A REVIEW OF PAST AND PRESENT FISH CONSUMPTION ADVISORIES

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INTRODUCTION

Transition from fish consumption advisories based on U.S. Food and Drug Administration (FDA) action levels to advisories based on U.S. Environmental Protection Agency (EPA) risk assessment procedures has caused confusion among fishery professionals, anglers, and the public. What are the differences between the older and newer advisories? How are these recommendations established, and how accurately do they actually measure the health risk associated with eating fish? Anglers and the public deserve some basic answers to these questions so that they can understand the process that can have a severe impact on sport fisheries.

RISK ASSESSMENT AND RISK MANAGEMENT

Fish consumption advisories are based on the concepts of risk assessment and risk management. Risk assessment is a scientifically based procedure used to estimate the probability of adverse health effects from a specified source under particular exposure conditions. Several methods for conducting risk assessments exist; each has different underlying assumptions. Risk management is the process of integrating risk assessment data with social, economic, and political information to decide how to reduce or eliminate the potential risks identified.

DIFFERENCES BETWEEN F.D.A. AND E.P.A.-BASED FISH CONSUMPTION ADVISORIES

Although FDA action levels and the EPA risk assessment procedures both use the principles of risk assessment and risk management, they are designed to protect different segments of the population. The purpose of FDA action levels established under the authority of the Food, Drug, and Cosmetics Act is to protect the general public from contaminants in fish shipped in interstate commerce (USEPA 1989). Action levels are developed in response to national needs and are based on national patterns of consumption that are often different than those of local sport or subsistence anglers (USEPA 1989). In contrast, the purpose of the EPA risk assessment procedure is to provide the states with a means for informing sport and subsistence anglers about the health risks associated with contaminated fish they catch from local waters (USEPA 1989). These subpopulations of anglers are potentially at greater risk than the general population because they tend to eat larger quantities of fish and because they often fish the same locations repeatedly.

Fish consumption advisories derived from the newer EPA risk-based-assessment approach generally give a much higher estimate of health risk for a given level of contaminant than those based on the FDA tolerance guidelines for several reasons. The two agencies use different risk assessment methodologies based on different assumptions (USEPA 1989), fish consumption rates vary in scope from national (FDA) to local (EPA). Also, FDA action levels are based not only on risk assessment but also on risk management considerations such as economic impacts likely to accrue to the commercial fishing industry (USEPA 1889). For example, the FDA clearly indicates that its rationale for the current 2 ppm action level for PCBs was a balance between public health protection and the economics involved in the loss of commercial fish to the consumer (USFDA 1984). In contrast, the EPA approach for fish consumption advisories gives full priority to protection of public health. That some states use different combinations of the FDA and EPA procedures to formulate their advisories further adds to the disparities in consumption advisories among states.

RISK ASSESSMENT MODELS AND CALCULATION OF RISK ASSESSMENT VALUES

Because there is a lack of reliable human epidemiological cancer data involving environmental exposures, animal bioassays provide most of the information used to predict carcinogenic effects on humans. Mathematical models are used to extrapolate from effects of the high doses administered to experimental animals to the effects of low doses on humans corresponding to levels found in

the environment. There are a number of possible models. Depending on the one chosen, the estimated increase in cancer incidence can differ by several orders of magnitude (Brown 1982; State of California 1985). The model used by EPA is a version of the linearized, multistage no-threshold model developed by Crump (USEPA 1980). This model leads to estimates of cancer risk that are very conservative (i.e., yields the highest risk values) (USEPA 1980; USEPA 1989). In addition to its conservatism in extrapolating from high to low doses, the EPA model is also conservative in extrapolating from rodents to humans and differs from the FDA in the approach used to compensate for the size difference between humans and rodents.

In any dose-response curve there is a degree of uncertainty. Thus, scientists calculate confidence limits, based on the quantity and extent of the data, that are the upper and lower estimates within which the estimate of mean risk or "best estimate" should fall. The EPA reports the increased cancer risk as the 95% upper bound estimate of the slope factor (USEPA 1980). This procedure generally leads to the highest (most conservative) estimate of the risk. If the best estimate or the lower bound estimate were used, the risk value would be much lower and could even be zero or close to zero. Thus, the numbers reported as an estimate of increased cancer risk include margins of safety and are conservative estimates of risk to human health.

The 95% upper bound is expressed mathematically as Q1*, the carcinogenic potency factor or slope factor (USEPA 1989). The formula $P=(X)(Q1^*)$ represents the increased lifetime cancer risk (P) caused by exposure to a daily dose (X) of carcinogen with a potency factor (Q1*) for 70 years. With this information, plus the size of each meal and the body size, one can calculate the EPA-derived number of meals that can be safely consumed over a given time.

QUESTIONS ABOUT RISK ASSESSMENT PROCESS

Extrapolation to Humans. Animal bioassays that use high doses of chemicals are coming under increasing criticism because many chemicals that cause cancer at high doses may not cause cancer at the low doses more comparable to human exposure (Cohen and Ellwein 1990). Extrapolation from rodents to humans has also been questioned because of differences in lifespan and metabolic rate, and biochemical and pharmacokinetic differences (State of California 1985; Ames et al. 1987).

Threshhold. The actual shape of the lower end of the carcinogen dose-response curve is hotly debated. A noncarcinogen has a threshold dose below which there is no observable detrimental effect on the animal. Conversely, a cancer may in theory develop from a single transformed cell. Therefore, cancer could develop from a non-threshold effect initiated by very small doses of a carcinogen reaching the right cell at the right time (State of California 1985).

However, even if there is no threshold, marked alterations in metabolic pathways that occur at high doses but not at low environmental doses could result in nonlinearity of the dose-response curve for some animal carcinogens at low doses (Gehring and Blau 1977; Hart and Fishbein 1986). These alterations could result in disproportionately high incidences of cancer at high doses. If a carcinogen does have a threshold or if the dose-response curve is not linear at low environmental concentrations, the cancer risk could be much less than predicted by the model.

About the only assumption on which all factions involved in the risk-assessment controversy agree is that decreasing the dose decreases the risk. EPA's present conservative approach assumes that any detectable amount of a carcinogen has the potential for inducing cancer (i.e., there is no threshold). The EPA takes this stance because cancer researchers cannot determine with any degree of certainty the minimum levels at which substances cause cancer. Another argument suggested in favor of the conservative approach is that exposure to low concentrations of a variety of substances could have an additive or synergistic effect (State of California 1985). Viewed in this manner, EPA assumes that at low environmental levels the dose-response curve is linear and, therefore, no level of exposure is free from risk.

Dose Pattern. Another conservative assumption in the EPA risk assessment process is that humans consume contaminated fish for 70 years at a constant dose (USEPA 1989). However, many compounds listed as animal carcinogens (e.g., chlorinated hydrocarbon insecticides and PCBs) have only come into existence over the last 30-50 years. Also, concentrations of many of these contaminants in aquatic systems are declining because of regulatory actions taken over the last 20 years.

A decrease in concentration has a dramatic effect on life-time cancer risk estimations. For example, average PCB levels in coho salmon (*Oncorhynchus kisutch*) fillets from Lake Michigan declined from 1.93 ppm in 1980 to 0.39 ppm in 1984 (De Vault et al. 1988). Using the EPA linear model, this decline leads to about a five-fold decrease in the estimated cancer risk. Average DDT levels in Lake Michigan bloater chubs (*Coregonus hoyi*) declined from 9.94 ppm in 1969 to 0.67 ppm in 1986 (Hesselberg et al. 1990). This decline would result in about a 15-fold decrease in the estimated cancer risk.

Types of Health Effects. Although cancer is the only health risk estimated in the EPA risk assessment procedure, other types of health risks may be associated

with eating contaminated fish. Principal among these are possible reproductive and developmental effects in young children and offspring of women who consume large amounts of fish (Jacobson and Jacobson 1988). As a result, fish consumption advisories in many states suggest that women of childbearing age and children should consume fewer fish than the rest of the population.

Uncertainty. Because of the high degree of uncertainty associated with the risk assessment process, there is a temptation to delay issuing fish consumption advisories until more reliable information is available. Waiting, however, is something agencies charged to protect human health cannot afford to do. Chemicals that cause cancer in experimental animals are in the environment and are accumulated by fish. Because of the lack of knowledge about the low-level effects of these chemicals on humans, EPA has adopted a very conservative approach to their estimates of increased cancer risks in the interests of public health. Despite the many shortcomings of interspecies extrapolation models, at present they are the main tool for predicting effects of environmental and dietary levels of animal carcinogens on humans.

Because use of the EPA risk assessment process in state fish consumption advisories is relatively new, and because of the many associated uncertainties, the process is under constant review by EPA and the states. In the future, more states probably will use some form of risk assessment process in their fish consumption advisories. Also, as new techniques for predicting cancer risk and other health risks such as reproductive effects are developed, the states will incorporate them into their fish consumption advisories. Consequently, even where concentrations of contaminants in fish remain the same, health risks suggested by the advisories may change.

PUBLIC PERCEPTIONS AND HEALTH ADVISORIES

Risk Comparisons. State fish consumption advisories can also lead to the perception that fish are the only food source that contain cancer-causing substances. The sensitive instrumentation now available makes it possible to detect trace amounts of carcinogens in most foods. Ames et al. (1987) and Schuplein (1990) suggested that dietary risks from natural carcinogens may be much more important than risks from synthetic pesticide residues or contaminants in food. The same risk assessment techniques discussed earlier can be applied to any food. Based on these calculations, the lifetime cancer risk associated with drinking one pint of milk per day is estimated to be 1 in 7,143 (Bro et al. 1987). One contaminant in milk is aflatoxin, produced by a mold that grows on corn and peanuts that may be used in feed grains. Similarly, eating 4 tablespoons of peanut butter per day, which also contains trace amounts of aflatoxin, results in an estimated increased lifetime cancer risk of 1 in 1,666 (Bro et al. 1987). Risk comparisons included in fish consumption advisories should include dietary risks from other foods, specifically alternative protein sources. These comparisons would be helpful to persons who needed to replace sportfish in their diet if they followed fish consumption advisories (Wendt 1986; Clark et al. 1987).

Many anglers also perceive the only risk involved with fishing is the health risk from eating contaminated fish. Indeed, a risk factor can be associated with everything we do including driving to the lake and going fishing. Estimates of such risks are derived from actuarial tables. Thus, they are a different class of risk than the lifetime cancer risks associated with eating contaminated fish that are based on extrapolations from animal data. However, such risks do offer anglers a way to place the health risks of eating contaminated fish in perspective with a variety of risks encountered in daily life. The lifetime risk of death due to motor vehicle accidents is 1 out of every 59 and deaths due to boating are 1 out of every 400 (Clark et al. 1987). Thus, driving to the lake and being out on the water also involve risk.

Food Preparation to Reduce Risks. A simple way anglers can decrease the cancer risk associated with eating fish is to trim and cook the fish properly (Skea et al. 1979; Foran et al. 1989; Gall and Voiland 1990;). Because many organic contaminants are stored primarily in fish fat, removing fatty areas can greatly reduce the amounts of these contaminants ingested and consequently reduce the health risk. Skinning fish removes the fatty layer between the skin and the flesh. Filleting removes fatty areas around the fins. Other fatty areas an angler can remove are those along the top of the backbone, the lateral line, and the belly. Baking, broiling or grilling fish on a rack drains off fats containing organic contaminants. Puncturing the skin also helps fats drain off. Although these methods may also result in some reduction of heavy metals in fishes, the reduction will not be as significant as it is for organic contaminants because heavy metals are more generally concentrated in muscle tissue.

CONCLUSIONS

There are several strategies that can be used to increase the understanding and adherence to fish consumption advisories by anglers. First, the states need to develop a more uniform approach to their formulation of fish consumption advisories. Anglers also need to be made more aware of the assumptions used in the development of advisories. For example, the EPA risk assessment procedure assumes a 70-year lifetime consumption of fish. With this information, anglers might choose to adjust their consumption of fish according to their lifetime consump-

tion history. The use of a single number as the estimate of health risk (e.g., an increased cancer risk of 1 in 100,000) implies a degree of certainty that, in fact, does not exist (Fessenden-Raden et al. 1987). Risk assessments that contain the full range (i.e., upper bound, best, and lower bound estimates) of risk estimates produced by interspecies extrapolation models would provide risk managers with a more complete view of the risk. However, risk managers must find effective means of communicating this complex array of information to the angler who only wants to know if the fish are safe to eat. Television, radio, newspapers, and magazines are important sources of information for anglers (Cable and Udd, 1990). Risk communicators need to do a better job of using these media to inform anglers about fish consumption advisories.

Risk communication problems associated with explaining fish consumption advisories involve us in a classic "bad news, good news" situation. The bad news is that we live in a world contaminated with chemical compounds. Aquatic systems are sinks for these compounds, and fish have a remarkable capacity to concentrate them. Relatively few of the hundreds of chemicals that have been identified in aquatic systems are monitored routinely. Finally, we have little information about the chronic effects of many of these compounds on fish and humans and even less information about their additive or synergistic effects. The good news is that aquatic systems are remarkably resilient. If a contaminant is prevented from entering these systems, its concentration in water, sediments, and fish declines. Dramatic declines in DDT and PCB concentrations in the Great Lakes over the past 20 years are good evidence of this (De Vault et al. 1986, 1988; Hesselburg et al. 1990).

The good news about fish consumption advisories is that they increase public interest in and concern about water quality. Proper application of risk communication can increase anglers' understanding of fish consumption advisories and help channel their legitimate concern into actions that will result in stricter water quality regulations. The end result of such actions will be improved water quality, which will benefit the health of the fish and the health of the people who eat them.

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