1. Cecil et al. PCBs in Laying Hens
164th ACS Meeting NYC August 1972

The effects observed by Cecil with regard to egg production and hatchability do not greatly differ from those seen in the original study at BIO-TEST. In our study egg production was decreased by 1242 and by 1254 at 100 ppm and hatchability was adversely affected by 1254 at 100 ppm and by 1242 at levels as low as 8 ppm with a marginal effect at 4 ppm. Cecil claims effects of 1242 and 1254 on both egg production and hatchability at 20 ppm, the effects of 1242 being the greater. Neither we nor Cecil observed effects with the more highly chlorinated Aroclors, 1260 at 100 ppm at BIO-TEST and Aroclor 1268 at 20 ppm by Cecil, except for a very questionable effect of 1268 on egg production. We confirm no effect of 1242 at 2 ppm but judged 10 ppm to be a no-effect level for 1254.

In the BIO-TEST study there were relatively more deaths during the earlier stages of incubation, particularly at the higher levels of Aroclor 1242.

In the BIO-TEST study the normality of the unhatched embryos was not determined but we did observe some gross abnormalities among hatched chicks from the control group and from some treated groups but no differences were judged to exist among control and treated groups. Growth and survival may have been affected by 1242 at 10 ppm and there was a suggestion of decreased viability of hatched chicks from groups treated with Aroclor 1254 at 10 ppm or with 1242 at levels lower than 10 ppm.

Cecil observed no abnormalities among hatched chicks but claims a 34% incidence of abnormalities among dead embryos. The data on abnormalities are not classified by compound or dose level and many of the so-called abnormalities might be judged to be manifestations of metabolic, nutritional or biochemical derangements rather than teratogenic responses in the sense that, to me at least, a teratogenic response implies that a genetic alteration has occurred with a consequent derangement of development. A teratogenic agent is thereby something quite different from a material which is simply functionally toxic. It is, furthermore, strange that no defect of any degree permitted survival through hatching.

Table 10 implies a very high teratogenic index but it is not possible to determine the quantitative basis for the statement that the Aroclors are teratogenic. A teratogenic index should be the number of abnormalities related to genetic changes divided by the total sample. In this case we do not know the total sample and do not know which abnormalities are considered to be genetic.

For example:

> Mean hatchability for 1232, 1242, 1248 and 1254 (from graphs Slide 6) = Ca. 53%. Unhatched = 47%. Total sample = $\frac{843}{.47}$ = 1792

If one considers rotated ankle, beak defects and others to be true teratogenic type deformities (and this is questionable), the teratogenic index would be: $\frac{55}{1792} \times 100 = 3\%$

Defects among hatched controls in the BIO-TEST study numbered 5 of 93 or 5+%.

In my estimation there is no evidence for teratogenic activity but whether or not others would consider edema, runting, etc. to be teratologic effects and whether or not the Aroclors are judged to be in fact teratogenic, the method used for presentation of the data is patently biased to support what must have been a preconceived opinion.

Cecil also concludes that growth of chicks from groups treated with Aroclors 1232, 1242, 1248 and 1254 was impaired but that survival was unaffected. We did not observe an effect on growth except perhaps for 1242 - 10 ppm. Our weight gains in 30 days were considerably higher than the 4-wk gains reported by Cecil.

I have no particular criticism of the protocol for the study but I feel that one can differ with the analysis and interpretation of the data, particularly with that relating to teratology.

I have not seen the data for the repeat studies in chickens and have not studied the residue data in an attempt to correlate effects with tissue residue levels.

2. Cecil et al.

No additional comments. This is a more formalized version of the data reported at the ACS.

3. Press Release - ACS News Service. No comments.

4. Fries et al.

No comments except to observe that unacceptably high levels of 1254 can occur in milk (19 ppm) as a result of consuming silage held in a silo painted with a paint containing 1254.

5. No comments.

6. All DDT & DDE papers

I don't see that these papers have any bearing on the Aroclor problem except to

1. Suggest protocols for studies to compare Aroclors to other chlorinated hydrocarbons

2. Take Aroclors off the hook as the culprit in the eggshell thinning problem. Peakall (1971), based on his own work and on that of Anderson et al. (1969) and Vermeer et al. (1970), has concluded that PCB's do not play a significant role in the phenomenon of eggshell thinning.

Peakall, D.B. (1971). Effect of polychlorinated biphenyls (PCB's) on the eggshells of Ring Doves. Bull. Environ. Contam. Toxicol. 6, 100-101.

Anderson, D.W., Hickey, J. J., Risebrough, R.W., Hughes, D.F. and Christensen, R.E. (1969). Significance of chlorinated hydrocarbon residues to breeding pelicans and cormorants. Can. Field Natur. 83, 91-112.

Vermeer, K. and Reynolds, L.M. (1970). Can. Field Naturalist 84, 117.

The PCB's probably do induce enzymes involved in steroid metabolism but this has been adequately covered in my earlier review of the general toxicology of the Aroclors.