CONCEPTS IN MARINE POLLUTION MEASUREMENTS

Edited by Harris H. White

A Maryland Sea Grant Publication
University of Maryland
College Park

Kepone Uptake: A Comparison of Field and Laboratory Data

Michael E. Bender, Robert J. Huggett and Harold D. Slone
Virginia Institute of Marine Science
Gloucester Point, Virginia 23062

INTRODUCTION

The ability to predict the environmental fate and effects of pollutants in the marine environment is of utmost importance in assessing the hazards posed by a compound's use and/or disposal. Most commonly utilized methods to establish potential environmental effects have involved an assessment with bioassays of a compound's acute and chronic toxicity.

For compounds that have the ability to bioaccumulate, the potential dangers from this process must also be determined. A "first cut" estimate can be made by determining the partition coefficients between an organic solvent, usually n-octanol, and water. If this coefficient exceeds 25,000, EPA requires a report of the potential hazard (under the Toxic Substances Control Act), and further study is required. The next step is to expose some likely target animal to the suspected contaminant through food and/or water.

Transfer by these routes is calculated as (1) a bioconcentration factor from water = concentration in the animal/concentration in water, and (2) a dietary accumulation factor from food = concentration in the animal/concentration in food. Further food chain accumulation studies are sometimes conducted in the laboratory if the importance of the compound indicates a need. Studies, both laboratory and field, are also performed to estimate the transfer of the contaminant from its usual reservoir in the field (i.e., sediment) to food organisms and water. With these data, an assessment can be made of the potential for a compound to accumulate in marine organisms.

The above procedures have been developed over the past ten years and have gained a general degree of acceptance in the discipline; however, their shortcoming lies in the lack of field verification. This is particularly true in the marine environment where field data on many aspects of the environmental compartmentalization are lacking.

The introduction of Kepone (1,1a,3,3a,4,5,5a,5b,6-decachloroctahydro-1,3,4-metheno-2H-cyclobuta (cd) pentalen-2-one) into the James River estuary in Virginia has resulted in a large number of investigations which provide in part the basis for an assessment, although an after-the-fact one, of how well the predictive system works in the marine environment.

The toxicity of Kepone to marine and estuarine life has been studied by Bookout et al. (1980), Bourquin et al. (1978), Couch et al. (1977), Hansen et al. (1977), Ninmo et al. (1977), Provenzano et al. (1978), Rubinstein (1979), Schimmel and Wilson (1977), Walsh et al. (1977), Fisher (1980), Roberts and Bendl (1982).

Studies on bioconcentration, bioaccumulation, depuration and transfer have been conducted by Bahner and Oglesby (1979), Van Veld (1980), Drifmeyer et al. (1980), Schimmel et al. (1979), Huggett and Bender (1980), Huggett et al. (1980), Slone and Bender (1980).

In addition to these investigations, various agencies of the Commonwealth of Virginia have been conducting monitoring studies on the river, biota, sediments and water.

METHODS

Samples of blue crabs (Callinectes sapidus), oysters (Crassostrea virginica) and two species of bottom-feeding fishes, croaker (Micropogon undulatus) and spot (Leiostomus xanthurus), were collected between 1976 and 1980 and analyzed for Kepone residues. Crabs were collected in the lower 10 km portion of the river, and oysters were collected from discrete "oyster rocks" shown in Figure 1. Fish were collected within the zones shown also in Figure 1.

Laboratory studies have shown maximum bioconcentration factors of 7,200 for fishes (Hansen et al. 1977). Transfer from food to fish is estimated to be on a 1:1 basis (Bahner et al. 1977; Van Veld 1980; Stehlik 1980). Uptake of Kepone from sediments by benthic food organisms occurs, and concentrations reached by the organisms approximate those of the sediments (U.S. EPA 1978). Estimates of the uptake of Kepone by fishes can be made by utilizing these data and field observations on the distribution of Kepone in the sediments, which serve as the reservoir for the aqueous phase.

Estimates of contamination levels in fishes and sediments for the period between 1976 and 1979 were made from the monitoring data of the Virginia State Water Control Board and the Virginia In-

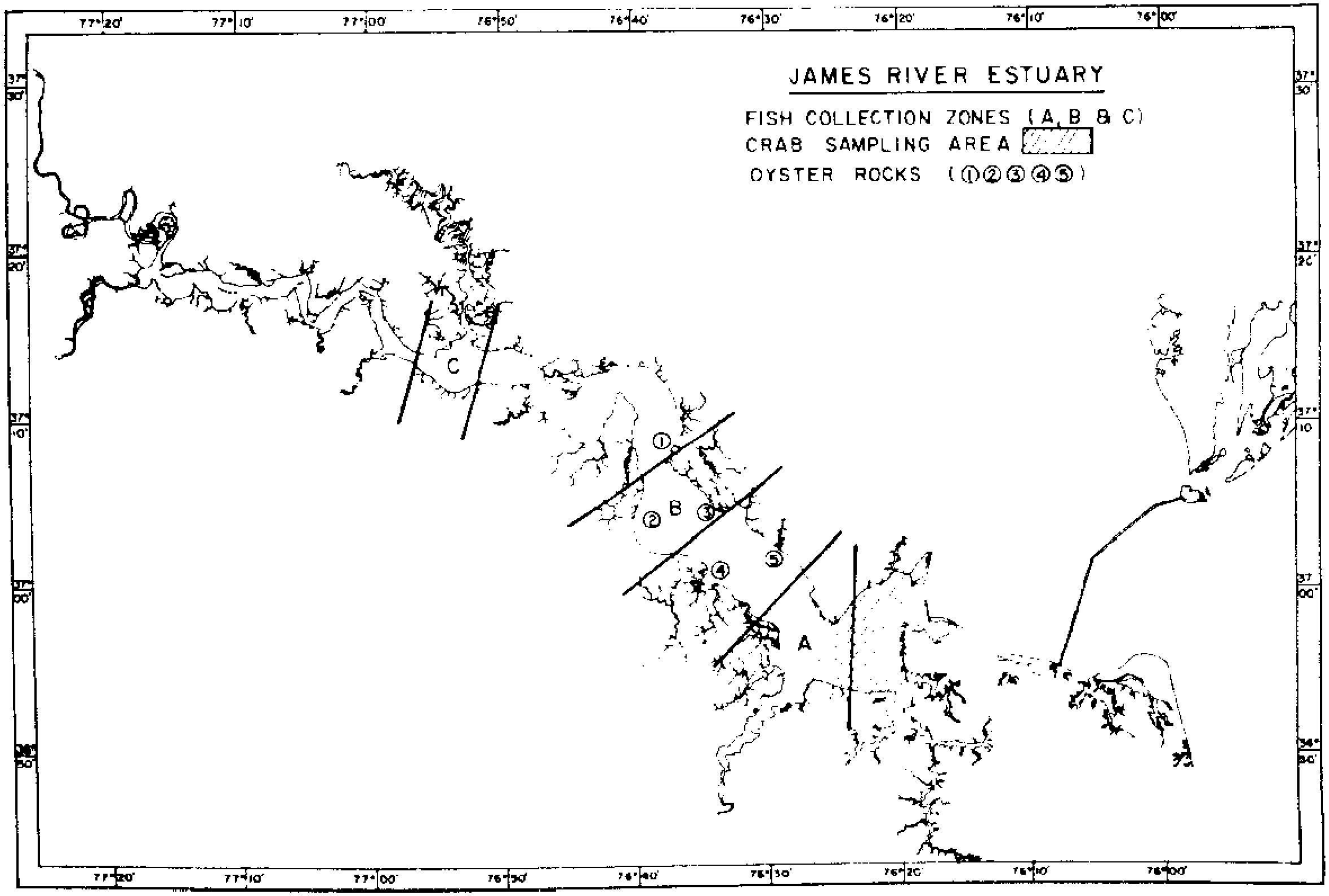

Figure 1. Sampling locations.

stitute of Marine Science. For the purposes of this study, we averaged the surface (upper 2 cm) sediment contamination levels over the zone in the river where the fish residue data were collected. Results from three zones are presented. Each zone is about 10 km long and the zones are approximately 20 km apart (Table 1). Dissolved Kepone levels were predicted by utilizing a partition coefficient between sediments and water of 5,000:1 (Strobel et al. 1981). Field data relating dissolved Kepone to sediment concentration are limited, but studies that have measured both parameters indicate that dissolved levels can be accurately estimated using this coefficient (Slone and Bender 1980).

RESULTS AND DISCUSSION

The residue levels of Kepone in fishes, predicted from laboratory exposures to contaminated water and food, are compared with field data in Figure 2. Significant deviations from the predicted values are apparent. There are several possible explanations for these differences:

1. The predicted bioconcentration factors are too low.
2. The actual levels of Kepone in the water are greater than predicted from the sediment-water partition coefficients.
3. There is greater accumulation through food than estimated from laboratory studies.

In the prediction, we utilized the maximum bioconcentration factor observed for fish (i.e., 7,200 vs. a mean value of 3,500 obtained from averaging the results of five different laboratory investigations). We believe, therefore, that the laboratory predictions used were not in error, at least on the low side.

Table 1. Mean Kepone in sediments (ng g^{-1}) and third quarter $\bar{x}$ for bottom feeding fish (spot and croaker) $\mu g\ g^{-1}$.

Year	Zone A		Zone B		Zone C	
	Seds.	Fish	Seds.	Fish	Seds.	Fish
1976	15	.72	67	1.6	117	-
1977	16	.47	56	1.8	144	-
1978	5	.36	19	0.9	37	-
1979	1	.35	16	-	65	0.6

Two studies have measured dissolved Kepone in the lower James. These investigations have shown levels of approximately 5 ng L^{-1}. Predictions from sediment concentrations at these sites indicate that the dissolved levels should have been approximately 7 ng L^{-1}. We feel confident, therefore, that actual levels of dissolved Kepone are not higher than the predictions and are, in fact, in remarkably good agreement.

If we are correct in our assessment of two possible explanations for error in the predictions, the most likely explanation is an underestimation in uptake through the food chain to account for the difference between predicted and measured amounts of dissolved Kepone.

Blue crabs bioaccumulate Kepone mainly from their food. Uptake from water is limited with a bioconcentration factor of only 8 (Schimmel and Wilson 977). Fisher (1980) determined the uptake of Kepone by juvenile blue crabs over a 2-month period. Utilizing his data, one can predict concentrations in crabs with the following equation:

$$C_a = DAF \times C_n \, (W_f/W_a)$$

where

C_a = predicted concentration in the crab
DAF = dietary accumulation factor = $C_e \, W_e / C_f \, W_f$
C_e = experimental equilibrium concentration in crabs
W_e = average weight of experimental crabs
C_f = experimental concentration in the food
W_f = weight of food consumed per day
C_n = concentration in natural food
W_a = average weight of crabs

The mean dietary accumulation factor derived from Fisher's studies is 30, when one utilizes the average weight of the crabs during his experiments. If we assume the blue crabs eat approximately 5% of their body weight per day (the ration used in Fisher's studies), then we can calculate the expected residue concentration in nature if we have an estimate of the level of food contamination.

Figure 3 compares field residue data collected from 1976 through 1980 with predictions based on the above equation. In this prediction we used, as an estimate of food contamination, the average residue level for all species of fish in the lower river. The agreement between laboratory predictions and the field residue data is very good for male crabs. Roberts and Leggett (1980) have shown that female crabs lose Kepone when they spawn, and since the predictive equation did not consider this loss, one could not expect agreement for females.

Oysters bioaccumulate Kepone from both dissolved and particulate phases. Uptake from particulate matter appears to be consid-

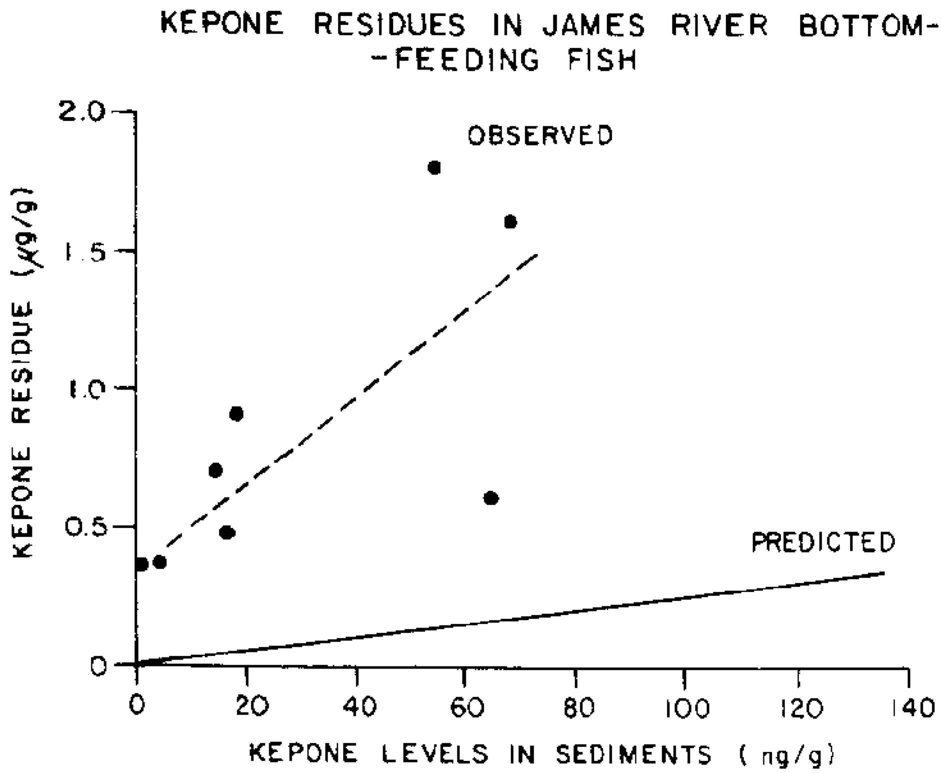

Figure 2. Kepone residues in fish vs. predicted uptake.

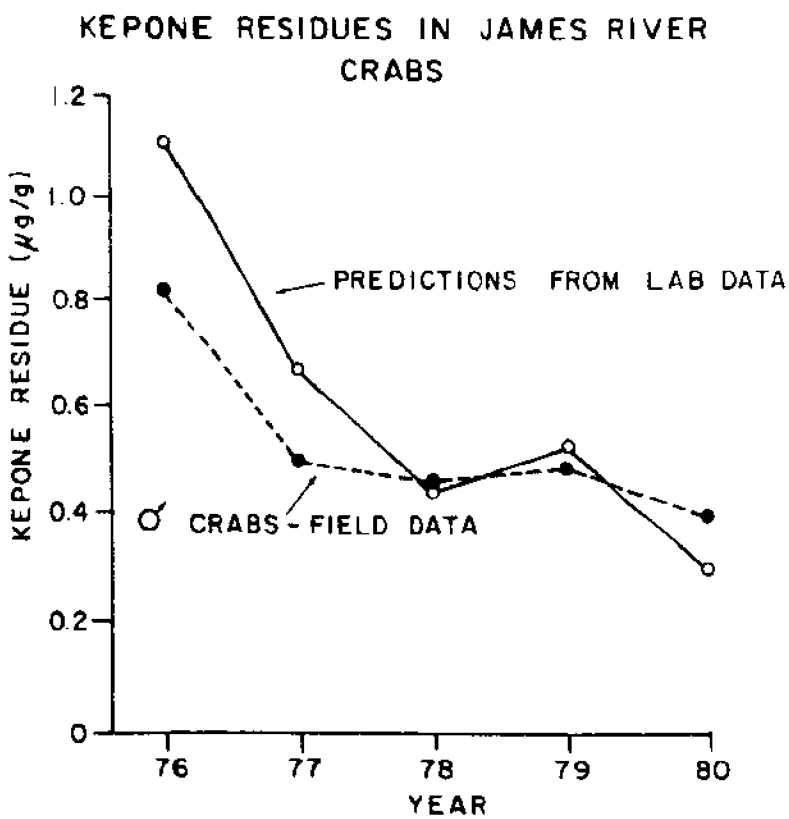

Figure 3. Kepone residues in blue crabs vs. predicted uptake.

erably less efficient than accumulation from the dissolved phase. Bahner et al. (1977) showed bioaccumulation factors from algae to oysters of 0.007, while Haven and Morales-Alamo (1977) demonstrated bioconcentration factors from sediments between 0.06 and 0.25. Bioconcentration factors from the dissolved phase averaged about 9,300 (Bahner et al. 1977).

Comparisons between laboratory uptake and field data for oysters are hampered to a certain degree by the oysters' natural variability as a function of season (Figure 4). However, if we normalize the field residue data for oysters to the water temperature at which the laboratory experiments were conducted, a reasonable fit is obtained (Figure 5). To construct this figure, we adjusted the observed residue levels for oysters at the time the sediment concentrations were determined to those that would be expected at 14°C. The predicted line was constructed utilizing a bioconcentration factor from the dissolved phase of 9,300.

Actual field observations of both water concentrations and oyster residues were made during 1979 when water temperatures approached 15°C. These values are shown on Figure 5. Unfortunately, our ability to measure dissolved Kepone was not developed until residues began to decline. Therefore, field confirmation is limited to the lower portion of the curve.

In the case of Kepone, our ability to predict residue levels in nature from laboratory data appears to be fairly good for crabs and oysters, while predictions for fish are poor. This is true not only for average residue predictions in migratory species but also for resident species. This limitation presents a serious practical problem in that fisheries are closed when residue levels of Kepone are excessive. The length of time such closures exist can of course have serious economic and legal consequences. In addition, evaluations of the cost versus benefit of cleanup must take into account the predicted length of closure with various cleanup alternatives including the no-action alternative. To answer questions of this nature for fisheries, it is clear that we need to develop a better understanding of what factors determine residue levels in the field.

Our ability to make predictions from laboratory data needs to be evaluated for other compounds; very few investigators have attempted to make comparisons of the kind discussed in this paper. We believe, at least for Kepone, that more attention needs to be given to uptake from food. Dietary accumulations may vary with the type of food consumed and/or with the actual rates of feeding in nature.

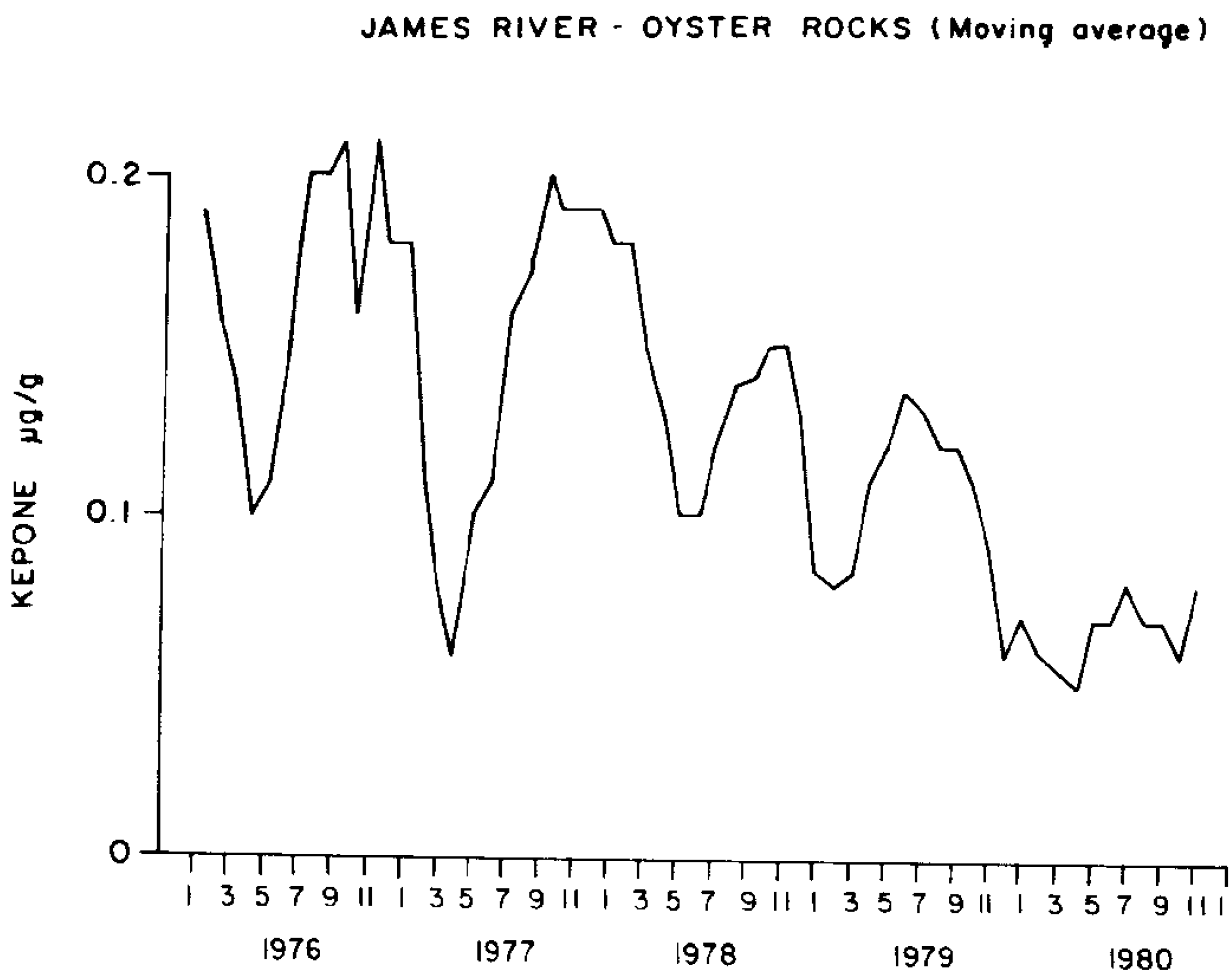

Figure 4. Kepone residues in oysters vs. time.

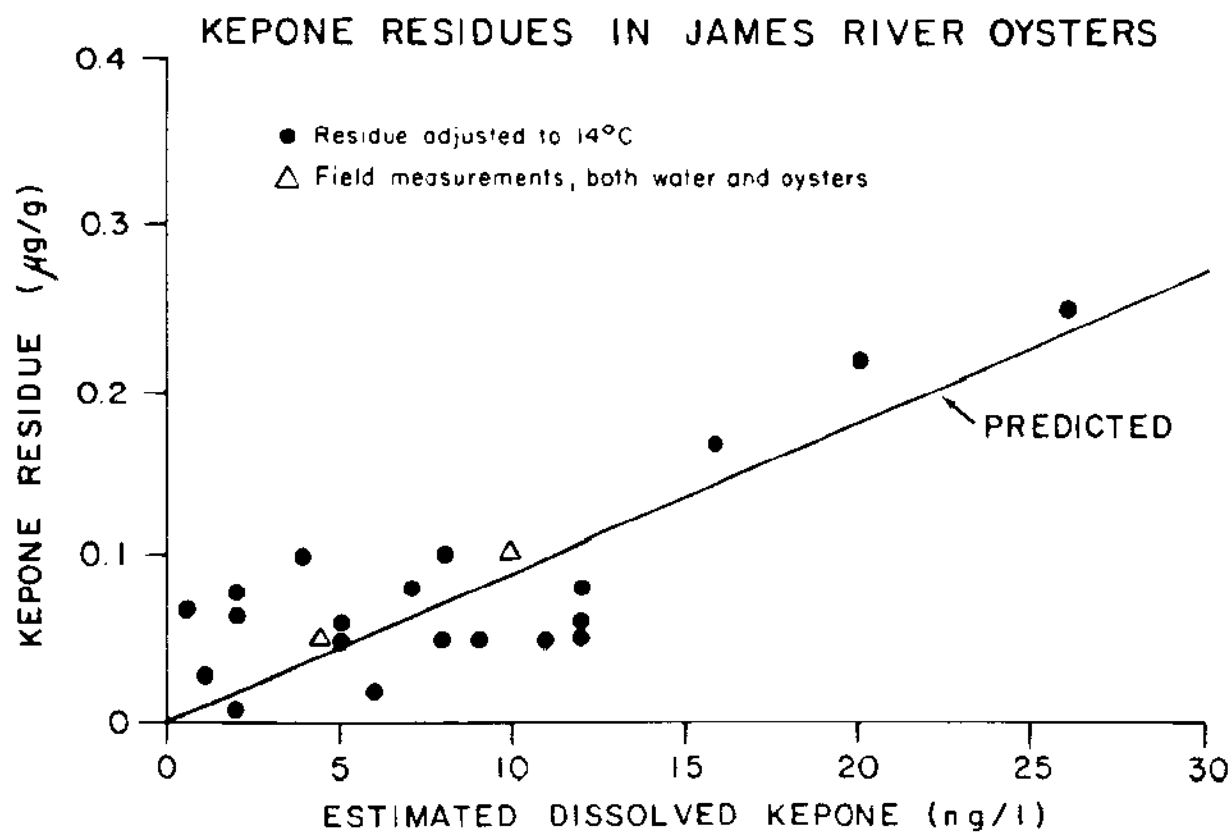

Figure 5. Kepone residues in oysters vs. predicted uptake.

ACKNOWLEDGMENTS

This paper is VIMS Contribution No. 1065.

REFERENCES

Bahner, L.H. and J.L. Oglesby. 1979. Test of model for predicting Kepone accumulation in selected estuarine species. In: L.L. Marking and R.A. Kimerle (eds.), Aquatic Toxicology. ASTM STP 667, American Society for Testing and Materials, Philadelphia, Pa.

Bahner, L.A., A.J. Wilson, Jr., J.M. Sheppard, J.M. Patrick, L.R. Goodman and G.E. Walsh. 1977. Kepone[R] bioconcentration, accumulation, loss and transfer through estuarine food chains. Chesapeake Sci. 18: 299-308.

Bookhout, C.G., J.D. Costlow, Jr. and R. Monroe. 1980. Kepone effects on larval development of mud-crab and blue-crab. Water Air Soil Pollut. 13: 57.

Bourquin, A.W., P.H. Pritchard and W.R. Mahaffey. 1978. Effects of Kepone on estuarine microorganisms. Dev. Ind. Microbiol. 19: 489-497.

Couch, J.A., J.D. Winstead and L.R. Goodman. 1977. Kepone-induced scoliosis and its histological consequences in fish. Science 197: 585-587.

Drifmeyer, J.E., C.L. Rosenberg and M.A. Heywod. 1980. Chlordecone (Kepone) accumulation on estuarine detritus. Bull. Environ. Contam. Toxicol. 24: 364.

Fisher, D.J. 1980. Effects of ingestion of Kepone (at environmental levels) on juvenile blue crabs (Callinectes sapidus Rathbun). M.S. Thesis, Coll. of William and Mary, Williamsburg, Va.

Hansen, D.J., L.R. Goodman and A.J. Wilson. 1977. Kepone chronic effects on embryo, fry, juvenile and adult sheepshead minnows (Cyprinodon variegatus). Chesapeake Sci. 18: 227-232.

Haven, D.S. and R. Morales-Alamo. 1977. Uptake of Kepone from suspended sediments by oysters, Rangia and Macoma. U.S. EPA Annual Report, Gulf Breeze Environmental Research Laboratory.

Huggett, R.J. and M.E. Bender. 1980. Kepone in the James River. Environ. Sci. Technol. 14: 918-923.

Huggett, R.J., M.M. Nichols and M.E. Bender. 1980. Kepone contamination of the James River estuary. In: R.A. Baker (ed.), Contaminants and Sediments, Vol. 1, pp. 33-52. Ann Arbor Science Publ., Ann Arbor, Mich.

Nimmo, D.W.R., L.H. Bahner, R.A. Rigby, J.M. Sheppard and A.J. Wilson. 1977. Mysidopsis bahia: An estuarine species suitable for life-cycle toxicity tests to determine the effects of a pollutant. In: F.L. Mayer and J.L. Hamelink (eds.), Aquatic Toxicology and Hazard Evaluation. ASTM STP 634, American Society for Testing and Materials, Philadelphia, Pa.

Provenzano, A.J., K.B. Schmitz and M.A. Boston. 1978. Survival, duration of larval stages and size of post-larvae of grass shrimp, Palaemonetes pugio, reared from Kepone contaminated and uncontaminated populations in Chesapeake Bay. Estuaries 1: 239-244.

Roberts, M.H. and R.E. Bendl. 1982. Acute toxicity of Kepone to selected freshwater fishes. Estuaries.

Roberts, M.H. and A.T. Leggett. 1980. Egg extrusion as a Kepone-clearance route in the blue crab, Callinectes sapidus. Estuaries 3: 192-199.

Rubinstein, N.I. 1979. A benthic bioassay using time-lapse photography to measure toxicants on the feeding behavior of lugworms (Polychaeta: arenicolidae). In: W.B. Vernberg, F.P. Thurberg, A. Calabrese and F.J. Vernberg (eds.), Marine Pollution: Functional Responses, pp. 311-351. Academic Press, New York.

Schimmel, S.C. and A.J. Wilson, Jr. 1977. Acute toxicity of Kepone to four estuarine animals. Chesapeake Sci. 18: 244.

Schimmel, S.C., J.M. Patrick, Jr., L.F. Faas, J.L. Oglesby and A.J. Wilson, Jr. 1979. Toxicity and bioaccumulation in blue crabs. Estuaries 2: 8-15.

Slone, H.D. and M.E. Bender. 1980. Kepone monitoring at Skiffes Creek. DACW65-79-C-0027, U.S. Army Corp of Engineers, Norfolk District, Norfolk, Va.

Stehlik, L.L. 1980. Bioaccumulation and effects of Kepone on spot, Leiostomus xanthurus. M.S. Thesis, Coll. of William and Mary, Williamsburg, Va.

Strobel, C.J., R.E. Croonenberghs and R.J. Huggett. 1981. The suspended sediment-water partitioning coefficient for Kepone in the James River, Va. Environ. Pollut. (Ser. B). 2: 367-372.

U.S. Environmental Protection Agency. 1978. Mitigation feasibility for the Kepone-contaminated Hopewell/James River areas. Office of Water and Hazardous Materials Criteria and Standards Division, Washington, D.C.

VanVeld, P. 1980. Uptake, distribution, metabolism and clearance of Kepone by channel catfish (Ictalurus punctatus). M.S. Thesis, Coll. of William and Mary, Williamsburg, Va.

Walsh, G.E., K. Ainsworth and A.J. Wilson, Jr. 1977. Toxicity and uptake of Kepone in marine unicellular algae. Chesapeake Sci. 18: 222.