Toxicity of Coal-Conversion Gasifier Condensate to the Fathead Minnow

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The increasing need to supplement our dwindling supply of natural gas and yet maintain environmental quality has generated a need for bioassay of aquatic effluents from coal-conversion systems. One of the more promising coal-to-gas schemes is the Synthane process currently under study at the Bruceton Laboratories of the Pittsburgh Energy Research Center (Pittsburgh, Pennsylvania). This laboratory-scale Synthane operation provides product streams considered to be representative of those which will eventually be produced by commercial procedures (FORNEY et al. 1974). The major effluent and potential source of environmental pollutants is the contaminated condensate from the 40-atm fluid-bed gasifier (FORNEY et al. 1974). Chemical analyses of this aqueous effluent show that its composition varies with the type of coal used, but its major organic components are phenolic compounds and ammonia (FORNEY et al. 1974; HO et al. 1977).

The purpose of the present paper is to examine the toxicity to the fathead minnow of the condensate water from the gasifier.

MATERIALS AND METHODS

Fathead minnows, Pimephales promelas, 4.5–6.5 cm standard length, were exposed to various concentrations of Synthane condensate at 18 ± 1°C during a 16/8-hr (L/D) photoperiod of grow-lux fluorescent lighting. Stock fish were maintained in aerated charcoal-filtered tap water and fed ground trout pellets. The gasifier condensate water, provided by the Pittsburgh Energy Research Center, was from the sample analyzed by HO and his co-workers (1977). Each of 5 replicates of 10 fish was placed in 1500 ml of nonaerated test solution in a glass aquarium. The initial pH of the test

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solutions varied from 7.8 for controls to 8.7 for 0.5% condensate. Fish were fed daily during the test period, and the test solutions were changed after each feeding. The number of survivors was recorded daily.

Concentrations resulting in 50% mortality (LC50) at 24, 48, and 96 hr were computed following the procedure of KOPPERMAN et al. (1974). In addition, the lethal threshold concentration was estimated by visual inspection of the toxicity curve. The incipient LC50, the percent concentration where the curve becomes asymptotic to the time axis, was more accurately estimated using the method of SPRAGUE (1969). Finally, the mortality rate of fish exposed to the various concentrations of product water was examined by linear regression (SPRAGUE 1969).

RESULTS

When unidirectionally exposed to gasifier condensate, fathead minnows displayed an avoidance reaction. However, once the toxicant was mixed, no directional swimming was observed. Although the behavioral pattern of minnows exposed to the product water was irregular, it was rather characteristic and dose-dependent. Soon after exposure, at concentrations of 0.3% and greater, the fish displayed short periods of rapid swimming; shortly thereafter they lost their sense of balance, rested on their sides, and swam erratically. Respiratory movements were also irregular, with periods of rapid opercular movement alternating with longer periods of weak or no movement. This was followed rapidly by death. In concentrations between 0.2% and 0.25% the fish lost their sense of balance less quickly. However, the periods of swimming on their sides were alternated with periods of what appeared to be normal activity. Finally, the fish stopped swimming, rested on their sides, exhibited occasional weak tail and opercular movements, and died. No initial burst of activity was noted as with higher concentrations. Fish which progressed to the stage of resting on their sides recovered normal activity within 30 min when placed in fresh water. The condensate often caused discoloration of the fish, especially at higher concentrations. This was concomitant with reduced activity, but the responses of individual fish varied widely. Fish exposed to lower concentrations (0.01%) of condensate water for longer periods of time (5–10 days) did not display any of the behavioral alterations described above, but they did exhibit a general inflammation of epithelia, especially of the head, gills, and areas at the base of the fins. Death in this instance was accompanied by extensive hemorrhaging in these areas.

The LC50 at 24, 48, and 96 hr exposure to gasifier condensate was calculated to be 0.163, 0.084, and 0.051% from a least-squares linear regression of the probit of survival against log concentration of the data in Table 1. SPRAGUE (1969) in his review of the methodology of fish bioassay favored the use of the incipient LC50 rather than the LC50 at some specific time. However, he did suggest the use of the 96-hr LC50 as an alternative since these are often equivalent (SPRAGUE 1969). To determine if the 96-hr LC50 is a useful standard in judging the toxicity in this study, the log median

TABLE I

Mean Percent Survival of Fathead Minnows Exposed to Synthane Laboratory-Scale Gasifier Condensate at 18°C

Dose (%)	Percent survival for exposures of: 24 hr	48 hr	96 hr
0.2	28	2	—
0.15	68	24	—
0.1	80	26	—
0.075	82	66	38
0.05	94	70	52
0.025	94	76	64
0.01	96	94	80
Standard error of the mean	4.3	5.7	7.1

lethal times were plotted against the log concentration (Fig. 1). This curve is linear well past the calculated 96-hr LC50 of 0.05% product water, indicating that the 96-hr LC50 is not a useful criterion in reporting the toxicity

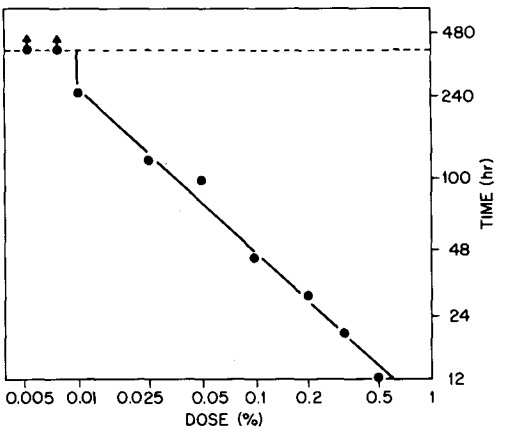

Figure 1. Effect of dose on median lethal time of fathead minnows exposed to Synthane gasifier condensate at 18°C.

of the condensate when the fish are exposed to fresh toxicant daily. A more accurate estimate of the incipient LC50 was made from the 7-day toxicity data. The mortality-versus-time curves at concentrations of 0.005 to 0.025% are typically sigmoid, 7 days being the upper extreme of the linear death rate. When the percent mortality at day 7 was plotted against the concentration using a probit–log transformation, the incipient LC50 was calculated to be 0.012%.

To determine if more than one mode of action was involved in the toxicity of the gasifier condensate, the percent mortality for each concentration was plotted against the length of exposure using a probit–log transformation (Fig. 2). A distinct slope change was observed between 0.1 and 0.075% condensate indicating a change in the mode of action (SPRAGUE 1969). The fact that the lines for 0.15 and 0.05% are closer to 0.1 and 0.075%, respectively, indicates that the slope change is real and not due to variability among fish.

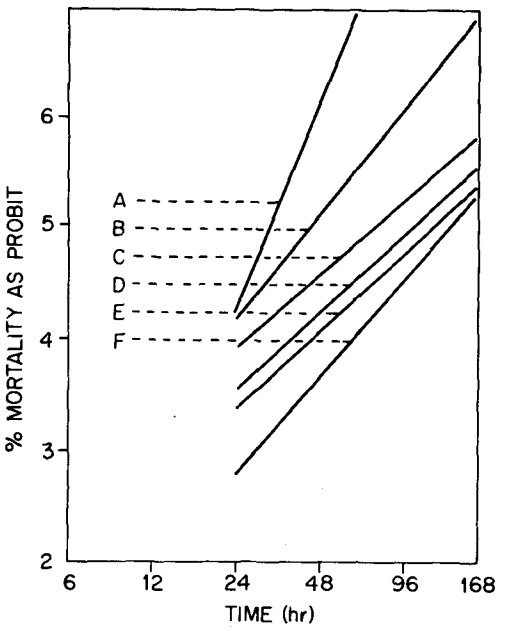

Figure 2. Comparison of time mortality for fathead minnows exposed to gasifier condensate at 18°C (A = 0.15%, B = 0.10%, C = 0.075%, D = 0.050%, E = 0.025%, F = 0.01%). Slopes (mortality rates) are 8.13, 4.13, 2.23, 2.31, 2.34, and 2.30 respectively.

DISCUSSION

Although the laboratory-scale Synthane gasifier condensate used in this study is not a final effluent, the size of the commercial plants envisioned — 15,000 tons of coal per day — makes such water effluents potential environmental hazards. The major organic condensate components are phenol (35% by weight of the total organics present), its monomethyl (cresols) and dimethyl (xylenols) derivatives (HO et al. 1977), and ammonia (FORNEY et al. 1974).

The most obvious effect following exposure of fathead minnow to high concentrations of condensate is the rapid loss of balance and irregular swimming and respiratory patterns. These reactions have been observed in other fish exposed to phenols (for review see EIFAC WORKING PARTY ON WATER QUALITY CRITERIA 1973). Such effects have led to the conclusion that the acute toxicity of phenols can be attributed to a breakdown in neural function. The general inflammation of exposed tissues of fathead minnows surviving long-term exposure to low concentrations is similar to that described by MITROVIC and his co-workers (1968) for fish exposed to phenol.

If one attempts to relate the effects of this potential Synthane condensate on fathead minnow to any single compound or group of compounds present, one is immediately drawn to the fact that very high concentrations of phenols (5220 mg/liter; HO et al. 1977) and ammonia (~9000 mg/liter; FORNEY et al. 1974), are present in the sample. While other components in all probability elicit effects, it is interesting to compare the data presented here with those previously obtained with pure compounds and mixtures (see EIFAC WORKING PARTY ON WATER QUALITY CRITERIA 1973).

Lethal concentrations of phenolic compounds are not consistent between species. However, trends are evident, at least in regard to phenol and the isomers of cresol; there is little difference between these compounds, but m-cresol appears to be the least toxic. SOUTHGATE (1932), who examined the toxicity of mixtures of phenolic compounds, came to the conclusion that, while p-cresol was slightly more toxic than phenol, mixtures of phenol and p-cresol are "interchangeable" — that is, when toxicants with the same mode of action are mixed, their toxicity is enhanced. Thus toxicity of phenol and cresols, both of which are present in product water, are additive.

Based on 24-hr LC50 values ALBERSMEYER and ERICHSEN (1959) found the toxicity of dimethyl phenols to vary significantly within the group. For example, 2,5-xylenol (24-hr LC50 10 mg/liter) was consistently about five times more toxic than 3,5-xylenol (24-hr LC50 50 mg/liter). Both of these isomers are found in gasifier condensate in quantities greater than 200 mg/liter (HO et al. 1977). While ALBERSMEYER and ERICHSEN (1959) found 2,4- and 3,4-xylenol as toxic as phenol and cresols, the behavioral data of SOUTHGATE (1932) suggest that xylenol has a different mode of action than phenol and cresols. This difference was further supported by the toxicity curve of a cresol–xylenol mixture which showed that the two components exert independent effects (SOUTHGATE 1932). SOUTHGATE (1932) further stated that "... small, sub-lethal concentrations of xylenol added to toxic concentrations of cresol cause an appreciable increase in their toxicity." Since

phenol and cresols are found in higher concentrations (4570 mg/liter) than dimethyl phenols (650 mg/liter; HO et al 1977), the conditions described by SOUTHGATE could be achieved at very low concentrations of Synthane gasifier condensate.

An additional problem which must be considered is the toxic effect of un-ionized ammonia. In five of the six coals tested, FORNEY and his co-workers (1974) found that the ammonia concentration was very high (~9000 mg/liter). The toxicity of ammonia to fish is directly related to the amount of undissociated ammonium hydroxide formed when the ammonia reacts with water. At pH 7 the ratio of ammonium ions to ammonium hydroxide molecules is 180:1, but it decreases sharply to 18:1 at pH 8 (MCKEE and WOLF 1963). Thus, the high concentration of ammonia in the condensate at the high pH (8.7 for 0.5%) makes its toxicity a real problem. LLOYD and HERBERT (1960) state that for most practical purposes the threshold toxicity may be assumed to be only a little below the concentration corresponding to the median lethal time (LT50) of 500 min.

In conclusion, it is worth noting that the gasifier condensate is unstable: a precipitate forms and the sample turns dark with time (HO et al. 1977). This undoubtedly affects the toxicity, but how and to what extent is unknown. Nevertheless, we feel the toxicity observed over the concentration range tested is almost exclusively due to the high concentration of phenol and cresols and that toxicity is significantly increased by the small amount of dimethyl phenols present. Ammonia toxicity is not a significant problem, since the LT50 for the concentration range tested is much greater than 500 min. This is supported by the behavioral and gill histopathological data, both of which parallel the effects of phenol and cresols.

The mortality rate of minnows exposed to various concentrations of condensate suggests two modes of action. Based on our behavioral observations and gill pathology, we feel that the fish die at high concentrations (0.5 to 0.1%) due to nervous paralysis and at lower concentrations due to general tissue necrosis and/or organ failure.

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