornsson *et al*, (1998, Ex. 500–119–7) used data collected over 24 years for its cohort study. 252 women and 232 men were randomly selected from 2500 for medical examination (a 62% participation rate). In 1969 these subjects had a questionnaire-based interview and an examination. LBP was defined as pain, aching, or stiffness in the lower back in the past 12 months. There was a follow-up a re-examination in 1993. Exposure assessment was based on a questionnaire from 1969 using a dichotomous scale for 11 work factors (*e.g.*, high mental load (hectic work, exhaustion at end of day), poor supervisor social support, monotonous work, full time work; night or shift work, overtime work, high physical load (40 kg for women, 60 kg for men or physical exhaustion at end of day), severe vibrations, and non-working conditions, using a dichotomous scale. (Insufficient or unsatisfactory leisure time, few or unsatisfactory social contacts, additional domestic workload). Risk factors for back pain during 1972–1992 included: for women, unsatisfactory leisure time (OR=1.5, 95% C.I. 1.1—2.0); for men, 1972–1993: high physical load (OR=1.4, 95% C.I.1.0—2.0), vibrations (OR=1.4, 95% C.I. 1.0—2.2), and unsatisfactory leisure

time (OR=1.5, 95% C.I. 1.1—2.0). Cumulative incidence ratios for 1972–1993, adjusted for age, and earlier back pain were 38% for women and 43% for men.

Lifting and Forceful Movements (LFM)

The NIOSH summary reviewed the 18 higher quality studies that address the association between LFM and LBP (Ex. 26–1, pgs. 6–13 to 6–21). Of the eighteen studies, 13 were cross-sectional, two were prospective, and three were case-control designs. Only the one case-control study of back pain in auto workers (Punnett *et al.* 1991, Ex. 26–39) fulfilled all four of their quality evaluation criteria. Besides auto workers, among the study groups which showed increased risks to workers with high lifting or manual materials handling (MMH) demands were nursing aides, baggage handlers, workers in manufacturing and electronics, crane operators, and concrete fabricators, although several studies focused more on the actual stresses within the job rather than job title. In all 10 of the eighteen studies showed at least some statistically significant associations between LFM and LBP, with the significant risk estimates generally ranging from 1.2 to 5.2 (Ex. 26–1, pg. 6–41). For the most part, higher ORs were observed in high-exposure populations. The highest risk estimate (OR=10.7) was from a group of workers in a cross-sectional study by Marras *et al.*, (1993, Ex. 26–170; 1995, Ex. 26–171). The MMH workers with this highest OR had the highest combination of exposure measures relating to five specific risk factors associated with lifting, twisting, frequency, angle, and force, again strongly suggesting synergism among the risk factors. The 5 studies reviewed for this chapter which showed no association between lifting and back disorder used subjective measurements of exposure, had poorly described exposure assessment methodology, or showed little differentiation within the study group.

With regard to temporality, both the prospective studies which assessed exposures prior to identification of MSDs, had positive association. Also, of the four (three cross-sectional and one case-control) studies which attempted to address temporality, three found positive relationships between lifting and LBP. OSHA also notes that of the eight studies which examined exposure-response relationships in some manner, six found positive associations, including Punnett *et al.*, 1991, (Ex. 26–39) while two others did not (Ex. 26–1, pg. 6–20).

Since OSHA's Ergonomics proposal was published, several other studies on LFM have been put into the record. Some are more recent, and these are discussed first, while several older studies, not part of the original review, are also discussed below.

With respect to the more recent studies, published since 1996, the studies of LFM and LBP in a wide variety of industries provide substantial additional evidence that repetitive lifting is associated with low back disorders.

There are a limited number of negative studies which provide little evidence to weaken the overall conclusion from the much large number of positive studies. Other reportedly negative studies of lifting and low back disorders have limitations. For example, Feyer, Herbison *et al.* (2000, Ex. 26–1499) conducted a prospective study of low back pain among nursing students, but there was no evaluation of the physical demands of jobs and there was a 1/3 dropout from the study.

In addition to the more recent studies, six older studies, not in the proposal, also discussed the relationship between LFM and LBP. Mandel and Lohman (1987, Ex. 500–41–92) showed an increased risk of back pain with lifting more than10 patients per week (OR=1.39, 95% C.I. 1.05—1.84) in a cross-sectional study in which 428 registered nurses in a Midwestern hospital participated (rate was 65%). Fifteen percent of the nurses had reported experiencing LBP for the first time during the study year, with most episodes occurring in younger workers. However, while intensive care unit nurses lifted significantly more patients, LBP was not associated with work area. The most significant associations were having LBP prior to the study year and having pain in another part of the spine. The limitations of this study are its participation rate and both its exposure assessment and health outcome definition. However, despite these limitations, it provides support for patient lifting as a risk factor for LBP in nurses.

Larese and Fiorto (1994, Ex. 38–130) in a cross-sectional study compared 425 general nursing staff from an urban hospital to 198 oncology nurses (participation rate: 91.4%). LBP cases were based on clinical examination or X-ray findings. Exposure measurements included the analysis of working conditions, which revealed both groups of nurses had to do frequent and heavy lifting, lowering, and pushing-pulling. Differences were found when analyzing the number of patients assisted by the different nursing groups: the staff nurses cared for double the number of patients compared to the oncology nurses. Calculating crude odds ratios showed that general nurses had an OR=1.9 (95% C.I. 1.32—2.76) for LBP and an OR=2.4 (95% C.I. 1.35—4.27) for back pain sick leave compared to the oncology nurses. The authors used the Mantel-Haenzel chi-square statistics to control for age and for occupation among the two groups, but multivariate analysis to control for both factors simultaneously was not done. The authors concluded that ''comparison between the two hospitals suggests factors associated with the disorders: work tasks and particularly nurses/patients ratio are more important than age and length of exposure.'' The authors did not present the data from which they drew these conclusions.

Stobbe *et al.* (1988, Ex. 500–41–45) carried out a retrospective study of three hospital groups at a major medical center including 143 licensed practical nurses, 252 nurses aides, and 20 attendants. Two groups were identified, one exposed to frequent patient lifting, one not. Health outcome was defined as back injuries, including both lost-time and non-lost-time injuries. Lifting frequency was determined through interviews with the nursing director, the head nurse, and nursing supervisors. High frequency lifting was defined as an average of more than 5 patient lifts per shift. Low frequency lifting (control group) was defined as average of less than two patient lifts per shift. Nursing personnel with estimated exposures of 3–5 patient lifts per shift were excluded. Lifting frequency (OR=2.7, p=0.009), and length of employment (p=0.0085) remained significant in the logistic regression model, while occupation did not. The authors used a survivor type conditional analysis which assumed that when a person with a back injury report resumed work, the future probability of injury was the same as if there had been no previous injury. This assumption has not been supported in other studies.

Kuh *et al.* (1993, Ex. 500–41–80) in their longitudinal study of 3262 same age Great Britain natives (born the first week, 1946), looked at risk factors for LBP, mainly the association with stature and height, but also lifting. The study population had been followed every 2 years in childhood, and every 5 years as adults. Participation rate for this study was only 60.8%. Exposure was assessed using job title and occupational histories. A matrix assigned jobs to three levels of lifting—low, intermediate and high. The interaction of height and occupational lifting as a risk factor for LBP was investigated for men. The onset

of back pain symptoms was significantly more common in men whose jobs were likely to involve heavy lifting (RR=1.3, 95% C.I. 1.0—1.7). The main occupations of heavy lifting associated with LBP were farming and construction. There was discussion of reporting bias, recall bias, lack of direct information about lifting at work. The weakness of this study is using ''job title'' as surrogate for exposure, but a bias here is likely to mask true associations.

Smedley *et al.* (1995, Ex. 500–41–40) conducted a cross-sectional survey of 2,405 nurses using a self-administered questionnaire to investigate the risk factors associated with low back pain. The response rate was sixty-nine percent. Among those who responded to the survey, 1616 were women. Due to the low number of male respondents, this study focused on female workers. Low back pain was defined as pain lasting for longer than a day in an area between the twelfth rib and the gluteal folds (indicated on a chart). Questions about non-musculoskeletal symptoms, included in the questionnaire, were designed to investigate whether psychological factors that influence reporting of other symptoms also affect reporting of LBP. After adjustment for age, height and non-musculoskeletal symptoms, significant associations were found for: frequency of manually moving patients around on the bed; manually transferring patients between bed and chair; and manually lifting patients from the floor.

Frequency	OR	95% C.I.	Factors controlled
Manual Movement of Patients on Bed			
5–9 moves	1.5	1.1–2.2	Age/height.
5–9 moves	1.6	1.1–2.3	Age/height/non-musculoskeletal symptoms.
10+ moves	1.7	1.2–2.3	Age/height.
10+ moves	1.7	1.2–2.4	Age/height/non-musculoskeletal symptoms.
Manual transfer of patients between bed and chair			
5–9 moves	1.7	1.2–2.3	Age/height.
5–9 moves	1.8	1.3–2.5	Age/height/non-musculoskeletal symptoms.
10+ moves	1.5	1.1–2.1	Age/height.
10+ moves	1.5	1.1–2.1	Age/height/non-musculoskeletal symptoms.

Evaluation of the task of manually lifting patients from the floor resulted in similar significantly elevated risks regardless of whether age and height alone or all three factors, *i.e.*, age, height, non-musculoskeletal symptoms, were controlled for (OR=1.3, 95% C.I.1.0—1.6). In this study, nurses who often report non-musculoskeletal symptoms, such as low mood or stress, were significantly more likely to report low back pain. For example, frequent low mood was strongly associated with subsequent back pain (OR=3.2, 95% C.I.. 2.2—4.8). Specific manual handling tasks were associated with an increased risk of back pain while no such association was found in this study among nurses using mechanized patient transfer (with hoists).

A study of personal and job-related factors that may affect the incidence of back injuries among 5,649 nurses was conducted by Venning *et al.* (1987, Ex. 500–41–49). A ''back complaint'' was defined as any work-related injury or complaint of discomfort in the back and reported through an employee health office. Nurses were surveyed by questionnaire and then observed for a 12-month study period. As annual injury rate of 4.9% was observed. Four factors were found to be highly statistically significant ($p<0.01$) predictors of back injury. Risk estimates for all four factors (service area, lifting, job category, and previously reported back injury), remained significantly elevated when a forward stepping model of logistic regression was applied. The observed adjusted odds ratios were: 4.26 for service areas where lifting occurs most often as compared with areas where lifting occurs least; 2.19 for daily lifters as compared with light, occasional, and nonlifters; 1.77 for nursing aides as compared with registered nurses and supervisory personnel; and 1.73 for individuals who have previously reported back injury as compared with those who have not reported previous injury. No other factors, including age, physical activity, availability of lifting aids, height and weight, and instruction in back care and lifting procedures, were significantly associated with reporting of back injury. The influence of service area is not easily explained. The authors chose to define service area as a work activity. With an attitudinal measurement, job satisfaction may have also proven to be a significant factor. The question would then be one of temporality and association between those factors. It is clear, however, that service area assignment is a major risk factor. When two employees who are similar in job category and history of back injury are assigned to different service areas, the risk of back injury is dependent on that ward assignment.

In summary, seven of the eight new studies, and all six of the older studies (all of nurses and nursing assistants who did more frequent patient lifting), found at least one statistically significant association between LFM and LBP. When considered with the 10 studies originally reviewed by NIOSH which found statistically significant associations, this epidemiology data base provides strong evidence for a causal association between LFM and LBP.

Bending and Twisting/Awkward Postures (BT)

The NIOSH summary reviewed the 12 higher quality studies which addressed the association between BT and LBP (Ex. 26–1 pgs. 6–21 to 6–26). Of the twelve, nine also examined the effects of occupational lifting, although for all but the Marras *et al.*, (1993, Ex. 26–170; 1995, Ex. 26–171) analysis discussed above the presented comparisons for LFM and BT are different. As with the analysis for BT above, only the Punnett *et al.*, 1991 case-control study fulfilled all four of the quality evaluation criteria. Nine studies were cross-sectional, two were case-control and one was prospective. Of the twelve studies seven reported statistically significant associations, with the significant risk estimates generally ranging from 1.2 to 3.5. However, two of these ORs were higher; in addition to the previously mentioned OR of 10.7 in the Marras *et al.* (Exs. 26–170, 26–171) study, Punnett *et al.*, 1991, (Ex. 26–39) using a multivariate analysis that adjusted for covariates, found a statistically

significant OR=8.09 (95% C.I. 1.4–44) for time in a non-neutral position for auto workers. Several studies suggested that both lifting and awkward postures were important co-contributors to risk of low back disorder.

With regard to temporality and exposure-response, three studies—one prospective, one case-control, and one cross-sectional—attempted designs and analysis to investigate temporality. Only the case-control study of Punnett *et al.*, 1991 (Ex. 26–39) found a strong association between exposure to awkward postures and back pain. The Riihimaki *et al.*, 1994 (Ex. 26–1188) prospective study comparing heavy equipment operators with office workers found a three year prevalence ratio for LBP of 1.4 (95% C.I. 1.0–1.9) (Ex. 26–1, 6–86). For exposure-response relationships between posture and low back disorder, five of the six studies which attempted such an analysis found significant relationships between some incremental index of LBP and exposures relating to awkward postures.

Since OSHA's Ergonomics proposal was published, three other recent studies on BT and LBP have been put into the record. These are discussed below:

With respect to the two most recent studies, both Latza *et al.*, 2000, (Ex. 38–424) and Vingard *et al.*, 2000 (Ex. 502–410) have been discussed above, in both the HPW and LFM sections. The Latza *et al.* study, in a logistic regression analysis controlling for several covariables, found that risk factors for LBP included working in a bent position, for men, with an OR =1.89 (95% C.I. 1.03–3.46). This OR was greater than those, computed in the same regression analysis, for carrying heavy loads, OR=1.47 (95% C.I. 0.97–2.24), and heavy physical work OR=1.77 (95% C.I.1.06–2.93). For the Vingard *et al.* study, there were statistically significant associations for both men and women when related to both heavy and cumulative exposures. When the combined physical exposures of ''heavily exposed to forward bending'' and ''manual handling over the last ten years'' were added to current exposures, the estimated RRs in men was 2.8 (95% C.I. 1.1–7.5) and in women 2.9 (95% C.I. 1.2–6.8). Multiple logistic analyses adjusting for a wide range of variables including age, social support at work and outside work, low back pain earlier, and negative life events, did not identify many physical or psychosocial factors as significant predictors. However, for ''forward bending greater than one hour'' the RR in men was 1.8 (95% C.I. 1.1–3.1), and in women 1.2 (95% CI 0.7–1.8).

The third recent study, Xu *et al.*, (1997, Ex. 500–119–9), examined bending and twisting, as well as physically hard work in the Danish population in a cross-sectional survey conducted in 1990. A random sample of 5,185 workers with similar sex, age, and occupational distributions as in the Danish population was selected, with a response rate of 89.3%. The health outcome was defined as symptoms of back pain in the past 12 months, assessed by structured interview, and included conditions of pain, ache, discomfort localized in the lower back, regardless of intensity and severity. Occupational exposure information included duration of daily exposure, vibrations affecting the whole body, physically hard work, frequently twisting or bending, sitting down, standing up, walking a lot, working with hands raised, concentration demands, repetition, and lifting heavy loads. The psychosocial factor ''concentration demands'' was also included in the model. Confounders controlled for included gender, age group, educational level, and duration of employment. There was a significant dose-response trend towards the greater prevalence of LBP with a greater proportion of the day exposed to the risk factors, for two physical factors—physically hard work (OR=1.28, 95% C.I. 1.08–1.52), and frequent twisting or bending (OR=1.71, 95% C.I. 1.51–1.93). Concentration demands and standing up were also significantly positively associated with the occurrence of low back pain. The results indicate that the associations of risk factors with LBP were stronger among those required to work for 37 or more hours/wk. The authors addressed issues of recall and participation bias.

In summary, the statistically significant associations of BT and LBP seen in seven of the 12 NIOSH reviewed studies and in all three of the more recent studies, provide by strong evidence that the associations observed are real.

Recent Epidemiology Reviews of Work-Related Low Back Disorders

Since the NIOSH 1997 review, there have been three published reviews which bear on the epidemiology of the work-related risk factors for back pain discussed above. The first is the NAS report, discussed elsewhere in this Health Effects section, which reviews and affirms the appropriateness of the methodology and the conclusions of the NIOSH 1997 review (Exs. 26–37). The other two are recently published reviews relating specifically to risk factors, especially physical stress factors, for back pain. One of these reviews also examines psychosocial factors (Ex. 500–71–24). These are discussed below.

The Burdorf and Sorock (1997, Ex. 500–71–24) review assessed the epidemiologic evidence of occupational risk factors for back disorders. They included only those published studies that clearly described exposure measures, had quantitative estimates of risk for work-related factors, and did not have evidence of a serious methodological problem. In all they included thirty-five articles, which they assessed for associations with physical factors at work, psychosocial factors at work, and individual factors. Of the 19 cited studies reporting on associations between back disorders and lifting or carrying of loads (LFM), sixteen were positive. The risk estimates ranged from 1.12 to 3.07, with attributable fractions estimated between 11% and 54%. Nine out of ten studies reported positive associations with frequent bending or twisting of the trunk (BT), three of which reported exposure-response relationships. Seven studies examined heavy physical load (HPW); six of these demonstrated increased risks of 1.54 to 2.58; however the one large longitudinal study did not demonstrate an association between physical load and the incidence of back injury claims during the study period (Ex. 26–1242). For static work postures (SWP), seven studies were considered and three of these had positive associations, The authors found some evidence of an association between the psychosocial factors of job dissatisfaction and low job decision latitude and back pain, but the evidence was not consistent across different studies and study designs. The review found that age, smoking habit, and education may be important confounders, while the individual characteristics of gender, height, weight, exercise or sport, and marital status were consistently not associated with back disorders. The finding that exercise or sport, the one physical individual characteristic examined, was not associated with back disorders provides supporting evidence that the physical work-related risk factor findings are real and are not confounded by leisure time physical factors.

In making their causality determination, Burdorf and Sorock acknowledged that the majority of cross-sectional design studies in the data base precluded a firm determination of the temporal and specificity criteria of the Hill criteria; they also expressed some concern that ''the state of the art does not allow unequivocal conclusions about the contribution of specific work-related risk factors to the incidence of

back disorders.'' (Ex. 500–71–24, pg. 253). Nevertheless, they concluded that:

> Despite these methodological concerns, the available literature has presented persuasive evidence for several risk factors for work-related back disorders. Various studies with clear differences in design, methodology, and populations have consistently produced comparable findings for MMH, frequent BT, heavy physical load and WBV. With regard to MMH, sufficient biomechanical and physiological evidence is available to support the biological plausibility of lifting as a risk factor for back disorders. The results on lifting do not distinguish between the effect of infrequent lifting of heavy loads and frequent lifting of light loads. The studies among nurses indicate that a single lift of a patient is associated with an increased risk of back pain or back disability [cite to Exs. 500–41–92, 500–41–70, and 500–41–49]. This finding is consistent with biomechanical evaluations that predict high compression forces on the lower back during patient lifting [cite to Ex. 38–141]. Frequent BT of the trunk was consistently related to back disorders in various studies. In one case-referent study with detailed exposure assessment, a clear dose-response relationship was shown [cite to Ex. 26–39]. The findings for heavy physical load demonstrate that this is an important work-related risk factor. Several community-based studies have presented dose-response gradients [cite to Exs. 29–959, and others]. The strength of the gradients is difficult to assess since self reports have been applied to rank exposure to physical load on ordinal scales. A second problem is that this particular risk factor probably includes MMH and frequent BT. Hence, in epidemiologic surveys, heavy physical load might be a surrogate measure for other risk factors rather than a separate risk factor (Ex. 500–71–24, pg. 253).

Finally the review concludes:

> This review concludes that there is a clear relationship between back disorders and physical load, that is, between back disorders and MMH, frequent BT, HPW, and WBV. * * * the evidence presented indicates that preventive measure reducing the exposure to these risk factors will decrease the occurrence of back disorders.

Hoogendoorn *et al.* (1999, Ex. 500–71–32) conducted systematic reviews of the literature for physical load as risk factors for back pain. A rating system was used to assess the evidence based on methodological quality and consistency of the findings; under this scheme cross-sectional studies were excluded based on the authors' quality criteria. The review of studies addressing physical load examined 28 cohort and 3 case-referent studies. For physical load, the review found that strong evidence exists for work-related MMH, BT, and whole-body vibration as risk factors for back pain. Moderate evidence exists for patient handling (LFM) and HPW, and no evidence was found for standing, walking, sitting, sports, and total leisure time physical activity.

OSHA finds that the consistency of findings in the NIOSH 1997 (Ex. 26–1) and the two other recent reviews, all using different study selection and evaluation criteria, provides confirmation of OSHA's emphasis on NIOSH's methodology and conclusions for work-related causes of back pain. The assessment on physical load factors was insensitive to slight changes in the assessment of findings and the methodological quality of the studies. Burdorf and Sorock (Ex. 500–71–24), in their review, also commented that comparable findings were consistently found for heavy physical work, lifting, twisting and bending, and whole body vibration at work in various studies with clear differences in design, methodology, and populations.

Dr. Tapio Videman's Testimony on Twin Back Studies

Dr. Tapio Videman, DrMedSci, University of Helsinki, testified that a weakness with the OSHA proposal was that in the studies OSHA examined, the role of genetic factors was not taken into account in studies estimating the effect of work-related stress factors (Tr. 16996). To make this point, Dr. Videman presented a slide in his testimony (Tr. 16997) that referred to a published paper he had co-authored on the determinants of lumbar disc degeneration in a retrospective cohort study (Ex. 26–71). The study design attempted to control for the role of genetics by comparing disc degeneration scores between identical twins with different exposure factors thought to be associated with back pain. Among the factors examined in the paper were occupational workload, leisure time physical activities, measures of aerobic exercise and other sports participation, occupational driving, and smoking.

The study consisted of 115 pairs of identical twins selected from the Finnish Twin cohort, who were among the most discordant pairs in terms of the exposure factors mentioned above. The objective was to study whether differences in exposure factors correlated with the disc degeneration scores, controlling for genetic factors. Both observational and digital summary scores for disc degeneration, based on an MRI examination, were obtained for both the upper and lower back regions. Occupational and leisure physical activity responses were derived via personal interviews.

An important feature of the study design is that of the 115 pairs of twins only 23 pairs were discordant for heavy work before the age of 20. Also, based on a job scale rating of 1 to 4 to aggregate every job title and associated task descriptions during a subject's lifetime work history, the mean absolute job scale difference in these 115 twin pairs was 0.9. For mean hours working in bending/twisting positions the absolute mean difference within the 115 pairs was 1.6 hours. This means that this study had little statistical power to show differences among physical work factors, after adjusting for genetic factors, since only discordant pair results factor into an individual matched analysis.

The authors examined the associations between the several exposure factors and disc degeneration scores using both univariate and multivariate analyses, and both observational and digital summary scores for disc degeneration for both upper and lower bask disc degeneration scores. In the univariate analyses, which apparently did not factor in the twins matched pair design, only the heavier physical work job code score and mean total occupational lifting per day were significantly adversely associated with disc degeneration score, and then only for the high back discs. Most other increased physical activity and smoking scores were also associated with increased disc degeneration scores, but the associations were not statistically significant. Increased mean time sitting at work was associated with less disc degeneration for both high and low back, but only the high back scores showed statistical significance.

To attempt to control for the genetic effect, the authors also used multiple regression methods in an attempt to explain the observation summary disc scores. Their results found that, for the upper back, only the mean job code and age were jointly statistically significant, with no other specific environmental or behavioral factors contributing significantly. For the lower back, heavy leisure time physical loading was the only specific environmental factor of statistical importance; this one variable explained 2% of the variance in the multiple regression model.

In an attempt to consider the amount contributed by the genetic component in the study design, the authors inserted 114 co-twin (indicator) variables in the model and recalculated the estimates. They found that together, these 114 variables, ''those of familial aggregation, reflecting primary genetic and shared early environmental influences * * * explained nearly 75% of the variability in disc degeneration score in the upper region and nearly 50% in the lower lumbar region (*id.,* pg. 2608). The

authors concluded, as did Dr. Videman in his testimony, that these ''findings suggest that disc degeneration may be explained primarily by genetic and early environmental influences and unidentified factors. * * * If disc degenerative changes are associated with symptomatic conditions, these studies findings suggest a need to rethink future research and prevention strategies in this area.'' (*id.,* pgs. 2610–2611).

Dr. Videman and associates made similar findings on the importance of genetic factors in disc degeneration in a study comparing 20 pairs of twins with discordant smoking status (Ex. 32–241–3–89; Tr. 16994–16995). Using the same type of multivariate methodology, with one variable for smoking and 18 variables for co-twin status, they concluded, ''Whereas smoking status and age explained 0 to 15% of the variability on the various degenerative findings in the discs, 26% to 72% of the variability was explained with the addition of a variable[s] representing co-twin status. These findings are compatible with a marked genetic influence and warrant further investigation.'' (Ex. 32–241–3–89).

In his testimony at the hearings, Dr. Videman emphasized the relative importance of genetic factors over physical work factors, ''(W)e could conclude that, from a blood sample, I can predict MRI [disc] changes better than having a lifetime work history about another interview.'' (Tr. 16998).

OSHA has considered Dr. Videman's testimony and publications and disagrees with his conclusions about the relative importance of physical work factors and genetics in the prediction of MRI disc changes. Although the agency agrees that the discordant identical twin study design is useful to control for genetic and early environmental factors, other factors in the design are at least as important. As was seen in the first study discussed above (Ex. 26–71), in a matched control study the amount of discordance in the exposure variables within the twin pairs will determine the power of the study to detect an effect. For example, with little discordance in exposure variables and few discordant pairs, the study has little ability to detect a true effect. In fact OSHA believes that in such a situation degenerative disc summary scores between twins should be very similar. To carry this example further in that first study, which involved the 115 twin pairs with little co-twin difference in the exposure variables, it is not surprising that adding 114 co-twin variables to the analysis, it is absolutely no wonder that in total these 114 variables will explain most of the variation in the multiple regression model. OSHA concludes that Dr. Videman's conclusion on the importance of genetic factors in his studies is a function of his analysis and his study design. This type of matched-control study is designed to control for genetic effects, not to study them.

OSHA also notes that in Dr. Videman's smoking study with 20 twin matched-pairs and a mean discordance between siblings of 32 pack years, ''a very huge difference'' (Tr. 16994), the disc degeneration difference was statistically significant at all of the measured disc levels. Controlling for genetic traits was undoubtedly important, as suggested by the statistical significance of the 18 covariables (Ex. 32–241–3–89, pg. 1666).

In the hearings, Dr. Videman was questioned by Ms. Seminario about a study he co-authored that concluded, ''environmental factors [including physical work factors] account for more than 80 percent of the [etiology] of sciatica and more than 90 percent in the case of patients admitted to the hospital.'' (Tr. 17054, see also Dr. Videman's response to a similar question by Ms. Butterfield, Tr. 17128). Although Dr. Videman acknowledged the correctness of this statement, he appeared to contradict these findings by explaining that ''all the data from that study was based on questionnaire data, so the reliability of the diagnosis is unclear.'' (Tr. 17129). OSHA notes, however, that in the actual paper the authors note that ''the cumulative age-specific incidences of sciatica [were] based on both the questionnaire and the hospital discharge records,'' and that the results are in ''accord with the results of a previous Finnish study.'' (Ex. 502–227, pg. 397). Furthermore, the authors noted that the hospital discharge diagnoses are given by doctors based on the WHO manual of the International Statistical Classification of Diseases (*id.,* 394). The authors also cited studies on the reliability of the nationwide hospital discharge registry (id., 394).

Thus, because that Dr. Videman's conclusions about the relative importance of genetics and physical work factors in back disorders were based on the questionable methodology used in the two twin studies discussed above, and because Dr. Videman's testimony on another study which contradicted those conclusions was not supportable, OSHA is unable to give much weight to Dr. Videman's testimony on this issue.

The Bigos et al., 1991 Back Study

Bigos *et al.*published several papers on a study (see, *e.g.,* Exs. 500–121–8, 38–280, 26–1241) that assesses the role of work perceptions and psychosocial factors in predicting the report of back pain disability. The study group was a cohort of aircraft assembly workers at the Boeing Company in Everett, Washington who volunteered to participate. This longitudinal study ultimately analyzed 1326 out of a cohort of 4027 aircraft assembly workers (33% of the original solicited population) for the final models.

The health outcome studied was ''back pain disability lasting longer than 3 months,'' and the authors used three notification systems—reporting to the company medical department, filing an incident report, or filing an industrial insurance claim. The study did not investigate the actual presence of back symptoms or specific back disorders. At the beginning of the study, subjects answered a series of questionnaires which addressed demographics, psychosocial factors, and cardiovascular risks, as well as a take-home questionnaire including the 566 question Minnesota Multiphasic Personality Inventory (MMPI), the Health Locus of Control Questionnaire, and a modified Work Adaptation, Partnership, Growth, Affection, and Resolve (APGAR) survey (modified from the Family APGAR survey). Other information included previous medical history, previous back discomfort or problem, back injury claims in the previous 10 years, and work perceptions. Subjects were also given a physical examination to assess physical attributes including anthropometry, lifting strength, aerobic capacity, and sagittal flexibility. A back examination including reflexes, girths, sciatic tension, and posture was performed. Thus, each subject provided individual responses to questions concerning these physical and psychosocial factors.

In contrast to the above factors, which were collected for each worker individually, workplace exposure assessment was limited to all jobs that employed more than 19 workers and was not performed on individual workers. These jobs were analyzed for tasks that were heavy and tiring tasks in terms of maximum loads on the spine, based on some unspecified biomechanical mathematic model. Any worker in a job with fewer than 19 people did not get physically measured; also, the authors did not measure workers' cumulative loads. As with the psychosocial factors, workplace

exposure was also measured only at initial recruitment.

Subjects were followed for slightly more than four years, during which 279 subjects reported back problems. After analyzing the data to determine which factors could best predict these reports, the authors concluded:

> Other than a history of current or recent back problem, the factors found to be most predictive of subsequent reports in a multivariate model were work perceptions and certain psychological responses. * * * Subjects who stated that they ''hardly ever'' enjoyed their job tasks were 2.5 times more likely to report a back injury (p=0.0001) than subjects who ''almost always'' enjoyed their job tasks. These findings emphasize the importance of adopting a broader approach to the multifaceted problem of back complaints in industry, and help explain why past prevention efforts focusing on purely physical factors have been unsuccessful.

OSHA notes that one major problem with the interpretation by other researchers of these results in the Boeing studies is that within the Boeing studies, ''physical variables'' include only those physical attribute variables that deal with anthropometry, back examination indices, and physical capabilities (*e.g.* flexibility, lifting strength, aerobic capacity) (Ex. 38–280, Table 1, pg. 25). It is under the ''nonphysical variables'' that the authors included workplace factors—duration of employment, job classification code, and measured peak spinal loading—as well as psychological and psychosocial factors. Other researchers include workplace factors (*e.g.,* measured peak spinal loading and physical workload) as physical variables. Thus, when Bigos *et al.*conclude in their study that none of the physical variables was important in predicting back pain reports (back disability > 3 months)—they are not referring to the same types of work-related physical risk factors—lifting/forceful movements, bending/twisting and awkward postures, heavy physical work, or static work postures—that OSHA refers to in its standard. Bigos *et al.*did not directly address these factors in their study.

OSHA also notes that the overall participation rate for this study was low, which makes representativeness an issue, especially for the 25% of the group that initially chose not to participate. The longitudinal study ultimately analyzed 1326 out of a cohort of 4027 aircraft assembly workers (33% of original solicited population) for the final models. In an attempt to determine whether the voluntary aspect of the study would create a bias, the authors compared the reported injury rates for those who returned incomplete data (n=1451) on their modified APGAR and MMPI packets, with the 1,569 subjects who did complete the forms. The difference in injury report rates was not statistically significant, which suggests that this final study group may be representative of the total.

OSHA also notes that no individual exposure measurements were carried out, although extensive individual psychosocial and psychological measurements were done. Workplace exposure assessment was limited to jobs that employed more than 19 workers, and there was no accounting for individual inter- or intra-variability. Because the exposure data represented the ''exposure'' of a group of workers rather than the measured exposure of individual workers, the authors would not be able to determine the contribution of physical factors to the observed outcome in as robust a fashion as they would the contributions of medical history, psychological surveys, physical exam, or job satisfaction survey, which were all recorded as individual exposure data. The authors did not report nor provide information on the analysis of the exposure data. There was no report on the data collected on biomechanical loads of the spine. They also did not report nor provide information on the data collected on the workers' perceived physical exertion in their jobs.

Dr. Bigos, in his testimony to OSHA during the hearings, stated that the Schultz model (the only biomechanical model related directly to human intradiscal measurements) was applied to the evaluation of mechanical stress on the Boeing subjects, and it found no significant relationship between mechanical stress on the subjects and the report of back problems or disability (Tr. 6725–6727). OSHA is addressing back pain in its final standard, and intradiscal measurement changes, obtained from the Schultz model, are not directly relevant to the existence of back pain or back disability.

OSHA also notes that this study did not address heavy lifting, or even jobs at the moderate or high end of HPW exposure. Bigos *et al.*report, ''the study was done in a diverse, highly sophisticated manufacturing industry where job tasks do not tend to be extremely stressful for the back.'' (Ex. 500–121–8, pg.5). As Bigos *et al.*(1991, Ex. 26–41) state, ''our study may not be representative of workers with extremely physically demanding jobs, where virtually no one remains active until retirement age.''

OSHA also has concerns about the interpretation of the results of the ''Work'' Adaptation, Partnership, Growth, Affection, and Resolve (APGAR) survey score. The authors added two additional untested items to the family APGAR: (1) ''I enjoy the tasks involved in my job,'' and (2) ''please check the column that indicates how well you get along with your closest immediate supervisor.'' (Ex. 26–1242, pg. 2). Results found the strongest statistically significant relationship between back disability and statement (1) ''I enjoy the tasks involved in my job.'' (id., pg. 3). However, this single initial response from a single point in time, rather than from more reliable repeated measures over time, was used to explain the outcome over a four-year period.

OSHA also has some concerns about a potential bias due to subjects who were excluded from strength testing if current back symptoms were present at the time of testing, or had caused them to miss work in the previous six months. This strongly influences the ability to draw from the study conclusions that are related to this variable, *i.e.,* eliminating the back pain subjects from the study population creates a healthy worker effect, which would bias results toward the null.

For the final predictive model, involving 33% of the original solicited population, the percentage of the overall variability explained by the model was 2.2% for job satisfaction, 1.9 for psychological factors, 1.2% for physical examination factors, and 3.3% for medical history; the sum of these individual components was 8.6%; 7% combined (Ex. 38–280, pg.29). This means that 93% of the variability was unexplained by this model for predicting industrial back pain reports (back disability > 3 months).

In sum, with the qualifications discussed above, OSHA acknowledges the importance of the Bigos *et al.*prospective study on the role of psychosocial factors in reports of back injuries. OSHA used this study in its weight of evidence determination for HPW as a risk factor for LBP, and found no association. However, OSHA concludes that physical risk factors were not as well determined in this study as were the psychosocial risk factors, making their relative contributions difficult to assess. Furthermore, the lack of truly HPW, according to the authors, among these workers would further limit the ability to study this physical risk factor. Thus, OSHA concludes that although this study found a significant relationship between psychosocial factors and LBP, this study lacked the ability to concurrently study the relative contribution of the physical work-

related risk factors of interest to OSHA. In Section G5 OSHA provides additional discussion of both the Bigos *et al.*study and psychosocial risk factors.

Biomechanical Factors and Laboratory Experiments

For a distilled summary of the literature describing laboratory experiments and biomechanical models of risk factors associated with low back pain in table format, see Table II–1 in the health effects appendices to the proposed rule (Ex. 27–1).

There is some debate as to the exact etiology of low-back pain, and some authorities suggest that it is possible to make a precise diagnosis in perhaps only 20% of patients presenting with acute low-back pain (Frymoyer 1988, Ex. 26–118; Nachemson 1976, Ex. 26–1147; White and Gordon 1982, Ex. 26–1160). Proposed etiologies for low-back pain that have been advanced include the roles of nerve compression, tissue ischemia, sensitization of nerve endings, inflammatory mediators, spinal instability, and other postulates (Frymoyer 1988, Ex. 26–118; Nachemson 1992, Ex. 26–490). The majority of cases of work-related low-back pain are attributed to mechanical causes, such as muscle and ligament strains and sprains and disc herniations. Degenerative disc or facet disease, spinal stenosis, spondylolisthesis and compression fractures have also been attributed, at least in part, to work. Additionally, back disorder is multifactorial in origin and may be associated with both occupational and nonwork-related factors and characteristics (Bernard 1997; Ex. 26–1).

One additional difficulty in evaluating the etiology of low-back pain is that roughly 50% to 60% of patients reporting an episode of work-related low-back pain note an insidious onset of pain rather than a single, point-in-time event with immediate low-back pain (Bergquist-Ullman and Larsson 1977, Ex. 26–933). This study also found that cases with an insidious onset experienced prolonged recovery. Part of the explanation for this may lie in the absence of nociceptors in the disc itself and the facet joints (except for the synovial lining) (Pope *et al.*1991, Ex. 502–502). These load-bearing structures may, therefore, become injured without immediate recognition (*e.g.,* sudden pain), and the eventual manifestation of low-back pain may only occur after a series of point-in-time events have sufficiently injured these spinal structures to the point where nociceptors become irritated (*e.g.,* in the outer one-third annulus or facet synovium).

Specific Low-Back Disorders

Low-back pain symptoms are caused by a variety of injuries and disorders. Although the underlying cause of back pain cannot be determined definitively in up to 90% of patients, work-related cases are believed to result from the following mechanisms: muscle or ligamentous (soft tissue) injury; herniation of the intervertebral disc with irritation of adjacent nerve roots; and degenerative changes (arthritis/spondylosis) in the intervertebral discs (Deyo, Rainville, and Kent 1992, Ex. 26–365). Evidence for work-relatedness for low-back disorders of these three sources of etiology is summarized below.

Soft Tissue/Mechanical Low-Back Disorders

As noted earlier, the exact etiology of low-back pain is unknown in many cases, and therefore, there is a lack of universal agreement on the contribution of muscle and ligament sprains and strains to work-related low-back disorders. In part, the difficulty in diagnosis relates to the inability to easily palpate deep low-back muscles, the lack of imaging information on low-back muscle disorders, and the absence of surgical pathologic specimens to evaluate.

However, in addition to an understanding of muscle anatomy, consideration of muscle function (static and dynamic loading), and repair mechanisms contribute to understanding the role of muscle and ligament sprains and strains in work-related low-back disorders.

Static Loading

In evaluating the pathogenesis of soft-tissue low-back disorders, there are considerations related to static and dynamic work activities. Simple maintenance of posture requires balancing of counteracting mechanical forces about the spine. Static loading affects muscle and connective tissue. During static trunk flexion, low-back extensor muscles must progressively increase their activity to maintain trunk flexion (Schultz *et al.*1982, Ex. 26–581).

Using myoelectric measurements, Andersson *et al.*(1974, Ex. 26–346) ascertained that activity of the erector spinae progressively decreased as the angle of the back rest advanced from 10 degrees of forward inclination to backward inclination. This results from a partial reduction of the lumbar spine load imposed by the upper body as the load is transmitted to the back rest (Andersson and Marras 1996, Ex. 26–412; Chaffin and Andersson 1991, Ex. 26–420). In addition, during unsupported sitting, the lumbar spine flattens, and the use of lumbar supports and back rests can reduce the loss of normal lordosis (Andersson *et al.*1979, Ex. 26–1553).

Using a back rest inclination of 110 degrees and a 4 cm lumbar support, the authors were able to demonstrate that lumbar posture could be similar to normal standing posture. Maintenance of adequate seated posture has further implications for the intervertebral disc, with lower intervertebral disc pressures noted during supported sitting as opposed to unsupported sitting (Andersson *et al.*1974, Ex. 26–346). Inadequate seating can contribute to the development of low-back pain. Individuals who sit in chairs that are too high and have their feet unsupported experience elevated pressure on the back of their thighs (Akerblom 1969, Ex. 26–522; Bush 1969, Ex. 26–455; Schoberth 1962, as cited in Chaffin and Andersson 1991, Ex. 26–420). Burandt and Grandlean (1963, Ex. 26–1569) observed the tendency of subjects in high seat pans to slide forward in their seats to support their feet, negating the benefit of a back rest.

Dynamic Loading

Dynamic loading of the lumbar spine has other implications for muscle and ligament. Stresses induced in the low back during manual materials handling relate to the load weight and the characteristics of the lift. As a result of their anatomic positions, large spinal movements are created from relatively small degrees of muscle shortening. Unfortunately, this results in the generation of relatively large muscle and joint forces, with potential for tissue overloading and injury. This could be particularly important during excessive or rapid movement (Andersson and Marras 1996, Ex. 26–412), or at the point of muscle fatigue.

A study by Hukins *et al.*(1990, Ex. 26–143) revealed that greater forces are exerted on ligaments as the speed of motion increases. In addition, elastic limits of the ligaments and disc may be exceeded (Adams and Dolan 1981, Ex. 26–1348). Bush-Joseph *et al.*(1988, Ex. 26–939) evaluated the effect of the speed of lifting on the external load moment. Subjects were asked to lift at slow, medium, and high speeds. There was a direct linear correlation between increasing speed of lifting and increased peak moment. Furthermore, a study by Marras and Mirka (1992, Ex. 26–982) revealed that muscles must generate a higher percentage of electromyographic (EMG) maximal activity to maintain a

constant muscle force as the speed of trunk velocity increases with bending.

Both lifting frequency and load weight affect back muscle work capacity, in part related to fatigue. Using EMG assessments, Kim and Chung (1995, Ex. 26–858) observed that lifting at 10% of maximum voluntary isometric strength (MVIS) at a rate of 6 times a minute was more fatiguing than lifting at 20% MVIS at a rate of 3 times per minute.

Frequent loading of the lumbar spine with moderate to heavy weights can also cause general physical fatigue with elevation in heart rate and energy expenditure. Uncoordinated muscle activation that could result from local and systemic fatigue could then place other tissues at increased risk with continued lifting (Garg 1986, Ex. 26–121).

Postural Issues

Additional postural factors during lifting significantly affect muscle function and risk of injury. Skeletal muscle is more likely to rupture during eccentric contraction (Friden and Lieber 1994, Ex. 26–546), a factor involved in many manual materials-handling tasks. In addition, muscle length affects the amount of force that muscle can generate, with maximal force produced when muscles are at their resting lengths (Andersson and Marras 1996, Ex. 26–412; Chaffin and Andersson 1991, Ex. 26–420). Therefore, lifting in positions where skeletal muscles are elongated or shortened can increase the risk of injury to these tissues.

Using EMG evaluation of muscle function during lateral flexion of the lumbar spine, Andersson, Ortengren, and Herberts (1977, Ex. 26–1570) demonstrated increased activity on the side contralateral to bending. Other researchers have determined that asymmetric loading in lateral flexion and axial rotation causes high levels of antagonistic activity in abdominal and back extensors. This is associated with increased myoelectric activity on the side of spine contralateral to the load, although there is still significant activity on the ipsilateral side (Astrand 1987, Ex. 26–527; Kelsey 1975, Ex. 26–1134; Magora 1970, Ex. 26–297; Merriam *et al.*1983, Ex. 26–299). Andersson (1977, Ex. 26–449) noted that increased intervertebral disc pressure and intraabdominal pressure occurs when the trunk is loaded in lateral flexion and axial rotation, with rotation being the greater factor.

Muscle Velocity and Acceleration

Marras (Ex. 26–1412) has indicated that several trunk muscle characteristics and demands associated with dynamic lifting may better assess the risk of developing a low-back disorder from manual materials handling. The authors analyzed 400 lifting jobs in 48 industries using a triaxial goniometer (Lumbar Motion Monitor or LMM) that was worn by working subjects. A combination of five trunk motion and workplace factors was able to reasonably predict jobs posing high risk for low-back disorders (Marras *et al.*1995, Ex. 26–1412). These factors include the lift frequency, load moment, trunk sagittal range of motion, trunk lateral velocity and trunk twist acceleration (Marras *et al.*1995, Ex. 26–1412). A recent NIOSH Health Hazard Evaluation provided additional verification that the LMM has predictive capacity equal to the NIOSH Lifting Equation in job analysis (NIOSH 1993, Ex. 26–521), with perhaps greater ease of administration.

Recently, Marras *et al.*(1990, Ex. 26–1523; 1993, Ex. 26–170; 1995, Ex. 26–171) studied the trunk angular motion characteristics of normal and chronic low-back pain subjects. Used in a clinical setting, the LMM appears to have good ability to accurately distinguish between normal individuals and those with chronic low-back pain or structural disease. The authors used anatomic and pain categories previously selected by the Quebec Task Force Study on Spinal Disorders (1987, Ex. 26–494). Normative trunk motion values for age and gender were derived in a study of 339 males and females from ages 20 to 70 years who had never experienced significant low-back pain. While wearing the LMM, subjects performed trunk flexion and extension in five symmetric and asymmetric motion planes (0 degrees, 15 degrees and 30 degrees right and left) while trunk angular position, velocity, and acceleration were recorded with the LMM. In a repeatability study, 20 healthy normal subjects who had never experienced a low-back disorder were tested with the LMM once a week for 5 weeks. No statistically significant differences were observed among the trunk motion characteristics between the five weekly test sessions using multivariate analysis of variance. Correlation coefficients were computed to select reliable trunk motion variables to be used in the next phase of the study. Correlations varied as a function of the angle of asymmetry and measured variables, with motion characteristics in the zero plane demonstrating correlation coefficients of 0.88 to 0.96 (number of conditions performed, twisting range of motion, sagittal range of motion at 0 degrees, sagittal extension velocity at 0 degrees, sagittal extension acceleration at 0 degrees, continuous velocity, continuous acceleration, lateral right range of motion at 0 degrees).

In the next phase, the eight highly reliable trunk motion characteristics evaluated in the healthy subjects were compared with measurements in subjects with chronic low-back pain (96 males and 75 females) who were recruited for study from secondary and tertiary referral practices. These individuals had been symptomatic for at least 7 weeks and had been sufficiently studied, including with appropriate imaging studies, to permit accurate Quebec classification. Dynamic trunk motion characteristics were normalized for age and sex, and using quantitative discriminant analysis, the 510 subjects were correctly classified in 94% of cases as being either healthy or having chronic low-back pain(stage-one analysis).

In a stage-two analysis, nine variables (the eight previously mentioned and continuous position) correctly classified 80% of subjects into one of eleven groups (normal, low-back pain alone, low-back pain with proximal or distal radiation, disc herniation with high or low pain scores, spondylolisthesis, spinal stenosis, postoperative, nonorganic components, other) via modified classification using splines. It was also noted that trunk range-of-motion parameters commonly used to quantify impairment had poor ability to discriminate normal vs. chronic low-back pain, nor was it useful in classification. Furthermore, a characteristic pattern of recovery from low-back pain was noted, with normalization occurring first in range of motion followed by velocity and later acceleration of dynamic trunk motion. It was opined that the LMM's ability to quantify unloaded free-dynamic motion and account for the co-activation of additional structures (*e.g.,* internal and external obliques, lattissimus dorsi) affecting erector spinae function was in part responsible for its enhanced discriminating ability compared to alternate imaging techniques.

Disc Disorders/Disorders of the Three-Joint Complex (Disc and Two Facets) and the Nerve Root

The three-joint complex refers to the intervertebral disc and two facet joints. This complex permits the spine to absorb compression and resist torsion and shear, while permitting translation and rotation of the spine. Epidemiologic evidence suggests that work exposures involving heavy lifting or manual materials handling are associated with

low-back disorders, including disc disorders (Bernard and Fine 1997, Ex. 26–1).

Excessive or repeated spinal loading and inadequate rest periods to permit repair mechanisms to function may be associated with biomechanical stresses that damage intervertebral disc cartilage endplates. This may then disturb metabolic transport, hastening the development of degenerative disc disease and disc herniation with secondary nerve root compression or inflammation.

Rowe (1971, Ex. 26–319) opined that up to 70% to 80% of recurring, chronic low-back pain will eventually be diagnosed as discogenic. Discogenic pain can include clear and consistent symptoms and signs expected with lumbar disc herniation and specific nerve root pathology, as well as chronic low-back pain associated with increased pressure in the intervertebral disc or degenerative disc disease. In patients with lumbar disc herniations, approximately 90% to 95% occur at the lower three intervertebral disc spaces (lumbar ¾ disc or lumbar 4th nerve root, lumbar ⅘ disc or lumbar 5th nerve root, lumbosacral L5/Sl or sacral 1st nerve root) (Deyo, Rainville, and Kent 1992, Ex. 26–365). Increased compressive and torsional forces transmitted to the lower levels of the lumbar spine probably account for this observation. Peak incidence of lumbar disc herniation occurs in adults during the working years from ages 30 to 55 (Spangfort 1972, Ex. 26–502). The onset of symptoms may be acute, subacute, or chronic, and the relationship to a single lifting incident may not always be obvious (Berquist-Ullman and Larsson 1977, Ex. 26–933). Symptoms and physical findings depend on the location of the disc herniation and the degree of nerve compression.

An understanding of disc biochemistry and biomechanics assists in the understanding of the pathogenesis of work-related lumbar disc disorders. For ethical reasons the majority of observations on spinal tolerance have been derived from cadaver spines. However, in vitro and in vivo comparisons appear to validate these conclusions. There is a wide biologic variation in human disc and end plate tolerances (Brinckmann *et al.,* 1988, Ex. 26–1318) related to age, gender, genetics, prior injuries, and other factors. The maximum axial compressive force tolerated by the human cadaver lumbar spine has been measured by Brinckmann *et al.,* 1988 (Ex. 26–1318) to range from 2.1 to 8.8 kN (210 to 880 kg), with 30% fracturing at forces below 4 kN and 63% fracturing below 6 kN. Adams and Hutton (1982, Ex. 26–1379) studied cadaver discs from male subjects aged 22 to 46 years. The authors determined that most specimens could withstand an average of 10 kN on single loading prior to failure, usually at the end plate. In contrast, Bartelink (1957, Ex. 26–349) noted that discs were fractured from forces ranging between 1.6 and 6.7 kN, with a mean of 3.1 kN. The wide inter-individual variation in tissue tolerance makes it difficult to assign a single value of compressive force against which to engineer jobs to prevent lumbar disc.

When mechanical failure occurs, it is generally through the cartilage endplates (Adams and Hutton 1982, Ex. 26–1379; Armstrong 1985, Ex. 26–1070; Brinckmann el al., 1988, Ex. 26–1318; Erdil, Dickerson, and Chaffin 1994, Ex. 26–424) Disc height, spinal position, and frequency of bending appear to be risk factors. Creep results in loss of disc height, increased contact between load-bearing surfaces of the facet joints, diminished capacity to dissipate forces, and decreased ability of the spinal column to tolerate loading (Kazarian 1975, Ex. 26–379). Adams and Hutton (1982, Ex. 26–1379) observed maximal single loading tolerances of up to 10 kN; however, when the spines were flexed forward, 40% of discs prolapsed at an average of only 5.4 kN. Repeated lumbar spine loading can cause tissue fatigue with fracture at lower loads than the spine would tolerate for non-repetitive loading. Adams and Hutton (1985, Ex. 26–1315) determined that when repetitive loading was simulated, previously healthy discs failed at an average of 3.8 kN.

These studies support the clinical observation that the intervertebral disc is especially vulnerable when loaded in the flexed position or when subjected to repetitive loading. This becomes more significant when workers with lower tissue tolerance from prior injury, degenerative disc disease, or age lift at high rates for prolonged periods.

Armstrong (1985, Ex. 22–877) noted that small microtears most often occur in the region of the posterior elements of the annulus fibrosus and cartilage end plates. As noted, these are the areas subject to the greatest spinal compressive forces (Gracovetsky and Farfan 1986, Ex. 26–128; Hickey and Hukins 1980, Ex. 26–708; Pope *et al.*1991, Ex. 26–1296). With repeated lumbar spinal stresses and/or injuries, progressive microfractures in cartilage end plates and annular fibers (annulus fibrosus) may develop in the intervertebral discs (initially toward the center of vertebral bodies). This causes altered metabolism and fluid transfer with different mechanical behavior of the disc.

Eventually radial tears result in the development of degenerative disc disease and/or bulging. As a result of this damage, the capacity of the lumbar intervertebral discs to tolerate further compressive loads during lifting is altered. When these smaller tears extend and form complete annular tears, the nucleous pulposis can protrude (disc herniation) (Farfan *et al.* 1970, Ex. 26–113). Over time, sclerosis of cartilage endplates and altered disc loading can facilitate the development of facet arthropathy, osteophytic change, stenosis, or instability. Disc degeneration in combination with facet arthropathy may also lead to foraminal narrowing with resultant nerve compression and radicular pain. These observations are consistent with a cumulative trauma theory that could account for some types of low-back injuries and is supported by the research and opinions of other authorities (Erdil, Dickerson, and Chaffin 1994, Ex. 26–424; Pope *et al.* 1991, Ex. 502–502; Yong-Hing and Kirkaldy-Willis 1983, Ex. 26–405).

While many individuals with degenerative disc disease are asymptomatic, individuals with greater degrees of degeneration are at risk for low-back pain. In one study (Vanharanta *et al.* 1987, Ex. 26–225) 90% of subjects with severe disc degeneration experienced pain during discography, while only 23% of those without disc degeneration reported pain.

Arthritis/Spondylosis

Several studies have suggested a relationship between lumbar degenerative disease and work activities (*e.g.,* heavy work, repetitive lifting, and vibration). This association has come from both radiographic and pathological evaluations in association with work histories. One difficulty in these evaluations is the observation that lumbar spine x-ray changes are common, occurring in about 40% of all low-back x-rays (Rowe 1983, Ex. 26–699). However, the relationship of many x-ray changes with symptoms of low-back pain is unclear (Andersson 1981, Ex. 26–1480; Himmelstein *et al.* 1988, Ex. 26–962; Magora and Schwartz 1976, Ex. 26–389; Rowe 1963, Ex. 26–317; 1969, Ex. 26–318). Videman, Nurminen, and Troup (1990, Ex. 26–1023) noted an increase in vertebral osteophytosis in autopsy specimens from workers who performed heavy work. Of interest is that the heavier work exposures also were observed in association with increased rates of low-back disability.

Riihimaki *et al.* (1991, Ex. 26–966) performed a radiographic study of the lumbar spine in concrete workers and house painters. Lateral lumbar x-rays were obtained in 216 concrete reinforcement workers and 201 house painters aged 25 to 54 years. Disc space narrowing was noted 10 years earlier and spondylophytes 5 years earlier in the concrete workers. Risk ratios for the univariate effect of occupation on disc space narrowing was 1.8, and for spondylophytes it was 1.6. Potential cofounders such as age, prior back accidents, body mass index, and smoking had minimal effect. The authors concluded that heavy physical work with materials handling and postural loading enhances the degenerative process of the lumbar spine.

Wickstrom, Nummi, and Nurminen (1978, Ex. 26–1161) evaluated degree of lumbar flexion, presence of pain, and x-ray findings of degenerative disc disease in 295 concrete reinforcement workers aged 19 to 64 years. These workers commonly perform work involving spinal loading in stooped postures. Radiographic evidence of degenerative disc disease was noted in two-thirds of the 110 individuals with restricted flexion and in one-third of those (n=185) with normal flexion.

Kirkaldy-Willis (1983, Ex. 26–431) described a pathophysiologic spectrum of changes that lead to the development of lumbar spine degenerative disease. In the first phase, there are early and mild changes in the posterior complex, with facet synovitis, joint effusion, capsular stretch, and thickening. Inflamed synovium may become entrapped in the joint between the cartilage surfaces and initiate cartilage damage. Meanwhile, the intervertebral disc develops some circumferential tears in the annulus fibrosus. Tears in the periphery have at least some potential to heal because of the proximity to vascularity, but these deeper tears lack this ability by virtue of their distance from blood flow or metabolic diffusion. As these circumferential tears enlarge, they develop into large radial tears. As a result, the nucleus pulposus begins to lose proteoglycan and exhibits structural changes with grade 1 or 2 degenerative disc disease. Loss of water and disc height as well as a decline in annular resistance can cause increased compression forces on the facets. Individuals may be asymptomatic or have vague low-back pain. However, due to the lack of nociceptors in the disc and facet joints (except the synovium), a significant degree of degenerative disease may occur before pain develops. Lumbar disc herniation may occur at this juncture with symptoms and signs or radiculopathy.

In the next phase, the posterior joint capsule and annulus fibrosus develops laxity and instability. The intervertebral disc progresses to grade 2 or 3 degenerative disease. It may be possible to detect instability on dynamic x-rays. Subperiosteal bone formation, calcification of the ligaments, and capsular fibers manifest as peripheral osteophytes and traction spurs (Dupuis 1987, Ex. 26–1299) in an attempt to stabilize the motion complex (MacNab 1977, Ex. 26–1367). If laxity predominates over repair processes, the degenerative spondylolisthesis (facet laxity) or retrolisthesis (disc laxity) may occur (Dupuis *et al.* 1985, Ex. 26–108).

In the final phase, there is fibrosis of the posterior facet joints, loss of disc material (grade 3 or 4 degenerative disc disease), and progressive osteophyte formation (Wedge 1983, Ex. 26–1035). This increases the load-bearing surface of the three-disc complex, although it decreases motion and results in increased stiffness. The repair process may create narrowing of the central canal (central spinal stenosis) from facet arthropathy, disc bulging, and hypertrophy of the ligamentum flavum. Lateral stenosis may also result from facet arthropathy and osteophyte formation adjacent to the neuroforamina. Spinal stenosis is a diagnostic entity that has only recently been described. A few patients have congenitally small spinal canals; however, most present with this type of acquired spinal stenosis secondary to longstanding degenerative disease. Most patients first become symptomatic after 50 years of age (Turner *et al.* 1992, Ex. 26–1455). By virtue of its long-term degenerative nature, spinal stenosis is not often considered a work-related disorder; however, patients with spinal stenosis may present with co-existing lumbar disc herniation or other degenerative changes that have been exacerbated by work factors.

Conclusions

OSHA finds convincing evidence from the confluence of many investigation on biomechanical models, laboratory research and epidemiology studies that work related risk factors including (1) heavy physical work, (2) lifting and forceful movements, (3) bending, twisting and awkward positions, and (4) static work positions are causally linked to low back disorders and pain. Work often involves several of these risk factors concurrently and there is evidence that the first three of these factors may act together in a synergistic way to increase the risk. However, OSHA considers that each factor, by itself, can increase the risk of back disorder.

F. Disorders of the Lower Extremities

Work-related disorders of the lower extremities have not received the same scrutiny as those of the upper extremities and back. However, existing information from pathophysiology, epidemiological studies, and biomechanical investigations implicate physical work factors related to repetitive, forceful exertion and awkward posture to these disorders, especially osteoarthritis of the knee and hip. As more completely described in Health Effects Appendix III.D (Ex. 27–1), osteoarthritis is considered a disorder of the movable joints characterized by the disintegration of the articular cartilage that covers the end of the bones. The articular cartilage and subchondral bone that lies just beneath the cartilage provide opposing structures and surfaces that are matched in such a way as to allow transmission of joint loads at the lowest and most uniform pressures, (Meisel 1984, Ex. 26–1562).

The arthrosis process is thought to begin with disruption at the thin surface overlying the load-bearing cartilage (Meisel, 1984, Ex. 26–1562). This disruption results in progressive erosion of the cartilage layer and a joint surface less able to withstand normal loads and forces. Continual loading on the joint then disrupts the process of bone/cartilage repair and regeneration, leading to formation of marginal bone in the shape of spurs (osteophytes). The degenerative process continues until the cartilage has been completely destroyed; there is bone-on-bone contact, and the structural integrity of the joint is lost. The clinical manifestations are joint stiffening, pain and loss of movement (Meisel 1984, Ex. 26–1562).

It is well recognized that acute trauma can trigger osteoarthritis, but there is also evidence that less substantial, but repetitive, forces to the joints can lead to microfractures of the articular cartilage and subchondral bone. The disruption in structural integrity results in the onset of the degenerative changes described above (Radin *et al.,* 1994, Ex. 26–578). This process has been observed in animals subjected to repetitive impact loading of one or more limbs (Moskowitz, 1992, Ex. 26–1547). Damage to the joints in these animals involve fibrillation and splitting of the cartilage, evidence of chondrocyte activity as bone remodeling occurs, progressive erosion of the cartilaginous layer, and formation of osteophytes.

Other MSDs of the lower extremity that may be caused by physical work-related factors include bursitis and tarsal tunnel syndrome. Joint overuse may lead to bursitis, an inflammation of a fluid-filled sac or sac-like cavity that serves to reduce friction in a joint (Ex. 502–317). Repetitive use of the foot may be related to tarsal tunnel syndrome, a nerve entrapment syndrome of the lower extremity analogous to carpal tunnel syndrome in the wrists (Day 1996, Ex. 26–615).

In addition to acute and repetitive trauma, MSDs of the lower extremities have been linked with congenital abnormalities, underlying genetic or metabolic disorders, and chronic conditions, such as cancer, diabetes and collagen-vascular disease (Felson 1994, Ex. 26–544; Meisel 1984, Ex. 26–1562).

Epidemiological Evidence

Epidemiological evidence of an association between workplace factors and MSDs of the lower extremities was discussed in Health Effects Appendix I. A summary of the risk factors is presented in Table C–1 (for osteoarthritis of the knee) and Table C–3 (for the hip). Several work-related activities, such as squatting and kneeling for more than 30 minutes per day, were significantly associated (OR≥3) with osteoarthritis of the knee in a population-based case-control study (Cooper *et al.,* 1994, Ex. 26–460). This study also showed that a combination of these activities along with lifting loads greater than 25 kg (which places an additional load on the lower extremities) resulted in an even stronger association (OR≥5) with this knee disorder. Other epidemiological studies associated occupations such as construction work, farming, firefighting, laundry/dry cleaning, and manual labor, with knee osteoarthritis (Anderson and Felson, 1988, Ex.26–926; Vinguard *et al.,* 1991, Ex. 26–1500).

Three case-control studies reported positive associations between MSDs of the hip and work tasks involving biomechanical factors (Coggon *et al.,* 1998, Ex. 26–1285; Croft *et al.,* 1992, Ex. 26–1503; Vinguard *et al.,* 1997, Ex. 26–1617). One study found that jobs requiring lifting over 25 kg more than ten times in an average week for more than 20 years raised the odds of developing hip osteoarthritis (Ex. 26–1285). Farmers, mail carriers, firefighters, and meat processors were occupations reported to be significantly associated with hip osteoarthritis in a registry-based cohort study (Ex. 26–400). Repetitive kneeling, squatting, and lifting are all activities involving the biomechanical risk factors of repetition, forceful exertion, and awkward postures of the lower joints. Table V–8 summarizes some key aspects of these investigations, including: Occupations examined; biomechanical risk factors involved; whether or not exposures were directly observed during the study, whether the health outcomes were verified by medical tests, whether evidence provided of an exposure-response or other temporal relationship between the risk factor and outcome; and the measure of relative risk used along with the results of this measure.

In addition to the evidence previously reviewed, Table V–8 includes five additional studies submitted to the docket that address physical work factors and disorders of the lower joints, primarily the knee (Ex. 500–41–114; Ex. 500–121–44; Ex. 500–41–69; Ex. 502–317; Ex. 500–41–68; Ex. 500–121–18. Three of the studies examined the prevalence of knee disorders among carpet- and floorlayers who spend a substantial amount of time working in knee straining postures. Kivimaki (1992, Ex. 500–41–78) compared 96 floor- and carpetlayers to 72 painters with regard to disorders of the knee. An analysis of videotaped work tasks indicated that floor- and carpetlayers assume a kneeling posture in their job 42% of their work time, compared to 3% of work time by painters. Ultrasonographic examination indicated changes in the prepatellar or superficial infrapatellar bursa in 49% of the carpet and floor layers compared to 7% of painters. On a symptom questionnaire, the floor- and carpetlayers reported a significantly greater prevalence of bursitis in front of the knee cap, knee pain in a kneeling posture, sudden and intense swelling of the knee, aspirations of the knee, and injections to the knee than painters.

TABLE V–8.—SUMMARY OF EPIDEMIOLOGY STUDIES EXAMINING MSDS OF THE LOWER EXTREMITIES

Study	Job type studied	Physical factors	Exposure basis	Diagnosis/body part	Other attributes	Risk measure (95% CI)[1]
Kivimaki (1992) Ex. 500–41–78.	carpet laying; floor laying.	F/R/P	observation questionnaire.	questionnaire ultrasound/knee.		NR*
Jensen (1997) Ex. 500–41–69	carpet laying; carpentry.	F/R/P	questionnaire observation.	questionnaire radiology/knee.	exposure response	OR=1.5–6.4* (3.2–8.9)
Tanaka (1986) Ex. 502–317 ..	floor laying; tile setting.	F/R/P	questionnaire	questionnaire knee	exposure response	PRR=1.1–5.0* (3.2–7.8)
Sandmark (2000) Ex. 500–41–114.	prosthetic knee patients.	F/R/P	questionnaire	surgery/knee	exposure response	OR=0.7–3.2* (2.0–5.2)
Cooper (1994) Ex. 26–460	general population	F/R/P	questionnaire	questionnaire X-ray/knee.		OR=0.8–6.9* (1.8–26.4)
Anderson (1988) Ex. 26–926	general population	F?/R/P	job title questionnaire.	questionnaire X-ray/knee.		OR=0.8–3.5* (1.2–10.5)
Vingard (1991) Ex. 26–1400 ..	various occupations.	F/R?/P?	job title	hospitalization knee or hip.		RR=0.6–3.8* (1,2–12.1)
Coggon (1998) Ex. 26–1285	patients case/control.	F/R/P?	questionnaire	hip replacement		OR=1.0–2.1* (1.1–3.9)
Croft (1992) Ex. 26–1503	patients case/control.	F/R?/P?	questionnaire job title.	joint measurement/hip.		OR=0.8–2.5 (1.1–5.7)
Vingard (1997) Ex. 26–1617 ..	patients case/control.	F/R/P?	questionnaire	hip replacement		RR=0.8–2.3* (1.5–3.6)
De Zwart (1997) Ex. 500–121–18.	Various occupations.	F/R/P	job title	questionnaire lower limbs.	temporal relationship.	NR*

F=forceful exertions; R=repetitive motion; P=awkward posture; ?=presence of risk factor unclear;
RR=relative risk; OR=odds ratio; PRR=prevalence rate ratio
*=p<0.05

[1] 95% confidence interval expressed for the upper end of the risk measure range.

Jensen *et al.* (1997, Ex. 500–41–69) conducted a larger cross-sectional study of knee disorders among current and former floor- and carpetlayers (N=133), carpenters (N=506), and compositors (N=327). Based on telephone interviews and video recording of work activities, the authors determined that floor- and carpetlayers spent 56% of their working time in knee-straining postures. Carpenters were reported to have spent 25% of their working time in such postures, while compositors did not spend any working time in knee-straining positions.

Response to a questionnaire revealed that carpenters experienced a significantly increased frequency of knee complaints within the last 12 months (OR=3.8, 95% CI: 2.7–5.5), within the last seven days (OR=3.6, 95% CI: 2.3–5.8), and for more than 30 days over the preceding 12 months (OR=2.5, 95% CI: 1.6–3.9) when compared to compositors. Floor- and carpetlayers, the highest exposed group, also reported a significantly increased frequency of knee complaints within the last 12 months (OR=6.4, 95% CI: 4.0–10.1), within the last seven days (OR=5.7, 95% CI: 3.3–10.1), and for more than 30 days over the preceding 12 months (OR=5.3, 95% CI: 3.1–8.9) when compared to compositors; the odds ratios reported for floor- and carpetlayers were uniformly higher than those reported for carpenters. Age, weight, body mass index, smoking, and sports activities were reported to have had no significant effect on the incidence of knee complaints. Among 50 floor- and carpetlayers, 51 carpenters, and 49 compositors who had radiological examinations of their knees, an increased prevalence of osteoarthritis was found in floor- and carpetlayers (14%) when compared to carpenters (8%) and compositors (6%).

A third cross-sectional study involving floorlayers by Tanaka *et al.* (1986, Ex. 502–317), and also reported by Thun *et al.* (1987, Ex. 26–60), examined the relationship between work activities involving strain on the knees and the development of knee disorders. Floorlayers (N=112) and tilesetters (N=42) who reported frequent kneeling in a survey questionnaire were compared to a group millwrights, bricklayers, and decorators (N=243) who did not commonly kneel.

The floorlayers reported more frequent bursitis of the knee (20% vs. 6%) and more needle aspirations of knee fluid (32% vs. 6%) than the millwrights and bricklayers. Tilesetters also reported bursitis (11%) and knee aspirations (31%) in excess of those reported by millwrights and bricklayers.

In this study questionnaire responses were compared to responses given by a representative sample of white males to standardized questions about symptoms of knee disease. When compared to sample, floorlayers, tilesetters, and millwright and bricklayers all reported a higher age-adjusted prevalence for each of the seven symptoms than the sample. This result suggests that the relative risk of knee disorders in the highly exposed groups may be understated when millwrights and bricklayers are the reference group since they may, themselves, be at increased risk relative to the general population.

Physical examination that included radiological tests of a subset of the workers was performed to validate the questionnaire. The questionnaire was reported to show low sensitivity (38–44%), but moderate specificity (82–89%), for both bursitis and arthritis.

Other studies examined the relationship between lower limb MSDs and physical work factors in more diverse occupational settings. Using a case-control study design, Sandmark *et al.* (2000, Ex. 500–41–114) compared individuals who had received prosthetic knee replacements due to osteoarthritis to control subjects to examine the relationship between lifetime physical load from work and the risk of knee osteoarthritis. A total of 625 individuals who had received prosthetic knee replacements due to osteoarthritis, and who were between the ages of 55 and 70 at the time of surgery were compared to 548 age- and gender-matched individuals randomly selected from the population of the same geographical area who had not reported osteoarthritis or other dysfunction of the knee.

Through telephone interview and written questionnaire, the subjects provided information on workloads from occupational and non-occupational activities, personal characteristics, and general health status. The duration and frequency of activities (*e.g.,* kneeling, sitting, number of stairs climbed) were computed for each individual. Subjects were then divided into three exposure groups: No or low exposure comprising the lower quartile; medium exposure comprising the middle two quartiles; and high exposure consisting of the top quartile.

Analysis of the data revealed that, among men, lifting at work (OR=3.0, 95% CI: 1.6–5.5), squatting or knee bending (OR=2.9, 95% CI: 1.7–4.9), kneeling (OR=2.1, 95% CI: 1.4–3.3), and jumping (OR=2.7, 95% CI: 1.7–4.1) were significantly associated with osteoarthritis of the knee. Individuals who had spent ten or more years in an occupation considered to involve high physical load on the knee were also more likely to undergo knee replacement due to osteoarthritis than those who had not worked in such occupations (men, OR: 2.5, 95% CI 1.7–3.6; women, OR: 2.5, 95% CI: 1.6–3.9). The analysis controlled for confounders such as age, body mass index, smoking, and sports activities.

The findings of Sandmark *et al.* (Ex. 500–41–114), Jensen *et al.* (Ex. 500–41–69) and Tanaka *et al.* (Ex. 502–317) indicate an exposure—response relationship between the frequency of work involving strain to the knees and osteoarthritis, bursitis and other signs of injury to this joint.

In a longitudinal survey study, de Zwart *et al.* (1997) (Ex. 500–121–18) investigated changes in musculoskeletal complaints among workers performing mentally demanding work (N=4686) and heavy physical work (N=7324). Job demands were determined by occupational title. Mentally demanding work was described as sedentary, while heavy physical work involved tasks such as lifting heavy objects, handling heavy tools, and stooping in combination with standing or walking. The subject groups were stratified by age (20–9, 30–9, 40–9, 50–9 years old). The occurrence of musculoskeletal complaints were compared between two surveys having a mean interval of approximately four years. No physical examination or examination of medical records was performed.

The incidence of musculoskeletal complaints of the lower limbs on the second survey was higher among those who had not reported complaints on the first survey for all age groups. However, the incidence was only statistically significant for the youngest three age groups. The authors concluded that younger and middle-aged employees develop musculoskeletal complaints as a result of exposure to heavy physical work, and that a healthy worker effect served to mask this effect for the oldest age group. Because of its prospective design, this investigation provides a temporal link between MSDs of the lower extremities and heavy physical work.

Lemasters *et al.* (1998) (Ex. 500–121–44) examined the prevalence and risk factors for work-related MSDs among carpenters. (N=522) who completed a

questionnaire on musculoskeletal symptoms, work history, and psycholsocial factors. The symptom questions assessed if they experienced pain, numbness, or tingling in a particular body region.

Generally, as duration of employment increased, the prevalence of symptoms increased. An adjusted logistic regression analysis showed that duration of employment in carpentry for at least 20 years was significantly associated with work-related MSDs of the knees (OR: 3.5, 95% CI: 1.3–9.2). Carpenters who indicated they felt exhausted at the end of day experienced significant increases of work-related MSDs of the knees (OR: 1.8, 95% CI: 1.1–3.1). Having minimal influence over their work schedule was also reported to be a risk factor for work-related MSDs of the knees (OR: 2.3, 95% CI:1.2–4.1).

A subset of the subject group received a physical examination including examination of the knees. The authors concluded that reported disorders, including those of the knee, were significantly associated with positive findings upon physical examination.

An examination of the reliability of questionnaire responses was performed by Booth-Jones *et al.* (1998) (Ex. 500–121–9). Ten percent of the subjects examined by Lemasters *et al.* (1998) (Ex. 500–121–44) were subsequently randomly selected and administered the original questionnaire for a second time. All positive responses were categorized as ''yes'' answers and all other responses were categorized as ''no'' responses. Comparison of the results of the first and second administrations of the test indicated that the responses were largely consistent, with overall agreement reported to be 85.6%. This result provides a strong indication that the questionnaire responses examined by Lemasters *et al.* (1998, Ex. 500–121–9) are a reliable representation of the recollections of the subjects examined.

A significant concern when evaluating studies in which exposure measurements and health outcome are based on self-reports is the possibility of recall bias. Among the studies pertaining to the lower extremities that are described here, those of Sandmark *et al.* (Ex. 500–41–114), Jensen *et al.* (Ex. 500–41–69), Tanaka *et al.* (Ex. 502–317), and Lemasters *et al.* (Ex. 500–121–44) each depend to a greater or lesser extent upon the accuracy of self-reported exposures to ergonomic risk factors. Such self-reports have been criticized as being unreliable (Exs. 30–276, 500–118). Evidence submitted to the docket regarding the studies discussed above, while not eliminating concerns about the reliability of self-reports, generally support their accuracy.

The validity of self-reporting as a means of measuring knee-straining work postures was examined by Jensen *et al.* 2000, Ex. 500–41–68). Self-reports were compared to timed video recordings for 39 carpenters and 33 floorlayers. The carpenters and floorlayers were videotaped while working and, then immediately afterwards were requested to estimate the amount of time spent in knee-straining postures. A close association was reported between the observed and self-reported durations (Spearman's correlation coefficient: 0.88). While this report provides evidence that immediate self-reports are largely accurate, recall bias associated with self-reports of historical work activities remains a concern.

Biomechanical Evidence

Bhattacharya *et al.* (1985, Ex. 502–270) examined the biomechanical forces associated with different working postures involved in carpet installation when using a knee kicker. The knee kicker is a device consisting of a plate with a set of teeth in one end that grips the carpet while an installer kicks the padded end with a knee to stretch the carpet. A job analysis indicated that carpet installers spend approximately 75% of their time in a kneeling position, and use the knee kicker an average of 141 times per hour. Postures were reported to require near-maximum knee flexion. Knee-flexion angles at impact averaged about 58°, while normal daily activities involve less flexion (*e.g.,* sitting, 87°; tying shoe laces, 74°; walking upstairs, 97°). Workers performing the heaviest of the knee kicks produced peak impact forces averaging over 3000 newtons, equivalent to approximately four times their body weight. The authors suggested that the biomechanical demands of installing carpet may be responsible for the high incidence of knee disorders among these workers.

Conclusion

OSHA concludes that strong evidence is available showing that steoarthritis of the knee and other MSDs of the lower extremities can result from exposure to the combined physical work-related factors of repetition, force, and awkward posture. This evidence comes from the consistently positive associations in epidemiological studies of carpet- and floorlayers who spend considerable amounts of time in knee-straining postures. Biomechanical evidence indicates knee flexion and impact forces can be substantial during installation of carpet. Other occupational activities that involve excessive squatting, kneeling, and climbing stairs have also been shown to be associated with osteoarthritis of the knee and hip. Some studies indicate an exposure—response or temporal relationship between physical risk factor and health outcome. Therefore, it is biologically plaucible that repetitive impact loading on the joints is consistent with the degenerative pathophysiology of osteoarthritis. OSHA concludes that the evidence reviewed in this section demonstrates that workers who perform job tasks requiring repeated forceful flexion of the knee or other joints of the lower extremities are at increased risk of serious musculoskeletal impairment such as osteoarthritis.

G. OSHA's Response to Health Effects Issues Raised in the Rulemaking

1. Comments on OSHA's Use of the NIOSH (1997) and NAS (1999) Reviews

Several commenters (Ex. 30–1722; Ex. 500–109; Ex. 32–368–1; Ex. 32–241–4; Ex. 500–197) criticized OSHA's reliance on the 1997 NIOSH review (Ex. 26–1) and the 1999 NAS report (Ex. 26–37) of the evidence for work-related MSDs. First, the commenters considered the methodology used by NIOSH to evaluate the epidemiological evidence that work-related factors were associated with MSDs to be seriously flawed. Second, they accused OSHA of ignoring obvious limitations of the NIOSH review and then misrepresenting its conclusions. Finally, the commenters claimed that the NAS workshop report did not support the OSHA position with regard to biomechanical risk factors and MSDs. A more detailed description of each assertion will follow along with OSHA's response.

The criticisms of the NIOSH methodology were aimed at nearly every level of evaluation. It was said that NIOSH exercised a ''publication bias in favor of positive studies'' in its study selection (Ex. 500–197, pg. I–146). It was said that the NIOSH criteria used to assess study quality ''emphasize[d] biased and unreliable methodology at the expense of sound scientific approaches.'' (Ex. 32–241–4, pg. 109). It was said that there was ''no indication of any systematic method for assigning weight,'' (Id. pg. 109), and that the weighting could not be ''replicated and, therefore fails to satisfy one of the most basic tenets of scientific inquiry.'' (Ex. 23–109, pg. 23). It was said that NIOSH ''failed to adequately consider other confounding factors in their analysis'' (Ex. 32–368–1, pg. 40). Finally, it was said that NIOSH was ''forced to draw its conclusions from a larger body of

literature that included studies meeting only some, or even none of these criteria.'' (Ex. 500–197, pg. I–148). One commenter summed up the NIOSH evaluation process as follows:

> The report did not conform to the generally accepted scientific methods for critical analysis. It did not use a weight of the evidence approach. For example, there is no explanation of how studies which met NIOSH's criteria standards were regarded differently than studies which did not. In essence, NIOSH put the 2000 studies into a black box, and out popped 600. Then the 600 went into another black box, and out popped the conclusions (Ex. 32–368–1, pg. 36–37).

OSHA strongly disagrees that the approach used by NIOSH to evaluate the epidemiological studies was flawed or that the conclusions in the 1997 review are weakly supported by the evidence. In the first chapter of its report, NIOSH describes, in detail, where it retrieved information on epidemiological studies, how studies were selected for more detailed review, the procedure used to analyze the overall strength of work-relatedness, the six criteria (strength of association, consistency, temporality, exposure-response, coherence, and role of confounders) employed to evaluate the evidence of causality, and the four categories to classify the evidence. The 600 studies reviewed by NIOSH [out of more than 2,000 identified in initial database searches] were published or accepted for publication in the scientific literature or government reports that had undergone peer review and were widely available. These had to meet some minimum requirement in terms of defined study groups, measurable health outcomes, identifiable exposures related to physical factors, and adequate study design. The NIOSH selection strategy was a common screening approach that has been successfully employed by OSHA and many other groups. There was no bias toward the selection of positive studies; rather NIOSH selected those only studies that met the above criteria. OSHA believes that the NIOSH selection process captured the best epidemiological studies available at the time on which to evaluate the evidence for a causal association between work-related risk factors and MSDs.

NIOSH analyzed the reviewed studies in terms of well-accepted epidemiological principles, such as participation rate, blinded study design, exposure method, and case definition and gave greater weight in its evaluation process to those that minimized selection and observation bias and confirmed the existence of exposure and health outcome by qualified experts. NIOSH applied the highly-regarded Bradford Hill criteria (see six criteria above) for judging the evidence for causation in classifying work-relatedness. These criteria were not applied to any single investigation but to the entire database of studies as a whole. NIOSH judged there was evidence of work-relatedness between biomechanical factors and MSDs when there existed convincing evidence from several studies for a causal relationship using the epidemiologic criteria, and for which chance, bias, and confounding factors were not the likely explanation. OSHA believes that NIOSH clearly did not use a ''flawed'' methodology and their evaluation process represents a systematic weight of evidence approach that relies on an unbiased set of sound and reliable scientific principles.

NIOSH concluded there was evidence that MSDs of the neck, shoulder, upper extremities, and back that have been subjected to epidemiological investigation were associated with at least some biomechanical factors or combination of factors. In several instances, the evidence was judged to be strong. For most MSDs, there were situations in which the epidemiological evidence was judged insufficient for certain biomechanical factors in isolation (*e.g.* CTS and extreme posture; epicondylitis and repetitive motion). However, these factors were usually found to be associated with the MSD when present in combination with other biomechanical factors (*e.g.* strong evidence of posture/force combination and CTS; strong evidence of repetition/force and epicondylitis). For several MSDs, OSHA found that the strength and consistency of the associations between biomechanical factors and MSDs was even stronger, if the evaluation was restricted to studies where exposure was directly observed or measured and the health outcome was confirmed by physical exam or medical tests (see Health Effects Section V). It is important to note that the NIOSH analysis focused primarily on the epidemiological evidence. OSHA believes these conclusions were reasonable and based on the selected evaluation criteria.

Since the evaluation process involved expert judgment, weighting of individual studies cannot be precisely ''replicated'' in the same way as a scientific measurement, however, substantial evidence in the rulemaking record supports NIOSH's conclusions. There were a number of written submissions and oral testimony from scientific experts supporting the position that sufficient evidence exists that biomechanical factors can increase the risk of MSDs (*e.g.,* Exs. 30–3805, 32–57, Tr. 9819, 16317, 17358, 17687). Some notable testimony on the epidemiological evidence from distinguished experts were as follows:

> There is a significant body of epidemiological and case study literature that indicate that a high rate of work-related MSDs, carpal tunnel syndrome, bursitis, tendinitis, and epicondylitis are significantly higher in jobs that involve repetitive motions, localized stress, awkward positions, vibrations, and forceful exertions.
>
> Dr. Robert McCunney (Tr. 17566–67)

> OSHA's conclusion that there is an epidemiological evidence of an association between many work factors and certain MSDs is consistent with the literature that I've read and my clinical experience as an occupational medicine physician treating thousands of patients with MSDs over the past 20 years.
>
> Dr. Michael Erdil (Tr. 1112)

> We have, first of all, lots of epidemiological studies that show physical factors are involved in MSDs. We have actually no epidemiological study that shows, that proves there is no physical factor involved.
>
> Dr. Niklas Krause (Tr. 1367)

Some commenters thought that OSHA misrepresented the findings from the NIOSH review in order to support its own conclusions that exposure to work-related biomechanical factors increase the risk of serious musculoskeletal impairment. It was claimed that OSHA had seriously overstated the NIOSH conclusions as ''having established causation'' (Ex. 32–241–4, pg. 98) between biomechanical factors and MSDs regardless of the length and intensity of exposure, instead of the true NIOSH goal of drawing conclusions about the evidence of an association between risk factor and health outcome under conditions of prolonged exposure. Commenters argued that OSHA ignored the restricted scope of the NIOSH analysis that was limited to ''certain objectively defined MSDs'' and ''examined only certain very specific stressors of highly repetitive and forceful work, lifting and forceful movements, awkward and prolonged sustained postures and exposure to vibration.'' (Ex. 500–109, pg. 24). On the other hand, it was claimed that OSHA used the NIOSH findings to ''support causal inferences for all other MSDs * * * which include not only those MSDs studied by NIOSH but also DeQuervain's disease, trigger finger, Raynaud's syndrome and tarsal tunnel syndrome'' and ''attempts to broaden the NIOSH exposure associations to include not only the factors that NIOSH studied, but also a wide range of other so-called ergonomic risk factors including among others, contact stress and cold temperatures.'' (Ex. 30–1722, pg. 43).

OSHA does not agree that the findings of the 1997 NIOSH review have been misrepresented in any way. The Agency has not stated that the epidemiological evidence established that MSDs are caused by exposure to work-related biomechanical factors. Epidemiological studies rarely, if ever, prove causation. They are designed to identify associations between two study variables. Depending on the strength and consistency of the associations and whether the association shows aspects of temporality and exposure-response, epidemiological data can provide evidence of a causal relationship. OSHA has stated that there is convincing scientific evidence that biomechanical factors, usually in combination, increase the risk of several specific MSDs. These conclusions are often based, not on epidemiological studies alone, but also on the pathophysiology of the disorder and biomechanical and psychophysical research that are able to link ergonomic risk factors to biomechanical and subjective measurements under a more controlled set of simulated work conditions.

In general, the conclusions drawn by OSHA based on the entire body of scientific evidence track closely with those of NIOSH. OSHA does not stretch the NIOSH findings ''far beyond the breaking point'' to support causal inferences of the existence of vast numbers of MSDs that are not examined by the epidemiological studies (Ex. 30–1722, pg. 44). For example, DeQuervain's disease and trigger finger are forms of hand tendinitis specifically examined in epidemiological studies (Ex. 26–48; Ex. 26–53; Ex. 26–897) relied on by NIOSH to conclude evidence of an association between repetition, force, and awkward posture and hand/wrist tendinitis. In fact, NIOSH states in its review that ''DeQuervain's disease and other tenosynovitis of the hand, wrist, and forearm have been associated for decades with repetitive and forceful hand activities as one of the possible causal factors.'' (Ex. 26–1, pg. 5b–8).

The other two MSDs cited as not being supported by NIOSH findings are Raynaud's phenomenon and tarsal tunnel syndrome (TTS). Raynaud's phenomenon refers to blanching of one or several fingers and is a characteristic sign of vascular damage that occurs in Hand-Arm Vibration Syndrome (HAVS) due to segmental vibration (Ex. 502–18). NIOSH concluded that there was strong evidence of a positive association between segmental vibration and the vascular symptoms of HAVS. TTS is an MSD of the foot and, therefore, was not addressed in the NIOSH review. However, it is a nerve impingement disorder analogous to CTS in the wrist. Like the carpal tunnel, the tarsal tunnel is a relatively ''tight'' compartment filled with flexor tendons and the tibial nerve that may be susceptible to compression in response to increases in intra-tarsal pressure as a result of repeated flexion/extension of the ankle.

In the Final Rule, OSHA does not broaden the set of biomechanical risk factors associated with MSDs beyond the four (force, repetition, posture, and vibration) supported by the 1997 NIOSH review (contact stress, which is covered by the standard, is a particular combination of force and repetition). Although OSHA believes that evidence exists that cold temperatures can aggravate some MSDs, this environmental factor principally operates to modify exposure to some of the biomechanical factors listed above and is not regarded as a primary risk factor. OSHA included contact stress in the final rule's Basic Screening Tool because there is reasonable evidence that repeated impact, such as hand hammering, increases the risk of the MSD known as hypothenar hammer syndrome (see Part D of the Health Effects section). In addition, repetitive knee hammering has been shown to be associated with a high risk of bursitis (''carpet layers knee'') (see Part F of Health Effects section). The final rule makes clear that it is prolonged and regular exposure to a combination of biomechanical work factors that presents the greatest potential hazard.

It should also be noted that workplace intervention is not required by the ergonomic standard unless there is an MSD incident that the employer has determined to be work-related and there is evidence of exposure to the biomechanical risk factors defined by the OSHA basic screening tool. This action trigger serves to limit the number of stressors and disorders that require action under the OSHA rule.

For the above reasons, OSHA finds that its conclusions with regard to work-related biomechanical factors and risk of MSDs do not misrepresent, but are entirely consistent with, the findings in the 1997 NIOSH review. This view was confirmed by written testimony from the Director of NIOSH, Linda Rosenstock:

> OSHA builds on the evidence of the association between workplace risk factors and the development of MSDs provided in the 1997 NIOSH review and strengthens the evidence with the supporting data provided by laboratory and psychophysical studies * * * NIOSH concurs with OSHA's conclusion from the discussion of the evidence from the epidemiological studies. OSHA concludes that ''In sum, although not all of the epidemiological studies reviewed demonstrate significant associations, the overwhelming majority justify a conclusion that the risk factors noted in this section, with effects adjusted by the four modifying factors, cause or exacerbate work-related MSDs.'' Thus the data justify the conclusion that these factors cause or exacerbate work-related MSDs (Ex. 32–450–1, pg. 7–8)

The commenters also claimed that OSHA misrepresented the findings of the NAS workshop and that the conclusions in their 1999 report ''simply do not support OSHA's broad conclusions linking physical work-related factors to musculoskeletal complaints.'' (Ex. 32–241–4, pg. 117). They allege numerous inadequacies of the workshop, such as the fact that the participants included ''only a few scientists who seriously questioned OSHA's ergonomic hypothesis'' (Ex. 32–368–1, pg. 33). Despite this, the workshop participants supposedly seriously questioned the NIOSH study and, unlike OSHA, ''admitted that the evidence of a link between MSDs and physical risk factors at the workplace is inconclusive at best,'' (Ex. 32–241–1, pg. 118). This led one NAS panelist, Dr. Howard Sandler, to state ''that the NIOSH approach to their review of the evidence was sufficiently flawed to make the conclusions questionable.'' (Ex. 32–241–4, p. 112). Presumably the NAS report ''actually undermines OSHA's decision to limit its analysis to physical, work-related factors'' since it cites ''individual, organizational, and social factors * * * which are possible influences on physiological pathways that lead from soft tissue to impairment and disability.'' (Ex. 32–241–4, p. 118). The argument for the OSHA misrepresentation of the NAS report is summarized as follows:

> In sum, the [NAS] Steering Committee advised *against* doing exactly what OSHA does in its analysis—focusing exclusively on physical work-related factors: ''Non-biomechanical factors *must* [emphasis added] be considered if understanding of the relationship between biomechanical work factors and MSDs is to expand and inform in the design of workplace interventions to reduce or prevent such disorders.'' (Ex. 32–241–4, p.120).

OSHA does not believe the NAS report seriously questions findings of the NIOSH review or undermines the OSHA position on the evidence that exposure to biomechanical factors increases the risk of MSDs. Regarding the epidemiological evidence, the NAS Steering Committee Report states:

> Restricting our focus to those studies involving the highest levels of exposure to biomechanical stressor of the upper

extremity, neck, and back and those with the sharpest contrast in exposure among the study groups, the positive relationship between the occurrence of musculoskeletal disorders and the conduct of work is clear. The relevant studies have not precisely determined the causal mechanical factors involved nor the full clinical spectrum of the reported MSDs (which are often lumped together nonspecifically as MSDs of a body region); nonetheless, those associations identified by the NIOSH review as having strong evidence are well supported by competent research on heavily exposed populations (Ex. 26–37, pg 15–16).

There is compelling evidence from numerous studies that as the amount of biomechanical stress is reduced, the prevalence of musculoskeletal disorders at the affected body region is likewise reduced. This evidence provides further support for the relationship between these work activities and the occurrence of musculoskeletal disorders (Ex. 26–37. p 16).

OSHA believes these NAS conclusions are not ''inconclusive at best'' but as the commenters claims, instead clearly support those associations between work-related biomechanical factors and MSDs identified in the NIOSH review where evidence is strong, namely combinations of forceful exertions, repetitive motions, awkward postures, vibration and heavy lifting. The above biomechanical exposures are the same ones that the OSHA standard seeks to reduce.

The NAS Steering Committee did point out some limitations to the epidemiological evidence, particularly that ''it was difficult to make strong causal inferences on the basis of evidence from any individual study.'' (Ex. 26–37, p. 15; emphasis added). They acknowledged that ''the occurrence of MSDs among populations exposed to low levels of biomechanical stressors was less definite. * * * In case of low levels of biomechanical stress, the possible contribution of other factors to MSDs is important to consider.'' (Ex. 26–37, p. 16). OSHA agrees with these statements and has not ignored the contribution of individual, organizational, and psychosocial factors in the etiology of MSDs. The Health Effects section of the rule emphasizes the multifactorial nature of MSDs. Substantial evidence in the rulemaking record, however, demonstrates that biomechanical risk factor show strong associations with elevated MSD risk when other non-work-related factors are controlled for. Thus, OSHA does not believe that the existence of other risk factors should prevent actions that reduce exposures to those work-related biomechanical stressors.

OSHA agrees that the majority of the NAS participants supported the ergonomic hypothesis that OSHA is espousing. This is not because the NAS selection process excluded those with other views, as implied by the commenters. The NAS prides itself on and is regarded world-wide as an organization that renders impartial and unbiased expert judgment on scientific issues. The reason for the NAS participants' support is simply that most ergonomic experts around the world agree there is clear evidence that biomechanical work factors increase the risk of MSDs.

OSHA is aware that one member of the six person panel addressing physical factors and epidemiology, Dr. Howard Sandler, was critical of NIOSH's methodology and findings. OSHA does not agree with Dr. Sandler's statements, and neither did the majority of the other panel members. In the NAS workshop summary, the consensus of the panel was that NIOSH had not overlooked any important body of epidemiological evidence. The panelists generally agreed that the NIOSH analysis resulted in the review on of high quality studies. With the exception of Dr. Sandler, the panelists unanimously agreed that a reassessment of the epidemiological literature would not alter the conclusions drawn by NIOSH regarding the work-relatedness of MSDs.

Finally, it is important to note that in evaluating all the evidence, not just the epidemiology, the NAS Steering Committee made the following conclusions:

Thus, while there are many points about which we would like to know more, there is little to shake our confidence in the thrust of our conclusions, which draw on converging results from many disciplines, using many methods:

- There is a higher incidence of reported pain, injury, loss of work, and disability among individuals who are employed in occupations where there is a high exposure to physical loading than for those employed in occupations with lower level of exposure.
- There is a strong biological plausibility between the incidence of MSDs and the causative exposure factors in high exposure occupational settings.
- Research clearly demonstrates that specific interventions can reduce the reported rate of MSDs for workers who perform high risk tasks. No single intervention is universally effective. Successful interventions require attention to individual, organizational, and job characteristics, tailoring the corrective actions to those characteristics (Ex. 26–37)

OSHA believes the above NAS conclusions support, not undermine, the premise that there is convincing evidence that exposure to work-related physical factors increases the risk of MSDs. There is a higher incidence of MSDs in exposed individuals; there is strong biological plausibility that relates these disorders to biomechanical risk factors; and interventions that reduce exposure to those factors have been demonstrated to reduce the incidence of the MSDs.

In summary, the methodology used by NIOSH to arrive at its findings that there is evidence of an association between a number of work-related physical risk factors and MSDs of the neck, upper extremity, and back is not a flawed ''black box,'' but a scientifically sound approach based on well-accepted epidemiological principles. By NIOSH's own testimony, OSHA's conclusions regarding biomechanical factors and the risk of MSDs in the workplace reinforce and do not misrepresent the 1997 NIOSH findings. Finally, the conclusions in the 1999 NAS report are supportive of both the NIOSH analysis and the OSHA position. In addition, to the NIOSH and NAS, the European Agency for Safety and Health at Work (Ex. 500–71–28) and Washington State (Ex. 500–71–93) have evaluated the scientific evidence and also reached similar conclusions regarding the evidence linking work-related biomechanical factors with the development of MSDs.

2. Issues Relating to Causal Inference in Epidemiology

Several commenters to the Proposal argued that OSHA had failed to show causality between exposure to workplace factors and MSDs; one group of comments emphasized that the types of studies used by NIOSH and OSHA to evaluate causality of the various MSD risk factors were inadequate for that purpose because of the studies design (see, *e.g.,* Ex. 32–241–4, pg 86–91). Specific comments were:

Only repeated longitudinal prospective studies can establish causation; OSHA relies instead on methodologies prone to error and bias. * * * Cross-sectional studies, upon which OSHA heavily relies, are incapable of providing evidence of cause and effect, because correlation does not establish causation (Id. pg. 86). Case-control studies are highly prone to bias. Prospective cohort studies are the best method of studying etiology, * * * retrospective studies [are prone to] the hazards of * * * ''recall bias.'' (Id. pg. 87). * * * In the case of musculoskeletal pain, which OSHA [has] linked to ''awkward postures'' and other biomechanical exposures, recall bias [in any retrospective design] can be extreme. * * * Cross-sectional studies are necessarily retrospective and prone to recall bias. (Id. pg. 87). [Cross-sectional studies] are useful for observing patterns and correlations, but can only generate hypotheses. A review seeking evidence of causation must exclude all cross-

sectional studies, because their methodology is inadequate to test a hypothesis. (Id. pg. 88)

With respect to case-control study designs, the comments continued:

[C]ase-control studies generally measure exposure to various hypothesized risk factors retrospectively, and consequently are prone to a number of biases, particularly in the recall of exposure to suspected risk factors. * * * Case-control studies are most suitable for examining rare diseases * * * Musculoskeletal complaints are hardly ''rare,'' of course, making OSHA's reliance on retrospective studies particularly unwarranted and puzzling (Id. pg. 89).

With respect to combining studies for a total weight-of-evidence assessment, critics were somewhat divided. Some noted that:

[In order to do a proper assessment] only prospective cohort studies reliably establish etiology, that is, valid scientific evidence of cause and effect. (Id. pg. 89). * * * Adequate science, however, requires more than mere association. It demands clinically accepted, rigorously controlled studies. (Ex. 32–241–3–1, pg.3),

while others, including Dr. Stanley Bigos, felt that case-control studies could also be used:

To infer causal relationships, one would look for consistent findings in a number of case-control and prospective cohort studies, as well as other supporting scientific information. Bradford Hill published an influential set of guidelines for causal inference. (Ex. 32–241–3–4, pg. 9).

However, another commenter cautioned about drawing conclusions for a group of studies:

It should be noted that weaknesses of individual studies cannot be overcome by synthesizing a large number of studies with different weaknesses that suggest the same conclusion. (Ex. 32–241–4, pg. 89).

Still another commenter, Dr. Lloyd Fisher, noted a methodology using a statistical approach for combining studies. This methodology is termed meta-analysis:

The process for properly formally synthesizing information from multiple studies of the same thing is described in a textbook I coauthored. Requirements for a valid meta-analysis include that (1) *all* studies in the area be considered, without ''publication bias'' based on treatment effect indicated in the studies; (2) a careful assessment of study quality should be performed; and (3) study results should reflect a homogeneity of results. This was not attempted where possible in the material that I reviewed.

Perhaps the most notable example of meta-analysis discussed by OSHA is [the NIOSH report]. However, it is not clear that the NIOSH report satisfies any of the three conditions. Some relevant studies (such as the Boeing back-injury study) are not included. The quality of the studies is not directly assessed to any great degree. (Ex. 32–241–3–7, pg. 3).

OSHA has carefully considered these comments on the criteria and methodology for selecting and combining studies for a weight-of-evidence approach to evaluating causality and has concluded that OSHA's approach and the approach used in the NIOSH report (Ex. 26–1) are scientifically sound. First, with respect to the NIOSH methodology, OSHA notes that NIOSH did prioritize studies by type of design and did discuss each design's inherent capabilities, weaknesses, and potential biases (Ex. 26–1, App. A). NIOSH also included in its criteria for evaluating the weight of a study the study's population, health outcome, and exposure: ''the greatest qualitative weight was given to studies that had objective exposure assessments, high participation rates, physical examinations, and blinded assessment of health and exposure status.'' (Ex. 26–1, pg. 1–9 and 1–10).

NIOSH then evaluated the data base of studies using guidelines to assess causal inference made famous by Bradford Hill (Ex. 26–726). These consisted of (1) strength of association; (2) consistency of association; (3) specificity of association; (4) temporality; (5) exposure-response relationship; and (6) coherence of evidence (a combination of consistency with other information and biological plausibility). These guidelines are endorsed in the Reference Manual On Scientific Evidence (Federal Judicial Center, 2000) that assists federal judges in interpreting scientific reasoning as it pertains to litigation and is held up by Gibson, Dunn & Crutcher as an authoritative source. The *Manual* states the following about the application of the Hill criteria:

There is no formula or algorithm that can be used to assess whether a causal inference is appropriate based on these guidelines. One or more factors may be absent even when a true causal relationship exists. Similarly, the existence of some factors does not ensure that a causal relationship exists. Drawing causal inferences after finding an association and considering these factors requires judgment and searching analysis, based on biology, of why a factor or factors may be absent despite a causal relationship and vice versa. While the drawing of causal inferences is informed by scientific expertise, it is not a determination that is made using scientific methodology. (pg. 375)

NIOSH witness Dr. Larry Fine stated in his testimony:

Again, it's always hard to talk in generalizations, but in a situation where you have evidence of a biologically plausible explanation for the relationship between exposure and disease, where you had a body of cross-sectional studies that had accurate exposure assessment and accurate health outcomes; in that setting, we believe that you may well infer causality, particularly if you see, in studies with a wide range of exposure, a dose-response relationship (Tr. 2095).

Second, OSHA has considered the NAS review of the NIOSH criteria for study inclusion and weighting (Ex. 26–37). In the NAS review seven epidemiologists specializing in ergonomics were asked about the NIOSH assessment's selection and weighting of studies. Each provided individual comments (*Id.,* pgs. 152–174). In general they concurred with the NIOSH approach. Dr. Frederick Gerr, Associate Professor, Rollins School of Public Health, Emory University, thought that NIOSH had included all important epidemiological evidence in its review (*Id.,* pg. 159), an opinion shared by Dr. Laura Punnett, Professor, University of Massachusetts, Lowell (*Id.,* pg. 162), Dr. Alfred Franzblau, Associate Professor of Occupational Medicine, University of Michigan School of Public Health (*Id.,* pg. 155), and Dr. David Wegman, Professor, University of Massachusetts Lowell (*Id.,* pg. 172). With respect to the four criteria NIOSH chose to use to further qualitatively weight each study, some of the NAS participants found that these ''criteria for identifying studies of relatively greater methodological rigor are reasonable and appropriate'' (*Id.,* pg. 159), and ''that the studies most heavily relied on by NIOSH in its assessment of workplace factors and MSDs are of good quality.'' (*Id.,* pg. 156); and ''[t]he quality of the studies that were most heavily weighted was generally quite high because they met the multiple criteria set out by NIOSH for weighting. (*Id.,* pg. 172). One panelist, however, Dr. Howard Sandler (in a study co-authored with non-panelist Dr. Richard Blume), thought that this weighting method was neither fully explained nor tested and validated. (*Id.,* pg.168). Dr. Sandler was scheduled to appear at the OSHA hearing as an expert for Keller/Heckman but never did so.

Because of the NIOSH assessment's use of cross-sectional studies, the comments of Dr. Alfred Franzblau in discussing NIOSH's weighting of cross-sectional studies should be noted:

What some researchers have done is to perform cross-sectional studies among workers (and jobs) that are known to have been stable for some minimum period of time (*e.g.,* six months or one year). This type of cross-sectional design overcomes some of the shortcomings of cross-sectional studies relative to prospective studies, and serves to greatly strengthen the confidence one can

have in the conclusion. Many of the studies that were most heavily weighted in the NIOSH assessment fall into this category (Ex. 26–37, pg. 156).

Dr. David Wegman provided the following summary comments:

There is no ''correct'' way to carry out a literature review particularly with as large a scope as the one undertaken by NIOSH. The authors of the NIOSH report are to be commended for developing a methodology that is reasonable, understandable, clearly presented, open and conservative. It is hard to imagine a more effective way to summarize this literature (Ex. 26–37, pg. 173).

Third, several witnesses and commenters on OSHA's ergonomics proposal also addressed the use of multiple types of epidemiological studies to determine causality. Dr. John Frank, Professor of Public Health Sciences, University of Toronto, stated in his testimony:

The best design cannot be read from a cookbook which automatically requires there to be a rank ordering of study design qualities for all circumstances. Prospective studies can actually make some mistakes that are overcome in well designed case-control studies (Tr. 1472).

Dr. Laura Punnett, Professor, University of Massachusetts Lowell, in support of the conclusions of the NIOSH report pointed out that:

Almost all of the studies considered in the review have been published in the peer-reviewed scientific literature, meaning that they had already been through the standard scientific quality control process prior to their publication and review by NIOSH. (Tr. 864).

In a statement that contradicts the view of several witnesses stating that medicine must rely on randomized clinical trials (RCT) for determining causality (*e.g.,* see Ex. 32–241–3–4, pg. 7–10), Dr. Niklas Krause, of the Public Health Institute, discussed the necessity of doing a careful evaluation of all the evidence:

So there are design problems in any study. And there is no gold standard, not even the randomized control trial is the gold standard as some people say. Epidemiologists say it. It is not the gold standard. You have to use all the available evidence. It is a careful evaluation of all the methodological features from measurement to control group to the timing and going through criteria that are important for causation as laid down by Hill and others. There is a discussion among us, you know, [about] which are the most important ones. But I think we all agree * * * we have established temporality in another way than doing a longitudinal study. And it can be established. We have repeated that. Then, all study designs are equally important. (Tr. 1476). * * * If you disregard all the cross-sectional studies for causal inference, you would not have medicine. (Tr. 1411).

When questioned about the cross-sectional design's inability to establish temporality, a key factor for determining causality, Dr. Krause further stated that in his studies this was not the case:

To give you an example, in our cross-sectional studies of the bus drivers, we measured the years of occupational driving. These years clearly occurred before they said to us I have back pain now. I have no doubt that these risk factors are [temporal], in a [temporal] relationship or coming before the back pain. And so this study qualifies for causal inference as a cross sectional study. I would not disregard this. (Tr. 1411).

The AFL–CIO post-hearing comments provide their analysis of the OSHA record with respect to the evidence for causality (Ex. 500–218). In discussing the types of studies that can be used to determine causality, they stated:

The record evidence clearly establishes that cross-sectional and case-control studies have been and can be used to identify causal relationships between exposures to risk factors and adverse health outcomes. In fact, the record demonstrates that cross-sectional and case-control studies have been used with great success to infer causal relationships addressing some of our nation's most important public health issues, such as smoking and lung disease, which have led to life-saving intervention measures in the absence of prospective studies. The record also does contain prospective epidemiological studies which have confirmed findings from cross-sectional and case-control studies that exposure to biomechanical/physical factors in the workplace cause MSDs among exposed workers.

(*Id.,* pg. 30)

In summary, with respect to the selection, use, and weighting of studies of multiple designs to make a determination of the causality between work-related stress factors and MSDs, OSHA concludes that the NIOSH approach is sound.

With respect to Dr. Fisher's comment that a formal methodology for combining study results to derive a weighted estimate of effect is a meta-analysis and that NIOSH did not perform a proper meta-analysis, OSHA agrees that NIOSH's analysis was not that of a formal meta-analysis. However, neither Dr. Fisher nor anyone else has provided a formal meta-analysis of the epidemiological literature to the record. Furthermore, OSHA notes that a necessary criteria for combining studies in a successful meta-analysis is that only studies measuring similar factors and estimating very similar effects should be analyzed together. OSHA's review of the database has determined that comparisons both between and within occupations with higher versus lower risk factors can be made in the various studies in a basic weight-of-evidence approach. However, a rigorous meta-analytic approach for a combined risk estimate is much more problematic because of the many factors being studied and the different response measures.

In addressing NIOSH's reliance on a qualitative evaluation of the epidemiology rather than a formal meta-analysis, Dr. David Wegman, Professor, University of Massachusetts Lowell, stated in his review for the NAS:

Meta-analysis is not appropriate when the question under study is as broad as the one NIOSH addressed. In my judgement [another writer] * * * provides the answer which, in his words is: ''I question whether quantitative methods can ever be as thoroughgoing, probing and informative as qualitative methods'' [Ex. 26–37].

The NAS Panel's Steering Committee concluded, with respect to the findings of the seven epidemiology experts on the NAS panel about combining studies for an overall risk estimate:

Methods used for the assessment of exposures and health outcomes vary [among studies], rendering the task of merging and combining evidence more challenging than in some other areas of risk assessment. But this variability does provide the benefit of multiple perspective on a common set of problems [Ex. 26–37].

In summary, OSHA finds no support for Dr. Fisher's comment that NIOSH erred by not performing a proper meta-analysis. Neither Dr. Fisher nor anyone else has provided any specific evidence to support his contention that a meta-analysis approach would be appropriate in this case. Instead, OSHA concurs with the National Academy of Science's conclusion that a formal meta-analysis would not be the best methodology in this case.

Gibson, Dunn & Crutcher also claimed that OSHA did not properly evaluate the epidemiological evidence according to the Reference Manual On Scientific Evidence (Ex. 500–197). Gibson, Dunn & Crutcher cited the following alleged weakness: that OSHA characterized the epidemiological evidence as proving cause while the *Manual* makes clear that epidemiological studies address association not causation, and that OSHA relied on studies of ''employee's recollection of the details of past job duties * * * and measures such as job titles coupled with the assumption that job duties were consistent across all job titles.'' (*Id.,* pg. I–55). The *Manual* criticizes studies that rely on the memory of subjects and states a preference for measurement of exposure. The *Manual* says that the

outcome or health effect being studied must be clearly defined, yet OSHA relied on ''studies that examine subjective memories regarding an individual's experience with or personal tolerance for pain.'' (*Id.,* pg. I–56). While NIOSH found that many studies ''did not take into account [confounding] factors beyond job duties and produced odds or risk ratios that were not statistically significant'' (*Id.,* pg. I–57), OSHA ''just picked the ones that purport to show results favoring its hypothesis'' and ''routinely relied on studies reporting associations or odds ratios well below 9–10 and indeed often below 2.'' (*Id.,* pg. I–59). According to Gibson, Dunn & Crutcher, the Manual ''indicates that where risk ratios are significantly below nine or ten there is a probability that unmeasured factors are the true causes of the effect or disease being studied.'' (*Id.,* pg. I–58).

Gibson, Dunn & Crutcher mischaracterized the nature of the epidemiological studies on which OSHA relied, the criteria used by OSHA to evaluate those studies, and the conclusions OSHA drew from those studies. They also misconstrue a key section of the *Manual.* OSHA did not simply rely on epidemiological studies in which exposures were assumed but never measured and in which the health outcome was simply self-reported memories of pain. For each MSD, OSHA relied primarily on a subset of studies in which exposure to work-related biomechanical factors was directly observed or measured and for which the health outcome was clearly defined by a combination of symptoms and physical exam. This meets the *Manual's* preference for objective and uniform exposure measures and case definition. It is also compatible with the 1997 NIOSH analysis, which quite properly give the greatest weight to studies that involved objective exposure assessments and physical examinations in their evaluation of the evidence (Ex. 26–1, pg. 1–10).

For example, in the case of epicondylitis and other elbow MSDs, thirteen epidemiological studies based case definition on physical examination and worker exposure determined by observational analysis (see Table V–3). In these studies, the diagnosis of epicondylitis was consistent and required both pain on palpation of the epicondylar area and pain at the elbow with resisted movement of the wrist. Exposures relied on videotaped analysis of job tasks to group exposed and unexposed workers, sometimes with quantitative estimates of cycle times (for repetition), static loading on the forearm (for force), and wrist posture. Nine of the thirteen studies found statistically significant associations between epicondylitis and exposure to work-related physical factors (see, *e.g.,* Exs. 26–907; 500–41–131; 26–53; 26–1117; 26–1364; 26–1433; 500–41–116; 26–945; 26–1473). Six of the studies reported odds ratios or other risk measures of five or greater (Exs. 26–907; 500–41–111; 26–43; 26–1117; 26–1364; 26–1433). One study found that the rate of repetitive exertions is highly predictive (p=0.002) of epicondylitis (Ex. 500–41–116). Two studies reported odds ratios greater than ten (Exs. 26–907; 500–41–111). This is a much different pattern of risk ratios than that presented by Gibson, Dunn & Crutcher, which claims that odds ratios are well below 9–10 and often around 2.

The *Manual* does not state risk ratios below 10 may indicate that confounding factors are responsible for the association, as implied by Gibson, Dunn & Crutcher. The *Manual* states ''a relative risk of 10 * * * is so high that it is extremely difficult to imagine any bias or confounding factor that might account for it.'' (pg. 376). The *Manual* goes on to say that ''although lower relative risks *can* (emphasis added) reflect causality, the epidemiologist will scrutinize such associations more closely because there is a greater chance that they are the result of uncontrolled confounding or bias.'' (Pg 377).

The *Manual* also discusses the Hill criteria previously cited. OSHA has evaluated the epidemiological evidence against these criteria. As mentioned above, the large number of studies reporting significant associations and risk ratios above five speaks to the strength of the association and the replicatibility of the findings for MSDs of the elbow. As further explained in the Health Effects section, there was one prospective cohort study of meat cutters that provided evidence of a temporal relationship between repetitive, forceful exertions of the forearm/elbow and epicondylitis (Ex. 26–53). In addition, several cross-sectional studies indicated an exposure-response relationship between the intensity or duration of repetitive exertions and the prevalence of MSDs (Exs. 500–41–116; 500–41–111; 26–1117; 26–697; 26–1473). Two studies reported ORs between 1 and 3 that were not statistically significant, probably because the workers were exposed to relatively low force directed at the forearm (Exs. 26–56; 26–697). Another study that did not find an association may have misclassified exposure, according to NIOSH (Ex. 26–1211). As a group, OSHA found that the studies relied on generally controlled for important confounders and bias, although not every individual study did so. Pathology information that epicondylitis is caused by microrupture of the tendons resulting from overuse of the forearm muscles, and the well-established connection between epicondylitis and racquet sports (*i.e.,* tennis elbow) establish the biological plausibility of the relationship.

The evidence briefly described above led OSHA to conclude that workers that perform job tasks requiring repeated forceful movements, especially flexion, pronation, or supination with the arm extended, are at increased risk of substantial and serious musculoskeletal impairment to the elbow. In its analysis of the epidemiological literature, NIOSH also concluded there was strong evidence for a relationship between exposure to a combination of work-related physical factors and epicondylitis (Ex. 26–1, pg 4–1 to 4–48). It should be noted that these OSHA and NIOSH conclusions do not, in fact, speak of causation as purported by Gibson, Dunn & Crutcher; both OSHA's and NIOSH's conclusions are careful to conform to the language of the *Manual.*

In Section V on health effects, OSHA evaluates the epidemiological evidence for MSDs of the upper extremity, shoulder, neck, back, and lower extremity, be focusing primarily on the most reliable studies. This usually means studies where exposures to physical work factors are directly observed or measured, not assumed based on job title, and the MSDs have been confirmed by a combination of symptoms, physical exam, and medical tests as appropriate. In addition to the evidence for epicondylitis cited above:

- Thirteen studies examined neck and neck/shoulder MSDs using physical exam and direct observation of exposure. All but one found significant associations between biomechanical risk factors and health outcome. At least three studies reported odds ratios greater than five (see Table V–1).
- Seventeen studies examined shoulder MSDs (mostly tendinitis) using physical exam and direct observation of exposure. All but one found significant associations between biomechanical risk factors and health outcome. At least six studies reported odds ratios greater than five (see Table V–2).
- Seven studies examined hand/wrist tendinitis using physical exam and direct observation of exposure. All but one found significant associations between biomechanical risk factors and health outcome. At least four studies reported odds ratios greater than five (Table V–4).
- Seventeen studies examined carpal tunnel syndrome using physical exam

and/or nerve conduction and direct observation of exposure. Thirteen found significant associations between biomechanical risk factors and health outcome. At least five studies reported odds ratios greater than five.

• Six studies examined hand/arm vibration syndrome using physical exam and vibration measurements. Four found significant associations between vibration and health outcome; all of which reported odds ratios greater than five.

OSHA has carefully evaluated the collective data base of studies for each MSD category using the criteria for causality cited in the *Manual* (pg. 374–378). OSHA used the epidemiological data, biomechanical research studies, and information addressing biological plausibility to draw its overall conclusions with regard to the evidence that the work-related biomechanical factors were responsible for the observed increase in the risk of health impairment. OSHA finds this evidence compelling and points to the need to take action to provide workers with necessary protection. OSHA does not believe that it is appropriate to wait for ''proof of causation'' since scientific evidence cannot ever establish causation beyond any doubt. As Sir Bradford Hill wrote over 35 years ago:

> All scientific work is incomplete—whether it is observational or experimental. All scientific work is liable to be upset or modified by advancing knowledge. That does not confer upon us a freedom to ignore the knowledge we already have or to postpone the action that it appears to demand at a give time (Ex. 26–726).

3. Evidence for Exposure Response Relationships

Several submissions, such as those submitted by the U.S. Chamber of Commerce and experts testifying on behalf of United Parcel Service (Exs. 30–1722, 32–241–3–19, 32–241–3–13, 30–4184, 30–1552), claimed that there is no epidemiologic evidence of exposure-response (or ''dose-response'') relationships between MSDs and the physical ergonomic stressors addressed by the OSHA standard. In their joint written testimony on the proposed rule, Kellie Truppa and Dr. Michael Vender, for example, stated:

> While it may seem very intuitive that decreasing reported ergonomic stressors would decrease disorders, there is no scientific study that has demonstrated a decrease in the incidence of true disease directly attributable to actual ergonomic changes. Unlike other risk factors to health (*e.g.*—smoking) there is no concept of threshold exposure or dose-response in relating ergonomic risk exposure to the development of disease. Therefore, there can be no predictability or guarantee of any benefit with reduction of ergonomic exposures * * * (Ex. 32–241–3–19).

In the preamble to the proposed rule, OSHA presented results of several studies that evaluated exposure-response trends; since publication of the proposal, OSHA has identified many more studies that provide evidence that, as the level (intensity, frequency or duration) of exposure increases, so does the risk of MSDs. OSHA summarizes this evidence in this section of the preamble. Based on these studies, OSHA finds that there is substantial evidence for a positive relationship between duration and intensity of exposure to biomechanical risk factors and the risk of developing MSDs, and that this evidence strengthens the causal relationship between exposure and risk.

One of the key criteria for demonstrating a causal relationship is evidence that the prevalence or incidence of a health outcome increases with an increase in the level of exposure to a hazardous condition. In occupational epidemiological studies, an exposure-response relationship is demonstrated when there is a statistical association between the prevalence or incidence of the health outcome in at least three groups of workers each with a varying degree of exposure (*e.g.,* no exposure, low exposure, high exposure). When exposure response relationships are based on groups of workers, the exposure variable is represented as an ordinal variable. Alternatively, statistical analysis can be performed on data for individual members of study cohorts to derive statistical functions that reflect the exposure-response relationship; in this case, the exposure variable is represented as a continuous variable. For this section, studies were included if the risk between musculoskeletal disorders and exposure to one or more biomechanical risk factors were examined using either of these two approaches. In the studies compiled here, the most common presentations of exposure response relationships are when the prevalence, incidence, odds ratio, or risk ratio for an MSD increases from one exposure category to the next. Typically these are accompanied by confidence intervals or a test of linear trend, as measures of statistical stability. In other studies, the exposure-response relationship may be expressed in the form of a statistically significant linear regression coefficient, or (partial) correlation coefficient, showing that, as exposure increases so does the prevalence or risk.

An exposure-response relationship, when present, is considered to strengthen the evidence of a causal relationship because it is believed to be a characteristic of cause-effect situations, in general, absent evidence to the contrary. In addition, it is thought that it would be more difficult for many or most forms of bias or confounding to produce an artifactual exposure-response relationship than to bias a simple association such as an odds ratio. However, it is not a sine qua non, in that an epidemiologic study can provide valuable information even if both exposure and outcome are represented only as dichotomous variables (*i.e.,* exposed versus unexposed), nor does it make unnecessary consideration of methodologic issues that must be addressed when evaluating a given study. Furthermore, the lack of an exposure-response relationship is not necessarily evidence against a causal effect.

The studies cited in this section utilized a wide range of exposure measures, including worker self-reports, observation, and direct measurement. As several authors have noted, even though exposure units and scaling vary, there is an overall consistency between self-reports and other, presumably more objective, measures in these studies (*e.g.,* Booth-Jones et al., 1998: Ex. 500–121–9; Jensen et al., 2000: Ex. 500–41–68; Neumann et al., 1999: Ex. 38–85; Pope et al., 1998: Ex. 500–71–67). This suggests that worker perception provides a useful guide to the identification of jobs involving high exposures to physical risk factors, and that, in general, the jobs that will be identified as potentially hazardous by workers' own evaluations will generally correspond to those that would be identified as potentially hazardous by other measures. The results of studies that have examined exposure-response relationships are summarized in Tables V–9 through V–13, and are summarized briefly below.

Work Pace and Repetition

There is substantial evidence of an exposure-response relationship for MSDs of the neck and shoulders. For example, in a case-control study of the general population in Sweden, the odds of neck/shoulder disorders increased markedly with work pace levels from slow to medium to rushed, as well as with hours per day of performing repetitive precision movements at work (Ekberg et al., 1994: Ex. 26–1238). Ohlsson *et al.*found positive associations with both the number of items handled per hour in repetitive assembly work and the number of years employed in such work, especially among younger employees (Ohlsson et

al., 1989: Ex. 26–1290). Johansson *et al.* studied blue- and white-collar manufacturing employees separately and reported exposure-response relationships with monotonous movements at work in each group (Johansson et al., 1994: Ex. 26–1331).

TABLE V–9.—EVIDENCE OF EXPOSURE-RESPONSE RELATIONSHIPS FOR MUSCULOSKELETAL DISORDERS WITH EXPOSURE TO REPETITIVE MANUAL WORK

Measure of repetitiveness (unit)	Health outcome/body region affected	Measure of effect	Reference
Neck and Shoulder			
Years sewing machine operator (4 categories).	Neck/Shoulder	Odds Ratio [unadj] 0 (control: 1.0 0–7: 2.3 (0.5–11.0) 8–15: 6.8 (1.6–28.5) >15: 16.7 (4.1–67.5)	Andersen *et al.*(1993: Ex. 26–1451).
Years sewing machine operator (4 categories).	Chronic neck pain	Odds Ratio [adj] 0 (control): 1.0 0–7: 1.9 (1.3–2.9) 8–15: 3.8 (2.3–6.4) >15: 5.0 (2.9–8.7)	Andersen *et al.*(1993: Ex. 26–1502).
Years sewing machine operator (4 categories).	Chronic should pain	Odds Ratio [adj] 0 (control): 1.0 0–7: 1.4 (0.9–2.4) 8–15: 3.9 (2.3–6.5) >15: 10.3 (5.9–17.9)	Andersen *et al.*(1993: Ex. 26–1502).
Years sewing machine operator (4 categories).	Chronic neck and/or shoulder pain.	Odds Ratio [adj] 0 (control): 1.0 0–7: 1.8 (1.2–2.6) 8–15: 4.3 (2.6–6.9) >15: 8.0 (4.7–13.8)	Andersen *et al.*(1993: Ex. 26–1502).
Data entry at video display unit (hours/week).	Neck (cervical diagnoses)	Odds Ratio [adj] 5–20 hr/wk: 1.2 (0.4–4.3) ≥20 hr/wk: 1.7 (0.7–4.3)	Bergqvist *et al.*(1995: Ex. 26–1195, 500–165–25).
Data entry at video display unit	Neck/shoulder	Odds Ratio [adj] Data entry: 1.4 (0.7–2.9) Data entry plus limited rest breaks: 4.8 (1.3–18.1)	Bergqvist *et al.*(1995: Ex. 26–1195, 500–165–25).
Typing speed	Neck	Prevalence [unadj] (test of trend): Slow: 10% Moderate: 14% Fast: 25% (p<0.001)	Burt *et al.*(1990: Ex. 26–698).
Percentage of time typing	Neck	Odds Ratio [adj] <20: 1.0 20–39: 2.0 (1.0–7.7) 40–59: 2.6 (1.4–5.0) 60–79: 2.2 (1.0–4.7) 80–100: 2.8 (1.4–5.4)	Burt *et al.*(1990: Ex. 26–698).
Typing speed	Shoulder	Odds Ratio [adj] Slow: 1.0 Moderate: 2.6(1.1–5.9) Fast: 4.1 (1.8–9.4))	Burt *et al.*(1990: Ex. 26–698).
Percentage of time typing	Shoulder	Prevalence [unadj] (test of trend): 0–19: 6% 20–39: 10% 40–59: 13% 60–79: 11% 80–100: 15% (p=.10)	Burt *et al.*(1990: Ex. 26–698).
Repetitive precision movements (hours/day) (3 categories).	Neck/Shoulder	Odds Ratio [adj] Low: 1.0 Medium: 3.8 (0.7–20) High: 15.6 (2.2–113)	Ekberg *et al.*(1994: Ex. 26–1238).
Work pace (3 categories)	Neck/Shoulder	Odds Ratio [adj] Low: 1.0 Medium: 7.6 (1.6–36) Rushed: 10.7 (2.2–52)	Ekberg *et al.*(1994: Ex. 26–1238)
Hour per day of video display terminal (VDT) use.	Neck, shoulder, upper back (''upper torso'').	Odds Ratio [unadj] per hour 1.4 (1.0–2.0)	Faucett *et al.*(1994: Ex. 38–256)
Monotonous working movements (duration of repetitive movements, static stress and sitting).	Neck (in white collar workers)	Partial correlation coefficient [adj] 0.38 ($p < 0.05$)	Johansson *et al.*(1994: Ex. 26–1331)
Monotonous working movements (duration of repetitive movements, static stress and sitting).	Shoulder (in white collar workers)	Partial correlation coefficient [adj] 0.32 ($p < 0.05$)	Johansson *et al.*(1994: Ex. 26–1331)

TABLE V–9.—EVIDENCE OF EXPOSURE-RESPONSE RELATIONSHIPS FOR MUSCULOSKELETAL DISORDERS WITH EXPOSURE TO REPETITIVE MANUAL WORK—Continued

Measure of repetitiveness (unit)	Health outcome/body region affected	Measure of effect	Reference
Monotonous working movements (duration of precision movements, repetitive movements, and static and stress).	Shoulder (in blue collar workers)	Partial correlation coefficient [adj] 0.15 (p < 0.05)	Johansson *et al.*(1994: Ex. 26–1331)
Years employed in repetitive assembly work.	Neck	Increasing odds (graphical presentation only)	Ohlsson *et al.*(1989: Ex. 25–1290)
	Shoulder	Increasing odds (p=0.03); below 35 years of age, p=0.01	
Work pace (items/hour) (4 categories).	Shoulder	Odds Ratio [adj] < 100: 1.0 100–199: est 8.0 (p=0.0006) 200–700: est 9.0 (p=0.0006) > 700: est 2.0 (p-value not given)	Ohlsson et al. (1989: Ex. 26–1290)
Hours per day of VDT use (4 categories).	Neck	Prevalence [unadj] (test of trend): 0 hr: 7% 0.5–3 hr: 7% 4–6 hr: 12% ≥7 hr: 19% (p<0.00001) Odds Ratio [adj] 0 hr: 1.0 0.5–3 hr: 1.8 (0.5–6.8) 4–6 hr: 4.0 (1.1–14.8) ≥7 hr: 4.6 (1.7–13.2)	Rossignol *et al.*(1987: Ex. 26–804)
Hours per day of VDT use (4 categories).	Shoulder	Prevalence [unadj] (test of trend): 0 hr: 6% 0.5–3 hr: 5% 4–6 hr: 10% ≥7 hr: 16% (p=< 0.00001) Odds Ratio [adj] 0 hr: 1.0 0.5–3 hr: 2.5 (0.7–10.8) 4–6 hr: 4.0 (1.0–16.9) ≥7 hr: 4.8 (1.6–17.2)	Rossignol *et al.*(1987: Ex. 26–804)
Sewing machine operation (years of employment).	Neck	Odds Ratio [unadj] < 8 yrs: 1.0 8–14 yrs: 1.1 (0.4–2.6) ≥15 yrs: 2.1 (0.8–5.6	Schibye *et al.*(1995: Ex. 26–1463)
	Shoulder	< 8 yrs: 1.0 8–14 yrs: 1.3 (0.5–3.4) ≥	15 yrs: 4.3 (1.5–12.5)
Arm and Elbow			
Data entry at video display unit (hours/week).	Arm/hand	Odds Ratio [unadj] 5–20 hr/wk: 1.6 (0.6–4.5) ≥ 20 hr/wk: 1.8 (0.8–3.9)	Bergqvist *et al.*(1995: Exs. 26–1195, 500–165–25)
Percentage of time typing	Elbow/forearm	Odds Ratio [adj] 20–39%: 1.2 (0.6–22.5) 40–59%: 1.7 (0.8–3.5) 60–79%: 1.9 (0.9–4.3) 80–100%: 2.8 (1.4–5.7)	Burt *et al.*(1990: Ex. 26–698)
Typing speed	Elbow/forearm	Prevalence [unadj] (test of trend): Slow: 7% Moderate: 11% Fast: 13% (p=0.02)	Burt *et al.*(1990: Ex. 26–698)
Hours per day of VDT use	Arm	Prevelance [unadj] (test of trend): 0 hr: 4% 0.5–3 hr: 2% 4–6 hr: 4% ≥7 hr: 7% (p=0.01)	Rossignol *et al.*(1987: Ex. 26–804)

TABLE V–9.—EVIDENCE OF EXPOSURE-RESPONSE RELATIONSHIPS FOR MUSCULOSKELETAL DISORDERS WITH EXPOSURE TO REPETITIVE MANUAL WORK—Continued

Measure of repetitiveness (unit)	Health outcome/body region affected	Measure of effect	Reference
Hand and Wrist			
Typing at video display unit (hours/day).	Hand/wrist	Odds Ratio [adj] 0–<2 hr: 1.0 2–<4 hr: 1.3 (0.6–1.8) 4–<6 hr: 1.3 (0.8–2.2) 6–≥8 hr: 2.1 (1.3–3.6) ≧8 hr: 3.3 (1.2–8.9)	Bernard *et al.*(1994: Ex. 500–165–21)
Typing speed	Hand/wrist	Odds Ratio [adj] Slow: 0.9 (0.3–2.3) Moderate: 1.3 (0.6–3.1) Fast: 2.5 (1.0–5.6)	Burt *et al.*(1990: Ex. 26–698)
Percentage of time typing	Hand/wrist	Prevalence [unadj] (test of trend): 0–19: 13% 20–39: 23% 40–59: 27% 60–79: 30% 80–100: 24% (p<0.01)	Burt *et al.*(1990: Ex. 26–698)
Hours per day of video display terminal (VDT) use.	Hand and arm	Odds Ratio [unadj] per hour 1.5 (1.1–2.0)	Faucett *et al.*(1994: Ex. 38–256)
Repetition rating (1 unit on 0–10 scale).		Odds Ratio [adj]:	Latko *et al.*(1999: Ex. 38–171)
	Dominant wrist/hand/fingers	1.17 (1.06–1.29)	
	Tendinitis (distal upper extremity)	1.23 (1.04–1.46)	
	Carpal tunnel syndrome	1.16 (1.00–1.34)	
Cycle length (seconds), in work performed 4–8 hours per day.	Carpal tunnel syndrome	Odds Ratio [adj] ≥1 min: 1.0 30–59 s: 1.03 (0.56–1.89) 10–29 s: 1.33 (0.75–2.37) <10 s: 1.90 (1.04–3.48)	Leclerc *et al.*(1998: Ex. 500–205–11)
Years employed in repetitive assembly work.	Hand	Increasing odds (p=0.002)	Ohlsson *et al.*(1989:Ex. 26–1290)
Repetitive wrist motions (years of exposure).	Carpal tunnel syndrome	Odds Ratio [unadj] <1 yr: 1.0 1–20 yrs: 2.3 (0.7–7.9) >20 yrs: 9.6 (2.8–33.0)	Wieslander et al.(1989: Ex. 26–1027)
Multiple Body Regions			
Piece-rate wage system (years of employment).	Musculo- skeletal diseases	Odds Ratio [adj] 0–4 yrs: 1.0 5–9 yrs: 4.3 (0.5–35.9) 10–14 yrs: 10.0(1.0–79.3) 15–19 yrs. 8.0 (0.8–76.8) ≥20 yrs: 11.4 (0.9–137.1)	Brisson *et al.*(1989: Ex. 26–937)
Hours per week of video display terminal use.	Upper extremity and back	Mean hours per week [unadj] 30 in cases, 27 in non-cases (p<0.05)	Knave *et al.*(1985: Ex. 26–753)
Percentage of recovery time per work cycle.	Upper extremity	Linear regression coefficient [unadj]: Ln(% recovery): 0.6 (r^2=0.49, p<0.001)	Moore *et al.*(1994: Ex. 26–1033)
Hours per day at keyboard	Hand, wrist, forearm and/or elbow	Prevalence [unadj] (test of trend): 3 hr: 21% 4 hr: 24% 6 hr: 45% 6 hr: 50% >6 hr: 86%(p<0.00001)	Oxenburgh (1987: Ex. 26–1367)
Keyboarding speed	Upper extremity	Prevalence [unadj] (test of trend): <40 wpm: 17% 40–60 wp,: 22% >60 wpm: 29% (p=0.025)	Polanyi *et al.*(1997): Ex. 500–41–106)
Daily time keyboarding (hours per day).	Upper extremity	Means (test of difference) [unadj]: Cases 3.9 hours/day, controls 3.2 hours/day (p<0.001)	Polanyi *et al.*(1997: Ex. 500–41–106)

Note: adj = adjusted for other covariate(s)
unadj = not adjusted for other covariates

TABLE V–10.—EVIDENCE OF EXPOSURE-RESPONSE RELATIONSHIPS FOR MUSCULOSKELETAL DISORDERS WITH EXPOSURE TO FORCEFUL MANUAL EXERTION

Measure of manual force (unit)	Health outcome/body region affected	Measure of effect	Reference
Neck and Shoulder			
Grocery checking: hours per week of checking work.	Shoulder	Odds Ration [unadj] <20: 1 20–25: 1 >25: 3.6 (p<0.05)	Baron *et al.*(1991: Ex. 26–697)
Forearm rotation while exerting very high forces (Frequency of exposure * Years of exposure).	Shoulder	Odds Ration [adj] per unit:	Hughes *et al.*(1997: Ex. 26–907)
	Interview	92 (7.3–±)	
	Examination	46 (3.8–550)	
Light materials handling [factor formed from frequency and duration of materials handling 0.5–<1 kg and 1–5 kg].	Shoulder (in white collar workers)	Partial correlation coefficient [adj] 0.18 (p < 0.05)	Johansson *et al.*(1994: Ex. 26–1331)
Years of carpentry work (<10, 10 to <20, 20+ years).	Shoulder	Odds Ration [adj] 10–<20 yr: 2.3 (1.0–5.4) 20+ yr: 3.2 (1.1–8.9)	Lemasters *et al.*(1998: Ex. 500–121–44)
Load lifted (cumulative exposure, in 3 categories: 0–709; 710–25,999; and >25,999 kg).	Shoulder: acromio-clavicular osteoarthritis.	Odds Ratio [adj] (per category) Right side: 1.55 (1.03–2.34) Left side: 2.55 (1.50–4.35)	Stenlund *et al.*(1992: Ex. 26–733)
Load lifted (cumulative exposure, in 3 categories: 0–709; 710–25,999; and >25,999 kg).	Shoulder tendinitis	Odds Ratio [adj] (per category) Right side: 1.02 (0.59–1.76) Left side: 1.81 (0.95–3.44)	Stenlund *et al.*(1993: Ex. 502–462
Arm and Elbow			
Grocery checking: hours per week of checking work.	Elbow	Elbows Odds Ratio [unadj] <20: 1 20–25: 1.4 >25: 2.8 (p<0.05)	Baron *et al.*(1991: Ex. 26–697)
Forearm rotation while exerting very high forces (Frequency of exposure * Years of exposure).	Elbow/forearm: Interview Examination	Odds Ratio [adj] per unit: 4 (0.2–4) 37.0 (3.0–470)	Huges *et al.*(1997: Ex. 26–907)
Strenuous exertions (years of high exposure).	Epicondylitis	Odds Ratio [adj] 0 yr: 1.0 1–14 yr: 1.8(0.6–5.9) 15–38 yr: 3.3 (0.9–12.5)	Ritz (1995: Ex. 26–1473)
Hand and Wrist			
Hand forces (finger flexor muscles on electromyography).	Carpal tunnel syndrome	Average force (test of difference in means): Cases: 4.3 ″ 3.5 kp Noncases: 3.8 ″ 3.2 kp (p<0.05)	Armstrong *et al.*(1979: Ex. 500–41–8)
Grocery checking (years of exposure).	Hand/wrist	Odds Ratio [adj] 0–5: 1 5–10: 2 10+: 6 (p<0.05)	Baron *et al.*(1991: Ex. 26–697)
Grocery checking (years of exposure).	Carpal tunnel syndrome	Odds Ratio [adj] 0–5: 1 5–10: 4 10+: 15 (p<0.05)	Baron *et al.*(1991: Ex. 26–697)
Grocery checking (hours per week of exposure).	Carpal tunnel syndrome	Odds Ratio [adj] <20: 1 20–25: 2.3 >25: 4.8 (p<0.05)	Baron *et al.*(1991: Ex. 26–697)
Forearm rotation while exerting very high forces (Frequency of exposure * Years of exposure).	Hand/wrist: Interview Examination	Odds Ratio [adj] per unit 17.0 (2.9–106) 9.3 (1.0–90)	Hughes *et al.*(1997: Ex. 26–907)
Years of carpentry work (<10, 10 to <20, 20+ years).	Hand and wrist	Odds Ratio [adj] 10 – lt;20 yr: 2.4(1.1–5.3) 20+yr: 3.1(1.1–8.4)	Lemasters *et al.*(1998: Ex. 500–121–44)
Biomechanical index from direct measurements of force and posture.	Carpal tunnel syndrome	Linear regression [unadj] Flexion 0.017(r=0.62) Extension: 0.035(r=0.26)	Loslever *et al.*(1993: Ex. 26–161)

TABLE V–10.—EVIDENCE OF EXPOSURE-RESPONSE RELATIONSHIPS FOR MUSCULOSKELETAL DISORDERS WITH EXPOSURE TO FORCEFUL MANUAL EXERTION—Continued

Measure of manual force (unit)	Health outcome/body region affected	Measure of effect	Reference
Mean relative finger flexor force (by EMG)/45–90 minute work sampling period.	Wrist	Linear regression coefficient [adj]: Mean relative deviation angle (p<0.05) Mean relative EMG signal (p<0.05) Seniority (years employed) (p<0.05)	Malchaire *et al.*(1996: Ex. 26–1473)
Manual force (as % MVC, in 5 categories).	Upper extremity	Linear regression [unadj]: Ln (Force: 2.0 (r^2=0.49, p<0.001)	Moore *et al.*(1994: Ex. 26–1033)
Forceful wrist motions (3 categories: low, medium, high).	Carpal tunnel syndrome By history.	Prevalence [unadj] (test of trend) Low: 0% Medium: 10% High: 63% (p=0.00006)	Osorio *et al.*(1994: Ex. 26–807)
	By nerve conduction velocity	Low: 0% Medium: 7% High: 33% (p=0.02)	
Forceful wrist motions (years exposed).	Carpal tunnel syndrome	Linear regression [adj], p<0.05 for: Right median nerve conduction velocity	Osorio *et al.*(1994: Ex. 26–807)
Grip >6 lb. per hand (3 categories of frequency).	Hand/wrist	Prevalence [unadj] (test of trend) None: 41% Some: 40% Frequent: 65% (p=0.30)	Stetson *et al.*(1993: Ex. 26–1221)
High load on wrist (years of exposure).	Carpal tunnel syndrome	Odds Ratio [unadj] <1 yr: 1.0 1–20 yr: 2.1 (0.8–5.2) >20 yr: 6.6 (1.4–14.7)	Wieslander *et al.*(1989: Ex. 26–1027)
Back			
Frequency of lifting per shift	Low back	Prevalence [unadj] 0/shift: 29% 1–5/shift: 33% 6–10/shift: 49% 11–20/shift: 55% >20/shift: 54%	Arad *et al.*(1986: Ex. 500–41–7)
Frequency of lifting >11.3 kg (times per day).	Prolapsed lumbar disc	Odds Ratio [adj] (test of trend): 0: 1.0 <5: 1.6 (0.4–6.1) 5–25: 2.7 (0.8–9.2) >25: 4.9 (0.5–47.6) (p=0.02)	Kelsey *et al.*(1984: Ex. 500–41–73)
Frequency of lifting >11.3 kg (times per day).	Prolapsed lumbar disc	Odds Ratio [adj] (test of trend): 0: 1.0 <5: 1.2 (0.7–2.0) 5–25: 1.3 (0.7–2.5) >25: 3.5 (1.5–8.5) (p=0.01)	Kelsey *et al.*(1984: Ex. 500–41–73)
Frequency of carrying 11.3 kg (times per day).	Prolapsed lumbar disc	Odds Ratio [adj] (test of trend): 0: 1.0 <5: 1.0 (0.6–1.9) 5–25: 2.1 (1.0–4.3) >25: 2.7 (1.2–5.8) (p=0.004)	Kelsey *et al.*(1984: Ex. 500–41–73)
Lifting 11.3 kg while twisting	Prolapsed lumbar disc	Odds Ratio [adj] (test of trend): Never or rare: 1.0 Moderate: 2.5 (0.9–6.8) Often: 3.1 (1.3–7.5) (p=0.002)	Kelsey *et al.*(1984: Ex. 500–41–73)
Load on spine (12 continuous biomechanical variables: peak and daily integraetd load).	Low back	Odds Ratio [adj] for inter-quartile spreads: Peak lumbar shear (N): 1.7 (1.0–2.9) Cumulative lumbar disc compression (N s/shift): 2.0 (1.2–3.6) Peak hand force (N): 1.9 (1.2–3.1)	Kerr *et al.*(2000: Ex. 500–41–74)
Index of stone load (weight*hours/day).	Low back	Odds Ratio [adj]: None: 1.0 Intermediate: 1.8 (0.3–9.3) High: 4.0 (0.8–19.8)	Latza *et al.*(2000: Ex. 500–19–6)

TABLE V–10.—EVIDENCE OF EXPOSURE-RESPONSE RELATIONSHIPS FOR MUSCULOSKELETAL DISORDERS WITH EXPOSURE TO FORCEFUL MANUAL EXERTION—Continued

Measure of manual force (unit)	Health outcome/body region affected	Measure of effect	Reference
Lifting demands index (''Job Severity Index'').	Back	Injury incidence rate, disabling injury incidence, and severity rate increased with JSI (graphical presentations)	Liles *et al.*(1984: Exs. 26–33, 500–41–88)
Dynamic trunk motions (31 continuous biomechanical.	Low back	Odds Ratio [adj] for combined weighted means of 5 variables: 10.7 (4.9–23.6)	Marras *et al.*(1993: Ex. 500–41–94)
Load on spine (12 continuous biomechanical variables: peak and daily integrated load).	Low back	Higher load in cases vs controls, by each variable (all p-values <0.04). Odds ratios [adj] computed both for full observed ranges of exposure and more conservatively for inter-quartile spreads: Peak shear (N) 1.5 (1.0–2.4) Peak trunk velocity (deg/sec) 1.6 (1.1–2.5) Integrated moment (MN m s) 1.4 (1.0–2.0) Usual hand force (N) 1.7 (1.2–2.6)	Norman *et al.*(1998: Ex. 38–84)
Transfer a patient on canvas and poles (frequency/average working shift).	Low back	Odds Ratio [adj] 0: 1.0 1–4: 1.0 (0.8–1.3) ≥5: 1.3 (0.8–2.1)	Smedley *et al.*(1995: Ex. 500–41–40)
Manually transfer patient between bed and chair (frequency/shift).	Low back	Odds Ratio [adj] 0: 1.0 1–4: 1.4 (1.1–1.9) 5–9: 1.8 (1.3–2.5) ≥10: 1.5 (1.1–2.1)	Smedley *et al.*(1995: Ex. 500–41–40)
Manually move patient around on bed (frequency/shift).	Low back	Odds Ratio [adj] 0: 1.0 1–4: 1.2 (0.8–1.7) 5–9: 1.6 (1.1–2.3) ≥10: 1.7 (1.2–2.4)	Smedley *et al.*(1995: Ex. 500–41–40)
Manually transfer patient between bed and chair (frequency/shift).	Low back	Odds Ratio [adj] 0: 1.0 1–4: 1.3 (0.9–1.7) 5–9: 1.6 (1.1–2.3) ≥10: 1.6 (1.1–2.3)	Smedley *et al.*(1997: Ex. 500–205–25)
Transfer patient between bed and chair with hoist (frequency/shift).	Low back	Odds Ratio [adj] 0: 1.0 1–4: 1.5 (1.0–2.0) ≥5: 1.6 (0.8–3.0)	Smedley *et al.*(1997: Ex. 500–205–25)
Manually move patient around on bed (frequency/shift).	Low back	Odds Ratio [adj] 0: 1.0 1–4: 1.3 (0.8–1.9) 5–9: 1.5 (1.0–2.3) ≥10: 1.7 (1.1–2.5)	Smedley *et al.*(1997: Ex. 500–205–25)
Lift patient in or out of bath with hoist (frequency/shift).	Low back	Odds Ratio [adj] 0: 1.0 1–4: 1.4 (1.0–1.9) ≥5: 2.1 (1.2–3.6)	Smedley *et al.*(1997: Ex. 500–205–25)
Frequent vs. infrequent lifting in patient care.	Back	Length of time at work without back injury longer for those with infrequent lifting demands (p<0.01 in survival analysis)	Stobbe *et al.*(1988: Ex. 500–41–45)
Lifting frequency (4 categories of hospital service area, from 1, lifting most, to IV, lifting least).	Back	Odds Ratio [adj] Area IV: 1.0 Area III: 1.26 (p>0.05) Area II: 1.73 (p>0.05) Area I: 4.26 (p<0.01)	Venning *et al.*(1987: Ex. 500–41–49)

TABLE V–10.—EVIDENCE OF EXPOSURE-RESPONSE RELATIONSHIPS FOR MUSCULOSKELETAL DISORDERS WITH EXPOSURE TO FORCEFUL MANUAL EXERTION—Continued

Measure of manual force (unit)	Health outcome/body region affected	Measure of effect	Reference
NIOSH Lifting Equation Lifting Index (LI) (4 categories).	Low back (severity rating, range 0–5).	Mean severity (standard deviation): LI:<1: 0.18 (0.15) 1≤LI ≤3: 3.57 (0.86) LI>3: 4.07 (0.73) RWL*=0: 3.86 (0.75) ANOVA (α=0.05) *Recommended Weight Limit	Wang *et al.*1998 (1998: Ex. 500–41–52)
NIOSH Lifting Equation Lifting Index (LI).	Low back	Odds Ratio [unadj] 0: 1.0 0<LI ≤1: 1.1 (0.2–5.3) 1<LI ≤2: 1.5 (0.6–3.8) 2<LI ≤3: 2.5 (1.3–4.9) LI ≥3: 1.6 (0.7–4.0)	Waters *et al.*(1999: Ex. 500–121–76)
Strenuous physical activity at work (hours per day).	Back	Odds Ratio [unadj] 0–<2 hr: 1.0 2–<4 hr: 4.2 4–<6 hr: 6.4 6–<8 hr: 5.6 ≥8 hr: 6.8 Odds Ratio [adj] per hour of strenuous work: 1.14 (1.11–1.17)	Wild (Ex. 26–1104; 26–1107)
Physically hard work	Low back	Odds Ratio [unadj] (test of trend): No or seldom: 1.0 ¼ of the time: 1.3 ½ of the time 2.3 ¾ of the time: 2.2 All of the time: 2.5 (p<0.001)	Xu *et al.*(1997: Ex. 500–71–53)
Lower Extremity or Multiple Body Regions			
Strength demand of job (3 categories: none, some, much).	Knee (radiographic osteoarthritis)	Odds Ratio [adj] Men, ages 55–64: 1.9 (0.9–4.0) Women, ages 55–64: 3.1 (1.0–9.4)	Anderson *et al.*(1988: Ex. 26–926)
Kneeling, squatting or stair-climbing, with and without heavy lifting.	Knee osteoarthritis	Odds Ratio [adj] Neither kneeling nor lifting: 1.0 Kneeling/squatting: 2.5 (1.1–5.5) Kneeling and lifting: 5.4 (1.4–21.0)	Cooper *et al.*(1994: Ex. 500–41–27)
Maximum compressive force (lb.) on L5/S1 lumbar disc.	''Overexertion incidents'' by clinic visit.	Incidence rate (per 200,000 hours): <1000 lb: 65 1000–1500 lb: 150 >1500 lb: 208	Herrin *et al.*(1986: Ex. 26–961)
Index of physically strenuous load	Overall MSD morbidity: Symptoms Findings	Linear regression coefficient [adj]: 0.127 (p=0.002) 0.091 (p=0.026)	Leino *et al.*(1995: Ex. 32–241–3–54)
Years of carpentry work (<10, 10 to <20, 20+ years).	Knee	Odds Ratio [adj] 10–<20 yr: 1.9 (0.9–4.1) ≥20 yr: 3.5 (1.3–9.2)	Lemasters *et al.*(1998: Ex. 500–121–44)
Lifting at work (kilograms per day)	Knee Men: Women:	Odds Ratio [adj] Medium: 2.5 (1.5–4.4) High: 3.0 (1.6–5.5) Medium: 1.2 (0.7–1.9) High: 1.7 (1.0–2.9)	Sandmark *et al.*(2000: Ex. 500–41–114)

TABLE V–11.—EVIDENCE OF EXPOSURE-RESPONSE RELATIONSHIPS FOR MUSCULOSKELETAL DISORDERS OF THE NECK AND SHOULDERS WITH EXPOSURE TO NON-NEUTRAL POSTURE

Measure of posture	Health outcome/body region affected	Measure of effect	Reference
Neck and Shoulder			
Height of video display unit keyboard relative to elbow height (centimeters).	Neck/shoulder	Linear regression coefficient [unadj] 0.18 (−0.03, 0.40)	Bergqvist *et al.*(1995: Ex. 500–165–24)
Duration of shoulder flexion or abduction >60 degrees (hours/day).	Shoulder/neck	Ratio for cases vs. controls: Right: 2.0 (p <0.005) Left: 2.4 (p <0.025)	Bjelle *et al.*(1981: Ex. 26–1519)
Frequency of shoulder flexion or abduction >60 degrees (times/day).	Shoulder/neck	Ratio for cases vs. controls: Right: 2.0 (p <0.001) Left: 2.2 (p <0.005)	Bjelle *et al.*(1981: Ex. 26–1519)
Arms lifted (hours per day, 3 categories).	Neck/shoulder	Odds Ratio [adj] Low: 1.0 Medium: 2.4 (0.8–7.1) High: 4.8 (1.3–18)	Ekberg *et al.*(1994: Ex. 26–1238)
Elbow flexed >1 time/minute (per hour/day).	Shoulder	Odds Ratio [adj] 1.10 (0.98–1.23)	English *et al.*(1995: Ex. 26–848)
Head rotation	Neck, shoulder, upper back (''upper torso'').	R-squared [adj] Pain: 0.11 (p<0.01) Stiffness: 0.18 (p<0.01)	Faucett *et al.*(1994: Ex. 38–256)
Keyboard height relative to elbow	Neck, shoulder, upper back (''upper torso'').	R-squared [adj] Pain: 0.05 (p<0.05) Stiffness: 0.06 (p<0.05)	Faucett *et al.*(1994: Ex. 38–256)
Years of exposure to repetitive shoulder flexion (angle ≥30 degrees, 600 times/hour) with high forces.	Shoulder impingement syndrome	Increasing prevalence ratio [adj] with cumulative exposure nonlinear trend, p=0.002 for quadratic term	Frost *et al.*(1999: Ex. 38–97)
Hands above shoulder level (hours per day).	Neck/shoulder pain with impairment.	Prevalence Ratio [adj] <1 Hr. 1.1 (0.8–1.5) 1–4 hr. 1.5 (1.2–1.9) >4 hr. 2.0 (1.4–2.7)	Holmstrom et al (1992: Ex. 500–41–64)
Stooping (hours per day)	Neck/shoulder pain with impairment.	Prevalence Ratio [adj] <1 Hr. 1.0 (0.8–1.3) 1–4 hr. 1.4 (1.1–1.8) >4 hr. 1.5 (1.1–2.1)	Holmstrom et al (1992: Ex. 500–41–64)
Bent work postures [factor=duration of precision movements and head bent foward; frequency and duration of trunk forward flexion (20°–60°)].	Neck (in white collar workers)	Partial correlation coefficient [adj] 0.20 (p<0.05)	Johansson *et al.*(1994: Ex. 26–1331)
Twisted work postures [factor=duration of trunk rotation (>45°) and head rotation (>45°)].	Neck (in white collar workers)	Partial correlation coefficient [adj] 0.23 (p<0.05)	Johansson *et al.*(1994: Ex. 26–1331)
Extreme work postures [factor=frequency and duration of trunk forward flexion (>60°); frequency of trunk forward flexion (20°–60°); and duration of head rotation (>45°), trunk rotation (>45°), and work with hands above shoulders].	Shoulder (in blue collar workers)	Partial correlation coefficient [adj] 0.14 (p<0.05)	Johansson *et al.*(1994: Ex. 26–1331)
Twisted work postures [factor=duration of trunk rotation (>45°) and head rotation (>45°)].	Shoulder (in white collar workers)	Partial correlation coefficient [adj] 0.16 (p<0.05)	Johansson *et al.*(1994: Ex. 26–1331)
Percentage of work cycle with shoulder elevated.	Cervicobrachial (neck to hand)	Odds Ratio [adj] 1.04 (p<0.05)	Jonsson *et al.*(1988: Ex. 26–969)
Neck flexion (percentage of work cycle).	Neck	Regression coefficient p-value [adj] p<0.01	Kilbom *et al.*(1986: Ex. 500–41–75)
Shoulder elevated (percentage of work cycle).		Regression coefficient p-value [adj]	Kilbom *et al.*(1986: Ex. 500–41–75)
	Neck	p<0.05	
	Shoulder	p<0.05	
Neck flexion (movements per hour)	Neck/shoulder	Ratio of median for cases vs. controls [unadj] Total movements: 1.3 (p=0.008) Flexions ≥30°: 1.3 (p=0.02)	Ohlsson *et al.*(1995: Ex. 26–868)

TABLE V–11.—EVIDENCE OF EXPOSURE-RESPONSE RELATIONSHIPS FOR MUSCULOSKELETAL DISORDERS OF THE NECK AND SHOULDERS WITH EXPOSURE TO NON-NEUTRAL POSTURE—Continued

Measure of posture	Health outcome/body region affected	Measure of effect	Reference
Frequency of shoulder flexion or abduction.	Neck/shoulder	Median elevation >30° (% of time) [unadj]: Cases=16, controls=9 (p=0.05) Median elevation >30° (movements per hour) [unadj]: Cases=60, controls=9 (p=0.004) Median abduction ≥60° (% of time) [unadj]: Cases=1, controls=0 (p=0.04) Median elevation ≥60° (movements per hour) [unadj]: Cases=47, controls=0 (p=0.04)	Ohlsson *et al.*(1995: Ex. 26–868)
Shoulder flexion or abduction >90 degrees (duration, as percentage of work cycle).	Left shoulder Right shoulder Either shoulder	Odds Ratio [unadj] (test of trend) >0%–<10%: 2.5 ≥10%: 5.1 (p=0.0001) >0%–<10%: 1.7 ≥10%: 2.8 (p=0.002) Ratio of mean duration in cases vs. controls [unadj]: 2.6 (p=0.003) Odds Ratio (95% CI) per 10% increment [adj]: 1.4 (1.1–1.8)	Punnett *et al.*(2000: Ex. 500–41–109
Twisted or bent postures (4 categories).	Neck/shoulder	Odds Ratio [adj] Little: 1.0 Moderate: 1.2 (1.0–1.5) Rather much: 1.6 (1.4–1.9) Very much: 1.8 (1.5–2.2)	Tola *et al.*(1988: Ex 26–1018)
Twisting of trunk (hours/day) (4 categories).	Neck	Odds Ratio [adj]: Not at all: 1.0 Little: 1.3 (0.7–2.4) Moderately: 1.9 (1.1–3.5) Much: 2.3 (1.2–4.3)	Viikari-Juntura *et al.*(2000: Ex. 500–41–50)
Working with hand above shoulder level (hours/day) (3 categories).	Neck	Odds Ratio [adj]: <0.5 1.0 0.5–1: 1.2 (1.0–1.3) >1: 1.4 (1.3–1.6)	Viikari-Juntura *et al.*(2000: Ex. 500–41–50)
Twisting or bending of trunk at work (3 categories).	Neck	Odds Ratio [unadj]: Very or rather little: 1.0 Moderate: 1.7 (0.9–9–3.2) Rather or very much: 1.9 (1.2–3.2)	Viikari-Juntura *et al.*(1994: Ex. 26–873)
Hand and Wrist			
Wrist bending or twisting (per 2 hours/day).	Carpal tunnel syndrome	Odds Ratio [unadj] 1.5 (1.2–1.7)	Blanc at al. (1996); Ex. 26–42 500–41–16)
Wrist flexion (hours/week) (hours truncated at 40).	Carpal tunnel syndrome	Odds Ratio [unadj] 0: 1.0 1–7: 1.5 (1.3–1.9) 8–19: 3.0 (1.8–4.9) 20–40: 8.7 (3.1–24.1)	De Krom *et al.*(1990: Ex. 26–102)
Wrist extension (hours/week) (hours truncated at 40).	Carpal tunnel syndrome	Odds Ratio [unadj] 0: 1.0 1–7: 1.4 (1.0–1.9) 8–19: 2.3 (1.0–5.2) 20–40: 5.4 (1.1–27.4)	De Krom *et al.*(1990: Ex. 26–102)
Shoulder rotation with arm elevated, >1 time/minute (per hour/day).		Odds Ratio [adj]	English *et al.*(1995: Ex. 26–848)
	Wrist/forearm Carpal tunnel syndrome	1.6 (1.2–2.3) 1.8 (1.2–2.8)	
Shoulder rotation with elbow flexed, >1 time/minute (per hour/day).	Finger	Odds Ratio [adj] 5.1 (2.0–12.8)	English *et al.*(1995: Ex. 26–848)
Wrist flexion or extension (per 20 repetitions/min).	Thumb	Odds Ratio [adj] 1.4 (1.1–1.8)	English *et al.*(1995: Ex. 26–848)
Ulnar abduction (degrees of ''typical'' work posture).	Forearm	Increasing percentage of operators w/medical findings vs. angle of ulnar abudction (graphical presentation only)	Hunting *et al.*(1981: Ex. 26–1276)

TABLE V–11.—EVIDENCE OF EXPOSURE-RESPONSE RELATIONSHIPS FOR MUSCULOSKELETAL DISORDERS OF THE NECK AND SHOULDERS WITH EXPOSURE TO NON-NEUTRAL POSTURE—Continued

Measure of posture	Health outcome/body region affected	Measure of effect	Reference
Relative angle of wrist ulnar or radial deviation/45–90 minute work sampling period.	Wrist	Linear regression coefficient [adj]: Mean relative deviation angle (p<0.05) Mean relative EMG signal (p<0.05) Seniority (years employed) (p<0.05)	Malchaire *et al.*(1996: Ex. 26–1473)
Wrist bending or twisting (mean hours/day) (5 categories).	Carpal tunnel syndrome	Odds Ratio [adj] 0: 1.0 0.25–1.75: 1.34(0.64–2.80) 2–3: 1.23(0.60–2.53) 3.5–6: 2.33 (1.24–4.36) 7–16: 2.47 (1.38–4.43) quadratic dose-response effect in alternative model, p=0.03	Nordstrom *et al.*(1997: Ex. 26–900)
Wrist deviation (3 categories of frequency).	Hand/wrist	Prevalence [unadj] (test of trend) None: 35% Some: 43% Frequent: 45% (p=0.43)	Stetson *et al.*(1993: Ex. 26–1221)
Back			
Postural load (index of frequency and/or duration of 4 postures, in 4 categories).	Low back pain	Odds Ratio [adj] (test for trend)	Bovenzi *et al.*(1994: Ex. 26–774)
	Lifetime	Mild: 1.0 Moderate: 1.3(0.8–2.4) Hard: 1.7(1.0–3.0) Very hard: 3.6(2.0–6.5) (p=0.001)	
	12 month pervalence:	Moderate: 1.8 (1.1–3.2) Hard: 2.2(1.3–3.8) Very hard: 4.6 (2.6–8.0) (p=0.0001)	..
Hands above should level (hours per day).	Low back (severe pain with impairment).	Prevalence Ratio [adj]: <1 hr: 1.1 0.8–1.5) 1–4: 1.5 (1.2–2.0) >4 hr: 1.6 (1.0–2.6)	Holmstrom *et al.*(1992: Ex. 500–41–65) ..
Stopping (hours per day)	Low back (severe pain with impairment).	Prevalence Ratio [adj]: <1 hr: 1.3 (0.9–1.8) 1–4 hr: 1.9 (1.4–2.6) >4 hr: 2.6 (1.7–3.8)	Holmstrom *et al.*(1992: Ex. 500–41–65) ..
Kneeling (hours per day)	Low back (severe pain with impairment).	Prevalence Ratio [adj]: <1 hr: 2.4 (1.7–3.3) 1–4 hr: 2.6 (1.9–3.5) >4 hr: 3.5 (2.4–4.9)	Holmstrom *et al.*(1992: Ex. 500–41–65)
Extreme work postures [factor formed from frequency and duration of trunk forward flexionn (>60°); frequency of trunk forward flexion (20°–60°); and duration of head rotation (>45°), trunk rotation (>45°), and work with hands above shoulders].	Low back (in blue collar workers)	Partial correlation coefficient [adj] 0.16 (p<0.05)	Johansson *et al.*(1994: Ex. 26–1331)
Monotonuous working movements [factor formed from duration of repetitive movements, static stress, and sitting].	Low back (in white collar workers	Partial correlation coefficient [adj] 0.22 (p<0.05)	Johansson *et al.*(1994: Ex. 26–1331)
Driving (hours/week)	Low back	Odds Ratio [adj] for prevalence: <10: 1.0 10–14: 1.5 (1.0–2.4) 15–19: 1.2 (0.8–1.9) 20–24: 2.0 (1.3–3.1) ≥ 25 2.1 (1.3–3.4)	Pietri *et al.*(1992: Ex. 29–309)

TABLE V–11.—EVIDENCE OF EXPOSURE-RESPONSE RELATIONSHIPS FOR MUSCULOSKELETAL DISORDERS OF THE NECK AND SHOULDERS WITH EXPOSURE TO NON-NEUTRAL POSTURE—Continued

Measure of posture	Health outcome/body region affected	Measure of effect	Reference
Driving (hours/week)	Low back	Odds Ratio [adj] for 1 year cumulative incidence: <10: 1.0 10–14: 4.0 (1.1–14.3) 15–19: 4.8 (1.4.8–16.4) 20–24: 3.3 (0.9–12.0) ≥ 25 3.7 (0.9–14.0)	Pietri *et al.*(1992: Ex. 38–309)
Percentage of work cycle in trunk flexion (3 categories).	Low back	Odds Ratio [unadj] (test of trend) Mild flexion: 0%: 1.0 1–10%: 4.2 ≥10%: 6.1 (p=0.014) Severe flexion: 0%: 1.0 0–10%: 4.4 ≥10%: 8.9 (p=0.003)	Punnett *et al.*(1991: Ex. 26–1289)
Percentage of work cycle in non-neutral trunk posture (mild flexion, severe flexion, twist or lateral bend).	Back	Odds Ratio [adj] 8.09 (1.5–44.0)	Punnett *et al.*(1991: Ex. 26–1289)
Twisted or bent postures (4 categories).	Low back	Odds Ratio [adj] Rather or very little: 1.0 Moderate: 1.3 (1.0–1.7) Rather much: 1.5 (1.2–1.9) Very much: 1.5 (1.2–1.9)	Riihimaki *et al.* (1989: Ex. 26–58)
Forward bending (minutes per day)	Low back	Odds Ratio [adj]: Men 1–59 min: 1.6 (1.1–2.5) ≥60 min: 1.8 (1.1–3.1) Women 1–59 min: 1.1 (0.8–1.6) ≥60 min: 1.2 (0.7–1.8)	Vingard *et al.*(2000: Ex. 500–41–51)
Repeated bending, twisting, and reaching at work (hours per day.	Back	Odds Ratio [unadj] 0 hr: 1.0 >0–<2 hr: 5.8 2+–<4 hr: 8.4 4+–<6 hr: 10.4 6+ hr: 14.1 Odds Ratio [adj] per hour of repeated bending, twisting and reaching: 1.09 (1.06, 1.13)	Wild (Ex. 26–1106; 26–1107)
Frequent twisting or bending	Low back	Odds Ratio [unadj] (test of trend): No or seldom: 1.0 1/4 of the time: 1.8 1/2 of the time: 1.9 3/4 of the time: 2.0 All of the time: 2.0 (p<0.001)	Xu *et al.*(1997: Ex. 500–71–53)
Lower Extremity			
Knee-bending demand of job (3 categories: none, some, much).	Knee: radiographic osteoarthritis	Odds Ratio [adj] Men, ages 55–64: 2.5 (1.2–5.0) Women, ages 55–64: 3.5 (1.2–10.5)	Anderson *et al.*(1988: Ex. 26–926)
Kneeling and/or squatting (Floor- and carpetlayers 56%, carpenters 25%, compositors 0% of working time).	Knee	Odds Ratio [unadj] Compositors: 1.0 Carpenters: 3.9 (2.7–5.5) Floor- and carpetlayers: 6.4 (4.0–10.1)	Kirkeskov Jensen *et al.*[Jensen, 1977#1975]
Standing (hours per day)	Knee	Odds Ratio [adj]	Sandmark *et al.*(2000: Ex. 500–41–114)
	Men	Medium: 1.5 (0.9–2.4) High: 1.7 (1.0–2.9)	
	Women	Medium: 1.2 (0.7–1.9) High: 1.6 (1.0–2.8)	
Squatting or knee bending (number per day).	Knee	Odds Ratio [adj]	Sandmark *et al.*(2000: Ex. 500–41–114)
	Men	Medium: 1.3 (0.8–2.2) High: 2.9 (1.7–4.9)	

TABLE V–11.—EVIDENCE OF EXPOSURE-RESPONSE RELATIONSHIPS FOR MUSCULOSKELETAL DISORDERS OF THE NECK AND SHOULDERS WITH EXPOSURE TO NON-NEUTRAL POSTURE—Continued

Measure of posture	Health outcome/body region affected	Measure of effect	Reference
Kneeling (minutes per day)	Knee	Odds Ratio [adj]	Sandmark *et al.*(2000: Ex. 500–41–114)
	Men	Medium: 1.4 (0.9–2.2) High: 2.1 (1.4–3.3)	
Jumping (number per day)	Knee	Odds Radio [adj]	Snadmark *et al.*(2000: Ex. 500–41–114)
	Men	Medium: (0.9–2.4) High: 2.7)1.7–4.1)	
Jumping (number)	Hip	Odds Ratio [adj]	Vingard *et al.*(1977: Ex. 26–1617) Medium: 1.0 (0.5–2.0) High: 2.1 (1.1–4.2)
Stairs climbed (flights)	Hip	Odds Ratio [adj] Medium: 1.3 (0.8–2.0) High: 2.1 (1.2–3.6)	Vingard *et al.*(1997: Ex. 26–1616)

TABLE V–12.—EVIDENCE OF EXPOSURE-RESPONSE RELATIONSHIP FOR MUSCULOSKELETAL DISORDERS WITH EXPOSURE TO SEGMENTAL VIBRATION, BY BODY REGION AFFECTED.

Measure of vibration exposure (unit)	Health outcome/body region affected	Measure of effect	Reference
Vibration exposure (energy equivalent frequency-weighted acceleration) for 4 hours/day.	Upper extremity	Odds Ratio [adj] <7.5 m/sec^2 2.7 >7.5 m/sec^2 14.1 (p<0.005)	Bovenzi *et al.*(1991: Ex. 500–41–18)
Daily vibration exposure (energy equivalent frequency-weighted acceleration).	Upper extremity	Odds Ratio [adj] per unit 1.29 (p<0.5)	Bovenzi *et al.*(1991: Ex. 500–41–18)
Daily vibration exposure (energy equivalent frequency-weighted acceleration).	Upper extremity muscle-tendon syndrome.	Odds Ratio [adj] per unit 1.42 (p<0.5)	Bovenzi *et al.*(1991: Ex. 500–41–18)
Daily vibration exposure (energy equivalent frequency-weighted acceleration).	Carpal tunnel syndrome	Odds Ratio [adj] per unit 1.73 (p<0.5)	Bovenzi *et al.*(1991: Ex. 500–41–18)
Lifetime dose (5 categories of acceleration^2 years).	Hand-arm vibration syndrome	Odds Ratio [adj] per unit 0: 1.0 0–19: 4.1 (1.1–16.4) 19–20: 4.7 (1.3–16.1) 20–21: 9.4 (3.1–28.4) >21: 34.3 (11.9–99.0)	Bovenzi *et al.*(1991: Ex. 500–41–17)
Riveting (years)	Wrist	Odds Ratio [adj] per year 1.12 (p<0.05)	Burdorf *et al.*(1991: Ex. 500–41–21)
Riveting (years)	Hand-arm vibration syndrome	Odds Ratio [adj] per year 1.07 (p<0.05)	Burdorf *et al.*(1991: Ex. 500–41–21)
Power tool usage	Forearm-hand (right)	Median values for workstations with high vs. low symptom prevalence [unadj] Holding time: 12 sec. vs 6 secs. (p<0.05) Total duration: 21 sec. vs 15 secs. (p<0.05)	Fransson Hall *et al.*(1996: Ex. 500–41–56)
Years of exposure to vibration (chain saw use).	Vibration-induced white finger	Positive association with duration of exposure Higher prevalence and earlier onset of symptoms with earlier first exposure (higher acceleration levels) (all data presented graphically)	Futatsuka *et al.*(1985: Ex. 26–1430)

TABLE V–12.—EVIDENCE OF EXPOSURE-RESPONSE RELATIONSHIP FOR MUSCULOSKELETAL DISORDERS WITH EXPOSURE TO SEGMENTAL VIBRATION, BY BODY REGION AFFECTED.—Continued

Measure of vibration exposure (unit)	Health outcome/body region affected	Measure of effect	Reference
Cumulative hours of exposure to vibration.	Median and ulnar motor and sensory nerve function.	Correlation coefficient [unadj] R median motor NCV: 0.274 (p=0.01) L median motor NCV: 0.123 (p>0.05) R ulnar motor NCV: 0.259 (p=0.05) L ulnar motor NCV: 0.389 (p>0.001) R median distal latency: 0.172 (p=0.05) L median distal latency: 0.214	Koskimies *et al.*[Koskimies, 1990 #1983]
Cumulative exposure to vibration (log hours).	Hand-arm vibration syndrome:	Odds Ratio [adj] per common log unit	Letz *et al.*(1992: Ex. 26–384)
	Vascular	2.9 (1.7–5.0)	
	Sensorineural	1.8 (1.2–2.9)	
Tool use (years)	Hand-arm vibration syndrome (Stockholm workshop scales):.	Odds ratio [adj] per year	McGeoch *et al.*(2000: Ex. 500–41–96)
	Neurological stage ≥ 1	1.09 (p<0.05)	
	Vascular stage ≥ 1	1.10 (p<0.05)	
Years of exposure to vibration	Hand-arm vibration syndrome	Odds ratio [adj] per year 1.11 (1.05–1.17)	Nilsson *et al.* (1989: Ex. 26–1148)
Years of exposure to vibration	Median nerve latency at carpal tunnel.	Odds ratio [adj] per year Right: 1.12 (1.02–1.23) Left: 1.09 (1.00–1.20)	Nilsson *et al.*(1994: Ex. 26–1190)
Cumulative vibration exposure (3 categories: 0–8999; 9000–255,199; and >255,199 energy-weighted hours).	Shoulder: osteoarthritis of the acromioclavicular joint.	Odds Ratio [adj] (per category) Right side: 1.3 (0.9–1.8) Left side: 1.8 (1.2–2.6)	Stenlund *et al.*(1992: Ex. 26–733)
Cumulative vibration exposure (3 categories: 0–8999; 9000–255,199; and >255,199 energy-weighted hours).	Shoulder tendinitis	Odds Ratio [adj] (per category) Right side: 1.7 (1.1–2.6) Left side: 1.8 (1.1–3.1)	Stenlund *et al.*(1993: Ex. 502–462)

TABLE V–13.—EVIDENCE OF EXPOSURE-RESPONSE RELATIONSHIP FOR MSDS WITH COMBINATION OF EXPOSURES (*e.g.,* REPETITION, FORCE AND POSTURE), BY TYPE OF EXPOSURE AND BODY REGION AFFECTED.

Exposure factors	Health outcome/body region affected	Measure of effect	Reference
Index of physical stress at work (sum of 6 items).	Neck	Odds Ratio [adj] Age 30–64 years: 1.26 (1.18–1.33) Age ≥ 65 years: 1.12 (1.00–1.26)	Makela *et al.*(1991: Ex. 26–980)
Index of mechanical workload (sum of 6 items).	Elbow: epicondylitis	Odds ratio [adj]: Model 2: 1.5 (1.0–2.3) Model 3: 1.7 (1.2–2.6)	Ono *et al.*(1998: Ex. 500–66–4)
Repetition; force (4 categories: LF = low force; LR = low repetition; HF = high force; HR = high repetition.	Hand/wrist: tendinitis	Prevalence Rate Ratio [unadj] LF LR: 1.0 HF LR: 4.8 (0.6–39.7) LF HR: 5.5 (0.7–46.3) HF HR: 17.0 (2.3–126.2)	Armstrong *et al.*(1987: Ex. 26–48)
Work at video display unit, with and without specific job features.	Arm/hand	Odds Ratio [adj] Data entry: 1.5 (0.7–3.4) Data entry plus keyboard too low: 2.8 (0.9–8.6) ≥ 20 hr/week: 0.5 (0.2–1.4) ≥ 20 hr/week plus limited rest breaks, no lower arm support: 4.6 (1.2–17.9)	Bergqvist *et al.*(1995: Ex. 26–1195 500–165–25)
Work at video display unit, with and without specific job features.	Arm/hand	Odds Ratio [adj] Limited rest breaks, plus no lower arm support, vs. one or neither: 10.1 (2.4–43.2)	Bergqvist *et al.*(1995: Ex. 500–165–24)

TABLE V–13.—EVIDENCE OF EXPOSURE-RESPONSE RELATIONSHIP FOR MSDS WITH COMBINATION OF EXPOSURES (*e.g.*, REPETITION, FORCE AND POSTURE), BY TYPE OF EXPOSURE AND BODY REGION AFFECTED.—Continued

Exposure factors	Health outcome/body region affected	Measure of effect	Reference
Force and repetition of hand activities (5 classes, from very light/low to very heavy/high).	Hand: Median nerve sensory conduction velocity.	Test of positive linear trend: p < 0.01	Nathan *et al.*(1988: Ex. 26–990)
Force and repetition of hand activities (5 classes, from very light/low to very heavy/high).	Hand: Median nerve sensory conduction velocity.	Linear regression coefficient [adj]: Class of hand activity: 0.011 (p < 0.05)	Nathan *et al.*(1992: Ex. 26–988)
Index of physical risk factors (sum of 3 items: force; 1 kg, cycle time < 30 sec, static hand work).	Hand: Radial tunnel syndrome	P <0.001, test for trend	Roquelaure *et al.*(1996: Ex. 500–41–111)
Index of physical risk factors (sum of 5 occupational items plus parity ≥ 3).	Hand: Carpal tunnel syndrome	Odds ratio [adj] ≤ 2 factors: 1.0 3 factors: 5.6 (1.6–24.5) 4 factors: 93.7 (13.4–93.8) ≥ 5 factors: 90.0 (8.0–366.5)	Roquelaure *et al.*(1997: Ex. 38–396)
Repetition; force (4 categories: LF = low force; LR = low repetition; HF = high force; HR = high repetition).	Hand/wrist	Odds Ratio [adj] LF LR: 1.0 HF LR: 5.2 LF HR: 3.3 HF HR: 29.1 (p < 0.05)	Silverstein *et al.*(1986: Ex. 26–1404)
Repetition; force (4 categories: LF = low force; LR = low repetition; HF = high force; HR = high repetition).	Hand: Carpal tunnel syndrome	Odds Ratio [adj] LF LR: 1.0 HF LR: 1.8 LF HR: 2.7 HF HR: 15.5 (p < 0.001)	Silverstein *et al.*(1987: Ex. 26–34)
Repetitiveness and forceful exertions of the upper limbs (Group I = neither, Group II = either, Group III = both).		Test of positive linear trend:	Chiang *et al.*(1993: Ex. 26–1117)
	Neck symptoms	p = 0.04	
	Shoulder symptoms	p = 0.000	
	Shoulder girdle diagnosis	p = 0.000	
	Elbow symptoms	p = 0.11	
	Epicondylitis	p = 0.14	
	Wrist symptoms	p = 0.03	
	Hand symptoms	p = 0.04	
	Carpal tunnel syndrome	p = 0.02	
Index of ergonomic stressors (sum of 9 items, range 0–25).	Upper extremity (neck, shoulder/upper arm, elbow/forearm, and/or hand/wrist).	Prevalence ratio [adj] 0–6: 1.0 7–12: 2.0 (1.2–3.4) 13–18: 2.6 (1.6–4.3) 19–25: 2.8 (1.6–4.8)	Punnett (1998: Ex. 26–38)
	Shoulder/upper arm	0–6: 1.0 7–12: 2.6 (1.1–6.2) 13–18: 3.6 (1.6–8.3) 19–25: 3.3 (1.3–8.3)	
	Wrist/hand	0–6: 1.0 7–12: 1.9 (1.0–3.8) 13–18: 2.4 (1.3–4.7) 19–25: 2.3 (1.1–4.7)	
Index of occupational physical stress (sum of 5 items, range 0–5).	Low back	Odds Ratio [adj]: 0: 1.0 1: 1.2 (0.9–1.6) 2: 1.7 (1.3–2.1) 3: 2.1 (1.6–2.7) 4: 3.2 (2.3–4.5) 5: 2.5 (1.4–4.7)	Heliovaara *et al.*(1991: Ex. 26–959) Lifting >11.3 kg while twisting Low back: Prolapsed lumbar disc Odds Ratio [adj]: Knees bent: 2.7 (0.9–7.9) Knees straight: 6.1 (1.3–27.9) Kelsey *et al.*(1984: Ex. 500–41–73)
Lifting > 11.3 kg while twisting	Low back: Prolapsed lumbar disc	Odds Ratio [adj] Knees bent: 2.7 (0.9–7.9) Knees straight: 6.1 (1.3–27.9)	Kelsey *et al.*(1984: Ex. 500–41–73)
Physical exposure index (sum of 3 items, range 0–3.	Low back	Odds Ratio [adj]: 0: 1.0 1: 1.41 (1.02–1.94) 2: 2.45 (1.63–3.68) 3: 3.18 (1.72–5.81)	Liira *et al.*(1996: Ex. 26–748)
Forward bending and manual materials handling (MMH) (highly exposed now, 5 and 10 years ago).	Low back	Odds Ratio [adj]:	Vingard *et al.*(2000: Ex. 500–41–51)

TABLE V–13.—EVIDENCE OF EXPOSURE-RESPONSE RELATIONSHIP FOR MSDS WITH COMBINATION OF EXPOSURES (*e.g.,* REPETITION, FORCE AND POSTURE), BY TYPE OF EXPOSURE AND BODY REGION AFFECTED.—Continued

Exposure factors	Health outcome/body region affected	Measure of effect	Reference
		Men Forward bending: 1.8 (1.0–3.3) MMH: 2.0 (1.0–4.3) Bending and MMH: 2.8 (1.1–7.5) Women Forward bending: 1.5 (0.8–2.6) MMH: 1.1 (0.6–2.1) Bending and MMH: 2.9 (1.2–6.8)	
Kneeling, squatting or stair-climbing, with and without heavy lifting.	Knee osteoarthritis	Odds Ratio [adj]: Neither kneeling nor lifting: 1.0 Kneeling/squatting: 2.5 (1.1–5.5) Kneeling and lifting: 5.4 (1.4–21.0)	Cooper *et al.*(1994: Ex. 500–41–27)
Kneeling, with (floor layers) and without (tile/terrazzo setters) use of knee kicker.	Knee: bursitis	Prevalence ratio [adj]: Floor layers: 3.2 (1.9–5.4) Tile setters: 1.8 (0.8–3.9)	Thun *et al.*(1987: Ex. 26–60)

In a cross-sectional study of newspaper workers, the risk of both neck and shoulder disorders increased with typing speed and with percentage of time working at the keyboard (Burt *et al.,* 1990: Ex. 26–698). Similarly, several investigators have shown exposure-response relationships for neck and shoulder disorders among video display unit operators with the number of hours per day (or week) of VDU work (Bergqvist *et al.,* 1995: Exs. 26–1195, 500–165–25; Faucett *et al.,* 1994: Ex. 38–256; Rossignol *et al.,* 1987: Ex. 26–804).

Two different studies of sewing machine operators in the garment industry have shown increasing prevalence of neck and shoulder disorders with cumulative years of exposure to repetitive work (Andersen *et al.,* 1993: Ex. 26–1451; Andersen *et al.,* 1993: Ex. 26–1502; Schibye *et al.,* 1995: Ex. 26–1463). (Note that Andersen 1993a (Andersen *et al.,* 1993: Ex. 26–1451) computed both crude and adjusted odds ratios, and the latter estimates were higher. However, in the adjusted model, each of the potential confounders had little association with the risk of neck/shoulder syndromes, so this model was deemed overly conservative and statistically inefficient, and the unadjusted ORs are shown in the table.) Andersen *et al.,* (Andersen *et al.,* 1993: Ex. 26–1502) also computed chi-square tests of trend with exposure for specific diagnoses. The following had a positive trend with years of exposure: cervicobrachial fibromyalgia ($p<<0.001$); rotator cuff syndrome ($p<0.01$); and cervical syndrome ($p<0.001$). The probability of having no MSD symptoms showed a negative trend with years of exposure ($p<0.001$). These findings are compatible with those of Brisson *et al.,* (Brisson *et al.,* 1989: Ex. 26–937), who examined long-term musculoskeletal disability in general, and specifically that due to arthritic and back disorders, including regular pain in the lower back, upper back/neck, shoulders, hands/wrists/elbows, or knees/ankles. The risk of long-term disability, both overall and for musculoskeletal disorders, increased with years of piece-rate garment work.

Elbow and forearm disorders are typically less prevalent, so there are fewer opportunities to evaluate exposure-response relationships with adequate statistical power. Nevertheless, several studies of VDU operators have shown such associations with speed or daily duration of VDU work (Bergqvist *et al.,* 1995: Ex. 26–1195, 500–165–25; Burt *et al.,* 1990: Ex. 26–698; Rossignol *et al.,* 1987: Ex. 26–804).

Intensity and duration of VDU work have shown similar exposure-response relationships with disorders of the hand and wrist region, including carpal tunnel syndrome (Bernard *et al.,* 1994: Ex. 500–165–21; Burt *et al.,* 1990: Ex. 26–698; Faucett *et al.,* 1994: Ex. 38–256), as well as with cases that include both proximal and distal regions of the upper extremity (Knave *et al.,* 1985: Ex. 26–753; Oxenburgh, 1987: Ex. 26–1367; Polanyi *et al.,* 1997: Ex. 500–41–106).

In the manufacturing sector, there is also evidence that the risk of hand and wrist disorders increases with work pace and repetitiveness (Latko *et al.,* 1999: Ex. 38–171; Leclerc *et al.,* 1998: Ex. 500–41–85) and with cumulative years of exposure to repetitive manual work (Ohlsson *et al.,* 1989: Ex. 26–1290; Wieslander *et al.,* 1989: Ex. 26–1027). Moore *et al.,* (Moore *et al.,* 1994: Ex. 26–1033) showed that the risk of reported upper extremity disorders decreased with the percentage of recovery time in each work cycle.

Force

Forceful manual exertions have been characterized by different investigators with a variety of metrics, some of them involving the combination of at least two of object weight, frequency of handling, and duration of exposure. These various approaches have yielded evidence of the risk of shoulder disorders increasing with exposure in white collar, construction, and manufacturing jobs (Hughes *et al.,* 1997: Ex. 26–907; Johansson *et al.,* 1994: Ex. 26–1331; Stenlund *et al.,* 1993: Ex. 502–462), and similar evidence for elbow disorders, even though limited by the smaller numbers of cases mentioned above (Hughes *et al.,* 1997: Ex. 26–907; Ritz, 1995: Ex. 26–1473).

Among grocery store workers, grocery checking has been identified as a job requiring forceful exertions. In two different studies, the risk of shoulder, elbow, and wrist/hand disorders, including CTS, was associated with the level of forcefulness required by each employee's job, the number of hours of checking work per week, and the cumulative number of years of checking (Baron *et al.,* 1991: Ex. 26–697; Osorio *et al.,* 1994: Ex. 26–807). Note that Osorio *et al.* defined three categories of exposure, but there were no CTS cases in the low exposure group, so in multivariate modeling only the odds ratio for low/medium vs. high exposure could be calculated. These dichotomous

estimates, adjusted for age, gender, medical history and alcohol consumption, ranged from 6 to 40.

In other studies of hand and wrist disorders, exposure-response relationships have been found for finger flexor forces, measured by electromyography, as well as for simpler estimates of force based on object weight and on self-report. In particular, these showed trends in risk of CTS that are compatible with the experimental evidence, as summarized recently by Viikari-Juntura and Silverstein (Viikari-Juntura *et al.,* 1999: Ex. 500–121–73).

There is a particularly large number of studies demonstrating that the risk of back disorders, including prolapsed lumbar disc, increases with the frequency or duration of manual material handling, with load weights, and with other indicators of physically strenuous work including but not limited to lifting and carrying tasks. Again, exposure has been variously characterized on the basis of observation, self-report, and bio-instrumentation measures and/or combined into indices. The volume of evidence is extremely impressive and demonstrates that such exposure-response relationships have been found in nursing and other health care work, in construction, in manufacturing, and in the wide range of jobs encountered in the general population. For example, Venning *et al.* (Venning *et al.,* 1987: Ex. 500–41–49) published a prospective study of a closed cohort, which showed the predictive value of work area classified *a priori* in terms of lifting demands. Kerr, Norman, and colleagues (Kerr *et al.,* Ex. 500–41–74 ; Norman *et al.,* 1998: Ex. 38–84) compared cases to controls on 12 continuous biomechanical variables, representing both peak and daily integrated load on the spine. There was a higher load in the cases by each variable (all p-values < 0.04). There was a moderate amount of correlation among these variables, so the final regression model was reduced to four, with adjustment for demographic and psychosocial factors. The odds ratios, computed both for full observed ranges of exposure and more conservatively for inter-quartile spreads, showed that several dimensions of load on the lumbar spine made independent contributions to risk of back disorders.

It is of particular interest that three different studies (Marras *et al.,* 1993: Ex. 500–41–94 ; Wang *et al.,* 1998: Ex. 500–41–52; Waters *et al.,* 1999: Ex. 500–121–76) showed such a relationship when lifting demands were characterized using the NIOSH lifting index (Waters *et al.,* 1993: Ex. 26–521). (It should be noted that Waters *et al.* (Waters *et al.,* 1999: Ex. 500–121–76) also estimated the odds ratios in a multivariate logistic regression model that included nine other covariates. These estimates so obtained were higher for the category of LI=1–2 and otherwise lower than the crude estimates. However, 7 of the covariates in the model had little association with LBP, so this model was deemed overly conservative and the unadjusted ORs were selected as summary measures of the study results.)

Studies of other, related health outcomes, including knee arthritis and ''overexertion incidents'' of any body part, provide compatible findings regarding the effects of strenuous work. In addition, Krause *et al.* (Krause *et al.,* 1997: Ex. 26–1281) found that disability retirement was increasingly frequent from jobs with heavy physical demands and also showed an exposure-response trend with an index of repetitive strain that included lifting demands, muscle effort, and non-neutral postures. The cases of disability retirement were due to any medical condition; however, a large proportion was caused by musculoskeletal conditions (see Table 2 of (Krause *et al.,* 1997: Ex. 26–1281)).

Posture

Studies of the effect of non-neutral postures also include a wide range of exposure measures, including estimated frequency or duration of specified postures, as well as tasks that imply specific postural demands (*e.g.,* driving as an indicator of highly constrained static sitting) and workstation characteristics that directly influence posture (*e.g.,* VDU keyboard too high). Since the anatomic segments of the body form a kinematic chain, non-neutral postures may affect not only the same joint region but also other joints along that chain. For example, if the work layout requires the trunk to be twisted while the eyes are facing forward, the neck will also be twisted and health effects may be found all along the spine. Work with the arms elevated may alter wrist posture or impose a biomechanical disadvantage on the arm muscles; it will increase the torque exerted by an object held in the hands, which in turn increases the compressive forces experienced in the lumbar spine (Chaffin *et al.,* 1991: Ex. 26–420).

There are a very large number of studies showing that neck and shoulder disorders exhibit an exposure-response relationship with arm and neck postures, especially arm elevation to form an included angle of at least 30° flexion or abduction. Both Bergqvist *et al.* (Bergqvist *et al.,* 1995: Ex. 500–165–24) and Faucett *et al.* (Faucett *et al.,* 1994: Ex. 38–256) showed an increasing risk as the height of the VDU keyboard increased relative to seated elbow height. In a case-control study within a single automobile assembly plant, Punnett and colleagues found an increasing risk of shoulder disorders with the observed proportion of the work cycle in which the included angle at the shoulder was at least 90 degrees (Punnett *et al.,* 2000: Ex. 500–41–109). This association was not confounded by gender or other demographic or medical history factors.

Viikari-Juntura *et al.* (Viikari-Juntura *et al.,* 2000: Ex. 500–41–50) carried out a longitudinal study with four repeated questionnaires among 5180 workers in a large forest industry enterprise. The authors used a modified Nordic questionnaire (Kuorinka *et al.,* 1987: Ex. 38–204) for the health outcome of '' radiating neck pain'' and validated exposure assessment and psychosocial questionnaires. There was a statistically significant dose-response relationship for radiating neck pain with the frequency of ''twisting movements of the trunk during a work day'' (ORs from 1.0 to 2.3), as well as a dose-response relationship for hands above the shoulder. These estimates were adjusted for body mass index and high mental stress.

English *et al.* conducted a study of patients in the general population seeking medical care for upper extremity disorders (English *et al.,* 1995: Ex. 26–848). Conditions affecting the wrist and hand showed exposure-response relationships with several different shoulder and wrist postures (Table 3b). The degree of ulnar deviation has been reported to be associated with the risk of forearm and wrist disorders (Hunting *et al.,* 1981: Ex. 26–1276; Malchaire *et al.,* 1996: Ex. 26–1473). Several authors have found that the risk of carpal tunnel syndrome increases with the number of hours per day or week in which the wrist is flexed or extended (Blanc *et al.,* 1996: Exs. 26–42, 500–41–16; de Krom *et al.,* 1990: Ex. 26–102; Nordstrom *et al.,* 1997: Ex. 26–900).

In studies of back disorders, a number of investigators have reported exposure-response relationships with trunk forward flexion, lateral bending, and rotation. These studies address non-neutral postures in both seated and standing work, and they cover a range of industries and occupations from tractor driving to construction to automobile assembly. Similar data for the U.S. general population were obtained from analysis of the National Health Interview Study (Exs. 26–1106, 26–1107). There is also evidence of

increasing risk with static sitting, both assessed directly and through estimated time or distance driving per week (although the latter may also involve exposure to whole-body vibration). In the study by Pietri *et al.* (Pietri *et al.,* 1992: Ex. 38–309), the odds ratios for both prevalence and one-year cumulative incidence of low back pain showed increases with hours of driving per week in multivariate models adjusted for age, gender, comfortable car seat (y/n), carrying loads (y/n), standing (y/n), tobacco consumption, and psychosomatic factors.

With regard to disorders affecting the lower extremity, knee-bending, kneeling, squatting, jumping from one level to another, and stair-climbing are all found in these studies. In a series of Danish studies, direct observations showed that the average proportion of time that was spent kneeling and/or squatting by workers in three different trades (Jensen *et al.,* 1997: Ex. 500–41–69). The prevalence of knee disorders among the same three trades increased proportionately to the exposure prevalences. Anderson and Felson utilized the U.S. Department of Labor Dictionary of Occupational Titles and characterized each occupation on the basis of the proportion of job titles within it that required knee-bending (0%, up to 50%, more than 50%) (Anderson *et al.,* 1988: Ex. 26–926). Among subjects aged 55 to 64 years, there was a two to three-fold increase in risk of radiographic osteoarthritis with each category of knee-bending, adjusted for gender, race, education, and body mass index. These odds ratios represent the increase in risk across the three categories, *i.e.,* from no to some and from some to much knee-bending.

Vibration

Segmental vibration exposure to the distal upper extremity, especially through holding and operating power tools, is another area of research where exposure-response relationships have been reported by numerous authors. Some studies have shown the association with years of exposure, and others combined work history with direct measurements of frequency and acceleration to construct biologically informed cumulative exposure indices. Most of the evidence concerns neurological and circulatory impairment of the hand and wrist. Three different investigations reported an odds ratio of about 1.1 for each year of occupational exposure to hand-arm vibration, which represents a doubling of risk about every 7 years. In addition to those studies shown in Table 4a, Nordstrom *et al.* (Nordstrom *et al.*, 1997: Ex. 26–900) reported an ''alternative'' multivariate model of CTS in which there was a positive quadratic dose-response relationship (p=0.01) for use of power tools or machinery. While this variable was not conclusive regarding exposure to segmental vibration, it does suggest an exposure-response trend between segmental vibration and CTS.

In an historical cohort, Futatsuka *et al.* (Futatsuka *et al.*, 1985: Ex. 26–1430) found a positive association between the prevalence of ''vibration-induced white finger'' and the duration of exposure to vibration (chain saw use). In addition, there was an interaction with year of first exposure: higher prevalences and earlier onset of symptoms were observed among workers with earlier first exposure, when the acceleration levels were higher (all data presented graphically). One study team found similar associations for the risk of shoulder disorders (Stenlund *et al.,* 1993: Ex. 502–462; Stenlund *et al.*, 1992: Ex. 26–733).

Several statements contained in submissions by the Chamber of Commerce and others cited OSHA's statement in the preamble to the proposal that it had not constructed ''generalized quantitative exposure-response relationships'' for standard-setting (64 Fed. Reg. at 65927), and that the Agency's reluctance to set permissible exposure levels for risk factors provided evidence of a lack of exposure-response relationship in the epidemiologic literature (*e.g.*, Chamber of Commerce, Ex. 30–1722, p. 46 and Ex. 500–188, pp. 10–11; United Parcel Service, Ex. 500–197, pp. I–61 to I–62). Such arguments confuse exposure-response relationships *as evidence of a causal relationship* with the last stage of *quantitative risk assessment*, namely computation of a permissible exposure level.

It is critical to distinguish between these points. Exposure-response relationships have been demonstrated in the epidemiologic literature, using a variety of exposure metrics and for a variety of health outcomes, and a number of reviewers have cited this evidence in concluding that there are causal relationships (eg., Armstrong *et al.*, 1993: Ex. 26–1110; Bernard, 1997: Ex.26–1; Burdorf *et al.*, 1997: Ex. 500–121–13; Hagberg *et al.*, 1992: Ex. 8–1; Hales *et al.*, 1996: Ex. 26–896; Viikari-Juntura *et al.*, 1999: Ex. 500–121–73). At the same time, although the indicted exposures and their associations with MSDs are qualitatively similar across many studies, the variations in measurement approaches results in very limited numbers of studies with any single exposure metric. More importantly, there is substantial evidence of interactions among physical exposures, so that (for example) jobs requiring both repetitive and forceful motions have a higher risk than jobs requiring either exposure alone (Armstrong *et al.*, 1987: Ex. 26–48; Silverstein *et al.*, 1986: Ex. 26–1404; Silverstein *et al.*, 1987: Ex. 26–34). (Numerous examples of other additive or multiplicative effects between physical ergonomic exposures have been listed in Tables V–9 through V–13). Thus, the exposure-response curve for each exposure should ideally be described as a function of the level of *each* other exposure that might also be present in the same job. This represents an enormous number of *combinations* of exposure, of which only some have been studied epidemiologically to date. Given the available exposure-response relationships, plus evidence that exposures interact with each other, the decision not to attempt quantitative risk assessment calculations at this time is readily justifiable. However, this does not at all imply that the evidence for exposure-response relationships is insufficient to conclude that there is a causal relationship between exposure to risk factors and the risk of MSDs.

Another argument made in the testimony cited above is that if an exposure-response relationship existed, it would necessarily be linear or monotonic, and that it would necessarily provide an exposure level that could be used to differentiate between background risk of MSDs and an elevated risk (United Parcel Service, Ex. 500–197, pp. I–62 to I–67). This assertion is false. An exposure-response relationship need not take the form of a straight line through all data points; it may conceivably be better described as a logistic curve, or as a step-function, or as any other of a variety of mathematical functions. As one example, the analyses presented by Frost *et al.* (Frost *et al.*, 1999: Ex. 38–97) clearly show a non-linear exposure-response trend with cumulative exposure to repetitive and loaded shoulder flexion. Two among many other illustrations of non-linear, positive exposure-response relationships can be found in Liles *et al.*, 1984 (Liles *et al.*, 1984: Ex. 26–33 500–41–88), where the authors suggested that their graphs provided evidence of exposure thresholds, and Moore *et al.*, 1994 (Moore *et al.*, 1994: Ex. 26–1033), where a log-log transformation improved the fit of the model. A non-linear relationship, for example, accommodates the likelihood that some physical activity is beneficial and that only at more extreme levels do