bioaccumulation soon become extremely complex, and for that reason they are not treated in detail here. Hamelink (1977) and Moriarty (1984) provide good discussions of the strengths and weaknesses of bioconcentration models.

The purpose of such models is twofold. First, if they can be validated by experimental findings (i.e. if the predictions of the models correspond with actual observations), they provide the means for making useful predictions based on comparatively simple experimental measurements. In other words, they can eventually become a substitute for actual experimentation, which may be time-consuming and expensive. Second, they can provide valuable information about the mechanisms of bioaccumulation. If, for example, an experimental finding does not agree with a prediction of a model, it indicates that one or more of the assumptions in the model is wrong, and thus focuses attention on areas which require further investigation. Although it is by no means clear that any existing model is of general application, studies on bioaccumulation and its mechanisms are of great practical importance. For instance, Moriarty (1984) has convincingly argued that the lack of understanding of bioaccumulation processes in single species has serious implications for ideas about the passage of pollutants through food chains.

It is widely believed that many pollutants pass through succeeding trophic levels and accumulate in high concentrations in the tissues of long-lived predators. Although there are a small number of widely-accepted examples of this, it is by no means well established that this is a general phenomenon, even for persistent pollutants like heavy metals and refractory organics. Moriarty (1984) has discussed some of the inadequacies of current knowledge. For example, comparing tissue levels of pollutants in field populations is very likely to produce biased results if, as is usually the case, mean levels of pollutants are compared. This is because of the differences in pollutant concentrations between individuals of the *same* species; frequently, mean values are biased by a very small number of individuals which have very high concentrations; that is, the frequency distribution of pollutant concentration values is highly skewed. Further, although under experimental conditions a steady-state concentration of the pollutant in the tissues generally is eventually achieved, it is not clear that this is the case in the field. Field populations are generally exposed to lower, and more widely-fluctuating, pollutant concentrations in their environment. Clearly a comparison between tissue pollutant concentrations in two different species is invalid if they are not both at their respective steady-state concentrations. It is also true that the interpretation of field observations, and the design of experimental investigations, often rests upon unverified, and unwarranted, assumptions about what animals actually eat. Obviously in a simplified experimental system, predators will feed on prey which may not form their normal or natural diet in the field. A further example is the widespread assumption that large marine predatory fish such as tunas feed primarily

or exclusively on smaller fish such as mackerel, herring or sardine. However, the stomach contents of several hundred tunas caught in the Mediterranean during an international angling contest consisted almost entirely of crustaceans and plankton (Fowler *et al.,* 1979). This should not have been a surprise to anyone who knows anything about the biology of tunas, but this simple observation destroyed some elaborate theoretical models! Thus while the question of bioaccumulation and biomagnification along food chains is an important and interesting one, authoritative answers require the solution of several technical and conceptual problems, and a greater knowledge of the basic ecology and physiology of many species.

4.5 Evaluating Toxicological Data

The literature on the toxicity of pollutants to aquatic organisms is enormous, and yet there are several important questions which cannot yet be clearly answered. The range and variety of lethal and sublethal toxicity test methods available are huge, and increasing; as is the recorded occurrence of sublethal toxic effects whose significance can frequently only be guessed at. The number of chemicals requiring some form of risk assessment increases by hundreds each year, the majority of species have yet to be investigated, and every circumstance has its own environmental conditions which we do know will influence toxicity considerably.

Probably few readers of this book will ever themselves be actively engaged in toxicological investigations, yet probably the majority will at some time need to refer to toxicological data and evaluate its reliability and significance. It is unfortunate that a great deal of the information available is of poor quality and likely to be misleading. This section offers some simple guidelines to assist in the evaluation of toxicological data.

Measurements of lethal toxicity are, in principle, the most straightforward because the criterion of toxicity, death of the organism, is usually easily recognisable. However, much published data are of less use than it may at first appear. Probably the commonest mistake is to use simple test methods for purposes for which they are not suitable. For example, tests designed for routine screening and monitoring are of little value in comparing the toxicities of different poisons, the susceptibility of different species or the effects of environmental conditions, for reasons explained in Section 4.1. Similarly, data presented in the form of fixed-time LC50 values are of doubtful significance unless there is some indication of how close that time is to that at which the lethal threshold becomes apparent. It is still disturbingly common to see data presented in the form of cumulative mortality curves—often without even the log-probit transform, which allows median values and their confidence limits to be determined—which are incapable of distinguishing between the speed of reaction of the organisms and the inherent toxicity of the pollutant to which they are exposed. All reports should be routinely examined to see if adequate

information is given on the chemical composition of the dilution water, the volume of the test containers and how frequently the test solutions are changed; on the frequency of observations of the experiment; and whether any steps have been taken to *measure* the actual concentrations of poison in the solutions, and the other experimental variables which are known to have large influences on the final result. To the extent that such information is lacking, the significance which can be attached to the results is diminished. Finally, some checks can often be done, based on internal evidence of the report, on whether the results have been correctly computed. This should not be necessary, but experience unfortunately shows that it often is! For example, if an LC50 value is expressed as, say, 15.56±0.008 mg l^{-1}, a rapid back-calculation will probably show that to achieve this result, the experimental concentrations would have to be impossibly closely spaced. Therefore the investigator did not carry out the experiment properly, or does not understand the calculations, or both!

For sublethal toxicity experiments, the same considerations apply, but in addition there is the problem of evaluating the biological significance, if any, of the toxic effect used as the end-point of the experiment. At one extreme, any effect could be considered significant. On the other hand, arguably only those effects which are of ecological significance could be considered as important. This point has yet to be resolved by consensus, but it is important in view of the considerable demand for toxicity tests for regulatory purposes. There is a tendency, whenever a novel toxic effect is discovered, to suggest that it could be used as a toxicity test, particularly if the experimental procedure is relatively quick, simple and sensitive. However, it is important to distinguish between the study of sublethal toxicity *per se,* and the application of sublethal toxic effects as test methods for regulatory purposes. If we wish to know how poisons affect organisms at sublethal levels, what is the sequence of cause and effect at succeeding levels of biological integration from the passage of the poison molecule into the organism, its initial reaction within the cells, and the ultimate consequences of this at the level of the whole organism or the population, then we are legitimately interested in any response we can detect. If, however, we wish to develop a particular biological response into a test method for regulatory purposes, we should be interested in a much smaller range of effects. For this purpose, we require methods which offer economies of time and scale, which are ecologically relevant and which bear some relation to the behaviour of a range of key organisms exposed to a wide range of pollutants as determined by more conventional methods. Thus, since insecticides are designed to be harmful to insects and relatively harmless to other species, any sublethal response of an insect species to an insecticide is most unlikely to be of use as a general test method for regulatory purposes, since it will give little accurate idea of how non-insecticidal poisons might behave. Toxicity testing for regulatory purposes is one important branch of ecotoxicology, but no more than that. Similarly, not all sublethal toxic