

Determining What Constitutes An Occupational Disorder: Is the Horse Already Well Out of the Barn Door?

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Introduction: The Policy Challenge

Historically, workers' compensation plans have been designed to provide health care and wage loss indemnity coverage for workers with injuries and illnesses for which there has been little question of the "cause" being directly work-related. For example, acute trauma occurring at the workplace, and clear-cut effects of toxic exposures, especially where these health effects are quite specific to the toxicant in question, fit this historically simple model of "occupational" health problems. But today's pattern of workers' compensation claims in North America is very different, and rapidly becoming more so, in that a majority of claims are for soft tissue strains, sprains and related conditions, often of gradual onset with no history of acute trauma. For these conditions science is beginning to paint a much more complex picture of causation, a multifactorial one, in which workplace exposures play an important but only partial role. In addition, there is mounting epidemiological evidence that a number of equally multifactorial "degenerative" conditions of later life, such as osteoarthritis and coronary heart disease, are substantially accelerated and made more severe in their onset as result of various physical and "psycho-social" aspects of previous lifelong work. This paper: 1) reviews the current epidemiological evidence of work-relatedness for this range of conditions — which are either already important in workers' compensation statistics or likely to become so, should the accumulating scientific evidence of occupational causation be used to argue successfully for compensation coverage; 2) summarizes the key scientific issues in determining the extent to which workers' compensation for these sorts of conditions should be paid, and under exactly which circumstances; and 3) suggests and briefly analyzes a range of broad policy options for dealing with this looming crisis, before workers' compensation programs face the unsavory options of financial unsustainability or arbitrary exclusion of certain "occupational" conditions from entitlement.

Scientific Criteria for Assessing Evidence of Causation

Criteria by which to assess scientific evidence on causation, for any health condition, have been most fully developed by epidemiologists, the best-known of which is Sir Austin Bradford Hill (1897-1991). Bradford Hill put forward, in a now-famous paper published in 1965 (Hill, 1965), nine criteria for causation which could be applied to the scientific evidence available on any given question of the form: "Does exposure to X cause health outcome Y?" These criteria are listed in and described in Appendix 1, in an order slightly different than Bradford Hill used (for clarity). They are explained in non-technical language, with examples of exposure/disease combinations to clarify how the criteria are applied in practice. Hill himself suggested that none of these criteria, excepting perhaps "temporality", *must* be met for an exposure to be causal, rather they are considered collectively in relation to the whole body of evidence pertaining to a given causation question. Although some criteria are more solid indications of causation than others (see below), the more criteria are met, the stronger the evidence for causation.

While more recent revisions of these criteria exist (Susser, 1977), they have not replaced Bradford Hill's original formulation. Thus the present paper will utilize these classic criteria to determine whether sufficient evidence now exists to validly consider various work-related exposures to be causal for two ubiquitous conditions of later life: coronary heart disease and osteoarthritis, for which workers' compensation payments are rarely made in North America, and one exceedingly common condition of midlife, mechanical low back pain, for which compensation appears to virtually always be awarded on this continent.

Three Examples: Low Back Pain, Osteoarthritis and Coronary Heart Disease

Utilizing the standard epidemiological criteria for causation, described above, we will now review accumulating epidemiological and biological evidence which suggests that these three very common conditions are at least in substantial part caused by conditions of and exposures at work, acting over a number of years of adult life. We choose these conditions because they represent major burdens of gen-

eral population burden of illness and disability on which a great deal of new etiological evidence has been generated in recent years which would seem to argue for credible, if partial, occupational attribution. Furthermore they represent an instructive mix of one multifactorial condition, low back pain, where there is currently almost universal compensability in North America, despite, or perhaps because of, virtually no meaningful capability to validly discern with current medical methods which cases actually are due to work (Deyo, Cherkin, Conrad, et al. 1991; Frank, 1998; Frank, Kerr, Brooker, et al. 1996), and two equally multifactorial conditions, eventually affecting the majority of all elderly, for which workers' compensation payments are rarely if ever paid in North America.

Low Back Pain: Arguably the commonest cause of disability in adults under age 45 in modern societies, low back pain (LBP) has defied all efforts to define its exact pathophysiology, i.e. the nature of the underlying lesion (Agency for Health Care Policy and Research, 1994; Frank, Pulcins, Kerr, et al. 1995; Nachemson, 1994; Nachemson, 1992; Deyo, Cherkin, Conrad, et al. 1991; Frank, 1998; Frank, Kerr, Brooker, et al. 1996). Despite the advent of very sophisticated laboratory and imaging techniques, over 95% of LBP cases in the working-age population have no consistent pathological abnormalities demonstrated on any testing that physicians can currently order. Of course such patients do have abnormal functional capacity tests, but these only attempt to quantify the degree of impairment and disability associated with LBP, rather than illuminate its cause. Indeed, the absence of any such "objective findings" is the criteria for labeling such patients as having "mechanical" or "idiopathic" (unknown-cause) LBP, a diagnosis of exclusion (Agency for Health Care Policy and Research, 1994). Although the presumed underlying pathology is "soft tissue — muscle, tendon and ligament — strain or sprain," this has never been clearly demonstrated with scientific techniques currently available.

On the other hand, despite this failure to fully meet the "biological plausibility" criterion for causation by exposures at work, low back pain does meet a number of other Bradford Hill criteria for causation, as exhaustively reviewed by NIOSH in 1997: strength of association, dose-response relationship, consistency of association, temporal relationship and coherence of evidence (Bernard et al, 1994). These findings are briefly summarized in Table 2.

As the methodologically sophisticated and comprehensive NIOSH review pointed out — and is clear from Table 2 — the LBP causation evidence is, overall, much more convincing for biomechanical exposures at work (e.g. heavy lifting or forceful movements, prolonged non-neutral postures, and whole body vibration, as in prolonged driving), than it is for psychosocial exposures such as perceived time-pressure, low job control, negative social milieu at the workplace, and job dissatisfaction. However this difference in the currently available level of evidence should not be over-interpreted: biomechanical exposures have been studied much longer, and with better measurement methods, than psychosocial exposures. Thus the rate of accumulation of new, high-quality evidence on the latter may well surpass that on the former in coming years. As an example, a new case-control study, currently submitted for publication by the first author and colleagues at the Institute for Work & Health, shows that perceptions of a poor-quality social environment at work (e.g. frequent conflicts and unhelpful relationships), inadequate matching of worker skills and job tasks in the mind of the worker, and low job control all contributed independently (RRs 2.8, 2.3 and 1.9 respectively) to the risk of reporting LBP to a workplace nursing station in a large car assembly plant in Ontario in the mid-1990s (Kerr, Frank, Shannon, et al. 1998). Furthermore, these risk contributions were estimated after controlling for equally large risk contributions from three different, objectively measured biomechanical forces on the spine while working, and worker perceptions of job "heaviness" (Norman, Wells, Neumann, et al. 1998; Kerr, Frank, Shannon, et al. 1998). This is the first study to so clearly show the independent effect of both occupational biomechanical and psychosocial risk factors on LBP, indicating how young this field of research still is.

A special note is warranted on the question of "biological plausibility" evidence for LBP and occupational psychosocial factors. While it may be entirely intuitive to see a relation between LBP and biomechanical loads on the spine related to the physical tasks of work, it is more difficult to appreciate how the mental and emotional state of the worker, as affected by the workplace social environment, could also affect the risk of back injury, leading to LBP. The NIOSH review does point out a number possible mechanisms for the latter effect, based on our rapidly growing knowledge of the body's myriad connec-

tions between the brain, the endocrine system and the locomotor system (Sauter & Swanson, 1996; Bernard, Sauter, Fine, et al. 1994; Bongers, de Winter, Kompier, et al. 1993):

“First, psychosocial demands may produce muscle tension and exacerbate task-related biomechanical strain. Second, psychosocial demands may affect awareness and reporting of musculoskeletal symptoms, and/or perceptions of their cause..... Third, initial episodes of pain based on physical insult may trigger a chronic nervous system dysfunction, physiological as well as psychological, which perpetuates a chronic pain process. Finally, in some work situations, changes in psychosocial demands may be associated with changes in physical demands and biomechanical stresses, and thus associations between psychosocial demands and musculoskeletal disorders occur through either a causal or effect-modifying relationship.” [(Bernard et al, 1994), Chapter 7, page 3.]

Lastly, some may question the entry in the last line of Table 2, concerning the current dearth of high-quality scientific evidence that LBP at work can be effectively prevented by intervening on the putative causal factors listed in the top line of the table, particularly the ergonomic spinal loads of jobs. As carefully explained in our 1996 comprehensive review of this subject (Frank, Kerr, Brooker, et al. 1996), it is so difficult to design and execute controlled studies of any workplace intervention, that the lack of good intervention studies of this kind may simply reflect this difficulty. A lack of high-quality evidence on prevention effectiveness is not the same as high-quality evidence (e.g. from “negative” controlled trials) that a condition cannot be prevented by an intervention. Indeed, our conclusion in that review was that there are ample basic-science and epidemiological grounds for believing that well-designed ergonomic changes should reduce rates of LBP disability in high-exposure jobs, but that proof of this may remain elusive. All such interventions consequently deserve careful evaluation of their health effects.

Coronary Heart Disease: A number of biomedical risk factors for CHD have been known for over thirty years, and these all now meet several of the causation criteria described above. For example, smoking, high blood pressure and high blood levels of total/low-density-lipoprotein cholesterol all:

- carry relative risks of 2 to 3 for CHD in all studies;
- show dose-response relationships (which are also consistent and temporally appropriate across studies);
- have credible biological mechanisms by which they speed up the process of arteriosclerotic plaque formation in arteries;
- demonstrate patterns of occurrence in space and time related to CHD rates that are consistent with causation;
- have convincingly been (albeit only within the last decade, in the case of high blood pressure and elevated cholesterol) experimentally reversed so as to actually reduce CHD risks, either in smoking cessation cohort studies, or randomized control trials of drug and/or dietary treatment.

Family history, and a variety of new genetic markers of CHD risk, while long recognized clinically as important in CHD causation, now have met all but the last of Bradford Hill’s criteria, and experimental reversal of risk may soon be within reach as a result of promising new gene therapies. Thus all these risk factors are widely recognized as causal. Indeed, many other more recently discovered CHD risk factors, such as elevated blood levels of iron and homocystine (the latter thought to be correctable by increased dietary intake of folic acid) and low levels of selenium and Vitamin E, are well on the way to meeting these same causal criteria. However new research on various “psychosocial” aspects of work, as risk factors for CHD, is just beginning to meet the Bradford Hill criteria and many epidemiologists, let alone practicing physicians, are therefore unfamiliar with it.

By far the most solidly implicated of these aspects of work is the degree of “control” or “decision latitude” felt by workers concerning their jobs, based on their responses to a standardized questionnaire created by Robert Karasek and Tores Theorell (Kasl, 1996; Theorell & Karasek, 1996; Johnson & Hall,

1995; Karasek & Theorell, 1990; Karasek, 1979). Typically, studies record workers' responses to these questions and follow them for some years, with periodic examinations to detect new cases of CHD, or at least record linkage to detect deaths from CHD. However, a "prospective cohort" study of this kind needs to meet a number of specific epidemiological quality standards in order to provide valid and precise estimates of the relative risk of CHD in workers with "low job control" as compared to those with "high control," while taking into account other "independent" risk factors for CHD, utilizing multivariable risk regression analyses. These standards, reviewed in detail elsewhere (Rothman, 1986; MacMahon & Pugh, 1970), have been taken into consideration in the selection of the studies discussed below.

Perhaps the most convincing set of publications come from the Whitehall II UK Civil Service cohort of Michael Marmot and colleagues in London. In a series of papers published in the last year or so, they have documented an approximate doubling of the risk of new CHD cases in both male and female office workers with low job control (Bosma, Peter, Siegrist, et al. 1998; Syme & Balfour, 1997; Marmot, Bosma, Hemingway, et al. 1997; Bosma, Marmot, Hemingway, et al. 1997). Furthermore, comparative analyses in this same cohort reveal that there is just as strong an independent relationship (RR 2.15) between CHD and subjects' earlier responses to a recently developed questionnaire measuring the imbalance between a worker's perceived rewards from his/her job and the effort required to do it (Peter & Siegrist, 1997; Siegrist, 1996; Siegrist, Peter, Junge, et al. 1990; Bosma, Peter, Siegrist, et al. 1998). A possible shortcoming of the new Whitehall II studies is that not all of the CHD cases analysed were medically confirmed. However, they are at least compatible with CHD, as judged by questionnaire responses concerning chest pain. The cohort is still rather young for CHD, and future analyses based on the additional cases rapidly accruing should remedy this deficiency.

While such modest relative risks are clearly not in themselves strong evidence of causation, Marmot et al. have statistically controlled for such a large variety of other CHD risk factors and worker characteristics that even skeptical cardiovascular epidemiologists are paying serious attention to these studies (Davey Smith, 1997). Furthermore, there is consistency between the RR estimates in these studies and those of many others (see for example the review by Schnall and Landsbergis in 1994 (Schnall, Landsbergis, & Baker, 1994)) including a large and well-designed case control study of Swedish middle-aged males (Johnson, Stewart, Hall, et al. 1996). While there are some respectable studies which show smaller RRs (Steenland, Johnson, & Nowlin, 1997; Alterman, Shekelle, Vernon, et al. 1994), there tend to be technical reasons for these discrepancies, such as differences in the amount of job control for which the RR is calculated, or probable exposure misclassification due to the use of national-survey-based estimates of job control for each subject's occupational title, obtained from other times and populations.

These studies, taken together, also demonstrate a dose-response relationship for CHD risk across degrees of job control, in that workers with jobs that carry an intermediate level of control show an intermediate level of CHD risk. In the Swedish study, there is also a suggestion that worker perceptions of low social support at work, when combined with low job control, carry a stronger (2.6-fold) risk of CHD over 25 years of follow-up. This is comparable to the strength of association demonstrated for CHD and smoking, high blood pressure or high cholesterol levels. Thus it is possible to fill out a summary evidence table for CHD and low job control, demonstrating the fulfillment of over half of the nine Bradford Hill criteria, based on only an embryonic literature that is growing exponentially each year (Table 3). This again compares favourably with the performance of classic CHD "causal" risk factors, such as smoking, in such summaries of evidence for causation.

What of the biological plausibility of the relationship between job control and CHD? New research is beginning to suggest that each individual's characteristic pattern of physiological and biochemical responses to such psychosocial stressors begins early in life (Boyce, Chesney, Alkon, et al. 1995; Jemerin & Boyce, 1990; Evans et al, 1994). Furthermore, such responses likely involve not only the classically studied neuro-endocrine "fright or flight" (adrenal-gland-medulla/catecholamine-mediated) or generic "stress" (adrenal-gland-cortex-glucocorticoid-mediated) pathways, but also a host of newly discovered other connections between the brain and our immune systems, as well as our blood-clotting apparatus (McEwen, 1998; Evans et al, 1994). Taken together with new evidence that the

decades-long development of arterial plaques in CHD may be influenced by immune process (Gupta, Leatham, Carrington, et al. 1997; Gupta & Camm, 1997) and that critically narrow arteries may then be closed off in a heart attack or stroke by acute tendencies to clot more easily, that may in turn build on lifelong thrombotic predispositions (Jensen & Eenberg, 1996), these new studies allow us to complete our causation evidence table, for CHD and low work control, as showing a high degree of biological plausibility (Table 3).

Unfortunately, evidence meeting the very useful last criterion in the table, experimental reversal of risk, is not yet available. Again, as with workplace ergonomic intervention studies, there are immense logistic, scientific and ethical hurdles involved in demonstrating in a controlled experiment that workplace reforms to improve job control actually reduce CHD risks in workers over subsequent decades. A recent review of all types of workplace intervention studies found very few that did a good job of measuring even the short-term health effects of such changes (Polanyi, Eakin, Frank, et al. 1997).

Osteoarthritis: Osteoarthritis (OA) is usually a slowly developing “degenerative” condition of later life, characterized by joint pain and limited joint function that can affect any joint of the body. It predominately affects the hands, hips and knees, the joints which are most subjected to mechanical loading. The etiology of OA is multifactorial in that its pathologic correlate is an injured cartilage, most often resulting from chronic mechanical stress, acting in concert with a slow, age-related degradation and thinning of the joint cartilage. High levels of physical activity and overweight have been the main foci of epidemiological investigations into factors contributing to the development of OA. However, other factors, such as acutely traumatic joint injuries, smoking and educational attainment have also been investigated. Two systematic overviews were published in 1996 and 1997, summarizing the relationship between occupational risk factors and knee disorders including OA (Jensen & Eenberg, 1996), and radiologically defined knee and hip OA (Maetzel, Makela, Hawker, et al. 1997). Both reviews concluded that the evidence in favour of mechanical occupational exposure as risk factor for OA is strong and consistent.

OA has been subject to many definitions depending on whether the focus is on clinical symptoms and functional deterioration, or on pathological manifestations independent of clinical status. Further confusing matters, the public tends to label all pain and malfunction in and around the joints as “arthritis and rheumatism”. A safeguard used in epidemiological studies against the inclusion of inadequately defined cases has therefore been to rely on clinical diagnosis by a health professional, or radiological diagnosis according to agreed-upon criteria. A variety of definitions have therefore been used in epidemiologic studies, which make direct comparisons of their results difficult.

In the published etiological overview by one of the authors of the present paper and his colleagues, all studies to date dealing with mechanical occupational exposure and the development of radiologically documented osteoarthritis of hip or knee, were rated for their quality and strength of evidence according to the Bradford Hill criteria for causation (Table 4). The association between mechanical occupational exposure and knee OA is moderately strong, with odds ratios (estimates of relative risk) around 2.5, and dose-response relationships, documented in several, high-quality epidemiologic studies (Kivimäki, Riihimäki, & Hanninen, 1992; Anderson & Felson, 1988; Felson, Hannan, Naimark, et al. 1991). One of these studies was able to follow-up the Framingham cohort over decades, as is required to investigate causation in such a slowly developing condition. It is therefore the only prospective evidence currently available of an appropriate temporal relationship between mechanical occupational exposure and the subsequent development of knee OA (Felson, Hannan, Naimark, et al. 1991). It was found that individuals exposed to frequent knee bending at work, or to generally heavy labour, have a 2.5 times higher risk of developing knee OA. The association between mechanical occupational exposure and hip OA is less well studied than that for knee OA. Although studies examining hip OA show consistently positive results, very few of them are free of major scientific flaws (Roach, Persky, Miles, et al. 1994; Vingard, Hogstedt, Alfredsson, et al. 1991; Croft, Cooper, Wickham, et al. 1992).

The “biologic plausibility” evidence is convincing in the limited sense that we know OA to be accelerated by mechanical exposures that damage the cartilage of a joint, which acts much like a shock-

absorber (Bullough, 1998). However, little direct human biological evidence is available on the exact mechanisms of joint damage due to prolonged low-level mechanical stress, such as that occurring in lifelong exposure to heavy work. As for analogy, clinicians have known for centuries that some individuals, presumably with an unknown genetic or other basis for their susceptibility, develop rapid-onset OA, termed “post-traumatic degenerative arthritis,” within less than a decade after acute joint injury. This condition would seem to represent a naturally occurring human analog of more slowly developing, garden-variety OA that afflicts the majority of the elderly at some point in their lives.

Conclusion: The point of these three summaries of causation is that there is already, even on the basis of still-incomplete evidence of occupational causation, a problematic inconsistency in the legislated compensability and practical adjudication of the three medical conditions discussed above. On the basis of the epidemiological and human biological evidence of causation tabulated above, one could argue that there are comparable grounds for considering that a substantial fraction of cases of all three conditions, arising in *former* or current workers with a history of the relevant specific biomechanical or psychosocial exposures at work, could be considered to have had their clinical onset accelerated, and partially caused, by those work exposures. One rather simplistic measure (cf. below) of the extent to which a given cause contributes to an overall burden of multifactorial illness, often used by epidemiologists, is the “etiologic fraction in the exposed,” which is equal to $(RR-1)/RR$ (Rockhill, Newman, & Weinberg, 1998). This takes a value between one-half and two-thirds for these three conditions, in that all three have RRs between 2 and 3 for the work exposures described in Tables 2-4. In turn, this implies that one-half to two-thirds of cases of all three conditions, in adequately exposed workers, would not have occurred unless the exposures had. Thus previously “exposed” workers with these conditions, whose jobs can be shown to have had the attributes in question during their working lives, could reasonably claim a substantial proportionate compensation award for their pain and suffering from workers’ compensation insurance. Yet, paradoxically, only cases of low back pain are likely to have such claims taken seriously by current compensation adjudication processes in North America. Indeed, as has been pointed out (Deyo, Cherkin, Conrad, et al. 1991; Frank, 1998; Frank, Kerr, Brooker, et al. 1996) virtually all LBP cases presenting as compensation claims in most North American jurisdictions are automatically awarded benefits, without any consideration of the specific degree of exposure in the individual worker’s case, largely on the grounds that it is currently medically impossible to determine which of these cases are in fact caused by work. Sometime soon, one would think, a thoughtful lawyer and his client will convince a court that this is fundamentally unfair to victims of early severe osteoarthritis and/or heart disease who have experienced apparently causal work exposures over many years. This decision will, in turn, predictably have very challenging implications for the whole current workers’ compensation model, when such claims for these and other ubiquitous conditions of later life, increasingly linked to work exposures, then become widespread.

How Large A Compensation Bill for Such Conditions Might We See?

Just to be provocative, we have performed some preliminary conservative calculations of what the current total burden of illness, and associated compensation claim costs, might look like, should osteoarthritis become widely compensable in Canada. We have attempted to estimate the current prevalence of work-related OA using four different scenarios, based on information from Canadian census data, national and provincial population health surveys and other epidemiologic data from international sources. The prevalence estimates were conservatively calculated only for the working age-group between 35 and 64, based on a variety of possible case definitions for work-related OA:

- Scenario 1 — “arthritis and rheumatism” reported by the Canadian National Population Health/Ontario Health Survey respondents as having been diagnosed by a health professional and linked by that professional to their work;
- Scenario 2 — the work-related excess prevalence (defined as in Scenario 1) of OA in Canada, obtained from the etiologic fraction $[(RR-1)/RR] = 0.6$, based on the relative risk of 2.5 reported in Table 4] applied to the population exposed to “heavy work,” based on Canadian surveys;

- Scenario 3 — “disability”, in the sense of inability to carry with normal activities of daily living, associated with “arthritis & rheumatism”, reported in the Canadian National Population Health Survey as being diagnosed by a health professional, and attributed by him/her to the respondent’s work;
- Scenario 4 — “knee or hip OA” based on radiological population x-ray surveys in the US or Finland, multiplied by the same etiologic fraction used in the previous scenario, for the Canadian population exposed to heavy work according to survey responses.

The overall result is that anywhere between 33,000 and 165,000 Canadians between 35 and 65 years of age in 1997 could be expected to have work-related osteoarthritis, according to these various case definitions. And this, again, excludes the elderly, in whom the condition is much more prevalent, with some OA-related disability affecting perhaps the majority of those who have been retired for a decade or more, a proportion of which is very credibly due to previous lifelong physical work exposures.

Because these prevalence estimates don’t provide any sense of the potential annual compensation claims volumes or costs for work-related OA, as a reason for time taken off work, we have looked for and found data from the German statutory health insurance system showing that 1.01% of actively employed women age 35-65, and 1.11% of employed men of the same age go off work for OA-related reasons each year, with a mean duration of disability of 45 days (AOK-Bundesverband, 1996). We may take as the mean cost per episode/claim a very conservative \$CDN, 5000, which is a reasonable mid-point estimate for the relatively few workers’ compensation claims for OA and related conditions (mostly thought to be post-traumatic degenerative arthritis) which were paid out in Ontario in the mid 1990s. The result is a potential total annual compensation bill in Canada for OA of \$1,255,844,159.00. Because the German absences for OA are not only for work-related OA but also all other OA cases, we could prorate by the population attributable risk estimating the proportion of all OA due to heavy work in Canada (25.6%, given an RR of 2.5 and the prevalence of “heavy work,” reported by 23 % of the employed respondents in the 1990 Ontario Health Survey, and defined as “bending and lifting more than 50 times a day.”) The resultant figure, still over \$320 million annually, is substantial, and probably too low, for two reasons: 1) 45 days of lost time alone, not including medical costs, often leads to more than \$5,000 in payments, on average, at current wage-replacement rates in several provinces; 2) there is a great deal of public-sector-subsidized early retirement for health-related reasons in Germany, so that employees severely affected by OA, and other disabling conditions, are much more often given attractive early retirement packages than in Canada, implying in turn that there are fewer such disabled people left in the workforce requiring sick-leave annually.

Key Constraints in Determining Partial Causation and Proportionate Attribution:

To further complicate matters, the scientific methods currently available to calculate the proportion of a multifactorial burden of disability which is attributable to a given occupational (or other) exposure are simply not up to the task of determining the exact compensation to be appropriately given to a particular workers’ compensation case. In fact, there are currently no identifiable scientific tools for validly and feasibly making this determination in the individual case, so that legislation (as in the case of low back pain, versus osteoarthritis and heart disease) has generally been compelled to make each diagnosis either fully compensable or not at all compensable.

Some public health professionals and clinicians have presumed that *individual* compensation awards for multifactorial conditions can be fairly decided using the sort of “etiologic fraction” calculated above (1/2 to 2/3) for the proportions of LBP, CHD, and OA in occupationally exposed populations which are “attributable to” specific work exposures, with relative risks of 2 to 3. However, as is clearly argued in a recent review of this subject, these fractions lack an essential logical quality for their use in such legal settings: the fractions can and often do add up to more than 100% of the whole case burden being “explained” by all the known risk factors for the condition in question (Rockhill, Newman, & Weinberg, 1998). The explanation of why this is so has been provided more than once by epidemiol-

ogists (Rothman, 1986; Greenland & Robins, 1988; Walter, 1976) but that has not stopped the publication of articles that inappropriately attempt, for example, to add up the etiological fractions for several risk factors for a multifactorial disease, and then naively and artificially constrain that total to never exceed 100% [see Rockhill et al. 1998 for examples (Rockhill, Newman, & Weinberg, 1998)]. Suffice it to say here that there is no appropriate way to utilize these epidemiological *population-based* tools for assigning an exact proportionate cost to workplace exposures, in the *individual* workers' compensation case, especially when, as for CHD or OA, there are other non-workplace exposures (often poorly measured) also known to have credible causal links the same outcome. In short, epidemiologists have no simple answer to the problem of assigning proportionate "blame" to one of several causes in a given case of multifactorial disease. This does not mean that work exposures on the *population level as a whole* are unimportant in causing an increased burden of illness and injury for these conditions. It just means that more usual, and therefore arbitrary, legal means, such as judicial or jury judgment, must be used in such individual cases to decide what proportion of a given claimant's "costs" should be borne by the workplace or its insurer.

Possible Solutions

In this final section, we briefly consider three possible policy solutions, some of which have already been informally tried, in some jurisdictions, to deal with the problem outline above: namely, how to adjudicate the "work-caused" proportion of multifactorial health problems that should be paid for, to cover both medical and wage-loss costs, by workers' compensation insurance:

One approach involves giving up on adjudication entirely and fully compensating all claims, independent of a case's specific exposures. As previously implied in this paper, this is in essence what we have now for LBP in most of North America. This hardly seems a viable long-term solution. Indeed, one international commentator has referred to compensable LBP as the "end of the welfare state". This is especially a concern given the strong likelihood of ever increasing claims in future for other common chronic diseases such as CHD or OA, now being scientifically linked to one aspect of work or another. On the grounds of fairness alone, this approach would seem unlikely to stand up in court as defensible for use with one set of multifactorial conditions influenced in part, but only in part, by work (e.g. LBP), but not another (e.g. CHD or OA.)

The second approach suggests the approach of tightening up eligibility criteria so as to fit the monies available for compensation (e.g. by limiting compensability to low back pain due to clear-cut acute trauma "witnessed" at work, or to acute heart attacks suffered at work). In going this route, we would be essentially attempting to mimic current LBP coverage policy in Japan, Switzerland and a few other wealthy countries (Hadler, 1995). However, one cannot judge these alternative national policies fairly unless one knows a great deal about the rest of the local social welfare net of payments available to the disabled, that "backstop" workers' compensation per se. For example, such apparently hard-nosed policies, such as requiring clear evidence of acute trauma (a fall or blow) for LBP cases to qualify for workers' compensation, may result in little absolute hardship if there is generous "no-fault" sickness pay widely available as well. This would obviously be unlike the situation in North America, where the backstop when workers' compensation fails may be nothing but general welfare, especially for blue-collar workers without collectively negotiated short- and long-term disability benefits. While such tough policies may appear to provide improved natural justice for employers who now complain that they pay workers' compensation premiums to cover some low back pain claims that are not purely caused by work, they would inadequately penalize employers with truly adverse, and preventable, ergonomic exposures in their jobs that only gradually overload the spine in the longer term, which may soon have as strong a body of evidence linking them to LBP as any known causal factor (Table 2.) Finally, it is patently ridiculous to limit coverage for chronic diseases such as CHD or OA to those cases that manifest symptom onset when the patient happens to be at work, when the underlying pathological process, now thought to be accelerated by work exposures, is known to have been developing for decades.

The third approach would involve biting the bullet and accepting the fact that many aspects of work, and life outside of work, are inextricably entwined as causal factors for a wide range of chronic diseases and conditions, as modern epidemiology is now beginning to show, and that the long term solution is a fairer, affordable and responsive system of “no-fault” sickness and disability insurance coverage for all citizens, whether their health problem is partly attributable to past or present work conditions or not. We suggest that the increasing list of common diseases which recent research has shown to have at least partial attribution to work constitutes a looming crisis in workers’ compensation as we know it. There is no easy way out, if we persist with the current simplistic “adjudication” of these conditions as either “caused” or “not caused” by work. The problem is simply not soluble without committing to a longer-term plan for generic (both occupationally and non-occupationally related) disability insurance coverage, which meets the basic needs of the whole population. Fortunately examples exist of such programs elsewhere (Mashew et al, 1996). We also note that in B.C. there is at least a single public sector disability insurer for each of the two largest categories of disability: motor vehicle accidents (ICBC) and work-related ill-health (WCB). This should at least make it easier to integrate these disability insurance programs. This is especially true for dealing with disability in the elderly, in whom most of the delayed health effects of adverse lifelong work — and other — exposures inevitably become manifest. We cannot feasibly hold past or present commerce fully responsible for all the adverse physical and psychosocial conditions of work decades ago, that have, for example, contributed to their former employees’ elevated rates of heart disease and osteoarthritis now.

This is not to argue that there is no place for employer liability for clearly hazardous working conditions, especially those that obviously threaten safety, or involve unacceptable exposures to known toxicants. But, when it comes to largely chronic and recurrent multifactorial conditions of delayed onset, such as those discussed in this paper, it would seem more appropriate to use our newfound knowledge of causation to proactively prevent ill-health by improving sub-optimal job design and working conditions, rather than hope that punitive workers’ compensation premiums levied decades later, when the caused cases present, will act as a deterrent. In the end, we all can expect some of our health problems of later life to be attributable to our earlier worklife. The issue is how to provide adequately for our care and livelihood when that occurs. The current workers’ compensation model would not seem to be up to this task.

Table 1: Bradford Hill’s Criteria for Causation

- Strength of association
- Dose-Response Relationship
- Consistency Across Studies
- Temporality
- Biological Plausibility
- Coherence
- Specificity
- Analogy
- Experimental Risk Reversal

Table 2: Summary of Current Causation Evidence - LBP and Work-Related Biomechanical and Psychosocial Exposures

Causation Criterion Status of LBP and Work Psychosocial / Biomechanical Exposures Evidence*	
Strength of association	RRs of 1.5-2.8, in a few good studies each, for perceived: time-pressure/workload, low job control, negative work milieu, job dissatisfaction; RR's of 2-5 or more in many good studies for: forces involved in (or observed levels of) heavy lifting/forceful movements, awkward postures (out of neutral position), and whole body vibration/driving
Dose-Response Relationship	Convincingly demonstrated for most of the biomechanical exposures listed above — not yet clear for the psychosocial exposures
Consistency Across Studies	Excellent for the biomechanical exposures, much less for the psychosocial ones, but far fewer good studies have been done
Temporality	Established for most biomechanical exposures and some psychosocial exposures
Biological Plausibility	Pathological lesion unclear, but ample laboratory evidence of several possible tissue- damage mechanisms for biomechanical exposures; for psychosocial exposures less clear, but consistent with recent psycho-neuro-endocrinologic and other biological research
Coherence	General pattern of LBP across occupations suggests causal role for heavy physical loads at work, but is difficult to assess for psychosocial factors, due to absence of routinely collected data on these exposures
Specificity	Unclear — though acute physical loads' traumatic effects in back tissues may mirror the effects of chronic/repetitive lower loads
Analogy	Criterion not met and unlikely to be met
Experimental Risk Reversal	No convincing studies yet available

* see text for references

Table 3: Summary of Current Causation Evidence -CHD and Work-Related Psychosocial Exposures

Causation Criterion	Status of CHD-Work Psychosocial Exposures Evidence*
Strength of association	RRs of about 2 in many good studies , and 2.6 for “low control” combined with “poor support” in one excellent study
Dose-Response Relationship	Present in some good studies -e.g. Whitehall II
Consistency Across Studies	Moderate, but higher in high-quality studies
Temporality	Well-established by prospective studies
Biological Plausibility	Rapidly becoming convincing
Coherence	Unclear: no exposure data routinely collected
Specificity	Not obvious at present
Analogy	No likelihood of meeting this criterion
Experimental Risk Reversal	No high-quality studies yet available

* see text for references

Table 4: Summary of Current Causation Evidence: Knee and Hip Osteoarthritis and Mechanical Occupational Exposure

Causation Criterion	Strength of Evidence	Comments
Strength of association	A (knee) A (hip)	Odds ratio of ~ 2.5 for jobs involving frequent knee bending or heavy labour
Dose-Response Relationship	A (knee) B (hip)	Studies for both knee and hip OA generally find ORs increase with severity of exposure
Consistency Across Studies	A (knee) A (hip)	Consistent relative risk estimates were found for knee OA in good quality studies. RR's higher and consistent, but studies of lower quality, in hip OA.
Temporality	B (knee) D (hip)	Framingham study is the only prospective study showing a clear temporal relationship
Biological Plausibility	B	Reasonably convincing
Coherence	D	? -- Data on mechanical exposures not routinely collected, but archaeological evidence consistent with heavy-work/OA link
Specificity	D	Criterion not likely to be met
Analogy	B	Post-traumatic degenerative arthritis
Experimental Risk Reversal	D	Criterion not likely to be met for ethical and logistic reasons

see text for references

- A: Strong research-based evidence (multiple relevant and high-quality scientific studies);
- B: Moderate research-based evidence (one relevant, high-quality scientific study or multiple adequate scientific studies);
- C: Limited research-based evidence (at least one adequate scientific study)
- D: No research-based evidence.

APPENDIX - Bradford Hills' Criteria for Assessing Evidence of Causation

1. Strength of Association: By this is meant the magnitude of the *relative risk* (RR) that describes how much more often the disease, or other health outcome in question, occurs after the exposure of interest, as compared its frequency without that exposure. For example, lung cancer was shown in the 1950's to occur about ten times more frequently in persons who smoke about a pack of cigarettes a day than in non-smokers. This is considered a high relative risk, indicating a strong association. Most exposures thought to play causal roles in the commoner diseases of our time, such as coronary heart disease (CHD) and cancer, have much smaller relative risks, indicating associations of more moderate strength. For example, both smoking and high blood pressure carry approximately two-to-three-fold risks of CHD, depending on just how much one smokes or how high one's blood pressure is. Although relative risks below 2, and especially below 1.5, indicate that the risk of the outcome in question is 50% to 100% higher in those exposed (an RR of 1.0 indicates no association at all), they describe what epidemiologists refer to as "weak associations", and the quality of study required to estimate such small RR's accurately is much higher. [Detailed discussion of how to judge epidemiological study quality is beyond the scope of this paper. A number of standard epidemiological texts describe how the quality of the evidence can be assessed from any causation study which estimates relative risk — the reader is referred to these (Rothman, 1986; MacMahon & Pugh, 1970)] Thus strong associations, with RRs above 5, while not frequently found, constitute firm *prima facie* evidence of causation even if several of the other criteria listed below are not met. On the other hand, a weak association, while not ruling out a causal role for an exposure, requires a great deal more evidence to be adduced for causation to be established.

2. "Biological Gradient" (Dose-Response Relationship): Closely related to the first criteria (above) is the notion that causal exposures should increase the risk of a health outcome more if they are intense, or "high-dose", than if they are "low-dose." Thus the fact that smokers of two or more packs a day are known to suffer up to twenty times as much lung cancer as non-smokers, compared to the ten-fold increase in risk for smokers of a pack a day, constitutes additional evidence of causation. Note, however, that some exposures are inherently rather "all or nothing", such as having a "Type A personality", so that there is no possibility of fulfilling this criterion, and it is therefore unhelpful in assessing causation in these situations. Additionally, there may be "saturation effects" in nature, whereby additional exposure beyond a certain "threshold" simply cannot elevate the risk of the adverse health outcome any further, and studies of exposures above this threshold will therefore not demonstrate such a gradient.

3. Consistency Across Studies and Settings: Whether a given scientific literature on a specific causation question consistently shows the same strength of association, and dose-response relationship, is very important, *where enough high-quality studies have been done*. However there are many situations, such as that described for the causes of occupational low back pain later in this paper, where only a handful of high-quality studies exist, making this criterion unhelpful.

4. Temporality: This almost self-evident criterion refers to the logical necessity that causes must precede their effects in time. This is not in question in longitudinal "cohort" studies where subjects without the outcome are followed over time to find out who succumbs to the outcome, after measures of the exposure are taken at baseline. However, epidemiologists frequently use other study designs, such as case-control, where questions about exposures may be asked of subjects after some have experienced the outcome in question. In these circumstances it is sometimes not so straightforward to be sure that the disease has not influenced the assessment of exposure. For example, a study comparing the personality types of persons who have recently had a heart attack, due to CHD, to "controls" without CHD would obviously need to ensure that the personality type measurement instrument used (usually a questionnaire) does not give different results in the same person before and after a heart attack, a profound event in one's life with known psychological effects. Otherwise, one is studying the personality-related *effects* rather than *causes* of CHD, leading to a "chicken and egg" problem in interpreting study results.

5. (Biological) Plausibility: This refers to the degree to which the biological laboratory *in vitro* and *in vivo* evidence on the exposure's effects in living systems is compatible with the human health

effects observed in epidemiological studies. For example, we now know that cigarette smoke contains many chemical compounds capable of carcinogenesis — the causing of cancers by cigarette smoke constituents has been observed in a wide range of lab animal models and tissue samples under controlled exposure conditions. Thus it is not surprising to basic biologists that smoking leads to a ten-to-twenty-fold increase in lung cancer risk. However, it is important to note here that the epidemiological evidence of the risk elevation was obtained many years before firm laboratory evidence on the specific biological effects of the smoke components. This was partly because technical methods of demonstrating carcinogenesis in the lab lagged behind our ability to do epidemiological studies. Thus there arise situations, especially with new exposures, e.g. to newly synthesized chemicals, wherein no such laboratory evidence of causal mechanisms is yet available. However this lack of evidence is not to be interpreted as evidence against causation; it can simply be a statement of our biological ignorance at a point in time.

6. Coherence (e.g. with descriptive epidemiological and other knowledge): If an observed association between an exposure and a health outcome is causal, then there should be observable situations where the initial occurrence (or an increase in the frequency/intensity) of the exposure was later followed by an increased incidence of the outcome, and more generally, settings in time and space without the exposure should have less of the outcome in their populations than those with the exposure, *other things being equal*. For example, sharp increases in lung cancer were seen in the Western World about twenty-five to thirty-five years after major increases in population smoking rates. This modern epidemic of lung cancer occurred first for men, and is now past its peak, but is still peaking for women, since they began serious smoking decades later. Note however that this association in time would have been missed by an epidemiologist looking at lung cancer time-trends in, say 1950 — simply because the lag time required for lung cancers to develop after smoking is decades long. Furthermore, simple comparisons of lung cancer rates in global settings with and without much smoking are confused (epidemiologists would say “confounded”) by differences in other exposures between settings, such as occupational and environmental pollutants, radon etc., that are known to cause lung cancer, but may be hard to measure well and thereby “control for” in the comparisons. Thus there are many reasons why existing knowledge of an exposure’s and outcome’s occurrence may not obviously suggest a causal relationship between the two, even though other criteria listed here are met.

7. Specificity (of a given exposure-effect pair): It is truly strong evidence of causation if one finds that a given exposure leads only to one specific health effect, and there are some known examples, such the occurrence of angiosarcoma of the liver after vinyl chloride exposure, and specific clinical infections after adequate exposure to particular pathogens. However, as already suggested above, most human diseases have more than one cause, and most harmful exposures have more than one sort of health effect. For example, many sorts of cancer, CHD, stroke, peripheral vascular disease, emphysema/chronic bronchitis, to name only a few medical conditions, are now known to be at least partly caused by smoking, and yet each of these also has numerous other contributory causes, both genetic and environmental. Indeed, CHD, as we will explore in this paper, now has well over twenty “independent risk factors” associated with its occurrence, most of which meet a number of the causal criteria listed here. Generally only very uncommon exposures and outcomes meet this criterion, which is therefore rarely useful.

8. Analogy (with a known causal disease process): Again, where available, the known causal occurrence of a similar health outcome in humans or animals, after exposure to the same or a similar hazard, is usually considered strong evidence of causation. There are many infectious disease examples of this, such as the similarities between various animal models of viral hepatitis and specific forms of human hepatitis. Unfortunately, good analogies are uncommon for the main health outcomes of importance in our work insurance schemes today.

9. Experiment: This criterion refers to availability of evidence, from well-designed and conducted experimental studies such as randomized control trials, that removal of the exposure reduces the risk of the outcome in question. While undoubtedly the most powerfully convincing criterion of those listed here, this sort of evidence is often simply unethical to obtain (i.e. for exposures already strongly suspected as being harmful, even if all their specific health effects are not fully known) or else infeasible

(e.g. the randomization of humans to chronic unpleasant or demanding exposures thought to possibly have very delayed health effects). Thus, while always desirable in principle, meeting this criterion is often not practicable. As a second-best and often very illuminating alternative, “quasi-experimental” analyses of “natural experiments”, such as tracking the long-term health effects of the introduction of mass-smoking in whole populations, can often approximate this ideal, provided that competing alternative explanations for the resultant observations can be ruled out.

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