



UNITED STATES ENVIRONMENTAL PROTECTION AGENCY  
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SUBJECT: Risk Calculation Utilizing Recent Data on DBCP Levels in Drinking Water  
OFFICE OF TOXIC SUBSTANCES

FROM: Lois Rossi, Biostatistician  
Special Pesticide Review Division *Lois Rossi*

TO: Jeff Kempter, Project Manager  
Special Pesticide Review Division

THRU: Kevin Keaney, Acting Chief *K. Keaney*  
Regulatory Analysis, Lab Audit and Policy Branch  
SPRD

I have completed a brief cancer risk assessment for DBCP found in drinking water. The residue levels of DBCP in water for which you requested risk calculations were 23.0 parts per billion (ppb), 10 ppb, 1 ppb and 0.1 ppb.

I obtained a revised risk equation from the Carcinogen Assessment Group's June 12, 1979 memorandum, "Re-evaluation of DBCP Risks Incorporating Recent Chronic Testing Data." The revised slope parameter was  $6.5 \text{ (mg/kg/day)}^{-1}$  or  $0.65$

$\times 10^{-5} \text{ (ng/kg/day)}^{-1}$ . Thus, the lifetime probability (P) of cancer from ingestion to DBCP is:

$$P = (0.65 \times 10^{-5} \text{ ng/kg/day}) \times X$$

where X is the level of DBCP in water in ng/kg/day.

The conversion from ppb to ng/kg/day in the diet is:

$$\frac{(\text{ug/l})(2 \text{ l/day})^*}{60 \text{ kg}} = \text{ug/kg/day} \times 10^3 = \text{ng/kg/day}$$

where 2 liters/day is the average human consumption rate of water and 60 kg is the average weight of a person.

The attached table present the lifetime probability of cancer associated with the various levels of exposure to DBCP.

\*/ ppb is expressed as ug/l.

Table 1

LIFETIME PROBABILITY OF CANCER DUE TO EXPOSURE TO DBCP  
IN DRINKING WATER \*

<u>Levels of DBCP (ppb)</u>	<u>Levels of DBCP. (ng/kg/day)</u>	<u>Lifetime Probability of Cancer Per Person</u>
0.1	3.3	$2.1 \times 10^{-5}$
1.0	33	$2.1 \times 10^{-4}$
10.0	330	$2.1 \times 10^{-3}$
23.0	767	$5.0 \times 10^{-3}$

\* Assumes 2 liters/day daily consumption of water for 70 yr. lifetime.

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The mechanism of the anti-spermatozoal effects of EOB appears not to be known at present. Two major possibilities present:

(1) Interference with endocrine status: Normal spermatogenesis depends on hormonal regulation at several stages. Interference with endocrine status will impair normal regulation.

(2) EOB may directly affect spermatocytes by, for example, inhibition of DNA synthesis, mutagenesis, ~~and~~ inhibition of sugar synthesis, etc.

In the simplest possible case, we will want to know which of 180 subjects exhibit a decreased FSH, LH or testosterone level with decreased sperm concentration, which exhibit a decreased sperm concentration with normal endocrine status, and how these values compare to control expectations (both the 50 normal subjects and historic values).

The <sup>meaning</sup> ~~purpose~~ of a fertilization test will vary from an endocrine + / sperm + subject to an endocrine - / sperm - subject. If data processing is for simple correlations between test outcomes, then the biologic significance of this data can be suppressed. ~~Nothing~~ No statements exist in the "data processing and analysis" section that ~~address~~ my concern in this regard.

We still don't know how long the people around Kunia were exposed to DB E DB T DBC P - one week or 3 years? Most significant if the study results are negative.

Starr