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Conceptual and Definitional Issues in Occupational Injury Epidemiology

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This paper presents several models that further define the concept of occupational injury. While traditional models have proved successful in isolating specific research questions and health phenomena, the conceptual model presented permits a broader view of all injury morbidity. This model is based on both the level and frequency of energy transfers. A process model of occupational injury is also presented to describe the basic pathophysiological relationships associated with tissue effects/damage and recovery/repair. Numerous tradeoffs exist in variable selection, and a third model explores some of these tradeoffs. Differences in terminology and fundamental principles can limit the progress of occupational injury research. Accordingly, an argument is made for consolidation and consensus of terms. Finally, considerations for research are suggested, with an emphasis on the severity of the injury, the risk ratio, and the population at risk. Am. J. Ind. Med. 32:106–115, 1997. *© 1997 Wiley-Liss, Inc.*

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INTRODUCTION

The overall aim of research in occupational injury epidemiology is to describe the distribution and determinants of occupational injuries and to make and test inferences about their prevention. During the international workshop ''Methodological Challenges to the Study of Occupational Injury,'' researchers encountered challenges in harmonizing injury models and terminology [Courtney et al., 1997]. These challenges were considered significant, and researchers worked together to develop a new epidemiologic conceptual model for understanding occupational injuries and their circumstances. Additional effort was expended to reach common definitions or sets of definitions. The purpose of this paper is to appraise concepts, definitions, and issues involving approaches to the study of occupational injury. The improved concepts and definitions presented should facilitate the selection of research questions and the design and conduct of studies of the etiology of occupational injury.

TOWARD A COMPREHENSIVE EPIDEMIOLOGIC INJURY MODEL

History and Background of an Epidemiologic Injury Model

Gibson [1961] was the first to conceptualize injury as resulting from the interaction of energy with tissue and that injuries may be best studied according to the types of energy that create them. Haddon [1968] extended the fundamental principle of injury as resulting from the interaction of energy with tissue to the infectious disease elements of host, agent, vector/vehicle and environment. The conceptual breakthrough was in the consideration of energy as the agent and the objects in the environment as the vehicles or vectors. The occurrence of injury

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was related to the quality, quantity, and rate of transmission of the energy and the tolerance of the tissue receiving the energy. Haddon further suggested limiting the definition of ''injury'' to tissue damage manifested within 48 hr (though most often sooner). Haddon's original matrix, in elemental form, presents temporal (phases) and disease factors axes. The temporal phases of injury are described as either pre-event, event, or post-event. The factors are described as host (human), agent (energy), vehicle/vector (machine/animal) and environment (physical and sociocultural). Andersson and Menckel [1995] recently introduced a general model for prevention which distills the elements of 11 major prevention models (including Haddon's). Elements relevant to classification and modeling of injury as a phenomenon include a *trichotomy* of factors (i.e., host, agent/vehicle, and environment), a temporal axis of pre- and post-pathogenic, and the degree of specificity of the morbidity.

Interestingly, these frameworks are often emphasized as preventive (indicating levels and opportunities for intervention or control), although conceptual description of injury is a related use. The agent in injury may be necessary, but not sufficient, for the occurrence of injury [Robertson, 1992]. This concept reprises the issue of multiple causation raised earlier in the twentieth century by Heinrich [1931]. In Heinrich's model, a sequence of events involving potentially independent phenomena had to occur to present the conditions that would result in an accident and potentially in injury. Consider a robotic welder that applies a welding torch to a work product. The intercession of a maintenance person (host) in the work zone, combined with an apparently idle robot with torch (vehicle), and a procedural—perhaps design-driven—error (improper control setting) could place the thermal energy of the torch (agent) in close enough proximity to the host tissues to damage them. While the host and agent are easily identified, the independence of many of the exposures makes it difficult to specify a clear etiology for this injury and its antecedent events.

New Conceptual Model of Energy Transfer and Injury

Traditional models have proved successful in isolating specific research questions and health phenomena. The new conceptual model described below permits a broader view of injury morbidity which may serve these purposes, as well as assist in the assessment of resource and research allocation across the full scope of injury morbidity. In the following discussion, the term *injury* is used broadly to include certain work-related musculoskeletal disorders.

Figure 1 presents the conceptual model for injury caused by energy which illustrates relationships among various exposures and outcomes. Exposure to high-energy transfer with low frequency may result in an injury (e.g., a fracture due to a fall). High-energy transfer combined with high frequency may also result in an injury. The distinction between an *injury* and a *disorder* is often problematic. One solution is to define an outcome as injury based on a specified latency period (e.g., Haddon's 48 hr). If the energy and frequency combination results in damage and symptoms beyond the specified latency period, we then term the outcome a disorder. A special set of exposures associated with disorders are the ones transferring low energy with low frequency (the duration of the energy transfer is very long). An example is a musculoskeletal disorder arising from constrained posture.

The injury condition can present itself in three different modes singly or in combination: morphologic (e.g., an amputation, swelling), physiologic (e.g., impaired vascularization, slowing of nerve conduction), or subjective (e.g., pain or discomfort) or a combination of these modes. The adverse health outcome (the condition) due to energy transfer can be acute and self-limited as, for example, a discrete tendon or laceration strain that heals within a few weeks, but may become chronic and/or persistent if the healing process does not lead to full recovery of the condition (e.g., long-term pain—chronic, an amputation—persistent). Neither the mode of effect (morphologic, physiologic or subjective) nor the duration of effect (acute vs. chronic) can be used as the distinction between an injury and a disorder. In automobile collisions, the injury may be an ''objective'' condition, such as fracture, laceration, or puncture wound. However, a collision may also result in a ''subjective'' condition such as a painful, whiplash injury with no physical findings. The duration of the described injuries may be short (acute-self-limited) or persist for a long time (chronic).

The circumstances of the energy transfer and resulting impact of the injury are influenced by the milieu, which includes psychological, social, economic, cultural, personal, and organizational factors. The energy transfer and the resulting adverse health effect are dependent on all the described factors. This is sometimes referred to as the ''web of causality,'' since an adverse health effect can usually be attributed to a chain of events that are dependent on a great number of individual and environmental factors [Krieger, 1994].

Process Model of Occupational Injury

The concepts presented in Figure 1 can be further elaborated through an underlying pathophysiologic process model presented in Figure 2. This process model is intended to represent the pathophysiological processes associated with tissue effects/damage and recovery/repair.

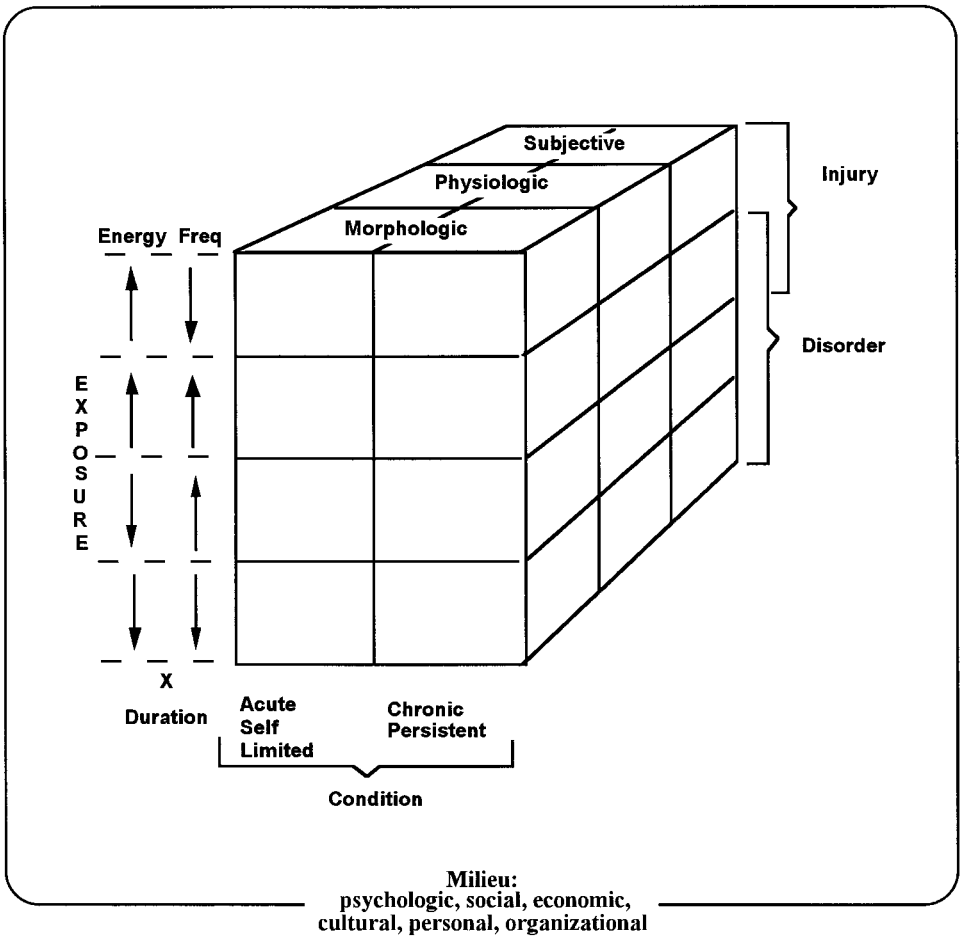

FIGURE 1. Conceptual model for occupational injury caused by energy transfer. See text for explanation.

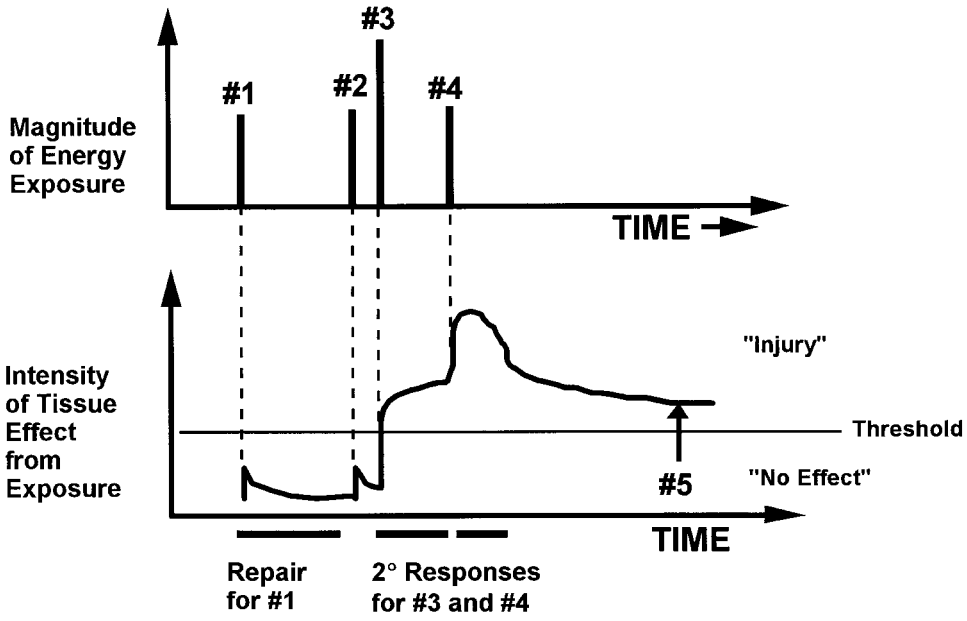

FIGURE 2. Process model for injury resulting from the interaction of forces and energy between the worker and a source/object. See text for explanation.

In the simplest case, there is a single exposure to high-energy mechanical force (e.g., a worker is struck by a forklift truck) and the tissues at the site of impact are damaged (e.g., cells are destroyed, resulting in skin penetrations and structural damage to underlying tissues). If there are no further exposures, the tissue will undergo a series of cellular processes. Initially, there are responses to the damage such as edema, bleeding, and release of cell mediators that recruit white cells and produce inflammation. As this initial response subsides, repair processes rebuild the damaged tissues, producing some residual scar tissue. The amount of damage and repair depends on the forces, their direction, and duration and on the characteristics of the impacted tissues. If the target tissue is affected by preexisting disease or an ongoing damage repair process, it will respond differently from unaffected tissues.

Some effects are not tissue damage; rather, they are intermediate responses to the absorbed energy. With continued exposure, tissue damage may result from an accumulation of the absorbed energy, as in the case of work-related musculoskeletal disorders. The pathophysiologic processes can be very complex in these situations and are not well understood.

A simple example of an effect-recovery process is shown in #1 in Figure 2. The tissue effect from the energy exposure is too small to be detected as an ''injury,'' and there is a simple recovery over a period of time. Where there are repeated exposures close in time (short relative to the recovery time), such as exposures #2–4, there may be interactions between the repair/recovery process and exposures. Early exposures produce no detectable effect, but the early effects interact with the subsequent exposures to produce disproportional responses, secondary responses, and eventually a detectable ''injury'' after exposure #3.

The process relationship shown in Figure 2 has several important features:

1. The magnitude of the tissue effect produced by a given exposure (exposure #1) depends on four basic factors: the magnitude, direction, and duration of the external force and the properties of the target tissues. Repeated exposures can produce very complex interactions because exposures are rarely exact replicates: force directions and durations can change, and tissue properties will change in response to the presence of effects and/or secondary processes, such as inflammation.
2. Effect/recovery processes will resolve the effect over time (exposure #1).
3. Additional exposure in the presence of effects or damage can produce an amplified response (exposures #3 and 4), so the relative timing of additional/repeated exposures to damage responses can be important in determining the eventual magnitude of the response. There can be feedback between exposures and responses. For example, if a severed tendon is exposed to seemingly normal work stresses before the recovery response has progressed sufficiently, the weakened tissue could interact with the stress to produce additional damage and a secondary response.
4. Effects/damage must exceed some threshold to be detectable as injury (exposure #3), and the threshold depends on the method of detection. Insensitive methods may only detect late and more complex results of the damage process.

As noted above, one of the major features of the effect–recovery process model is that the timing of effects and recovery is a critical determinant of the overall level of health impact. As a result, there may be no simple metric to represent the quantitative relationship between exposure over a period of time and the intensity of effects present at the end of that time interval. Exposures at #1, #2, and #4 have the same magnitude but produce different magnitude responses. Therefore, simple dose metrics such as cumulative exposure (sum of exposure magnitudes for #1–4 multiplied by the duration to time point #5) are not proportional to the intensity of effects observed at time point #5. The biological dynamics of each type of exposure–effect relationship need to be determined separately for each type of exposure, such as high-energy single exposure events versus low-energy repeated exposures, and for each type of effect.

These interactions are much more complex and varied than the standard toxicological types of exposures and responses and require appropriately more complex dose metrics. As with toxicological processes, the dose metrics should be based on the mechanism of the response obtained from basic science laboratory studies. For biomechanical interactions, the mechanisms of effect are much less well understood than for toxicological interactions, but a similar conceptual approach would likely produce useful dose metrics.

DEFINITIONAL AND DESIGN CONCERNS

Characteristics and Acceptance of Definitions

Some of the confusion and controversy in occupational injury research may be attributed in part to the lack of clearly defined terms and concepts. Consensus definitions of injury terms and concepts are rare, perhaps due to the traditional epidemiologic focus on chronic and infectious diseases. The wording of those definitions that do exist is often source dependent. The lack of consistent definitions creates a difficult challenge in even defining a research question in occupational injury.

This situation is complicated by variations in administrative definitions used in national reporting systems around the world. These definitions are often

TABLE I. Suggested Definitions of Outcome Terms in Injury Epidemiology

Term	Definition	Source
Injury	Damage to cells and organs from energy exposures that have relatively sudden, discernible effects	Robertsson [1992, p. 10]
Injury	The result of events and behaviors that have environmental, biological, and behavioral determinants that can often be reduced or eliminated	First World Conference on Accident and Injury Prevention [1989]
Occupational injury	Any damage inflicted to the body by energy transfer during work with a short duration between exposure and the health event (usually $<$48 hr)[a]	Present paper
Acute injury	All injuries are acute in the beginning—if they do not heal, they become chronic	Present paper
Chronic injury	A chronic condition, in a medical context, persists for a long time; time limit for a chronic condition after an injury varies between the type of injury and also the investigator—typical time limits used for chronic injuries: 3 or 6 mo	Present paper
Disease	Observable impairment in body configuration and function; an entity characterized by at least two of the following three criteria: recognized etiologic agent(s), an identifiable group of signs and symptoms, consistent anatomical alterations	Steadman [1976]
Occupational disease	In an occupational disease, there is a direct specific cause–effect relationship (e.g., exposure to asbestos—asbestosis)	WHO [1985]
Work-related disease	Work-related diseases are as multifactorial where the work environment and the performance of work contribute significantly, but as only one type of factors of a number of different types of factors to the causation of disease	WHO [1985]
Illness	The perception of being sick—the perception can be of abnormal function or in symptoms	Steadman [1976]
Disorder	A disturbance of function or structure, or both, that may be due to inborn error, development errors, exogenous factors, or disease	Steadman [1976]
Accident	An (unintentional) event that results, or could result, in an injury	First World Conference on Accident and Injury Prevention [1989]
Cumulative injury	An increase in severity of an injury due to new energy transfer	Present paper
Recurrent injury	A repeated injury after the first injury has healed	Present paper

[a]Viewed as the preferred term by the present authors when more than one definition is presented.

developed for classification in social insurance systems, regulatory systems or health care systems. These original premises can sometimes compete with or complicate surveillance and research uses of these data. For example, a country may experience a change in injury rates of 200% or more in a given year [National Safety Council, 1995], but this could have more to do with artifacts than with injuries, as reporting systems and registries open, close, and are redefined by their various societies. Similarly, some countries report ''injury'' statistics which include diseases, while others omit diseases. Even within the United States, definitions of injury vary between registers [Rubens, 1995]. Furthermore, differences often exist between the scientific and lay concept of ''injury'' and related terms. For example, the terms ''injury'' and ''accident'' may be considered synonymous by the layperson but distinctly unique by the epidemiologist (Table I).

Another problem with the terms defining exposures and outcomes is that they are dependent on the method used to detect the problem. Sensitive methods may detect subtle exposures and primary stages in the damage process. Less sensitive methods will require a greater manifestation of damage resulting from secondary rather than primary tissue responses. There is also the problem of using exposure-related definitions of outcomes (e.g., cumulative trauma disorder, repetitive strain injury, and accident). A sharper distinction between exposure and outcome would help, as would sharper definitions of both exposure (energy,

time course, repetition) and pathology (tissue damage, time course relative to exposure, primary vs. secondary responses).

In this document, priority has been given to terms that have been defined authoritatively in international practice standards, recognized texts, and/or published scientific studies. In addition, the authors have modified several of these terms to better harmonize them with the model presented in Figures 1 and 2. Scientific reports should include as a standard practice an explicit statement of definitions and presentation of the rationale for newly developed definitions.

Outcome Terms and Issues

Occupational injury.

Occupational injury is herein defined as ''. . . any damage inflicted to the body by energy transfer during work . . . .'' (Table I). In the new conceptual model, the damage should be caused by energy transfer and involve a short duration between exposure and the health event (usually less than 48 hr). Examples are a fracture after a fall or a burn after an explosion. In registries, the definition of occupational injuries varies. Usually, restrictions are made on duration of restricted activity and medical attention caused by the damage [Rubens et al., 1995]. The term *injury* should be distinguished from the term *accident*. *Accident* was defined by the First World Conference on Accident and Injury Prevention [1989] as an (unintentional) event that results or could result in an injury. *Injury* was defined by the same conference as ''the result of events and behaviors that have environmental, biological, and behavioral determinants that can often be reduced or eliminated'' [First World Conference on Accident and Injury Prevention, 1989].

Injury epidemiology outcomes are often characterized two-dimensionally. One dimension concerns the nature of body damage (e.g., pain, burn, fracture). The other dimension concerns the nature of the event or occurrence (e.g., slip, car crash) that resulted in the bodily damage. This may reflect a need to reduce a general set of outcomes to a more specific common subset. For example *fractures* can be reduced to *fractures resulting from slips and falls*. However, it is important to stress that this approach does not *per se* reveal the exposures for the injury. In the example, the exposures for the outcome class (fracture) and the event class (slips and falls) are still unknown though the outcome class has become more specific. An interpretive problem may occur, as discussed subsequently, when the outcome information is generalized while the event information remains specific (e.g., slip and fall injury, repetitive motion disorder). These types of descriptions may unintentionally overextend the existing etiologic certainty by suggesting an exclusive causal relationship.

The terms *acute injury* and *chronic injury* are confused by application to both the injury process and the injury condition. A chronic condition in a medical context persists for a long time. For example, a single exposure to a respiratory agent might produce acute bronchitis that persists and becomes chronic. An acute overexertion might lead to acute low back pain that may persist and become chronic. The time limit for a chronic condition after an injury varies depending on the type of injury and the investigator. Back injuries with pain lasting more than 90 days are sometimes regarded as chronic [Fordyce, 1995]. Bonica [1990] defines chronic pain as ''pain that persists a month beyond a reasonable time for an injury to heal.'' An international task force on taxonomy for classification of chronic pain stated that ''chronic pain is recognized when the process of repair is apparently ended'' [Merskey and Bogduk, 1994]. The task force also stated that a period of 3 months is often a chronologically convenient point of division between acute and chronic [Merskey and Bogduk, 1994].

It is likely that unless resolved, this taxonomic ambiguity will lead to analytic errors in future epidemiologic research. We recommend that the terms *acute* and *chronic* refer to the outcome rather than the exposure. *Acute* or *chronic injury* would therefore refer to the persistence of the damage or symptoms. Short duration, high-intensity exposures could be referred to as discrete or single exposures, rather than acute exposures.

Injury vs. cumulative trauma disorders.

In recent years, the use of terms such as cumulative trauma disorder (CTD) and repetitive strain injury (RSI) has been strongly criticized [Hadler 1990]. This critique is partly due to poor terminology and the lack of a conceptual model. Low-energy transfers may cause tissue effects that are subclinical, i.e., not evident to the person affected (#1 and #2 in Fig. 2). Incomplete recovery may allow similar energy transfers to accumulate to a clinical effect. These energy transfers may last months or years before an effect becomes evident. The specificity in effect and in exposure of low-energy transfers is low. For example, carpal tunnel syndrome (CTS) may be related to highly repetitive wrist motions but also to hormonal factors [Hagberg et al., 1992]. Furthermore, highly repetitive wrist motions may relate to both tendinitis and CTS [Hagberg, 1996]. Thus, terming a condition RSI or CTD could be misleading, since other occupational and non-occupational factors than repetitive strain and cumulative trauma may have contributed to the disorder. Instead, the correct medical diagnostic term (in medical or lay language) is preferred. The term ''work-related'' can be used to address associations to work. This term specifies neither a causal mechanism nor a single etiological factor (Table I).

TABLE II. Suggested Definitions of Exposure Terms in Injury Epidemiology

Term	Definition	Source
Exposure	Proximity and/or contact with a source of a disease agent in such a manner that effective transmission of the agent or harmful effects of the agent may occur	Last [1995]
Exposure	Any of a subject's attributes or any agent with which he or she may come in contact that may be relevant to his or her health	Armstrong [1992]
Exposure	Presence of a factor (substance) in the environment external to the worker[a]	Checkoway et al. [1989]
Cumulative exposure	Summation of intensity over time	Checkoway et al. [1989]
Burden	Amount of a factor (substance) that exists in the body (organ) at a point in time	Checkoway et al. [1989]
Dose	Amount of a factor (substance) that remains at the biological target at some specified time interval	Checkoway et al. [1989]

[a]Viewed as the preferred term by the present authors when more than one definition is presented.

The new model (Figs. 1, 2) is consistent with a ''cascade'' model for work-related musculoskeletal disorders presented by a multinational scientific group [Armstrong et al., 1993]. This model considered a cascade of responses to exposure and dose that may be immediate but in which the clinical presentation of disease may take weeks or months. It is recognized that there are also health promoting energy transfers. A basic principle in rehabilitation is to use physical training to improve function and capacity.

Exposure Terms and Issues

Exposure is usually defined as the ''presence of a substance (factor) in the environment external to the worker'' [Checkoway et al., 1989](Table II). *Dose* is defined as the ''amount of a factor (substance) that remains at the biological target for some specified time interval''[Checkoway et al., 1989]. This concept would also fit traumatic injury epidemiology. Energy transfers such as force and heat can be quantified in the body and regarded as dose. Exposure needs to include dose metrics for reversible damage processes. Energy delivered over a long period may not produce the same effects. Exposure must be measured in a time period relevant to the outcome and recovery.

In injury epidemiology, the concept of *exposure* is often unclear. In discrete traumatic injury, a sudden unforeseen event causing the injury may be treated as the outcome or as the exposure. In addition, the *exposure of interest* may not be the direct factor causing the injury (e.g., overexertion) but the chain of events necessary for the unforeseen sudden event to occur. Macroenvironmental factors such as organizational design and dynamics and the organizational creation and deletion of controls and control practices may combine to render a given sequence of events hazardous [Backström, 1996]. To assess exposure–response in discrete traumatic injury epidemiology, individual characteristics, the immediate chain of events and these other factors have to be considered as determinants. In analytical studies of discrete traumatic injuries, there has been little consensus and few (if any) successful examples of how to model interaction of determinants in an exposure–response model.

Exposure-based studies may be categorized into two classes: those that focus on hazard surveillance, and those that involve exposure assessment linked to outcome. Hazard surveillance is based on the principle that the collection of information on the distribution of hazards is useful for prevention. Information on the distribution of hazard may be useful for prioritizing intervention, targeting intervention, assessing the efficacy of efforts to control hazards, etc. A basic example of hazard surveillance is the evaluation of worksites by a potential insurer in order to assess potential risks. Where exposure assessment is linked to outcome (adverse effect), the direction of the study can be prospective in which a population exposed to a hazard is assessed for the occurrence of an adverse effect. Alternatively, the direction can be retrospective with the presence of the hazard evaluated among cases and controls. For example, the contribution of physical factors such as road conditions could be evaluated for vehicle crashes by assessing the road conditions a set distance in the direction from which the motor vehicle came [Haddon, 1968].

Latency is the delay between first exposure to a disease-causing agent and the manifestation of the disease. Other epidemiologists, [Rothman and Greenland, 1997] have defined *induction period* as the duration between first exposure and onset of disease, whereas the latency period represents the duration between onset of the disease and the manifestation of the disease. For acute injuries, the latency between the injury event and the injury is usually short and approaches zero; thus, the induction period is usually also short. Consideration of latency is therefore not common in

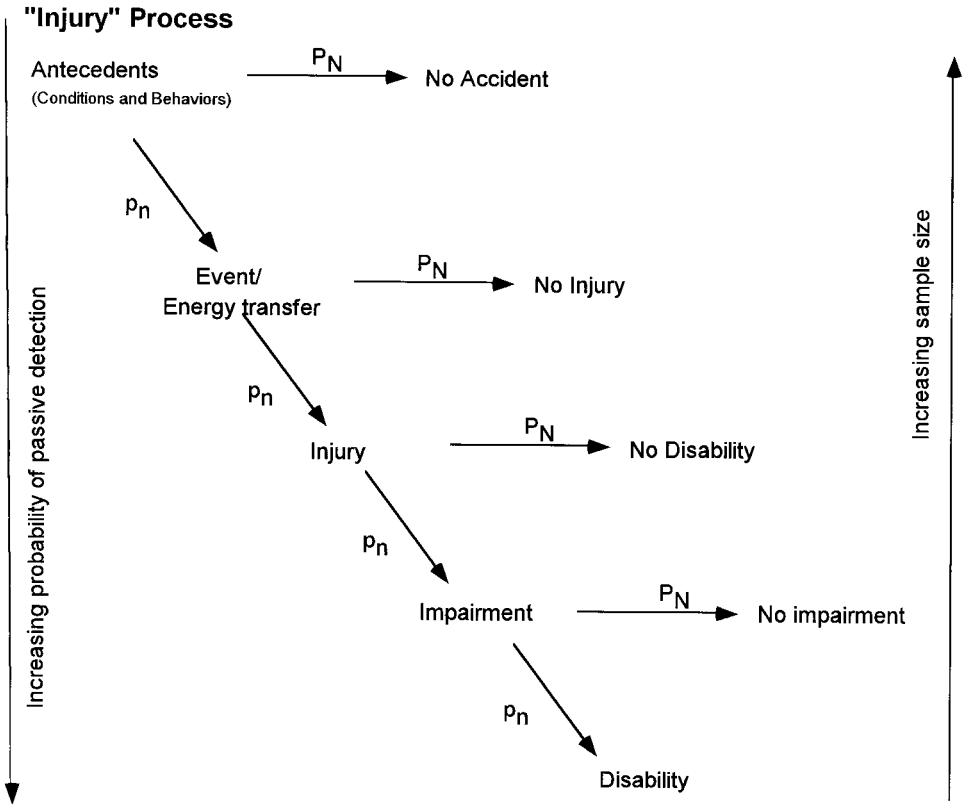

FIGURE 3. Simple model of tradeoffs in variable selection. The expectation is that $p_n \ll P_N$ for each fork in this decision tree.

epidemiologic studies of acute injuries. However, the concept of latency time needs to be further explored in injury epidemiology. In the process model (Fig. 2), it is evident that energy transfers not causing an injury may precede the actual injury causing energy transfer.

Whether the *latency of the sequence of events* before the injury event in discrete traumatic injury research should be considered is unknown [Kjellén and Larson, 1981]. Furthermore, in a system-analysis approach in studying discrete traumatic injuries, one might use the ''latency'' for an injury as the outcome and model the dependency on factors present in the work system [Backström, 1996] in the individual. The latency concept needs further consideration and investigation in injury research.

Misclassification may tend to operate the same way for injuries as for diseases. Nondifferential misclassification will tend to underestimate the effect and differential misclassification may over- or underestimate the risk factor of interest.

The healthy worker effect [Checkoway et al., 1989; Monson, 1990] should also be considered when studying injuries. Hence, cohort studies should consider injury experience for those who leave the job [Sorock and Courtney, 1996]. The healthy worker effect may operate differently for injuries than for chronic diseases. To date, there are no studies of the healthy worker effect for discrete traumatic injuries in the workplace. Moreover, there may be individual susceptibility attributes, whether genetic or acquired, which impact injury risk. Host characteristics, to the extent measurable, are potential risk factors and may constitute confounders or effect modifiers in analytical studies. Finally, risk perception may vary between individuals and may itself be a determinant for injury.

BROADER INJURY RESEARCH CONSIDERATIONS

The selection of the variables for an occupational injury study, intentionally or unintentionally, is influenced by the nature of the incident–injury–impairment–disability process. Assessment at each level in the process has different ramifications for study requirements and is influenced by prevailing scientific, political, and economic concerns.

The continuum of potential variables may include antecedent conditions, exposures, established risk factors, and the actual injuries experienced. This continuum may be characterized by decreasing n (number of events or occurrences) with an increasing specificity of findings. The potential increase in sample size may be visualized from Figure 3.

Moving downward in the model would increase the probability that the particular level is documented in some

passive data system (e.g., injury registry), with disabling injuries having the greatest likelihood, while moving upward would increase the potential sample size and decrease the probability of reporting. Thus, an unusual competition is established between sensitivity, specificity, power, and availability of data.

Influences which may result in the underreporting of injuries that have been identified in some studies of American recording practices [Olenick et al. 1993], including a variety of work organization, psychological, social, and socioeconomic factors. Cultural practices may play a part. For example, the societal concept of guilt or shame may influence individual reporting behaviors differently between cultures [Wokutch, 1992].

The disability that may result from an injury depends to an extent on the socioeconomic conditions of the individual as well as her or his specific job demands. For example, a low back injury sustained from a non-task-related slip or trip is likely to have a different disability potential for an office worker, than a similar injury sustained by a person involved in continuous manual materials handling. For the former, the level of impairment may not necessarily be disabling, while for the second it well may be.

The occurrence of an injury, as well as the severity or extent of the physical damage, appears to be largely a stochastic process, involving the conditions, locations, activities, and other factors, of the individual at the point of incident. The incident occurrence itself is frequently a rare combination of a subset of intrinsic behaviors and extrinsic conditions that, separately, can be universally found in workplaces.

Thus, the tradeoff appears to be between large samples and associated, relatively low specificity data and small samples associated with much more specific data. For example, a good accident report, although not widely available, will enable many detailed antecedent factors to be evaluated, together with the unique characteristics of the accident situation. Potentially, even the post-event factors relevant to the injury and of the progression of that injury into a disability may be established.

FACTORS INFLUENCING RESEARCH PRIORITIES IN OCCUPATIONAL INJURIES

Many factors may influence the setting of research priorities. The full scope of the problem considers the severity, the incidence rate, the risk ratio, and the population at risk. Occupational injury fatalities have a high research priority because of the loss of life and associated individual and societal costs. Even low risk ratio situations should result in a search for interventions to minimize occupational injury fatalities. Musculoskeletal injuries may not be as severe to individual health or have high risk ratios, but the population at risk is large and the excess number affected may still result in substantial costs as above. Persistent problems that are expected to remain should be given research priority.

Yet another consideration is whether the problem is preventable. In occupational injury, past experience shows clearly that many risks are preventable. Risk communication is another priority. If knowledge of the risks exists, then dissemination of prevention information may improve both organizational and individual decision-making in hazardous situations. It is also possible that more research is needed on risk perception. Generally, better research into the factors influencing the initiation and implementation of prevention efforts will increase the impact of injury research. Economic resources and availability of competent practitioners are also factors that should be reviewed when setting research priorities.

The relationship between any population of workers and their environment is dynamic, changing over time. Exposures vary over time, the workplace physical setting varies over time, the workforce ages, individuals alter personal habits, and individuals develop concomitant medical conditions that may alter their injury risk. Thus, it is important to prioritize studies trying to assess this complex mix of variables in an attempt to develop a new understanding of the effects of workplace conditions on injury (acute and chronic), disability, and death. The analytic methods used to assess epidemiologic data have become more sophisticated over the past two decades as the focus of occupational epidemiology has shifted to the detection of early health effects associated with various kinds of exposures. Innovations in computer technology have removed barriers to analyzing large data sets from population studies. However, there is a need for innovation in study design with regard to injury epidemiology. Such innovation may come from disciplines not traditionally associated with medicine or public health. For example, research methods from management sciences (process management or systems management) considering both management and work organization, from econometrics, or engineering may be applicable in collecting and analyzing data. Such alternative approaches should be explored.

CONCLUSION

The study of occupational injury and related morbidity is a challenging field in which many of traditional assumptions and definitions used in disease epidemiology are being tested. The models presented herein have attempted to address several dimensions of the problem conceptually while providing insight into problem definition and study design concerns. By suggesting a more specific terminology, the present paper has argued for increased precision in study design, execution, and reporting, which should increase our ability to apply meta-analytic techniques across a diverse

spectrum of morbidity. Given the multifactorial nature of injury phenomena, it may benefit researchers to expand the array of techniques and skills applied in injury research by adopting methods from allied fields. Ultimately, the way in which injuries are viewed and described will influence thinking about injury causation and the types of problems selected for study.

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