Sciences provide different approaches to the study of man: man can be scrutinized in terms of molecules, tissues and organs, as a living creature, and as a social and a spiritual person. Correspondingly, philosophy of science investigates the philosophical assumptions, foundations, and implications of the sciences. It is an enormous field, covering sciences such as mathematics, computer sciences and logic (the formal sciences), social sciences, the natural sciences, and also methodologies of some of the humanities, such as history. Against the backdrop of the sweep of the field, this chapter comprises a brief overview of the philosophical aspects salient to research in the medical and biological sciences. Consequently, discussion is limited to the natural sciences (Section 1.1) and the social sciences (Section 1.2). The formal sciences, such as logic and mathematics, are not discussed.

1.1 PHILOSOPHY OF THE NATURAL SCIENCES

What do we mean when we say that ‘smoking is the cause of lung cancer’? What counts as a scientific explanation? What is science about, e.g. what is a cell? How do we obtain scientific evidence? How can we reduce uncertainty? What are the
limits of science? These are only a few of the issues discussed in philosophy of the natural sciences, which are to be discussed in this chapter.

**Traditional philosophy of science**

The traditional philosophy of science has aimed to put forth logical analyses of the premises of science, and in particular the logical analysis of the syntax of basic scientific concepts. In the following sections, the principal traditional issues concerning reason, method, evidence and the object of science (the world) are discussed.

**The glue of the world: causation**

A pivotal task of the biomedical sciences is to find the causes of phenomena, such as disease. However, what is the implication of saying that something is the cause of a disease? According to Robert Koch (1843–1910), who was awarded the Nobel Prize in physiology or medicine for finding the tuberculosis bacillus in 1905, a parasite can be seen as the cause of a disease if it can be shown that the presence of the parasite is not a random accident. Such random accidents may be excluded by satisfying the (Henle–) Koch postulates:

- The organism must be found in all animals suffering from the disease, but not in healthy animals.
- The organism must be isolated from a diseased animal and grown in pure culture.
- The cultured organism should cause disease when introduced into a healthy animal.
- The organism must be reisolated from the experimentally infected animal.

As became clear to Koch, these criteria are elusive. If such postulates are considered to be general criteria for something to be a cause in the biomedical sciences, causation is unlikely.

Acknowledging that overly stringent criteria for causation minimize the chance of identifying causes of disease, the British medical statistician Austin Bradford Hill (1897–1991) outlined tenable minimal conditions germane to establishing a causal relationship between two entities. Nine criteria were presented as a way to
determine the causal link between a specific factor (such as cigarette smoking) and a disease (such as emphysema or lung cancer):

- **Strength of association**: the stronger the association, the less likely the relationship is due to chance or a confounding variable.

- **Consistency of the observed association**: has the association been observed by different people, in different places, circumstances and times (similar to the replication of laboratory experiments)?

- **Specificity**: if an association is limited to specific people, sites and types of disease, and if there is no association between the exposure and other modes of dying, then the relationship supports causation.

- **Temporality**: the exposure of interest must precede the outcome by a period of time consistent with any proposed biological mechanism.

- **Biological gradient**: there is a gradient of risk associated with the degree of exposure (dose–response relationship).

- **Biological plausibility**: there is a known or postulated mechanism by which the exposure might reasonably alter the risk of developing the disease.

- **Coherence**: the observed data should not conflict with known facts about the natural history and biology of the disease.

- **Experiment**: the strongest support for causation may be obtained through controlled experiments (clinical trials, intervention studies, animal experiments).

- **Analogy**: in some cases, it is fair to judge cause–effect relationships by analogy: ‘With the effects of thalidomide and rubella before us, it is fair to accept slighter but similar evidence with another drug or another viral disease in pregnancy’.

Hence, with regard to causality, these criteria are less pretentious than the Koch postulates. Nonetheless, there are many cases where we might refer to ‘the cause of the disease’, but where the criteria do not apply. However, the Bradford–Hill criteria admit that causation in the biomedical sciences is far from deterministic (as in the Koch postulates), and that it is an amalgam of more general criteria.

However, if the causes of phenomena studied in the biomedical sciences are not deterministic, what then are they? That is, what is the true nature of the causation with which we deal? In the deterministic version of causation, we know both the necessary and the sufficient conditions for an event. The Koch postulates require that there are no cases of disease without the parasites, and there are no parasites...
without the manifestation of disease. As Koch realized when he discovered the asymptomatic carriers of cholera, the requirement of both necessary and sufficient conditions for causation is overly rigorous.

Whenever there is a necessary but not sufficient condition for an event, we do not say that it causes the event. For example, having an arm is a necessary condition for having an inflammation of it, but having an arm is not said to be the cause of the inflammation. In this case, multiple factors are prerequisites for something happening, but no one of them alone is sufficient. There may be many necessary conditions for an event that are not considered causes of it. Nevertheless, necessary conditions are germane to causation, as without them, the event will not occur. Hence, necessary conditions are relevant through their absence: we can eliminate tuberculosis by eliminating one of its necessary conditions: *Mycobacterium tuberculosis*. It is also important to notice that necessity can mean two different things. Necessity can mean irreplaceability, that is nothing else than A could have resulted in B, e.g. the modification of the Huntington gene is the only thing that results in Huntington’s disease, but necessity can also mean non-redundancy, that is when many things can result in B, but one of these is A in combination with R and S (see Figure 1.1). In this case A is non-redundant. A virus infection is a non-redundant condition for having a cold, as there are many other conditions resulting in a cold, but when these are absent, and you do not have a virus infection, you will not have a cold. Under those circumstances, the virus infection is a necessary condition for having a cold.

![Figure 1.1 Two meanings of necessity](image)

The situation differs when there is a sufficient but not necessary condition. For example, when a person develops cancer after being exposed to ionizing radiation known to be of the sort and strength that results in cancer, we tend to say that the

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1. A is an irreplaceable condition for B, if and only if nothing other than A could have resulted in B.
radiation caused the cancer. The cancer could, of course, have been caused by other factors (sufficient conditions) in the absence of the radiation. Nevertheless, if a sufficient condition is present, we know that the event (the effect) will occur. Hence, sufficient conditions for an event are said to be its causes. In contradistinction to necessary conditions, they work through their presence.

Another situation is when there are two factors that individually are insufficient conditions for a certain event, but which together make an event occur. For example, alone being stung by a bee or being hypersensitive to bee venom does not cause an anaphylactic reaction. However, in certain circumstances, acting jointly, both may be sufficient and necessary for an anaphylactic reaction, so both are said to cause the event. In short, each of the factors is an insufficient but necessary part of a sufficient and necessary condition for the event. Although we seldom find single factors that are both sufficient and necessary for events in the biomedical sciences, we more often find cases where multiple factors together are sufficient and necessary.

Consider the scenario when a person drinks (a lethal dose of) poison, no antidote is taken, the stomach is not pumped and the person dies. What is the cause of death? Does the person die because poison is ingested, because no antidote is taken or because the stomach is not pumped? Ingesting poison alone is not sufficient, as many people drink poison without ensuing death (because their stomachs are pumped). However, drinking the poison is part of a concert of conditions that are jointly sufficient to cause death. Moreover, given this set of conditions and not another set sufficient for death, drinking the poison was non-redundant: deaths do not occur in such circumstances when poison is not drunk.

Accordingly, drinking poison is an insufficient and non-redundant part of an unnecessary but sufficient condition for death. This is called an INUS condition (Mackie 1974). It can be argued that many relationships in biomedical sciences, regarded as causal, satisfy INUS conditions. Hence, causation is given by the conditions of an event. If the conditions are both sufficient and necessary, if they are jointly sufficient and necessary, or if they are INUS conditions, then one could argue that they are causes. However, what about smoking and lung cancer: is smoking an INUS condition for lung cancer? INUS accommodates the fact that not all smokers develop lung cancer, and not all people with lung cancer have been smokers. However, it requires a concert of conditions for which lung cancer follows when smokers, but not when non-smokers, are subjected to them.
CHAPTER 1

Common to approaches defining causation in terms of sufficient (and necessary) conditions is that they hinge on scientific determinism, that is, that complex phenomena can be reduced to simple, deterministic mechanisms, and therefore in principle be predicted. In the case of smoking being an INUS condition for lung cancer, all the conditions are not known. Hence, we will have to assume the existence of hidden conditions in order to retain determinism. The belief in unidentified conditions, as well as the difficulty in explaining dose–response relationship, has challenged sufficient component conceptions of causation (sufficient condition, insufficient but necessary part of a sufficient and necessary condition, and INUS).

Rather than satisfying an INUS condition, we observe that smokers develop lung cancer at higher rates than do non-smokers. This leads us to believe that the increased probability of lung cancer among smokers is the causal link. This represents a probabilistic approach to causation. The central idea in probabilistic theories of causation is that causes raise the probability of their effects.

Despite their plausibility, probabilistic approaches to causation are challenged with regard to how much a probability must be raised in order to become a cause. We say that aspirin ‘causes’ Reye’s syndrome in children and that certain tampons ‘cause’ toxic shock syndrome, though the probabilities are low. Accordingly, it becomes difficult to differentiate between causation and non-causal associations.

Moreover, some scientists are uncomfortable with the propensity of probabilistic approaches to abandon determinism. Events are not determined as having occurred, although there may have been (probabilistic) causes for them. This may frustrate the aim of pursuing causality: circumventing certain events (disease) and promoting others (symptom relief, health). In other words, if an event is not determined to have occurred, then nothing can be part of a sufficient condition for it. Hence, some would prefer to say that smoking is an INUS condition for lung cancer, although we do not (yet) know the concert of conditions sufficient for its occurrence.

Another approach highlights that the presence or absence of a cause ‘makes a difference’. This is expressed by counterfactuals: a counterfactual draws on the contrast between one outcome (the effect), given certain conditions (the cause) and another outcome, given alternative conditions. C causes E if the same condition except C would result in a condition different from E, when all other conditions are equal (ceteris paribus). For example, ‘if I had taken two aspirins instead of just a glass of water an hour ago, my headache would now be gone.’ A counterfactual
conception of causation is considered to be more precise with respect to what distinguishes causation from mere association than the probabilistic approach, while it avoids referring to hidden deterministic conditions. Counterfactuals can also be probabilistic: ‘if I had taken two aspirins instead of just a glass of water an hour ago, I would be much less likely to have a headache now.’ However, in practice it is not easy to satisfy the *ceteris paribus* condition. The same individual cannot be observed in exactly the same situation as both a smoker and a non-smoker.

It is important to notice that the different conceptions of causation are not mutually exclusive. For example, a probabilistic approach does not exclude sufficient conditions altogether; a sufficient cause is one that raises the probability of its effect occurring to one. A counterfactual where a factor makes all the difference\(^2\) is equal to a necessary condition. (See Box 1.1 and Table 1.1.)

### Box 1.1 Some criteria for causation

- Sufficient conditions for an event
- Insufficient but necessary part of a sufficient and necessary condition for an event
- Insufficient and non-redundant part of an unnecessary but sufficient condition for an event (INUS)
- Raised probability for an event (non-deterministic)
- Counterfactual: the condition (cause) makes a difference with respect to the effect

### Table 1.1 Conceptions of causation with regards to determinism

<table>
<thead>
<tr>
<th>Deterministic conception of causation</th>
<th>Non-deterministic conception of causation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sufficient condition</td>
<td>Probabilistic</td>
</tr>
<tr>
<td>Insufficient but necessary part of a sufficient and necessary condition</td>
<td>Counterfactuals 2</td>
</tr>
<tr>
<td>INUS</td>
<td></td>
</tr>
<tr>
<td>Counterfactuals 1</td>
<td></td>
</tr>
</tbody>
</table>

2. If C does not occur, E does not occur (*sine qua non*).
Might the complications of causation be avoided by referring to explanations? Might there be an imperative to find explanations of phenomena significant in the biomedical sciences?

**Scientific explanation**

From the time of Aristotle, philosophers have realized that a distinction could be made between two kinds of scientific knowledge; roughly, knowledge *that* and knowledge *why*. It is one matter to know *that* myocardial infarction is associated with certain kinds of pain (angina pectoris); it is a different matter to know *why* this is so. Knowledge of the former type is descriptive; knowledge of the latter type is explanatory, and it is explanatory knowledge that provides scientific understanding of the world (Salmon 1990).

How, then, do we explain the phenomena studied in the biomedical sciences? For example, how do we explain the change in haematopoietic cell growth in a medium when its temperature changes? What criteria do we have for something to be acceptable as a scientific explanation? The standard answer to questions such as these is that we explain something by showing how we could expect it to happen according to the laws of nature (*nomic expectability*) (Hempel 1965). The haematopoietic cell growth is explained by the laws that govern haematopoietic cell growth and the initial conditions, including the type of medium, the humidity and the pressure. Accordingly, a singular event is explained if (a description of) the event follows from law-like statements and a set of initial conditions.

When a phenomenon is explained by deducing it from laws or law-like statements, the sequence of deductive steps is said to follow a deductive–nomological model (DNM) that turns an explanation into an argument where law-like statements and initial conditions are the premises of a deductive argument.

*Deductive nomological model of explanation:*

<table>
<thead>
<tr>
<th>Premise 1: Initial conditions</th>
<th>Type of medium, humidity, light, temperature</th>
</tr>
</thead>
<tbody>
<tr>
<td>Premise 2: Universal law(s)</td>
<td>Laws of haematopoietic cell growth</td>
</tr>
<tr>
<td>Conclusion: Event or fact to be explained</td>
<td>Greater growth due to temperature increase</td>
</tr>
</tbody>
</table>
In other words, we explain a phenomenon by subsuming it in a law. For this reason DNM is often referred to as ‘the covering law model of explanation’. One reason for the prominent position of DNM is its close relation to prediction. A deductive–nomological explanation of an event amounts to a prediction of its occurrence.

However, DNM incurs challenges. One is that DNM allows for symmetry. For instance, certain conditions of a growth medium for cells (temperature, humidity, light, etc.) can be explained by the growth rate of haematopoietic cells (in this medium), given the same laws. We like to think that there is an asymmetry between cause and effect (that is, what is considered to be a cause leads to an effect, and not the other way round).

Moreover, if the biomedical sciences can provide explanations only when phenomena subsume under deterministic laws of nature, then there are innumerable phenomena that cannot be explained. For instance, we tend to say that lung cancer can be explained by smoking, despite there being no strict law stating which smokers will develop lung cancer. The answer to this objection is straightforward and entails replacing deterministic laws with probabilistic statements. This engenders the deductive–statistical model (DSM) of explanation, which has the form:

**Deductive statistical model of explanation:**

<table>
<thead>
<tr>
<th>Premise 1: Initial conditions</th>
<th>Premise 2: Statistical laws</th>
<th>Conclusion: Event or fact to be explained</th>
</tr>
</thead>
<tbody>
<tr>
<td>Having sinusitis</td>
<td>Taking antibiotics probably leads to recovery</td>
<td>People taking antibiotics will recover</td>
</tr>
</tbody>
</table>

DSM is a version of DNM that supports explanations of statistical regularities by deduction from more general statistical laws (instead of deterministic laws). However, DSM cannot explain singular events, such as Mr Hanson recovering from a sinusitis after taking antibiotics. DSM can only explain why people taking antibiotics will recover (in general). In order to explain singular events in terms of statistical laws, one may refer to the inductive–statistical model (ISM) of explanation. Hence, ISM can explain likely events inductively from statistical models.
CHAPTER 1

*Inductive statistical model of explanation:*

<table>
<thead>
<tr>
<th>Premise 1: Initial conditions</th>
<th>Mr Hanson has sinusitis and takes antibiotics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Premise 2: Probability ( r ) of event, given 1</td>
<td>The probability of recovery in such cases ( r \approx 1 )</td>
</tr>
<tr>
<td>Induction: Event or fact to be explained</td>
<td>Mr Hanson will recover</td>
</tr>
</tbody>
</table>

Table 1.2 summarizes the traditional models of explanation, DNM, DSM and ISM. Common to all the models is that explanations are arguments (deductive or inductive) and that they are based on initial conditions and on law-like statements, either deterministic or statistical (номic expectancy). The standard form of each such argument is:

Premise 1: Initial conditions
Premise 2: Law-like statements
Implication: Event or fact to be explained

Most explanations in the biomedical sciences appear to fall under these models.

<table>
<thead>
<tr>
<th>LAWS</th>
<th>SINGULAR EVENTS</th>
<th>GENERAL REGULARITIES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Universal laws</td>
<td>DNM</td>
<td>DNM</td>
</tr>
<tr>
<td>Statistical laws</td>
<td>ISM</td>
<td>DSM</td>
</tr>
</tbody>
</table>

However, these models of explanation incur many challenges. One is that arguments with true premises are not necessarily explanatory. For instance, if Hanson takes birth-control pills and Hanson is a man (initial conditions), and if no man who takes birth-control pills becomes pregnant (law), it leads deductively to the conclusion that Hanson will not become pregnant. According to DNM, taking birth-control pills then explains why Hanson cannot become pregnant, but it is intuitively wrong, because the premises are explanatorily irrelevant.

As already indicated, DNM permits symmetry. For example, DNM enables us to use plane geometry and the elevation of the sun to find the height of a flagpole from the length of its shadow as well as predict the length of the shadow from the height of the flagpole. However, as the length of the shadow clearly does not
explain the height of the flagpole, DNM does not present a set of sufficient conditions for scientific explanation.

These challenges with regard to relevance and symmetry have made some philosophers of science argue that explanations should be based on causation: to explain is to attribute a cause. According to such a causal model of explanation (CM), one must follow specific procedures for arriving at an explanation of a particular phenomenon or event:

1. Compile a list of statistically relevant factors.
2. Analyse the list by a variety of methods.
3. Create causal models of the statistical relationships.
4. Test the models empirically to determine which is best supported by the evidence.

However, these procedures revert to some of the challenges of causation. Moreover, although it is intuitively correct that to explain a phenomenon is to find its cause, it is not necessarily so. Indeed, David Hume (1711–1776) argued that causation entails regular association between cause and effect. Hume's conception of causation as regularities adds nothing to an explanation of why one event precedes another. Accordingly, Bertrand Russel (1872–1970) claimed that causation 'is a relic from a bygone age, surviving, like the monarchy, only because it is erroneously supposed to do no harm' (Russell 1959, p. 180). Defining explanation in terms of causation would enhance our ability to predict, but not to understand the phenomena (Psillos 2002). Accordingly, explanation entails more than referring to a cause; it invokes understanding, and thus, one could argue, it must include the laws of nature.

Hence, DNM, DSM and ISM, the principal models relevant to the biomedical sciences, are but three of the many models for scientific explanation.

**Modes of inference**

The biomedical sciences tend to employ three modes of inference first set forth in 1903 by Charles Sanders Pierce (1839–1914): deduction, induction and abduction.

- **Deduction** entails inference from general statements (axioms, rules) to particular statements (conclusions) via logic. If all people with type 1 (insulin-dependent) diabetes are known to have deficiencies in pancreatic insulin production (rule), and Mr D has type 1 diabetes (case), then Mr D has deficiencies in pancreatic insulin production (conclusion).
CHAPTER 1

- **Induction** is inference (to a general rule) from particular instances (cases). If all people observed with deficiencies in pancreatic insulin production have the symptoms of type 1 diabetes, and the people are all from the general population (that is, not selected subjects with other deficiencies causing the symptoms), we conclude that all people with deficiencies in pancreatic insulin production have the symptoms of type 1 diabetes.

- **Abduction** infers the best explanation. When we make a certain observation (case) we find a hypothesis (rule) that makes it possible to deduce (the conclusion). If Mr D has deficiencies in pancreatic insulin production, and all people with type 1 diabetes have deficiencies in pancreatic insulin production, then Mr D has type 1 diabetes.

The crucial aspect of deduction is whether the axioms hold, while both inductive and abductive inference are knowledge enhancing (ampliative inference). In induction we infer from some cases (conclusion) to the general rule, and in abduction there could of course be other rules that could explain what we observe; that is, other explanations may be even better. Table 1.3 illustrates the differences between these three modes of inference.

Table 1.3 Modes of inference

<table>
<thead>
<tr>
<th></th>
<th>Deduction</th>
<th>Induction</th>
<th>Abduction</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Deduction</strong></td>
<td>All Fs are Gs (rule)</td>
<td>All Ss are Fs (case)</td>
<td>All Ss are Gs (conclusion)</td>
</tr>
<tr>
<td></td>
<td>All balls in this urn are red</td>
<td>All balls in this particular sample are taken from this urn</td>
<td>All balls in this particular sample are red</td>
</tr>
<tr>
<td><strong>Induction</strong></td>
<td>All Ss are Gs (conclusion)</td>
<td>All Ss are Fs (case)</td>
<td>All Ss are Fs (case)</td>
</tr>
<tr>
<td></td>
<td>All balls in this particular sample are red</td>
<td>All balls in this particular sample are taken from this urn</td>
<td>All balls in this particular sample are taken from this urn</td>
</tr>
<tr>
<td><strong>Abduction</strong></td>
<td>All Fs are Gs (rule)</td>
<td>All Ss are Gs (conclusion)</td>
<td>All Ss are Fs (case)</td>
</tr>
<tr>
<td></td>
<td>All balls in this urn are red</td>
<td>All balls in this particular sample are red</td>
<td>All balls in this particular sample are taken from this urn</td>
</tr>
</tbody>
</table>
What science is about

The biomedical sciences are about this world and its biomedical phenomena. However, what is this world? Many scientists find this question odd, even irrelevant. We deal with viruses, cells, substances and the effects of interventions, and it is clear to most of us that cells exist and that they more or less correspond to our theories. However, in history there are innumerable examples of situations where convictions of the reality of the entities of our theories, such as phlogiston (proposed by the German physician and alchemist Johann Joachim Becher, 1635–1682), ether and ‘cadaver poison’ (Ignaz Semmelweiss, 1818–1865), have been replaced by new entities and new convictions. How can we be sure that the world is as our scientists portray it, and how can we explain that our theories change?

Scientific realists hold that successful scientific research characteristically enhances knowledge of the phenomena of the world, and that this knowledge is largely independent of theory. Furthermore, realists hold that such knowledge is possible even in cases in which the relevant phenomena are not observable. According to scientific realism, you have good reason to believe what is written in a good contemporary medical textbook because the authors had solid scientific evidence for the (approximate) truth of the claims put forth about the existence and properties of viruses and cells and the effects of interventions. Moreover, you have good reason to think that such phenomena have the properties attributed to them in the textbook, independent of theoretical concepts in medicine.

Consequently, scientific realism can be viewed as the sciences’ own philosophy of science. On the other hand, scientific antirealism holds that the knowledge of the world is not independent of the mode of investigation. A scientific antirealist might say that photons do not exist. Theories about them are tools for thinking. They explain observed phenomena, such as the light beam of a surgical laser. Of course, the energy emitted from a laser exists, as well as the coagulation, but the photons are held not to exist. The point is that there is no way we can know whether the world is independent of our investigations and theories.

One may distinguish several levels of scientific realism. A weak notion of scientific realism holds that there exists a real world independent of scientific scrutiny, without advancing any claim about what it is like. A stronger notion of realism argues that not only does the world exist independently of human (scientific) enquiry, but the world has a structure which is independent of this enquiry.
An even stronger notion of scientific realism holds that certain things, including entities in scientific theories such as photons and DNA, exist independently of humans and their enquiry of the world. Accordingly, the scientific realist claims that when phenomena, such as entities, states and processes, are correctly described by theories, they actually exist.

Realism is common sense (and certainly 'common science'), as we do not doubt that the phenomena we study exist independently of our investigations and theories. However, how can this intuition be justified? This is where the philosophical challenges start. Three arguments justifying scientific realism may be advanced: transcendental, high-level empirical and interventionist.

- **The transcendental argument** asks what the world must be like to make science possible. Its first premise is that science exists. Its second premise is that there must be a structured world independent of our knowledge of science. There is no way that science could exist, considering its complexity and extent, if the things science describes did not exist (Bhaskar 1997). Hence, the argument reasons from what we believe exists to the preconditions for its existence. Even when science is seen as a social activity, how could this activity exist without the precondition that the world actually exists? That is, science is intelligible as an activity only if we assume realism. However, one premise of the argument is that science expands our knowledge of the world and corrects errors. But how do we know this? Furthermore, how can we reason from what we believe to exist to the conditions of its existence? The answer is that we do so through thought experiments. We could not think of the effects of certain microbiological events without the existence of DNA. From this we argue that the existence of DNA is a necessary condition for the microbiological events. But what guarantees that the reason that we cannot think of the microbiological events without the existence of DNA is not due to the limits of scientific imagination?

- **The high-level empirical argument** contends that scientific theories are (approximately) true because they best elucidate the success of science. The best way to explain progress and success in science is to observe that (1) the terminology of mature sciences typically refers to real things in the world, and (2) the laws of mature sciences typically are approximately true (Putnam 1981). However, this is an abductive argument, where we argue from the conclusion (science has
success) and the rule (if science is about real things, then it has success) to the case (science is about real things). Abductive arguments are knowledge expanding, and there may be other explanations that are better, but that are yet not available to us.

- The interventionist argument holds that we can have well-grounded beliefs about what is on behalf of what we can do (Hacking 1983). We can use intervention to test whether the entities of our scientific theories exist. If a theoretically induced intervention does not work, it does not exist, but 'if you can spray them, then they are real'. Hence, you can test whether something is real. One problem with the interventionist argument is that it is not robust with respect to explanation. If you test whether ghosts are real by spraying 'them' with red paint, you may conclude that ghosts are not real. However, how do you know that this is the right method to show that ghosts are real? Could it not be that red paint does not adhere to ghosts, whereas yellow paint does?

Scientific realism, which most scientists find common sense, is exasperatingly difficult to justify. One could, of course, dismiss the whole question by arguing that observable results are what matters, and whether entities of our theories, be it photons or arthritis, are real does not matter. However, at certain points a scientist may reflect upon the nature of being (ontology) of the entities studied.

**Scientific rationality**

Rationalism is the position that reason takes precedence to other ways of acquiring knowledge. Traditionally, rationalism is contrasted with empiricism, claiming that true knowledge of the world can be obtained through sensory experience. In antiquity ‘rationalism’ and ‘empiricism’ referred to two schools of medicine, the former relying primarily on theoretical knowledge of the concealed workings of the human body, the latter relying on direct clinical experience.

One might argue that the demarcation between rationalism and empiricism remains relevant in clinical practice but not in science. There are many examples of cases in which treatments established on rationalistic ground, such as ligation of arteria mammaria interna as a treatment for angina pectoris, have been revealed by empirical studies to be without effect (beyond placebo). Correspondingly, established treatments induced from experience have been revealed to be without
effect or even to be detrimental. However, modern biomedical scientists tend to rely on rationality as well as on experience in their work. Hypotheses may be generated on rational grounds (the substance S should have the effect E because it has the characteristics X, Y and Z), and theories are tested empirically, such as in animal models or in randomized clinical trials.

Nevertheless, the enduring rationalism–empiricism debate still seems relevant in the biomedical sciences because there are limits to scientific methodology. There may be ethical reasons, such as reluctance to use placebo surgery, which limits empirical research, or there may be lack of knowledge with respect to mechanisms, limiting a rationalistic approach, such as when we wish to test a substance that appears to have promise in eliciting a desired effect, but for which we lack the knowledge of why it should work.

Theory testing

The author of one of the most prominent Hippocratic writings, The Art (of medicine), identified three challenges to medical treatment and research: (1) the obtained effects may be due to luck or accident (and not intervention); (2) the obtained effect occurs even if there is no intervention; and (3) the effect may not be obtained despite intervention. In the terminology of causation, we are faced with the challenges that the intervention is not a necessary condition (2) and not a sufficient condition (3) for the effect, and that there may be a probabilistic relationship between intervention and effect or there may be other (unknown) causes of the effect (1). Today, almost three millennia later, we still struggle with the same kind of question: how can we be certain that our theories and hypothesis of the world are true, given the large variety of possible errors?

The standard answer to the question is to put the hypothesis to an empirical test according to the hypothetical–deductive method. The hypothetical–deductive method is the scientific method of testing hypotheses by making predictions of particular observable events, then observing whether the events turn out as predicted. If so, the hypothesis is verified (confirmed), and if not, the hypothesis is refuted (disconfirmed, or falsified). The steps of the hypothetical–deductive model are:

1. State a clear and experimentally testable hypothesis.
2. Deduce the empirical consequences of this hypothesis.
3. Perform empirical experiments (in order to compare their results with the deduced empirical consequences).
4. If the results concur with the deduced consequences, one can conclude that the hypothesis is confirmed, otherwise it is refuted.

According to the traditional interpretation of this model, hypotheses can be confirmed and scientific knowledge is accumulated through the verification of ever more hypotheses (verificationism) (Table 1.4).

| Table 1.4 Simplified comparison between the structure of verification and falsification |
|---------------------------------------------|---------------------------------------------|
| Verification                                | Falsification                               |
| 1. Hypotheses                              | A is better than B                          | B is better than A                          |
| 2. Deduced empirical consequences           | If A is better than B, we must observe that A gives better results than B in the empirical setting | If B is better than A, we must observe that B gives better results than A in the empirical setting |
| 3. Experiments and observations             | We observe that instances where A is used obtain better results than B | We observe instances where A is used obtain better results than B |
| 4. Conclusion                              | The experiment confirms the hypothesis      | The experiment refutes the hypothesis, and lends support to the alternative hypothesis (A is better than B) |
| Logical structure                          | If p, then q                                | If p, then q                                |
|                                            | q not q                                     | not q                                       |
|                                            | p (Confirming the antecedent)               | not p (Modus tollens)                       |

However, as Karl Popper (1902–1994) showed, this approach cannot avoid the challenges mentioned above. First, the verification of a hypothesis presupposes induction, which is not warranted. Secondly, the logical form of the model is not sound.

Moreover, Popper was critical of the early twentieth century lack of standard criteria for establishing scientific truth, and of the corresponding trend to use
(scientific) authority to decide what was true, which made it difficult to differentiate science from other social activities. Popper’s radical turn was to avoid stating explicit (authoritative) criteria for truth and to provide stringent procedures for testing hypotheses. Furthermore, he broke with the ideal of final determination of the truth, and provided a scientific knowledge base of non-truths (falsified hypotheses). Scientific knowledge progressed through enlarging the graveyard of falsified hypotheses. The method of refutation rather than that of verification makes all truth provisional, conjectural and hypothetical. According to Popper, experiments cannot determine theory, only delimit it. Theories cannot be inferred from observations. Experiments only show which theories are false, not which theories are true. (See Box 1.2.)

Box 1.2 Popper on ‘The success of refutation’

‘Refutations have often been regarded as establishing the failure of a scientist, or at least of his theory. It should be stressed that this is an inductivist error. Every refutation should be regarded as a great success; not merely a success of the scientist who refuted the theory, but also of the scientist who created the refuted theory and who thus in the first instance suggested, if only indirectly, the refuting experiment.

Even if a new theory (such as the theory of Bohr, Kramers, and Slater) should meet an early death, it should not be forgotten; rather its beauty should be remembered, and history should record our gratitude to it – for bequeathing to us new and perhaps still unexplained experimental facts and, with them, new problems; and for the services it has thus rendered to the progress of science during its successful but short life.’

(Popper 1963)

Hypothetico-deductive method

In empirical fields, the hypothetico-deductive approach (see Figure 1.2) is used almost daily, often without a thought. The control experiment is a typical example. Can a possible effect or an absent effect have a trivial explanation? Might changes over time...
or in titrations of solvents produce effects, or might the cells have failed to respond at all? Control experiments are included to rule out such trivial explanations.

In clinical research involving trials of new drugs, patients' symptoms may be strongly influenced by the treatment situation, and a placebo may cause an effect. So, a placebo group is included to rule out (falsify) this hypothesis. Correspondingly, tests are conducted double blind, to falsify the hypothesis that the observed effect of a treatment is due to the expectations of the experimenter.

Correspondingly, statistical tests are performed to falsify the hypothesis that a result is obtained owing to biased selection (as of patients). They include assessment of whether recorded differences between groups are random. This is done by setting up the contention of a null hypothesis $H_0$ that there is no difference between the groups and thereafter assessing the probability for its being true. If that probability is very small, the null hypothesis is rejected, which strengthens the principal hypothesis that there is a real, not random difference.

A hypothesis must have testable implications if it is to have scientific value. If it is not testable, and thus not falsifiable, then it is not science, as Popper contended. The lack of adequate methods often hinders scientific progress, because limited testability restricts what can be of scientific enquiry. Therefore, often, a new, more powerful method propels science ahead. Suddenly, new research areas open up. Outstanding instances include Kary Mullis' development of the polymerase chain reaction (PCR) in molecular biology, recognized by a Nobel Prize in 1993, and the development of the patch clamp in neurobiology by Erwin Nehr and Bert Sakmann, recognized by a Nobel Prize in 1991.

The development of hypotheses is closely associated with the development of models and the planning of experiments. Many hypotheses can be shown to be too imprecise and ambiguous to be rejected and consequently cannot be challenged as

Figure 1.2 The hypotheticodeductive method
CHAPTER 1

Popper requires. Formally, there should be two alternative hypotheses that mutually exclude each other. Then, a decisive experiment should be done to distinguish between them. If a hypothesis is falsified, this may lead to the development of new hypotheses, which in turn can be tested.

Moreover, a hypothesis should have the power to explain. It should relate to existing, generally accepted theoretical basis of the field. There must be good grounds to reject established theories, such as an accepted law of nature. A theory that flounders on the grounds of falsifying experiments may be defended by its remaining adherents who contend that it puts forward ad hoc hypotheses. Whenever newer observations so indicate, it is advisable to modify a hypothesis. In fact, that is part of the scientific process. However, an ad hoc hypothesis differs from a modified hypothesis in that it is not testable and often is more complicated and consequently usually hinders rather than promotes scientific development.

Although falsification has become common ground in empirical biomedical research, its strengths and weaknesses are not always appreciated. According to Popper, a theory or hypothesis should be bold and far-ranging. Its empirical content should be high, that is, it should have great predictable power. Furthermore, the hypothesis should be testable with a radical test. If the results from the empirical test support the hypothesis, it is corroborated (but not verified); if not, it is falsified.

Regardless of how influential Popper’s approach has been and still is in empirical research in the biomedical sciences, falsificationism has been severely criticized. Four challenges to it are frequently mentioned:

- First, when we falsify theories, we do not test their prospective robustness. We only test them on past evidence.
- Secondly, a severe test is one that is surprising and unlikely on present evidence. However, to set up a test that is unlikely, we base our knowledge on what is likely, and in so doing we rely on induction. Accordingly, if one really defies induction, there is no reason to act on corroborated theories or hypotheses, because doing so would be induction.
- Thirdly, when we falsify a theory it is on behalf of empirical observations. However, observational statements should also be fallible, and hence the falsification of a theory may be erroneous (if the observational statements are not true).
Fourthly, Popper’s method can lead to falsification of robust and fruitful theories with high empirical content, such as due to errors in the test procedure. In practice we do not falsify a potentially fruitful theory on the basis of only one observation. That is, a theory that is not corroborated is not necessarily falsified. We design new experiments and ad hoc hypotheses to investigate or explain the falsifying observation. Hence, in practice we falsify not single theories, but rather groups or systems of theories.

**Aim of science: reducing uncertainty**

The primary aim of science is to increase knowledge in order to explain, understand and intervene. We need scientific knowledge to reduce our uncertainty. It is convenient to differentiate among four kinds of ‘uncertainty’: risk, uncertainty, ignorance and indeterminacy (Table 1.5).

Table 1.5 The modes of uncertainty

<table>
<thead>
<tr>
<th>Probability/Outcome</th>
<th>Known Outcome</th>
<th>Unknown Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Known probability</td>
<td>Risk</td>
<td>Ambiguity</td>
</tr>
<tr>
<td>Unknown probability</td>
<td>Uncertainty</td>
<td>Ignorance</td>
</tr>
</tbody>
</table>

**Risk** is when the system behaviour is basically well known, and the chances of different outcomes can be defined and quantified by structured analysis of mechanisms and probabilities. It is a task of science to find the outcomes of a given situation or intervention and its probability; for example, the outcome with respect to survival rate (with respect to cardiovascular disease) when using statins prophylactic for patients with type 2 (non-insulin-dependent) diabetes.

**Uncertainty** is characterized by knowledge of the important system parameters, but not of the probability distributions. We may know the major outcomes of a certain intervention, but we do not know their respective probabilities. There may be many sources of uncertainty, such as uncertainty in reasoning: how to classify a single case with regards to general categories. There may also be uncertainty in biomedical theory, such as when all mechanisms in a certain field are not known in detail, or because of multifactor causation. Moreover, diseases may be complicated, and it can be difficult to know and understand all of their causes. In the case of uncertainty the main task of science is to provide the probability distributions.
Ignorance is the case when we know neither the outcomes nor their probability distributions. The aim of science is, of course, to find both. However, this is difficult, as we do not know what we do not know.

Even though we would be able to reduce all ignorance to uncertainty and all cases of uncertainty to risk, we still might be subject to indeterminacy. It is not always a question of uncertainty due to imprecision (which is assumed to be narrowed by more research), but also a question of how we classify things according to different properties or criteria. When we classify myocardial infarction according to a set of clinical criteria, we will have a different perspective than if we classify it according to the level of troponin in the blood. Likewise, if we investigate pain in terms of neural activity or according to a visual analogue scale (VAS), the risk, uncertainty and ignorance may differ. Processes may not be subject to predictable outcomes from given initial conditions, owing to imprecise classification.

The empirical turn in philosophy of science

Although many of the challenges within traditional philosophy of science (as discussed above) have been addressed, and progress has been made, interesting and fruitful contributions have been fuelled through empirical studies of sciences and scientists. Intimate empirical studies have revealed characteristic social aspects of science. In particular, the norms and activities of scientists have been shown to be basically similar to the norms and activities of other groups in society (Stengers 2000).

The traditional philosophy of science has been theoretical and focused principally on the products of science, that is, knowledge and its conceptual preconditions. The newer approaches are empirical and focus on the social processes of science (and its interaction with material matters). A seminal and famous study of scientific activity (Kuhn 1969) showed that knowledge is not accumulative and that science does not develop in a linear manner. Instead, it evolves in an abrupt way (scientific revolutions) with intervening quiescent periods.

Inspired by Kuhn’s paradigmatic conception of scientific progress and by Wittgenstein’s theories on rule following and language games (Wittgenstein 2001), a series of science studies, termed the sociology of knowledge (SoK) movement, emerged. The key issue is to show that science is a social activity that follows
social patterns in the same manner as do other groups in society. The question of how things are in the world cannot be addressed without the question of how the social group comprising scientists conceives of these things. Things, be it photons or DNA, cannot be attributed a role in our world independent of symbols and meaning.

Hence, while the traditional philosophy of science had procedural criteria of demarcating science from non-science, such as Popper’s criteria of refutation, SoK applies social criteria. Whereas the normative aim of traditional science studies was to free science from power inherent in the social structures among scientists and in society, SoK strives to disclose power within the scientific society and to emancipate.

In many respects, the key issue in the classical philosophy of science has been the relationship between scientific theories and nature. In SoK the focus is on the relationship between theory and culture. In what way do scientific theories reflect social structures (instead of structures of the world)? Nevertheless, what appears to be similar in both the traditional philosophy of science and SoK is the focus on epistemological issues: in both cases the key question is what scientific theories represent. In the first case they represent patterns in nature, in the second, they represent social structures.

Later studies of science have tried to avoid this representational pattern. Their empirical studies of science have investigated not only the relationship between theories and the social processes and structures in science, but the scientific process itself, including its material premises. How do scientists behave, and how do they produce the facts of science? This may be called a processual approach (PA), according to which science is the change, restructuring, making new, and stabilizing of things and theories. What characterizes the social process of science is an interaction of methods, material, activities and processes, where negotiations lead to stabilizing and generation of facts. When species of the *Helicobacter pylori* bacteria were found to be associated with gastric and peptic ulcers, scientific debate ensued on the bases of the residing theories, and negotiations on behalf of continued empirical work confirmed that *H. pylori* is a key factor.

There is no question about what the theory represents (either nature or culture), but rather it is a question of negotiation between different scientific groups with regard to what will be considered to compromise facts. Hence, according to
the issues are not the relationship between theory and nature/culture (epistemological and representational), but what scientists regard and treat as real (ontological and processual).

1.2 PHILOSOPHY OF THE SOCIAL SCIENCES

A significant part of the overall spectrum of healthcare problems comprises matters that principally are not biological. Should we wish to find why patients do not take prescribed medicines, why wrong medicines are given in hospitals, or why it is difficult to obtain fully informed consent for trials or treatment, we cannot search for answers in human biological research, but instead must turn to the methods of the social sciences.

So, it is essential to know the ways in which the philosophies of the social sciences and the biological sciences differ, so that we do not erroneously use the criteria of one area to judge another. In the social sciences, many different methods are used, and there are various schools of theory. So, the discussion here comprises a brief introduction and does not cover the broad scope of methods and schools of theory.

Interpretation, understanding and explanation

The social sciences differ from the biological sciences in two respects:

- they entail greater elements of interpretation that often enter into compilations of data
- in many cases, a result is an understanding, not an explanation.

Explanation and understanding

The principal goal of inquiry in the biological sciences is to elicit explanations of phenomena studied. One might, for instance, seek the cause of a particular manifestation of a disease.

Some projects in the social sciences also seek causal explanations of social phenomena, but many seek instead an understanding. Understanding is a form
of knowledge that enables us to know why a person or a group behaves in a particular way, why and how they experience a specific situation, how they themselves understand their way of life, and so on. We attain understanding through interpretation.

The distinction between explanation and understanding was first expressed by the German philosopher, psychologist and educator Wilhelm Dilthey (1833–1911), who believed that these two ways of understanding the world were characteristic of the natural sciences and the human sciences (Geisteswissenschaften), respectively. However, the distinction between explanation and understanding is not as distinct as many believe. Many theories of the social sciences include elements of both causal explanation and non-causal understanding.

Interpretation

All content-bearing objects and statements can be interpreted. People express themselves not just in speech, writing and deeds, but also in architecture, garden design, clothing, etc. If, for example, we enquire into where and why institutions for psychologically ill patients were built, we will find that the history reflects varying understandings of psychological illness. The architecture of the asylum is content bearing. However, here we will focus on the interpretation of texts and other linguistic statements, as it is germane in the discussion of the theory of interpretation, often called hermeneutics.

Interpretation may have many goals, but in general we seek to fathom the information content of the content-bearing material. The various theories of interpretation are based on differing concepts of the nature of content and how it should be located. Is there content in a statement itself, in the thoughts of the person making the statement, in the social structure in which the statement is made, etc.? These differences are germane when analysing the validity of specific methods of the social sciences, but are of lesser importance here in the general discussion of interpretation.

The question of whether one obtains a true interpretation of a text is old. All written religions have sets of hermeneutic rules for interpreting the content of holy texts. For example, in Christian theology, biblical exegesis concerns interpretation of the scriptures.
In modern times, interest arose in the interpretation of secular statements, first as part of literary and historical research, and then as a part of research in the social sciences.

The goal of the various hermeneutic methods that have been developed is to arrive at an understanding of content that can be defended as a valid, intersubjective understanding. That is, it is an understanding that can be substantiated and discussed rationally.

As Popper pointed out, the elements of interpretation enter into all observations and thereby into all forms of science. We lack direct access to the world ‘as it is’ through our senses. We always view the world through a theoretical filter, and all observations are theoretically loaded. For example, when we say that the sun rises, we reflect the influence of the old geocentric world view in which the sun circled the Earth. And the ‘description’ that a pathologist gives of a histological preparation seen by microscope is to a large extent an interpretation based on theories of cells, inflammation, etc.

The hermeneutic circle, understanding horizon and ‘double hermeneutics’

The hermeneutic interpretation of a text rests on individual parts as well as on the understanding of each individual part related to the whole. Neither an individual part nor the whole text may be interpreted without reference to each other. So, interpretation is circular, the hermeneutic circle. In principle, this circle cannot lead to certain closure, as we will never know whether a deeper analysis of the text may change our interpretation of it. The problem of attaining valid, intersubjective interpretation has long been and still is discussed, and optimistic interpretation theoreticians speak of a hermeneutic spiral that implies that interpretation gets better and better. At the pragmatic level, the problem of the hermeneutic circle is less worthy of attention, as agreement on the meaning of a text usually can be more easily attained.

The concrete interpretation is also influenced by the interpreter’s ‘horizon of understanding’, a concept from *Wahrheit und Methode*, the principal work of German philosopher Hans-Georg Gadamer (1900–2002). Gadamer argues that before I have begun a conversation with another person or begun to interpret a text, I already have bias about them based on my horizon of understanding, a collective
term for my world view. My horizon of understanding builds up throughout my life and comprises my understanding of particular words, the connotations that particular words and concepts hold for me, and so on. For a resident of London, the word 'city' connotes financial affairs, while for people elsewhere, it simply connotes an urban concentration of population. Two people engaging in a conversation may believe that they have understood each other without actually having done so. Full understanding is possible only when two conversing people have acquired each other's horizons of understanding ('fused horizons'). Hence there may be a problem of interpretation, as in interviews in the social sciences, which often are too short for the interviewer to understand the interviewee's horizon of understanding. Consequently, a vital part of the interview comprises an effort to find out how the interviewee uses and understands words and concepts in the area being discussed.

Furthermore, English sociologist Anthony Giddens pointed out that within the social sciences, research comprises a 'double hermeneutics' (Giddens 1976, 1990). In reality, the social sciences research interprets interviewees' interpretations of their own understandings, and parts of their understandings arise through concepts that they have acquired from the theories of the social sciences (such as the Marxist concept of class or the incest taboo of psychology). Hence, there is a complex interaction between the interpretations of the researcher and the interviewee, which is why an additional level of interpretation often may be needed to focus on how an interviewee's self-image is affected by the theories of social science. Consequently, an interviewee may be misunderstood if the interviewer does not take such reflections into account.

**Power, ideology and interests**

Our interpretations of the statements and deeds of others are influenced by aspects in addition to our horizons of understanding. The German philosopher Jürgen Habermas (1929–) pointed out that power, ideology and interests play leading roles. Usually, we are not neutral or objective observers, but interpret according to our power of position, our ideology and the interests we wish to further (Habermas 1986).

In Habermas' view, ideology is not restricted to political ideology. An ideology is simply a set of assumptions that further the interests of a particular group in
society. For example, the assertion that ‘an extensive hospital system is essential in healthcare’ is an apolitical ideology that in addition to safeguarding the interests of patients, furthers the interests of doctors and other healthcare professionals.

A difficulty with ideologies is that they are often concealed, as we neither are aware that we have them nor know where they came from. So, behind our backs, they influence our actions and our interpretations. Consequently, Habermas maintains that the principal task for the critical social sciences is to identify prevailing ideologies so we may be freed from them.

Validity

In the above, we have discussed problems widely recognized, that an interpretation and the understanding that we attain through interpretation can never be ‘a final truth’ concerning the meaning of a particular statement (unless the statement is extremely simple). So, we are obliged to ask how we can judge the validity of a scientific interpretation. The simple answer is that if a researcher has been aware of these problems and has taken the best possible steps to avoid or avert them (such as by trying to identify which ideologies and interests have influenced the various elements of the research process), there are grounds to rely on the interpretation; not because it is of necessity true, but because it comprises a well-founded hypothesis without significant sources of errors in the research process.

Reductionism and emergence

Some biological researchers contend that there is no need for social scientific interpretation because in the final analysis, all knowledge can be reduced to facts about physical conditions. Social phenomena can be reduced to group psychology, which in turn can be reduced to individual psychology, which in turn can be reduced to neurology, which in turn can be reduced to cellular biology, and so on, until we reach the physical level at which prevailing physical laws provide explanations for all phenomena observed at higher levels. This view, called reductionism, is in strong dispute.

So, here, it is crucial to distinguish between methodological reductionism and general reductionism. In some research projects, methodological reasons may
dictate the exploration of one or more factors that can influence the phenomenon of interest without indicating that other factors are unimportant. We have no methods that can acquire data and at the same time investigate ‘the whole’. Of necessity, our attention must be focused on something more specific. Methodological reductionism can be meaningful and necessary, even though we refute general reductionism. If, for instance, we wish to examine a biological relationship, it may be necessary to ignore an ancillary social relationship. Conversely, if we examine a social relationship, it may be necessary to ignore a biological relationship.

Methodological reductionism is itself straightforward, as long as the factors that we examine are sensible. It becomes problematical only when a set of factors is systematically excluded, such as by ignoring the correlation between poverty, social deprivation and disease.

There are many arguments against reducing social phenomena to physics, two of which are summarized here. The first problem confronting the reductionist is that it is doubtful that individual psychology can be reduced to neurophysiological processes. Dispute persists on the precise description of the relationship between psychological phenomena and cerebral activity, and today we seem no closer to solving the ‘mind–brain’ riddle than we were a century ago. If this link in the reductionistic chain fails, reductionism as a whole cannot be carried out.

The other problem for the reductionist is that many social phenomena are emergent, that is, they are socially not reducible as they occur at particular social levels and have no meaning when reduced to lower levels (individual psychology, neurology, etc.).

Paper money, for example, is an emergent social phenomenon. A £10 banknote has no value itself (unless you keep it for its portrait of scientist Charles Darwin). It cannot be exchanged for gold or other objects of value at the central bank. But it is integrated in social relationships that enable it to be exchanged for goods or services worth 10 pounds. Otherwise, it is just a small, rectangular scrap of paper.

Emergence at the social level also may be ascribed to a particular set of social conventions or formalized laws. For instance, most societies have the institution of marriage, but the concrete implications of being married and the social effects of it vary from society to society. The human penchant to form pair relationships
might be reduced to the biological level, but the concrete institution of marriage in a particular society cannot be similarly reduced. However, it is clear that the concrete, non-reducible institution of marriage affects human actions and considerations, so a full description of these actions and considerations is possible only on the social level.

If the antireductionists are right, scientific effort in the social sphere is useful, and it may employ methods that differ from those applicable at lower levels.

Generalization

Generalizing statistics are often useful in research projects that use quantitative methods. Whenever we take samples from a well-defined population, we express the statistical confidence interval of the results and consequently permit their general extension to other similar populations. In principle, that implies that results from research conducted in the USA may be directly applicable to choices of treatments in Norway. However, it is worth noting that such generalization of results is acceptable only when we have grounds to assume that the populations are in fact similar, as by assuming that there is no biological difference between Americans and Norwegians.

Generalization may be used in much the same way in quantitative social science research, but statistical methods cannot be used in research that is not quantitative. Does this imply that understanding attained in social science research cannot be generalized? Were statistical generalization the only form of generalization available, understanding could not be generalized. Yet there is a form of generalization that is not quantitative and is frequently used across all the sciences. It is theoretical or conceptual generalization, sometimes called transferability. We often generalize, not in exact numbers, such as the cure rate for a particular drug, but rather within a conceptual or a theoretical frame of understanding. For instance, when teleological explanations based on the theory of evolution are used in biology, they rest upon a theoretical generalization of the theory of evolution, not upon a statistical generalization. Social scientific concepts and theories may be generalized in the same manner.

In all forms of generalization, both statistical and conceptual, it is important to keep in mind that conditions change with time. Generalizations that
once were valid can be rendered invalid if there are changes in the supporting biological conditions, such as the resistance patterns in bacteria or the structures of families.

REFERENCES


FURTHER READING

CHAPTER 1