Chapter 7
On Causes and Correlations

Development of Western science is based on two great achievements: the invention of the formal logical system (in Euclidian geometry) by the Greek philosophers, and the discovery of the possibility to find causal relationships by systematic experiment (during the Renaissance).
A. Einstein

7.1 Causes Are INUS Conditions

When seeking a scientific explanation of a given phenomenon, we often look for its cause. We ask for the reason for why we have caught a cold and why the boiling-plate becomes hot after having switched on the electricity.

In situations such as these, if we possess a certain level of general knowledge of scientific matters we can conclude that a cold is typically caused by a virus and that the boiling plate becomes hot due to the friction of electrons in the conductive lines which reside within the metal. But what does it mean for one event to be the cause of another? A preliminary answer would be that the cause of an event is that which makes the event take place. But what do we mean by 'makes'? Is it not more or less synonymous with the word 'cause'? Have we really improved our understanding of causation just by replacing 'makes' for 'causes'? Furthermore, does each event have a single cause, or are there circumstances in which an event may have more than one cause?

People often say that a cause *necessitates* a certain effect. What do we actually mean by this? It cannot really be a matter of logical necessity, as the relationship between cause and effect is not purely logical. David Hume (1711–1776) argued against the idea of a necessary relationship between cause and effect, if this notion is taken to mean some sort of necessity in the nature. He asked what it is we in fact observe when we observe that a cause is followed by its effect. According to Hume, all we can observe is the following: (i) cause precedes effect, (ii) cause and effect are contiguous, and (iii) there is a constant correlation so that each time we observe the same type of cause, we can also find the same type of effect. Thus there is no logical connection between cause and effect, nor can we observe any other kind of necessary connection. Hume therefore concluded that our idea of a necessary connection between cause and effect is a strictly psychological habit, arising after

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having regularly seen a certain type of event followed by another type of event. We become conditioned to expect that a subsequent event of a certain type will follow from a prior event of another type, and it is this expectation that creates our belief in a necessary correlation between cause and effect. Strictly speaking, no cause is, in a modal or metaphysical sense, necessary. It is just a matter or regularity.

Many people have critically opposed this negative conclusion, yet many others have also accepted Hume's view as essentially correct. John Mackie is a modern philosopher who has further developed the notion of causality in the spirit of David Hume. In his book *The Cement of Universe*, Mackie defines a cause as a so-called *inus-condition*. In order to explain what this means, Mackie use a house fire as example. After such an occurrence, most people, especially the insurance company, would want to find out what caused the fire. The local Fire Department normally conducts a formal investigation, to determine the cause of the fire, so let us assume that the investigation concludes that the fire was caused by a short circuit in some faulty electrical wiring. A spark created at the time of short lit up nearby curtains and since nobody was home to put out the fire or call the Fire Department, the whole house was destroyed.

Based on this conclusion we can list several necessary factors that contributed to this event; the wires were in poor condition, the main circuit breaker for the house was 'on', the curtains were too close to the point in question, nobody was home, there was oxygen in the room, and so forth. It is evident that if one of these circumstances had not been present, the house would never have burnt down. Why did the fire engineer consider the bad wires as the real cause of the event? Why not the curtains? Why did he not claim that the cause of the fire was that the main circuit breaker was on, or that there was oxygen in the room? Each of these conditions is equally necessary for event's occurrence.

Intuitively, it is clear that in this case we would never say that the presence of oxygen, or the main power switch, or any of the other circumstances listed above caused the fire. The reason for this is probably that circumstances that appear natural and normal do not cause deviations from the normal course of events. It seems as if our thoughts follow the principle 'abnormal effects follow abnormal causes'. However, what is considered abnormal is certainly a function of our personal perspective and our personal experiences. What may be considered normal or abnormal in specific situation is not merely an objective question. The conclusion of this argument is that a 'cause' is one of the necessary conditions for a particular occurrence, but it is impossible to objectively determine which of the necessary conditions actually caused the event.

Furthermore, the concept 'necessary conditions' (see Appendix; *necessary condition* is *not* a modal concept!) implies that in order to generate a particular effect, all necessary conditions must obtain. We can therefore conclude that a cause is a necessary component of a complex set of conditions, which together bring about a certain effect.

It is, however, obvious that a house can burn down due to other circumstances other than a short circuit in the electrical system. Fires can be caused by pyromaniacs, lightning, or sparks from nearby fires. Thus the set of conditions, which

together caused the house to burn, is one of many possible sets. With this point in mind, one may offer the following definition of the concept of a cause:

> *Def*. A cause is a necessary component of a set of conditions, which together are sufficient for the occurrence of some effect.

This is a somewhat simplified version of John Mackie's definition of a cause, expressed in terms of the so-called *inus conditions*:

> A cause is an Insufficient but Necessary part of an Unnecessary but Sufficient condition for the effect.

I explained above that a selection criterion for a given cause is that it must deviate from what would be considered normal. Another basis for selecting a particular inus-condition as the cause of a given event is the possibility of manipulation. A third reason to refer to an inus-condition as a cause is that it has a high degree of specificity.

There are many medical examples that illustrate this point, such as the cause of the common cold. It is well known that the cause of the flu, or the common cold, is a specific type of virus. However, all who are exposed to the virus do not develop the illness. For the virus to cause an infection one needs to have a weakened immune system. So why can we not just say that the flu is caused by a weakened immune system? Well, that would be reasonable, but we typically point to a virus being the cause of a common cold or the flu because the virus has a specific effect. If the immune system is compromised, several illnesses can take hold, or, if you are lucky, you may not contract any.

We can easily imagine a somewhat different biological system where the flu virus is always in the environment and that we normally do not become ill, except for when the immune system has been compromised. In that case we would probably say that the cause of the flu is a weakened immune system.

In the medical field one often make a distinction between genetic causes and environmental effects. As the following example[1] will show, this distinction is not that clear.

Some breed of the species *Drosophila Melanogaster*, the common fruit fly, has a genetic defect that results in its wings being significantly shorter than normal. This occurs if the temperature is around 20° Celsius at the time the fly is maturing. But if we change the temperature to about 32 °C, we find that the wings grow to normal length. What is the cause of the shortened wings? (Fig. 7.1)

The answer to this depends on which contrast is being considered: if we compare two populations with the same defective gene pool at different temperatures we would naturally conclude that it is the low temperature which causes the shortened wings. However, if we compare two populations of flies at the same temperature, of which one has shortened wings and another does not, we would say that it is the genetic factor that caused the shorter wings.

[1] The example is taken from Hesslow's, G. (1984). In Lindahl & Nordenfelt (Eds.), *What is a genetic disease? On the relative importance of causes* (pp. 183–93).

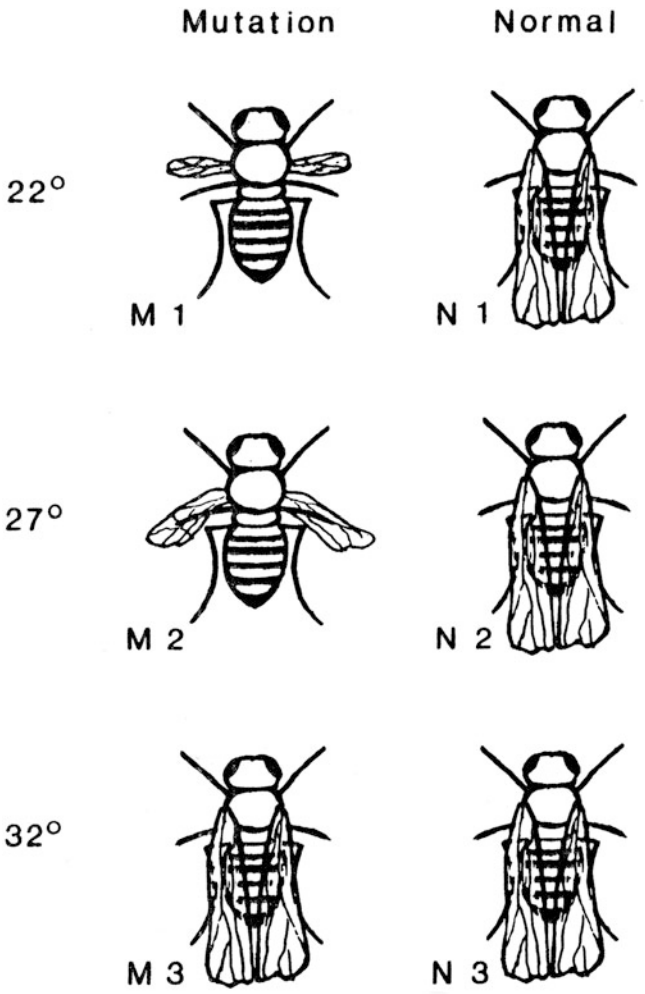

Fig. 7.1 Two populations of fruit flies matured in different circumstances (Picture adapted from Hesslow 1984)

Far from all defects which we typically consider to be genetic can be compensated for by manipulating environmental factors, but the example does demonstrate that a distinction between genetic and environmental causes is not as clear as one might have thought.

The general conclusion of this and other examples is the following. The answer to what is *the cause*, or what are the causes, of a specific event has both an objective and a subjective, or pragmatic, aspect. The objective part of the answer is to give the necessary conditions of the complex that, as a whole, is sufficient for the effect.

The pragmatic, subjective or biased part of the answer is the proposal of one of these conditions as *the cause*, as opposed the other conditions in the complex, i.e. the background conditions. This selection is generally governed by our various interests.

7.2 Cause-Effect and Order in Time

In the discussion above we have looked at examples of both singular occurrences and connections between classes of events. In the first case there are causal connections between singular events, and in the second case causal connections obtain between classes of events. Singular events always occur within a specific time frame. Cause-effect relation is asymmetrical, that is, if event A causes event B, it is impossible for B to be the cause of A. The basis for this asymmetry is that cause must precede effect. In general it is the case that the cause precedes the effect for every individual cause-effect pair in a class of event pairs (There are cases where we are prone to say that two events are related as cause and effect albeit they occur simultaneously, or very nearly so. But this is a limiting case.). The definition of a cause as an inus-condition does not include this aspect of causation; rather, it is taken for granted.

Why does a cause typically precede its effect? The better question is perhaps 'why have we formed the concept of a cause, which operates in a manner that cause precedes effect?' The answer to this question is that causes are typically used to identify nodes in the flow of events where we can step in and perhaps modify the course of those events; we want to find the cause of an event in order to prevent or recreate a similar event. Naturally, we cannot prevent an event that has already taken place. When we want to know the cause of an event, we typically want to know what to do.

There is thus a pragmatic aspect to selecting the right cause, as we explained in the previous section. It is worth noting that this view is consistent with the idea that a cause and an effect could be practically simultaneous.

7.3 Causes and Statistical Correlations

How do we research causal connections? The ideal method would be the following: to isolate the object one wants to study and then to vary the presence and strength of certain factors that may influence the state of the object, that is, the different components of the various possible complexes of sufficient conditions. By varying one factor at a time, and by measuring the variation in the state of the object, one can obtain quantitative descriptions of how each component influences the object.

In most disciplines, however, this is not feasible. Instead, one has to make do with statistical investigations of classes of objects and events. The type of information one can acquire from such statistical measurements is essentially correlations between various factors that answers questions of the form 'is there some statistical connection between variables A and B, and how strong is it' (See Sect. 4.2).

Before delving further into this discussion, we should note that a correlation can only be attributed to a pair of *sets of events*, which we can describe using stochastic variables, whereas a cause-effect relation basically obtains between two single events, and only indirectly between types of events.

Despite their name, stochastic variables are actually functions, i.e., mappings from sets of events to sets of numbers. As an example, consider the throwing of dice. Suppose we call the stochastic variable K. For each throw of the dice, K receives a definite value, the number on the dice. That a stochastic variable receives a certain definite value can be considered an event. Thus, when discussing possible conclusions one can draw from correlations, in regards to causal connections, the question becomes which causal connections exist between the two individual events *variable X has the value* x_i and *variable Y has the value* y_i, given these event types comprise two correlated variables.

Now suppose that there is a strong correlation between two variables A and B.[2] Does this mean that A-events cause B-events? No, of course not. For all we know, B-events could be the cause of A-events, since the statistics give us no way of knowing the temporal order of the events. There is also a third option, that A-events and B-events are caused by some unknown type of events, call them C-events, without there being any causal link between A-events and B-events. If two events have a common cause, it is likely that they will appear together more often than could be explained by mere coincidence; and so looking at sequences of the same type of events, they will be correlated. Hence if all we know is that A and B are statistically correlated types of events, no stronger conclusion than the following can be made:

- A is a cause of B, or
- B is a cause of A, or
- A and B have a common cause.

This statement, that every statistical correlation is based on one of these three types of causal connection, is called *Reichenbach's principle*, after the philosopher Hans Reichenbach (1891–1956).

A common objection to this principle is that the correlation in question might be purely accidental. This objection is valid only if by 'correlation' one refers the observed correlation of a sample test. Indeed, it is clear that one can find a correlation between two variables in a sample without there being any such

[2] Instead of talking about two stochastic variables, statisticians use the notion of a two-dimensional stochastic variable.

correlation in the entire population. As described in Sect. 3.6, it is not possible to draw any conclusion about the probability for an hypothesis, in this case the hypothesis that there is a correlation in the population, using only results of observation of a sample; either one has to start with a prior probability and uses Bayes' theorem, or determine the likelihood, i.e., the probability for the observed result, conditional on the hypothesis that there is a real correlation.

Then, if the probability for obtaining a correlation in the sample test is less than 5 %, conditional on the assumption that there is no correlation in the entire population, and one still obtains a correlation in the sample test, then one has obtained a significant result at the 5 % level. This result may be interpreted as showing that there is reason to believe that the hypothesis that there is a correlation in the entire population. But we haven't got any probability measure for this hypothesis.

In what follows, the word 'correlation' refers to correlations in reality, that is, in the entire population and not only in samples. It will be assumed that one has observed a correlation in a sample test, performed the significance test, and *correctly* concluded that there really exists a correlation in the entire studied population. Given this meaning of correlation, the objection above is invalid. This is clearly shown by the following thought experiment. Suppose we have chosen 10 objects out an unlimited supply of a certain type of object, and have subsequently observed two properties. Furthermore, we find that there is a correlation between the variables we use for measuring these properties. Of course, this could be a coincidence. Thus we perform a larger sample test, say of 100 objects, and find the same correlation, or nearly so. Of course, this could also be a coincidence, even if the probability that it is has diminished. We can continue this process ad infinitum. If the correlation obtains regardless of how large of a sample test we perform (which we can never know, but a sequence of larger and larger samplings showing a convergence to a certain correlation coefficient, may give strong support for the hypothesis that the real value is close to the convergent result), then it is, by definition, not a coincidence that the correlation obtains, provided the sequence of samples is randomly generated.

In order to determine which of the three alternatives in Reichenbach's principle is the case, we must acquire further information. One can often dismiss one of the alternatives from information about the temporal order: If the singular event B precedes the singular event A, then A cannot have caused B. But how does one determine whether A causes B, or if there is a third background factor that is the cause of both A and B? In some cases the background information is sufficient, as the following examples illustrate.

Example 1 An investigation conducted in West Germany during the years 1966–1980 showed a correlation between birth rates and the presence of storks; as the number of storks decreased, so did the number of babies. The correlation was so strong that the probability for a mere coincidence was calculated to be less than 0.1 %. Hence there are very good reason to believe that it is real correlation. But since we do not believe that storks deliver babies, or that new-born babies attract

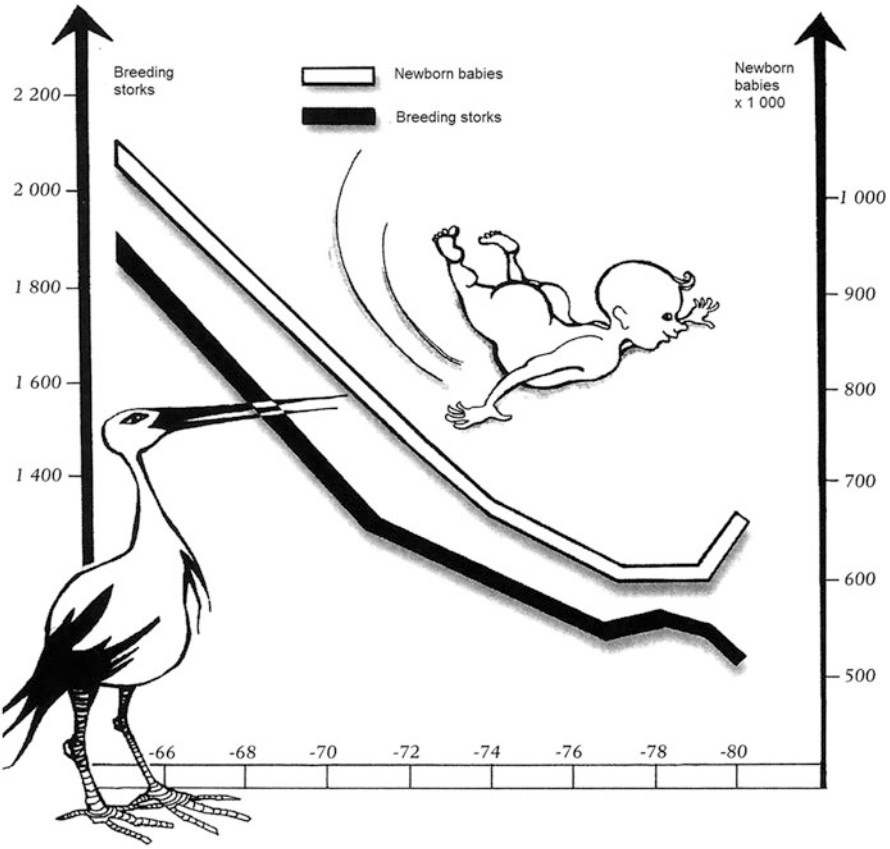

Fig. 7.2 Breeding storks and new-born babies in West Germany 1965–1980. The likelihood for the two variables not being correlated is less than 0.001 (*Source*: Nature, 7 April 1988. Reproduced with kind permission of Lou-Lou Pettersson)

storks, there must have been a hidden variable, a common cause. In my view, the most reasonable explanation is that both the decrease in the number of storks and the decrease in the number of babies are both the effects of an increase in German urbanization and industrialization during the period of observation (Fig. 7.2).

Example 2 An investigation of a cross-section of a certain population revealed a correlation between intelligence and shoe size. The bigger the feet, the more likely it was that one would perform better than the majority on an intelligence test. Is there in fact a correlation between foot size and the abilities of the brain? No, it is likelier that some background factor is at work here, namely age. Included in a cross-section of a population are a number of children of various ages, which have smaller feet and less likely to score well on an intelligence test than adults. This explanation can be easily tested by sorting out all children under the age of 18, and

then checking if the correlation stills obtains. If it does not, then the correlation was the result of the age factor.

Assume that we do not have any background information that would help us determine whether a correlation between X-events and Y-events depends on a causal connection between them, or whether they have a common cause. If an experiment is feasible, the simplest test is to vary the incidence of X (where X precedes Y) and observe whether the incidence of Y follows suit. If so, there is an argument for X being the cause of Y. If the incidence of Y does not vary with the incidence of X, then X cannot be the cause of Y, which indicates that both X and Y share a common underlying cause.

However, it is often not practically, or ethically, feasible to perform the required experiment. In such situations one has no other choice but to calculate various conditional probabilities and observe which correlations obtain. In order to precisely say what conclusions can be drawn from information regarding correlations we need to understand two statistical concepts, viz., *statistical independence* and *conditional probability*.

Def. of Independent events: Two events A and B are independent of one another if and only if the probability that both A and B occur is the product of the individual probabilities, i.e. P(A and B) = P(A)P(B).

If this obtains, we say that the probability of the combined event, A and B, is *factorable*.

Def. of *Conditional Probability*:

$$P(A|B) = \frac{P(AB)}{P(B)},$$

where P(AB) stands for P(A and B). We express this equation as follows: the conditional probability of A given B is the probability of A in a population where B has occurred. The difference between the probabilities P(A) and $P(A|B)$ is that they are calculated in two different populations.

If two events, A and B, are independent of each other, it follows that the coefficient of correlation $r_{AB} = 0$ (see Sect. 4.2) i.e. they are not correlated. By Reichenbach's Principle, neither A nor B can cause the other. On the other hand, if A causes B (or B causes A), then a correlation exists which leads to A and B being dependent, i.e. $P(AB) \neq P(A)P(B)$. The converse, however, is not valid. Two events can be uncorrelated while still being statistically dependent.

(Note the difference between converse and contraposition. Given a proposition of the form 'if A, then B', we can form the converse 'if B, then A'. However, the contraposition to 'if A, then B' is 'if not B, then not A'. The contraposition of a proposition, as opposed to the converse, is a logical consequence of the original proposition.)

Now suppose that we have a correlation between variables X and Y and we know that X-events always precede Y-events. At this point we ask ourselves, are the X-events one of the causes of the Y-events, or do X and Y-events share a common

cause? Suppose that we believe Z to be this common cause. Using conditional probabilities we calculate P(XY|Z), P(X|Z) and P(Y|Z) and we get either

$$P(XY|Z) = P(X|Z) \cdot P(Y|Z)$$

or

$$P(XY|Z) \neq P(X|Z) \cdot P(Y|Z)$$

(In practice, of course, we will never get an exact equality; if P(XY|Z) is close to P(X|Z) · P(Y|Z), we have to make a decision: equal or not?) Assume that P(XY|Z) is factorable, as in the first alternative above. What conclusions can be drawn? One possibility is that Z actually is a common factor of X-events and Y-events. Another possibility is that the factor Z stands for an event in between an individual X and Y-event (Note that even in this case P(XY|Z) is factorable.). If this is case, it follows that X-events are indirect causes of Y-events, a possibility that cannot be excluded.

If it so happens that P(XY|Z) is not factorable, as in the second alternative above, then Z is neither a common cause of X and Y, nor an intermediate factor. This result does not exclude the possibility of some other common cause of X and Y. All it means is that we cannot know for sure that X is a cause of Y.

Unfortunately, it seems very difficult draw any definite conclusions about causal connections from information about statistical correlations only, even if that information is significant and we have correctly concluded that these correlation obtain in the entire population (i.e., we have not made an error of the first kind.) More information is needed. Nancy Cartwright, using arguments different from those presented here, has come to the same conclusion, formulated as 'No causes in, no causes out' in her (1989).

7.4 Risk Factors and Conditional Probabilities

Risk factors for various diseases are often discussed in medicine. Fore example, smoking, high cholesterol and a certain genetic disposition are considered to be factors that increase the risk of a heart attack. This means that the probability of having a heart attack is increased if any of these factors are present. Thus the conditional probability of a smoker having a heart attack is higher than the non-conditional probability. This means that the number of heart attacks in a group of smokers is higher than in the entire population, which can be expressed as

$$P(\text{heart attack}|\text{smoking}) > P(\text{heart attack}), \text{or } P(H|S) > P(H).$$

Applying the definition of conditional probability,

$$P(H|S) = \frac{P(HS)}{P(S)}$$

we get

$$\frac{P(HS)}{P(S)} > P(H)$$

Rearranging the terms, we get $P(H|S) > P(H) \cdot P(S)$. H and S are evidently statistically dependent, which implies that there is a correlation between smoking and heart attacks (In this case there are only two values for each of the two variables, i.e. smoker/non-smoker and heart attack/no heart attack, which means that the correlation cannot be calculated in terms of the product–moment presented in Sect. 4.2; but a different form of correlation can be calculated.). My previous conclusion, that a correlation should not be automatically interpreted as a causal connection, also applies to risk factors. *A risk factor need not be a cause*. Rather, it could be a side effect of some circumstance, which both causes the disease, and, in another causal chain, causes the risk factor. In such cases, risk factors function as warning flags. It follows that if such is the case, there is no point in trying to decrease or eradicate the risk factor in order to decrease the impact of the disease. Doing so would be like turning off a warning light in order to avoid the catastrophe that the light warns us about. It is therefore essential to determine whether or not a risk factor is a cause (But in this case it is well established that smoking is an inus-condition, and can thus be said to be a cause, of lung cancer.).

However, it is worth pointing our that one does not often use the term 'risk factor' for factors that we believe not to be causal, even if they correlate with the disease. Indeed, there seems to be a shift in use of the word 'risk factor' from meaning 'statistically relevant factor' to 'statistically relevant factor, which comprises a part of the total cause'. This unclarity is deplorable and Olli Miettinen at Harvard suggests a remedy:

> Determinants of incidence are commonly referred to as risk factors. This term is a misnomer. Since the relation of an occurrence parameter to a determinant need not be the result of a causal connection, and since the term 'factor' (from the Latin word for doer) suggests causality, 'risk factor' is not a proper substitute for 'determinant of risk' A proper synonym is 'risk indicator' – analogously with 'economic indicator', 'health indicator' and so on.[3]

[3] Miettinen, O. (1985). *Theoretical epidemiology* (p. 10). New York: Wiley.

7.5 Direct and Indirect Causes

If A causes B, and B causes C, then A is an indirect cause of C. An indirect cause is still a cause, according to the above definition, since if B is a necessary part of a complex of sufficient conditions for C, and A is a necessary part of sufficient conditions for B, then A is also a necessary part of sufficient conditions for C. Thus A is a cause of C. The causal relation is hence transitive, which means that we can talk about causal chains. Every causal description is, therefore, infinite in principle. It also means that when we say that some event A causes another event B, there can, in principle, be infinitely many intermediate events. Thus the cause of some effect can occur long before that effect. In medicine there are many such examples, e.g. skin cancer, which can be caused by too much exposure to the sun decades before the disease arises.

Likewise, historians may discuss distant causes of important historical events. A. J. P. Taylor argues (in his *War by Time-table*) that the 'the man who pulled the trigger' and caused the first world war (1914–1918) was count Alfred von Schlieffen, German chief of general staff 1891–1906. How could he have 'pulled the trigger', he retired 8 years before WW1 began?

Schlieffen had construed a mobilisation plan for the German army on the presumption that France and Russia were allies and if war broke out Russia would take much longer time to mobilize than France. In case of war, therefore, the German army should first fight France with most of its force and then move to the eastern front. Hence, according to the plan, immediately after mobilization the major part of the German army was transported by train to Aachen, near the Belgian border. There is no place for millions of soldiers in Aachen so every division has, immediately after debarking the train, to march into Belgium in order to leave place for new troops coming. And since Belgium was neutral and England and France had promised to defend it, a German decision to mobilize was also in practice a decision to invade Belgium, which meant to start war. This was not fully realized by the Kaiser and his government; the German government's motive for deciding mobilization in August 1914 was to put political pressure on Russia, not to start war. So Taylor concludes that Schlieffen's mobilization plan was the cause of WW1 (And he shows, quite convincingly, that none of the great powers really wanted war.).

My own view is that Taylor has provided strong reasons to say that von Schlieffen's plan is an inus-condition for the first world war; and since no one really wanted war, according to Taylor, it is not unreasonable to say that the Schlieffen-plan was the cause (The causes of the first world war is a hot topic of debate, and by rehearsing Taylor's view I do not want to say he is right. But it is a prima facie plausible account of the causes and an interesting illustration to the notion of indirect causes.).

7.6 Causes as Physical Effects

According to David Hume, as we saw in Sect. 7.1, (i) a cause precedes, or at most occurs simultaneously with, its effect, (ii) causes and effects are always in close proximity, or connected by an intermediate chain of events also in close proximity, and (iii) a cause is regularly followed by its effect. One can characterize Hume's thesis as that he has given the necessary conditions for the use of the term 'cause'. Many philosophers have accepted Hume's thesis with the modification that a cause makes an effect more probable. But if we accept Reichenbach's principle, it follows that Hume's view cannot be correct, since the third condition, that a cause is regularly followed by an effect with a certain probability, is not able to distinguish between true cause-effect relations and common-cause correlations in which one factor happens to arise sooner than its correlated partner. Those who accept Reichenbach's principle thus require a deeper analysis of causation than what we get from Hume. Thus Reichenbach[4] and later Salmon[5] have further developed Hume's analysis of causation. Since Hume's second condition of proximity only applies to physical bodies, we need an analysis of causal connection that does not view links as made up of such objects.

A natural idea is to define a causal process as a transmission of energy (Prime example: the sun transmits energy to the earth and causes life.). That A causes B simply means that there is a physical connection, a signal of some kind carrying energy, which is transmitted from A to B. As far we know, there are only four fundamental kinds of interaction in nature: gravitation, electromagnetism, week and strong nuclear interaction. If an event A causes another event B, then these events must be connected by one of these four kinds of interaction, that is, there must be some exchange of particles doing the signalling from A to B. For example, in the human body, all connections between cause and effect, such as neural transmissions, are of an electric nature and neurons exchange charges. Mechanical interaction, such as friction or pressure, are also at bottom electromagnetic interactions: when two molecules collide with each other they exchange energy and momentum in the form of electromagnetic quanta, photons.

Since no signal can travel faster than the speed of light, causes must precede their effects no matter what coordinate system the observer uses. This fact agrees with our concept of a cause. If a cause and its effect appear to occur simultaneously, it is actually the case that the time it took for the interaction to take place is so small that it cannot be measured without sophisticated equipment.

From Mackie's analysis, it follows that a cause increases the probability that some effect will ensue. This is because, in most cases, a cause is part of a complex of necessary conditions, which must all be met for the effect to take place. Thus the

[4] Reichenbach, H. (1956). *The direction of time*. University of California Press.

[5] Salmon, W. (1984). *Scientific explanation and the causal structure of the world*. Princeton University Press.

coupling of a cause and an effect is only occasionally strictly deterministic, viz., in cases where there is only one inus-condition.

7.7 Cause and Effect in History

When studying history one is often interested in causes. For example, one might ask what caused the fall of the Roman Empire, the disintegration of Yugoslavia, or the industrial revolution. Can one apply the analysis given above to answer these types of questions?

The answer is yes. Mackie's analysis in terms of inus-conditions fits quite well in these situations, since we usually assume that historical events depend upon a large number of interacting causes. This analysis tells us that it is incorrect to ask for only one cause. Rather, we should try to identify all, or most, of the necessary factors, which together give rise to an historic event.

However, the next step in the analysis of causation, reference to physical mechanisms, seems, at least at first, more problematic. Is it really legitimate to talk about physical interactions as the basis of causal connections between historical events? It does not seem reasonable, but such is the case. Historical chains of events are comprised of a complex net of natural events (climate change, disease, famine, etc.) and the conscious actions of humans. It is obvious that the natural events are ultimately connected by physical mechanisms, but what about conscious actions?

Actions have both an internal side (thoughts, feelings and opinions) and an external side (our behaviour). Our behaviour, like natural events, is comprised of physical (chemical and biological) events, which are thus connected to each other via one of the four fundamental kinds of interaction. But what about our thoughts and feelings? In fact, even these can be understood as having a physical description (see Sect. 12.6). According to this view, historical events, including the historical agent's conscious considerations and unconscious motives, can be viewed as connected via causal chains (which are physical in nature) to other events, since they are at bottom physical states. In any case, there is no conflict between the idea that all causal connections are achieved via one or more of the four fundamental interactions and the use of the concept of a cause to link historical events.

However, it should be stressed that this fact implies nothing about whether or not historical events *should* be described as chains of physical mechanisms. The argument is not methodological but ontological. In history the physical aspects of events are not often of much interest. My point is merely to show that it is possible to give a single unified analysis of cause and effect that applies equally to the natural sciences, social sciences and the humanities.

Counterfactual Accounts of Causation

Causation has been a heated topic of debate among philosophers for decades. One particularly popular idea is to analyse the causal relation using counterfactual formulations. The general schema is something like

'A causes B' means 'if A had not occurred, B would not have occurred'.

This appears intuitively plausible, but I'm inclined to respond that I understand the meaning of the word 'cause' much better than the meaning of counterfactual propositions. So those who argue that counterfactual analyses of causation need a viable account of the semantics of counterfactuals. Many have relied upon the notion of possible worlds, utilized in the semantics of necessity and possibility. Roughly, the idea is as follows: in our world A occurs and is followed by B, whereas in a possible world, fulfilling certain conditions, A does not occur and neither does B. The crucial problem is, of course, how to identify those possible worlds where neither A nor B occur without using intuitions about causation, which in my view seems difficult. In fact, much of the discussion has been about purported counterexamples built upon our intuitions about causation. In other words, our intuitions about causation are used as adequacy conditions for a counterfactual analysis. But, why, then bother about counterfactuals, if we have stronger semantic intuitions about the concept of cause than that of counterfactuals? If anything, it seems more reasonable to analyse counterfactuals in term of causes.

An alternative account of counterfactuals builds on Judea Pearl's notion of structural equations, see Pearl (2000). The structural equations in a model of a type of system describe the dynamical evolution of the system being modelled. There is a structural equation for each variable of the form

$$Y = f(X_1, \ldots X_n)$$

where $X_1, \ldots X_n$ are the variables in the model. This equation is read by e.g. Woodward (2003) and Hitchcock (2001) as

> If the variable X_1 would have the value x_1, X_2 the value x_2. . ..., then Y would have the value $f(x_1, \ldots x_n)$

How do we obtain knowledge about such equations? By doing experiments and observations and generalise from limited number of observations to these general statements. In other words, causation is in this approach related to our possibilities to manipulate systems. In this I wholly agree, most often are we interested in causes because we want to know what to do in a particular situation.

Repeated manipulations of a system thus may enable us to formulate structural equations that express observed regularities. This is thoroughly discussed in Pearl's (2000). What this means, basically, is that this is a version of Hume's regularity account of causation, with the amendment that it is observed regularities which are brought about by manipulations.

7.8 Summary

The discussion of the concept of cause, as it is practically used, has led to the concept of inus-conditions. In cases where we want to pinpoint some such factor as the central or most important cause, this selection was shown to be a pragmatic one. We even found that the way we use the concept of a cause requires that the cause precede its effect and that both must be in close proximity, or otherwise connected by intermediate causes.

Cause-effect relations give rise to statistical correlations. What we observe are correlations, but if two events are correlated we cannot validly infer that they are causally related, because two events that share a common cause are also correlated. An important task in many scientific disciplines is to determine which of the three possible causal connections, listed by Reichenbach, accounts for a particular observed correlation. In fact, this can only occasionally be done using *only* statistical methods. Thus if at all feasible, performing the right experiment is a significantly more effective approach.

Finally, we placed the concept of a cause in our scientific world-view by conceiving of the connection between cause and effect as physical in nature i.e. based on one of the four fundamental kinds of interaction found in nature.

Exercises

1. In the 1940s, a strong correlation was found between the consumption of sweets and the frequency of caries among children. Could we conclude from this that a similar investigation would yield a similar result today?
2. So far, all attempts to establish a statistical correlation between so-called 'oral galvanism' (a taste of metal in the mouth, pain, headaches etc.) and the presence of other galvanic currents in the mouth have been unsuccessful. Does this mean that we can exclude galvanic currents as the cause of oral galvanism?
3. The following information is known:

   (a) Large amounts of substance X is a risk factor for the disease Y.
   (b) Lowering the amount of X in a group of people with high values of X in their bodies, but who have not yet contracted Y, does not reduce the risk for Y in this group.

   What conclusion(s) can one draw from these observations?
4. Suppose that a positive correlation has been found between low levels of a substance F in the blood and the incidence of the disease S. Which conclusion or conclusions can one draw from this?

   (a) Low levels of F is a risk factor for S.
   (b) Treating a group of patients with a medicine that increase the level of F in the blood will reduce the incidence of S.
   (c) Either the lack of F contributes to S, or S contributes to the lack of F.
   (d) There is a hidden factor causing both the low levels of F and the incidence of S, though the level of F in the blood has no effect on the incidence of S.

(e) None of the above conclusions can be drawn from the given information.

5. Given that the presence of factor F in the blood exceeding a certain limit causes disease G, which of the following is true?

   (a) If no amount of F is present in the blood, then one will not contract G.
   (b) If the amount of F in the blood exceeds the limit, one will contract disease G.
   (c) If the amount of F in the blood exceeds the limit, then the risk of contracting G is increased.
   (d) If one has G, then the amount of F present in the blood must have exceeded the limit.
   (e) If the amount of F exceeds the limit, the risk of contracting G will decrease if a medicine is administered to lower the amount of F in the blood.

6. The following example is fictitious. Out of a population of Swedish students, 31 % decide to seek higher education. Among students who have parents with academic training, 70 % seek higher education. Does this higher probability for higher education depend upon cultural factors, or does it depend on the fact that more educated parents have higher incomes? In order to answer this question, the following investigation was undertaken. The families whose income was 50 % higher than average were sorted out. Out of this group of families, 58 % of students went on to higher education. Furthermore, from this high-income group, 72 % of the students who chose to go on to higher education had academically trained parents. The families that contained at least one academically trained parent made up 80 % of the high-income group.

   What does this statistical data show? Is it higher education or higher income that causes a child from an academically trained family to seek higher education more often than other children?

7. Suppose you want to know whether there is a causal connection between two variables X and Y in a population. You select a sample from the population, measure the two variables and find no correlation. It is of course possible that there is a correlation in the population, so you make a statistical test. It shows that it is highly unlikely that there is a correlation in the population and you conclude there is not. Suppose this is true. Could one then infer that there is no causal connection between X and Y? Hint: Google ‘Simpson’s paradox’.

Further Reading

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