OPINION ARTICLE

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Determination of causal associations in occupational medicine and the medico-legal context: references and standards

Determinação de nexo causal na medicina do trabalho e na perícia judicial: referências e critérios

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ABSTRACT | Causality is a transdisciplinary topic with the medical-legal field representing one of its most exciting aspects. Since medicine and law have different roots and objectives, this article provides references to support occupational physicians and medico-legal experts in the difficult task of establishing occupational causation. In addition to the traditional Bradford Hill criteria and Schilling's Classification, additional standards are provided to enhance critical assessment and contribute to the responsible use of the concept of causation in both the legal and medical-occupational fields.
Keywords | causal nexus; work-relatedness; causation; occupational medicine; medico-legal expertise.

RESUMO | Causalidade é um tema transdisciplinar, sendo a interface médico-legal uma de suas vertentes mais instigantes. Levando em consideração que a medicina e o direto possuem raízes e objetivos distintos, este artigo traz referências para auxiliar o médico do trabalho e o perito médico na árdua tarefa de caracterização do nexo causal ocupacional. Além dos clássicos postulados de Bradford Hill e da Classificação de Schilling, são apresentados outros norteadores capazes de aprimorar o pensamento crítico e permitir o uso responsável do conceito de concausalidade tanto no universo jurídico quanto no ambiente médico ocupacional.
Palavras-chave | nexo causal; nexo ocupacional; causalidade; medicina do trabalho; perícia médica.

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INTRODUCTION

The search for a reason for facts and events is an integral part of the human journey. Etiology is a major branch of philosophy, as classically expressed in the work of Aristotle, including his theory of the four causes.[1] The scope of this article is to analyze medical causation/causality from both technical and occupational perspectives. Our goal is to provide a variety of references on which to base the definition of a causal relationship, especially in individual situations, without delving into metaphysical or epistemological questions.

This issue is embedded in the medico-legal field. On the one hand, medicine is rooted in science, based on observation and experimentation, and concerned with multifactorial conditions, which are better explained by probabilistic models than attributable to a single definite cause. On the other hand, the law seeks to achieve justice by establishing a causal link between illicit acts and harm, in an attempt to establish legal accountability.[2,3] Additionally, medicine and law do not base their decisions on the same criteria: what is considered acceptable evidence for medicine may be unacceptable in law, and vice-versa. This discrepancy can have a significant impact on litigation, leading to the dissatisfaction of the parties involved, uncertainty for judges, and conflicts with medico-legal experts.

As noted by the authors of the well-known *AMA Guides to the Evaluation of Disease and Injury Causation*[3]:

> The courts did not have their origins in science, and, therefore, the laws developed are historically—not scientifically—derived. Judges and legislatures have the power to substitute convenience for science. [...] One common method for doing so in workers' compensation cases is the establishment, by legislative or judicial decree, of presumptions that institutionalize societal choices.

In other words, even without a solid medical basis, laws and precedents may support the presumption of work-relatedness, thereby influencing rights and accountability. Regardless of legal aspects, medical accountability should be established with the consistency of the scientific method of causation. This article provides a non-exhaustive contribution to this process.[4,5]

THEORETICAL BACKGROUND

ADEQUATE CAUSATION THEORY

From a legal perspective, it is important to highlight the theory of adequate causation,[6] developed by the German physiologist Von Kries. It is often used in legal settings but also applies to the medical field.

An exclusive causal relationship is not required to establish causality: the presence of concurrent, simultaneous, or successive causes does not diminish their individual causal effects. For instance, individual pathological predispositions do not restrict the right to reparations as long as there is evidence to support the role of an occupational agent as a relevant contributing cause.

The theory of adequate causation excludes the possibility of causality in the case of deviant, extraordinary, or unpredictable events. It relies on the concept of predictability (that is, statistical regularity) to determine whether a factor constitutes a feasible, suitable, or most likely cause of harm. This doctrine is based on philosophical and legal aspects of probabilistic causality.[6] As such, in a concrete case, it is not sufficient for an act to be a *sine qua non* condition for an outcome: it must also be a theoretically plausible cause.[7]

BRADFORD-HILL POSTULATES

Austin Bradford Hill was a world-renowned British epidemiologist known especially for his paradigm-shifting collaboration with Richard Doll, in which the authors proved that smoking caused cancer and other severe diseases.[8] Hill was also a pioneer in the field of randomized clinical trials.

In 1965, under the influence of 18th (David Hume) and 19th (Stuart Mill) century British philosophers, Sir Bradford Hill published one of the most cited articles in the history of science. His greatest achievement was the establishment of standards for the interpretation of statistical findings that reduced doubts regarding causality in the biomedical field[9]: the higher the number of criteria met, the greater the likelihood of a causal association.

- Strength of the association: usually evaluated using a measure of association, such as relative risk or odds ratio;
- Consistency: consonance with the results of other studies;
- Specificity: the illness is caused by a specific exposure;
- Temporality: the cause precedes the outcome (illness);
- Biological gradient: dose-response or proportional effects;
- Biological plausibility: the association is supported by a plausible explanation based on current knowledge of physiopathology;
- Coherence: the findings adhere to current scientific standards;
- Experimental evidence: experimental evidence of an increased frequency of events;
- Analogy: findings based on other illnesses or exposures with similar characteristics.

The Bradford Hill criteria help determine causality in the biomedical field, with applications reaching far beyond the issue of occupational harm.

INUS (INSUFFICIENT BUT NON-REDUNDANT PART OF AN UNNECESSARY BUT SUFFICIENT) CONDITION

INUS conditions represent a sophisticated evolution of the traditional concept of a necessary and/or sufficient cause. The creator of this model was John L. Mackie (1974), an Oxford-based Australian philosopher, who, unlike Bradford Hill, also contemplated weak, dyssynergic, or unknown causal links.[10] This is an elegant construct, though it is little known in the medical-legal field.

In this framework, the cause of an illness can be defined as an event/condition/attribute that precedes and is necessary for the illness to occur, all other things being equal. If this event/condition/attribute differed in a particular characteristic, the illness would be milder, delayed, or absent altogether.[11] This model does not require etiological exclusivity or direct/immediate causality. It admits the occurrence of other events, contemporaneously or otherwise, as in the case of indirect causality, as long as the conditional etiological factor elicits the event that is directly responsible for the harm inflicted.[7]

INUS stands for an insufficient but non-redundant part of an unnecessary but sufficient condition, that is, an insufficient but necessary part of a set that, in turn, is unnecessary but sufficient for the occurrence of an outcome. This mechanism can be illustrated using the classical example described by Mackie and cited by Araújo, Dalgalarrondo, and Banzato[9]:

> Experts agree that the fire that partially destroyed a house was caused by a short-circuit. The short-circuit alone was not necessary or sufficient to cause the fire. It was unnecessary because the fire could have occurred for a different reason, such as a short-circuit somewhere else or intentional arson, etc. It was not sufficient because in the absence of oxygen or the presence of an efficient sprinkler system, the fire would not have occurred. Therefore, the short-circuit (heretofore referred to as A) was a necessary condition of the ABc set, where B represents positive factors such as the presence of oxygen and c represents negative factors such as the presence of a sprinkler. As such, if A is a necessary part of a minimally sufficient condition (ABc), then A is an INUS condition.
>
> The approach proposed by Mackie can address both individual cases and 'strong' causes as well as population-level phenomena and 'weak' causes. The latter are likely to be associated with several minimally sufficient conditions (ABc, DEf, GHi, etc.) including many INUS conditions (A, D, and H, for instance). In these cases, a probabilistic model can be used to try to determine the strength of the association between the INUS conditions and the outcome of interest. [...] Lastly, the analysis of INUS conditions does not preclude the subsequent use of probabilistic techniques to determine the influence of each individual factor on the outcome of interest.

This basic intuition is shared by researchers across several disciplines. Lawyers also discuss the concept of a NESS, which stands for a "necessary element of a sufficient set," and constitutes a more straightforward reformulation of Mackie's INUS condition. In epidemiology, Rothman (1976) proposed a similar approach (sufficient-component cause model/causal pie model)[12] to that of Mackie, which can be illustrated using a pie chart (Figure 1).

Causes in medicine are usually insufficient components of sufficient causal sets. For example, exposure to Koch's bacillus may not necessarily lead to tuberculosis. Additional elements such as malnutrition or immunodeficiency must be present to create minimally sufficient conditions where exposure to the microorganism can play its necessary role (since tuberculosis, by definition, involves infection by the bacillus).[13] The model also includes protective elements: if the subject is well-nourished, the absence of minimally sufficient conditions may prevent the outcome (i.e., tuberculosis), prolong the latency period, or diminish the severity of illness.[7] Additionally, several psychiatric illnesses are compatible with the INUS model, such as schizophrenia[14] and bipolar disorder[15] in the presence of cannabis use; and depression and post-traumatic stress disorder after domestic violence.[16]

In the realm of occupational illnesses, asbestosis is often used as an example. However, work-relatedness may not be as clear in the case of an inveterate smoker with lung cancer and a remote history of occupational exposure to asbestos. Nevertheless, according to the INUS and Mackie models as well as Rothman's causal pie, these factors would be considered (contributing) causes of lung cancer, even in cases involving no exposure to smoking or asbestos.

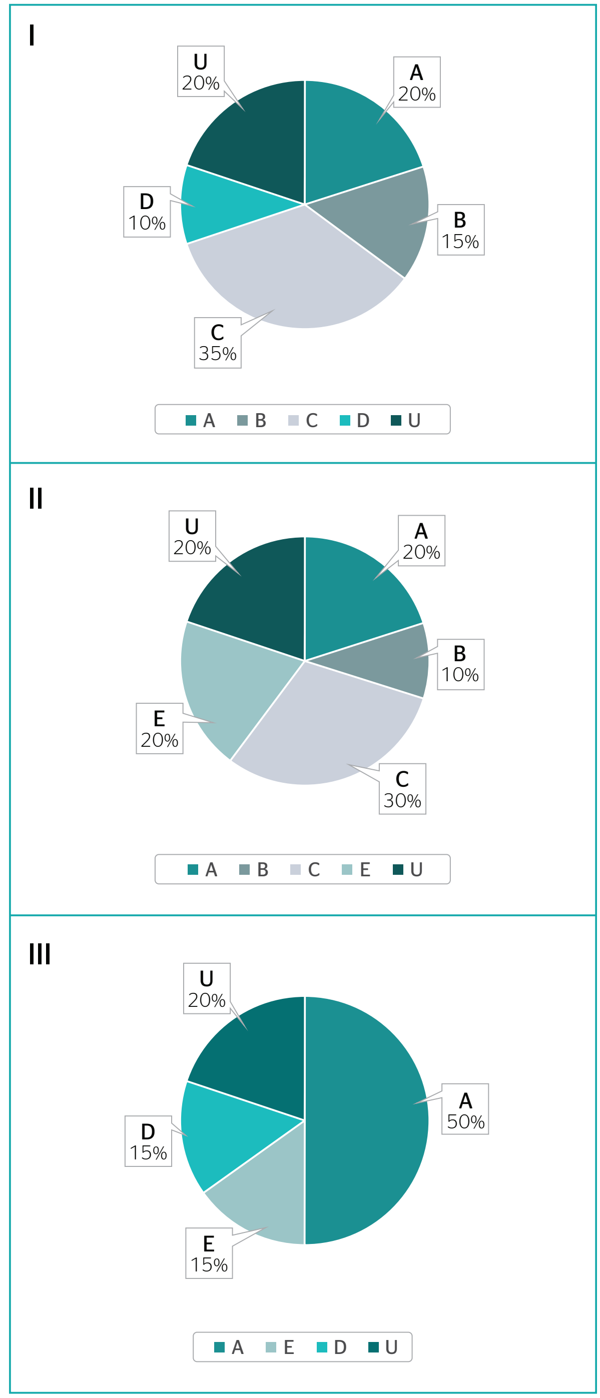

Figure 1. I, II, and III are sufficient sets of conditions for an illness to occur. A, B, C, D, E, and U are insufficient to induce the disease, and U represents unspecified or unknown elements. Therefore, according to the INUS model, A, B, C, and D can be defined as causes. If no other sets/combinations of conditions are sufficient to induce the illness, A is also considered a necessary cause in the traditional sense of the term, as it is always present. Adapted from Rothman et al.[11]

SIMONIN AND FRANCHINI CRITERIA

In France, Simonin (1960) revised and published the criteria developed by Muller and Cordonier decades prior.[3] Long afterward, Franchini (1984), a major figure in Italian legal medicine, issued similar criteria to those of Simonin, with the additional advantage of using more intuitive terms. Another strength of this theory was his reference to the importance of an epidemiological perspective. Franchini's criteria[18] are as follows:

- Chronological criterion: temporal order of alleged cause and effect; keeping in mind that the former must precede the latter and that the process must evolve chronologically.
- Topographical criterion: rational compatibility between the site of damage and the lesion, which does not necessarily imply anatomical coincidence (given the possibility of distant lesions, for instance).
- Lesion adequacy criterion: correspondence of the damaged structure to the alleged causative agent, including the lesion mechanism and the instrument or method that produced it.
- Phenomenological continuity criterion: the physiopathological course is consistent with a causal chain, as defined by Simonin's anatomoclinical correlation criterion and supplementary to the chronological criterion[7];
- Exclusion of other causes: differential diagnosis and knowledge of the individual's prior history;
- Epidemiological or statistical criterion: relationship between the frequency/incidence of damage and its potential cause provides evidence of association or dissociation.

Unlike the Bradford Hill criteria, Simonin and Franchini's standards were developed for the medico-legal field. Nevertheless, though they are based on the assumption of the trauma agent as the standard cause, these standards are suitable for general application.

SCHILLING CLASSIFICATION

The contributions of Richard Schilling mark a defining moment in the discussion of the relationship between illness and work. The comprehensibility of his ideas facilitated their dissemination beyond the biomedical field.

Schilling's original classification (1984) is composed of three groups[18]

- Group I: illnesses where work is a necessary cause, such as occupational accidents and legally recognized occupational diseases (such as lead poisoning and silicosis);
- Group II: illnesses where work is a contributing factor (for example, repetitive strain injury [RSI]/work-related musculoskeletal disorders WMSD; and
- Group III: illnesses aggravated by work or cases where work provokes a latent disorder (for example, asthma or allergic dermatitis).

The fourth category, though less well-known, is worthy of mention. It was introduced by the author in 1989 and encompasses illnesses where work offers easy access to potential dangers (for example, liver cirrhosis in bartenders or innkeepers; suicide in medical laboratory workers, intensive care physicians, and anesthesiologists).[19]

This is, in effect, an extension of the division described by Bernardino Ramazzini, father of occupational medicine, in 1700: professional diseases (technopathies) caused by factors inherent to occupational activities; and work-related illnesses (mesopathies), provoked by the circumstances of work. This perspective is related to the current concept of multifactorial pathogenesis, as it does not neglect the role of social determinants of health and disease, but still allows for a view of work as a suitable setting for health promotion and protection. Schilling's categories were developed based on social hygiene, and are well-suited to a public health framework.

On the other hand, in individual case analysis, Schilling's Classification must be complemented by other criteria to ensure that the causal role of work is adequately specified. Work must play a substantial role in inducing the illness; in other words, if employed in a different occupation, the individual would have likely had a less intense illness or never developed it at all based on all available knowledge of the condition and their preexisting deficiencies.[20]

NIOSH RECOMMENDATIONS

According to the United States National Institute for Occupational Safety and Health (NIOSH), the extent to which a disease is work-related can be determined based on the following six-step process[4]:

- Correct diagnosis of the disease - disease conceptualization; diagnostic criteria and evidence in the case at hand;

- Collection of epidemiological evidence - availability of relevant epidemiological data to support the relationship between the disease and work;
- Characterization of individual exposure - objective evidence that the exposure is consistent with an occupational cause based on frequency, intensity, duration, and temporal pattern;
- Consideration of other relevant factors - observing the presence of other etiological factors (comorbidities, habits, addictions, etc.);
- Assessing and reviewing validity - ensuring that information collected in previous steps, including sources, opinions, and expert testimony, is reliable and credible;
- Drawing conclusions - synthesizing information across the five previous steps.

These steps provide a basic framework for analysis, though the evaluation of the evidence requires additional discipline on the part of the investigator.

INSS/DC RESOLUTION NO. 10/1999

Published over 20 years ago, the INSS/DC Resolution No. 10/1999[21] summarizes the work of Hill, Simonin, and Schilling, in addition to the well-known CFM Resolution No. 1488/98[22] (now superseded by Resolution No. 2183/2018[23]). Item IV, titled "Medical procedures to determine a causal relationship," recommends that medical procedures and processes include answers to the following questions:

- Nature of the exposure: can the 'pathogenic agent' be clearly identified based on the individual's occupational history and/or information collected at their workplace and/or from reputable sources familiar with the work environment of the Insured?
- 'Specificity' of the causal relation and 'strength' of the causal association: could the 'pathogenic agent' or the 'risk factor' have a significant influence on the causes of the disease?
- Type of causal relationship to work: is work a necessary cause (Typo I)? A contributing risk factor to a multicausal illness (Type II)? A trigger or aggravator of a preexisting illness (Type III)?
- In the case of Type II work-related illnesses, were other (non-occupational) causes fully analyzed and, in the case at hand, either excluded or judged less relevant than occupational causes?
- Degree or intensity of exposure: is it consistent with the development of the disease?
- Duration of exposure: was it sufficient to induce the disease?
- Latency of exposure: was it sufficient for the disease to develop and manifest?
- Is there a record of the "prior status" of the insured worker?
- Does knowledge of the "prior status" contribute to the establishment of a causal link between the "current status" and work?
- Is there other epidemiological evidence to support the hypothesis of a causal relationship between the illness and the current or previous occupation of the insured?

If most of these questions are answered in the affirmative, technical analysis is likely to support a causal relationship between the disease and work.

DISCUSSION

This article provides some instruments that may be used by investigators, namely technical experts or occupational physicians, to reach solid conclusions. These references should be used together since each offers a different perspective on the issue of causality, even though some overlap is present.

Unlike a traumatic injury, some illnesses may not show a clear cause-and-effect relationship with a particular exposure. Most occupational diseases are insidious, and their symptoms are confused or intertwined with the effects of aging or other significant non-occupational factors. Information on previous occupational exposures, if available, is often inadequate or incomplete. Additionally, individual susceptibility to particular exposures can influence causal judgments, and occupational exposures can be either a primary or contributing cause.

It is important to consider epidemiological evidence, even though most studies on the relationship of work to non-acute injuries are of unsatisfactory quality. With this in mind, we return to the original question posed by Bradford Hill: "is there any other way to explain the set of facts before us; is there an equally or more likely explanation of cause and effect?" In other words, it is crucial to define a statistical threshold for the acceptance of a reasonable association between work and illness. However, on this point, the collective and individual viewpoints differ considerably.

For illustrative purposes, suppose a study had revealed a measure of association (relative risk [RR] or odds ratio [OR]) of 1.10, with a 95% confidence interval of 1.06–1.17, and $p < 0.05$. Similar results have been observed in other high-quality studies. Based on statistics alone, this would seem to indicate a significant risk factor. However, it is important to note that for every 110 cases of the illness, 100 are accounted for by the general population incidence, with occupational exposure only playing a role in the remaining 10; in other words, the odds of an employee developing the illness as a result of his work are 10/110, or only 9%.

From a public health standpoint, this finding may lead to the adoption of population-wide preventive measures, but when analyzing a particular case, it seems illogical to classify a condition as work-related based on these 9%, which is far from a "reasonable probability." Normally, this would correspond to a cutoff point of at least 50%, which would indicate that the association is "more likely than not." The corresponding RR or OR would then have to be equal to or greater than 2, as required by most courts in the United States.[2]

In fact, the lack of a cutoff point represents a serious inadequacy of the Technical Epidemiological Nexus (Nexo Técnico Epidemiológico Previdenciário; NTEP) of the National Social Security Institute (Instituto Nacional do Seguro Social; INSS), which cross-references the National Classification of Economic Activities (Classificação Nacional de Atividades Econômicas; CNAE) with all diagnostic groups in the International Classification of Disease (ICD).[24] On the one hand, the NTEP decreases the underreporting of occupational accidents and illnesses. On the other, even with additional statistical constraints, an OR of 1.0 is too lenient a cutoff for individual patients seeking social security disability.

The lawyer Sebastião Oliveira attempted to achieve a more equitable solution to this issue by establishing disability payments according to the degree of work-relatedness in Labor Law.[26] The resulting system is similar to the concept of apportionment employed in several locations in the United States[2] (Chart 1).

In the Brazilian context, it is also important to note the contributions of Penteado (Penteado criteria)[27] and Lenz (causal link equations and the concepts of "transcause" and "transoccupational illness").[28] However, both of these derive from the previously presented sources.

Chart 1. Degree of work-relatedness for social security purposes

Degree of causation		
Absence of occupational cause		
Cause is non-occupational; work may have had only a negligible, hypothetical, or clearly circumstantial influence on occurrence.		
Presence of occupational cause		
Degree	**Contribution of occupation**	**Contribution of non-occupational factors**
Grade I	Low - mild	High - intense
Grade II	Medium - moderate	Medium - moderate
Grade III	High - intense	Low - mild

Source: Adapted from Oliveira.[25]

CONCLUSIONS

Except for the case of typical accidents, work-relatedness is not easy to determine. The definition of a causal association must be based, at the very least, on concepts from medicine and law, which are based on widely different assumptions, and no absolute or failsafe formulas can be used to address this issue. In the humble words of Sir Bradford Hill, there are no strict rules for causal association[9]: although this is not ideal, some degree of subjectivity and art is expected on the part of the investigator.

The theoretical models presented in this article must be adapted to the circumstances and characteristics of each case.[29] These issues cannot be addressed using Schilling's Classification alone. The concept of an INUS condition can be integrated with prevalent legal theories and the context of occupational compensation, as it clarifies the issue of multiple causality. However, additional criteria must be applied to improve the consistency and coherence of the scientific reasoning behind etiological explanations in specific cases. In this context, the INSS/DC Resolution No. 10, issued 1999,[23] combines the standards proposed by Hill, Simonin/Franchini, and Schilling.

The conscientious professional must get used to the instruments in this article when explaining his reasoning in technical reports.[30] This will contribute to the enhancement of their critical thinking skills and help avoid fallacious inferences.

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