Causation in epidemiology: association and causation

Introduction

Learning objectives: You will learn basic concepts of causation and association.

At the end of the session you should be able to differentiate between the concepts of causation and association using the Bradford-Hill criteria for establishing a causal relationship.

Read the resource text below.

Resource text

A principal aim of epidemiology is to assess the cause of disease. However, since most epidemiological studies are by nature observational rather than experimental, a number of possible explanations for an observed association need to be considered before we can infer a cause-effect relationship exists. That is, the observed association may in fact be due to the effects of one or more of the following:

- Chance (random error)
- Bias (systematic error)
- Confounding

Therefore, an observed statistical association between a risk factor and a disease does not necessarily lead us to infer a causal relationship. Conversely, the absence of an association does not necessarily imply the absence of a causal relationship.

The judgement as to whether an observed statistical association represents a cause-effect relationship between exposure and disease requires inferences far beyond the data from a single study and involves consideration of criteria that include the magnitude of the association, the consistency of findings from other studies and biologic credibility [1].

The Bradford-Hill criteria are widely used in epidemiology as providing a framework against which to assess whether an observed association is likely to be causal.

The Bradford-Hill criteria (J Roy Soc Med 1965:58:295-300)

1. Strength of the association.
According to Hill, the stronger the association between a risk factor and outcome, the more likely the relationship is to be causal.

2. Consistency of findings.
Have the same findings must be observed among different populations, in different study designs and different times?

3. Specificity of the association.
There must be a one to one relationship between cause and outcome.

4. Temporal sequence of association.
Exposure must precede outcome.

5. Biological gradient.
Change in disease rates should follow from corresponding changes in exposure (dose-response).

6. Biological plausibility.
Presence of a potential biological mechanism.

7. Coherence.
Does the relationship agree with the current knowledge of the natural history/biology of the disease?

8. Experiment.
Does the removal of the exposure alter the frequency of the outcome?

According to Rothman [2], while Hill did not propose these criteria as a checklist for evaluating whether a reported association might be interpreted as causal, they have been widely applied in this way. Rothman contends that the Bradford - Hill criteria fail to deliver on the hope of clearly

distinguishing causal from non-causal relations.

For example, the first criterion 'strength of association' does not take into account that not every component cause will have a strong association with the disease that it produces and that strength of association depends on the prevalence of other factors.

In terms of the third criterion, 'specificity', which suggests that a relationship is more likely to be causal if the exposure is related to a single outcome, Rothman argues that this criterion is misleading as a cause may have many effects, for example smoking.

The fifth criterion, biological gradient, suggests that a causal association is increased if a biological gradient or dose-response curve can be demonstrated. However, such relationships may result from confounding or other biases.
According to Rothman, the only criterion that is truly a causal criterion is 'temporality', that is, that the cause preceded the effect. Note that it may be difficult, however, to ascertain the time sequence for cause and effect.

The process of causal inference is complex, and arriving at a tentative inference of a causal or non-causal nature of an association is a subjective process. For a comprehensive discussion on causality refer to Rothman.

References

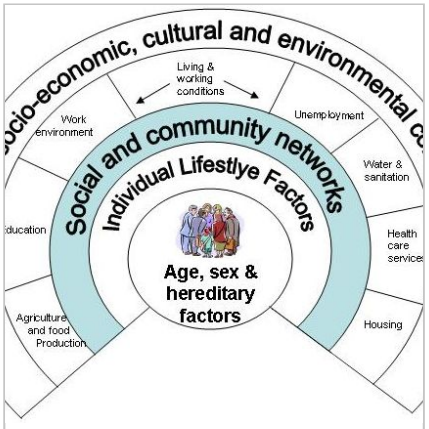
1. Hennekens CH, Buring JE. Epidemiology in Medicine, Lippincott Williams & Wilkins, 1987.
2. Rothman KJ, Epidemiology: An Introduction. Oxford University Press, USA, 2002.
3. Lucas, R M, and Mcmichael, Anthony J. Association or Causation: evaluating links between 'environment and disease'. Bull World Health Organ, Oct. 2005, vol.83, no.10, p792-795.

Further Reading

Rothman KJ, Modern Epidemiology, Lippincott Williams & Wilkins, 1998, p7-28.
Hill, AB, The environment and disease; association or causation? Proc R Soc Med 1965;58:295-300.

‹ Introduction to study designs - developing a questionnaire up Role of chance, bias and confounding in epidemiological studies ›

Recommended for you

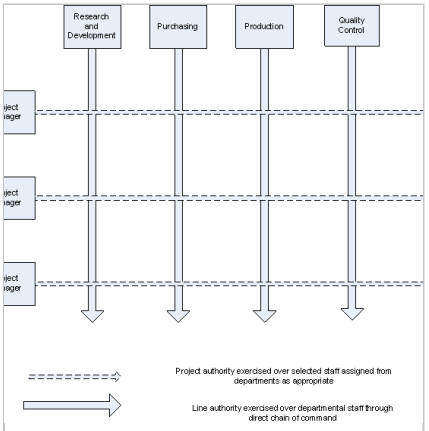


Collective and individual responsibilities for h...

www.healthknowledge.org.uk

The uses of epidemiology and other methods in de...

www.healthknowledge.org.uk



Understanding the internal and external organisational envir...

www.healthknowledge.org.uk