Levels of DDT and metabolites in breast milk from Kwa-Zulu mothers after DDT application for malaria control

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Concentrations of DDT, DDE and DDD were determined in the breast milk of Kwa-Zulu mothers residing in two different areas—with and without annual intra-domiciliary applications of DDT for the interruption of malaria transmission (exposed and control groups, respectively). While no significant change in levels with time was found in the control group, both DDT and DDE in breast milk of the exposed group increased after DDT application and this continued for three more months, after which it did not decrease appreciably. Percentage DDT increased from 42.57% (ΣDDT = 12.21 mg/kg milk fat) before spraying to 50.87% (ΣDDT = 13.79 mg/kg milk fat) following DDT application. At 6 and 9 months after the application it was 45.85% (ΣDDT = 19.49 mg/kg milk fat) and 43.27% (ΣDDT = 18.34 mg/kg milk fat), respectively. These results suggest a risk to the health of the infants in the exposed group.

Introduction

The concentration of DDT in human breast milk, which indicates the potential risk to the mother and infant, has been the subject of many studies (*1*, *2*). Hyporeflexia in infants, for example, has been associated with DDE levels as low as 4 mg/kg (milk fat) in breast milk (*3*). The need for a special approach to evaluate the health risk of chemicals during infancy has therefore been presented by the World Health Organization (*4*).

Little work has been done to establish DDT use in malaria control as a single source of contamination in breast milk. No information is available on the possible changes in levels due to yearly applications of DDT. Some studies have implicated agriculturally contaminated diet as the major route of uptake (*5*, *6*). Reports from countries where malaria control is practised frequently do not state the source of DDT, possibly because this is very difficult to establish. Whenever DDT is available for malaria control, it is also used frequently in agriculture, thereby increasing the number of sources.

The northern part of Kwa-Zulu in Natal is endemic for malaria. Since the 1950s, the dwellings of the more than 300 000 residents were treated every year (from January to March) with DDT at a rate of 2 g/m^2 on the inner wall surfaces (*7*). During the 1988 season, 26 018 kg of 75% emulsifiable DDT were used in 98 912 dwellings.[a] The aim of the present study was (*a*) to determine the changes in the mean levels of DDT in breast milk from mothers exposed to DDT during spraying for malaria control, (*b*) to compare this with a control group not exposed to such DDT, and (*c*) to try and determine the risk posed by DDT in breast milk to the health of the infant.

Materials and methods

Subjects

Milk samples were collected at two hospitals during 1986/7. The mothers attending clinics at Mseleni Hospital (exposed group) live in an area where DDT is used only for malaria control (other uses of DDT have been banned in the Republic of South Africa since 1976 (*8*)). Mseleni is situated on the shore of Lake Sibaya on the Makatini Flats and is removed from major routes of migration. In a pilot study it was found that migrants from Mozambique, where hardly any malaria control is practised, had very low serum levels of DDT (H. Bouwman, unpublished observations, 1987). The homes are usually constructed of mud, branches and thatch. Homesteads consist of three to seven such structures and house from 4 to 22 people. The major source of income is

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from migrant labour in mines or farms. The diet consists of staples such as maize and rice with fish (caught locally), and the flesh of goats, chicken or cattle. This is supplemented by fruits, nuts and roots.

The control group was drawn from the Port Shepstone area in southern Natal where malaria does not occur. No DDT has been used there since 1976 for any purpose (*8*). The population from this area is also stable, but more westernized than the Mseleni population. Dwellings here also are constructed from mud, but cement structures are more in evidence. Homesteads consist of two to four dwellings. Incomes were generated through working in transport, plumbing, carpentry and the tourist industry. The diet was essentially the same as for the Mseleni population; most of the food was obtained from retailers.

Sample collection

Four cross-sectional surveys were undertaken over a period of twelve months (November 1986, March, June and November 1987) (Nov'86, Mar'87, Jun'87 and Nov'87). Indoor application of DDT was completed just before the Mar'87 survey. This allowed comparison of the trough levels (Nov'86 and Nov'87, just prior to spraying) with levels found after application.

Informed consent was obtained from all the mothers on routine visits to the clinics and from mothers in the delivery wards of the two hospitals. A questionnaire was completed for each participant. Information obtained included ages of the mother and children, parity, previous residence, and occupational exposure to DDT or other pesticides. This was done in their own language.

The mothers were provided with clean 100-ml glass beakers and asked to manually express about 10 ml of breast milk. The milk was immediately transferred to 10-ml blood-collecting tubes, kept on ice and then frozen on the same day.

Sample preparation and chromatography

Samples were extracted using a method previously described (*9*). Briefly, this involved the denaturing of the milk protein using mercapto-ethanol and solubilizing the fat using deoxycholic acid. After liquid–liquid extraction with hexane, the extract was cleaned up using silica gel. The purified extract was analysed using gas-chromatography (electron capture detector) with aldrin as an internal standard. This method gave recoveries ranging from 107% for DDT at high levels to 74% for DDD at low levels. ΣDDT recovery was 98.6%. This method was developed to facilitate recovery of a fraction of the spiked compound that was not extracted after the spiked sample was frozen for longer than 24 hours. The results presented here are not corrected for recovery.

Results

Of the mothers approached, only those experiencing difficult or no lactation did not participate. A comparison of the study and control groups is presented in Table 1. At the 5% level there were no significant differences in parity, maternal age, infant age, or percentage of milk fat between the two groups. Mothers from the Murchison area (the control group) tended to practise more bottle-feeding than mothers attending the Mseleni clinics. (The percentages of mothers who practised bottle-feeding exclusively were included in the table for the sake of completeness, but were not included in the total number of participants or in subsequent statistics). Establishing maternal age was difficult as in many cases this was not known. An estimated age was used. The same was true for infant age, but here more accurate inference was possible.

Table 1: **Comparison of the two groups of mothers and children attending baby clinics**

	Exposed group		Control group	
No. of surveys	4		3	
No. of samples	132		88	
Maternal age (years)[a]	25.5	(5.6)[b]	25.5	(7.1)
Parity[a]	2.7	(1.8)	3.0	(2.1)
Neonatal age (months)[a]	8.6	(5.4)	7.7	(5.9)
% Breast-feeding	89		71	
% Bottle-feeding	1.5		4.6	
% Breast + bottle feeding	8.3		24.1	
% Milk fat[a]	3.95	(2.1)	3.76	(2.3)

[a] These do not differ significantly at the 95% level.
[b] Standard deviations are given in parentheses.

It became apparent that the levels of DDT in the breast milk of the control group were so low, and already (after the third survey) significantly different from the exposed group (at the 1% level), that there was no reason to conduct the last survey. The Poisson test for rates from the surveys of the two groups revealed no difference between them ($P = 0.0037$; rate ratio = 1.125).

DDE and DDT were detected in all the samples (of all four surveys) of the exposed group. DDD was not detected in three samples of the exposed group. The control group was 100% positive for DDE, five samples were positive for DDD, and only 19 had detectable DDT residues. Standard deviations of the ΣDDT concentrations in breast milk of the exposed group were relatively large (Table 2), sometimes as large as the mean value, indicating a wide distribution. The largest range for ΣDDT, 57.3 mg/kg, was for the Jun'87 survey. The concentration data were skewed, as indicated by the difference between the mean and median. The concentration data had a significant log-

Table 2: **Levels of DDT and metabolites in the milk fat of the exposed group**

	Concentration (mg/kg milk fat)					
	Mean	Median	Standard deviation	Standard error	Maximum	Mimimum
Nov'86 ($n=39$):						
DDE	6.86	5.90	4.95	0.79	18.50	0.60
DDD	0.43[a]	0.43	0.36	0.06	1.78	0.0
DDT	4.89[b]	4.70	2.47	0.40	11.00	0.42
ΣDDT	12.21	12.63	7.38	1.18	30.00	1.05
%DDT	42.57[a]	42.54	8.37	1.34	67.20	25.00
Mar'87 ($n=35$):						
DDE	7.06	5.15	5.95	0.99	30.80	0.30
DDD	0.13	0.11	0.12	0.02	0.55	0.0
DDT	6.59	5.30	3.57	0.60	18.50	0.70
ΣDDT	13.79	14.11	9.08	1.51	44.20	1.00
%DDT	50.87[b]	50.30	8.60	1.43	66.60	27.60
Jun'87 ($n=28$):						
DDE	10.95	5.50	10.96	2.07	46.90	0.50
DDD	0.58[a]	0.46	0.53	0.10	2.14	0.0
DDT	7.93	6.40	5.80	1.10	28.80	1.40
ΣDDT	19.45	19.45	16.10	3.04	59.30	2.00
%DDT	45.85	45.79	9.84	1.86	72.60	19.20
Nov'87 ($n=30$):						
DDE	9.98	7.70	7.99	1.46	36.90	1.30
DDD	0.49[a]	0.38	0.36	0.07	2.14	0.0
DDT	7.85[b]	6.50	4.80	0.88	20.30	1.22
ΣDDT	18.34	18.34	12.60	2.30	57.60	2.70
%DDT	43.27[b]	44.35	10.04	1.83	57.50	3.90

[a] $P<0.001$ (comparing log transformed data in a two-tailed *t*-test).
[b] $P<0.01$ (comparing log transformed data in a two-tailed *t*-test).

normal distribution (χ^2 goodness-of-fit test, $P>0.05$). The highest value for ΣDDT was 59.3 mg/kg sampled during Jun'87. DDT made up between 42% and 46% of the ΣDDT, except the Mar'87 survey, when it increased to over 50%. Percentage DDT had a significant normal distribution (χ^2 goodness-of-fit test, $P>0.05$).

The levels of DDT and metabolites in the control group (Table 3) were much lower than that of the exposed group, but the standard deviations were also of the same order as the respective mean values. The widest range for ΣDDT was 4.79 mg/kg for the Mar'87 survey. Data were slightly skewed, as indicated by the difference between the mean and median. Percentage DDT values fluctuated between 0% and 24.0% with the means between 4.2% and 7.5%.

For each survey the groups were highly significantly different ($P<0.01$) with respect to DDT, DDE, DDD, ΣDDT and %DDT. No significant change was found in breast milk levels of DDT or DDE between surveys for the control group. Levene's test was employed to test whether the variances of the surveys were significantly different. If the variances were not different, then the means were compared with one-way analysis of variance; otherwise the test by Welch was used. Specific differences between surveys were tested for by making use of an approximate *t*-test at the Bonferroni adjusted level of significance (Tables 2 and 3). DDD for the control group was not tested as the small number of samples with detectable amounts did not allow any meaningful interpretation of change.

For the exposed group no significant change was detected for DDE and ΣDDT (Table 2). Mean DDE varied between 10.95 mg/kg for Jun'87 and 6.86 mg/kg for Nov'86. DDD showed significant change, varying between means of 0.13 mg/kg for Mar'87 and 0.58 mg/kg for Jun'87. Mean DDT increased significantly from 4.89 mg/kg in Nov'86 to 7.85 mg/kg in Nov'87 ($P=0.0052$), but no other pairwise significant change was found for DDT. Percentage DDT increased significantly from 42.75% in Nov'86 to 50.87% in Mar'87 (Table 2; $P=0.0001$). The decrease in percentage DDT from Mar'87 to Nov'87 (43.27%) was also significant ($P=0.0019$). The Jun'87 value (45.85%) did not differ significantly from the Mar'87 and Nov'87 values.

Considering the percentage contribution of the various isomers, the increase in ΣDDT found directly after application was accompanied by an increase in percentage DDT (Fig. 1). The increase in ΣDDT from

Table 3: **Levels of DDT and metabolites in the milk fat of the control group**

	Concentration (mg/l milk fat)					
	Mean	Median	Standard deviation	Standard error	Maximum	Minimum
Nov'86 ($n=25$):						
DDE	0.608	0.540	0.375	0.075	1.56	0.10
DDD	0.001	0.000	0.006	0.001	0.03	0.0
DDT	0.048	0.040	0.074	0.015	0.36	0.0
ΣDDT	0.658	0.550	0.411	0.082	1.56	0.12
%DDT	7.464	7.273	6.860	1.372	24.00	0.0
Mar'87 ($n=29$):						
DDE	0.922	0.590	0.954	0.177	4.73	0.0
DDD	0.0	0.000	0.0	0.0	0.0	0.0
DDT	0.037	0.020	0.048	0.009	0.23	0.0
ΣDDT	0.957	0.600	0.969	0.180	4.79	0.0
%DDT	4.244	2.857	5.246	0.974	4.79	0.0
Jun'87 ($n=34$):						
DDE	0.497	0.375	0.458	0.079	2.06	0.03
DDD	0.0	0.000	0.0	0.0	0.0	0.0
DDT	0.025	0.020	0.027	0.005	0.23	0.0
ΣDDT	0.522	0.405	0.471	0.081	2.10	0.03
%DDT	5.297	4.074	5.593	0.959	21.70	0.0

Mar'87 to Jun'87 saw a reversal of this trend (a decrease in percentage DDT), with the percentage contribution of the various isomers looking very much like that of Nov'86 and Nov'87. No significant change in the levels or in the composition of the isomers in the breast milk of the control group was obvious (Table 3 and Fig. 2).

Discussion

The socioeconomic circumstances of the two groups were such that no activity (such as employment in a pesticide factory) or situation (such as nearby intensive farming using DDT) would have caused exposure to DDT other than from background levels or from use for malaria control. The mean maternal and infant ages, parity, and percentage milk fat did not differ significantly between the groups (Table 1), which were therefore considered as comparable.

The DDT levels in breast milk of the exposed group were significantly higher than for the control group. As DDT use for malaria control was the only difference between the groups, the assumption that yearly applications resulted in exposure, uptake and eventual elimination via milk is therefore confirmed.

Kalra & Chawla (*10*) were the first to report DDT levels in breast milk from India. The mean value for ΣDDT was 510 μg/l (whole milk) for 75 mothers, but no mention as to type of exposure was made. Attempts to relate malaria control activities (using DDT) to levels in fat tissue of the general population in India were not successful (*11*). House treatment with DDT was also implicated as a source of DDT detected in the body fat of people in Israel (*12*). Malaria control was mentioned by Hashemy-Tonkabony & Fateminassab (*13*), but no correlation with areas under such control in Iran were made (mean ΣDDT level = 24 μg/l for 131 whole-milk samples). Weisenberg et al. (*14*) mentioned "disease vector control" in Israel, but did not relate their findings to pesticide levels. Stacey & Tatum (*15*) established a clear connection between the levels of dieldrin in human milk and house treatment for termite control.

Slorach & Vaz (*16*) reported, in a survey of human exposures to selected organochlorine compounds, the mean national levels of DDT and DDE in milk from various countries. The highest median levels in milk fat were from China (4.4 mg/kg DDE and 1.8 mg/kg DDT) and India (4.8 mg/kg DDE and 1.1 mg/kg DDT) where DDT is still used in agriculture and for vector control. Samples were taken from mothers nursing their first or second child and residing in urban areas.

Perhaps the best examples of positive relationships between malaria control and DDT levels in breast milk come from studies in New Guinea, Guatemala and Kenya. Hornabrook et al. (*17*) presented evidence that malaria control contributed to elevated levels in whole milk (246 μg/l from a sprayed area compared to 3.32 μg/l from an unsprayed island). However, the possibility that DDT had been used for agriculture on the control island could not be excluded. Winter et al. (*18*) looked at 290 milk samples

Fig. 1. **Changes in percentage composition of DDT and metabolites in the breast milk of mothers exposed to DDT applications for malaria control (exposed group).**

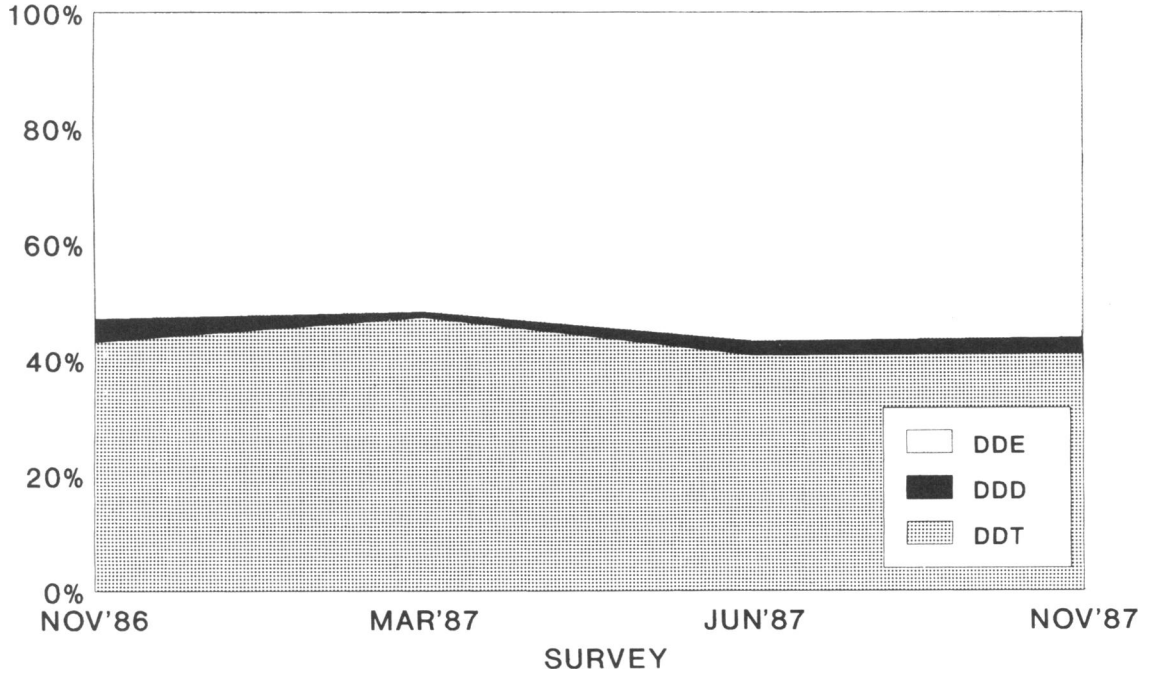
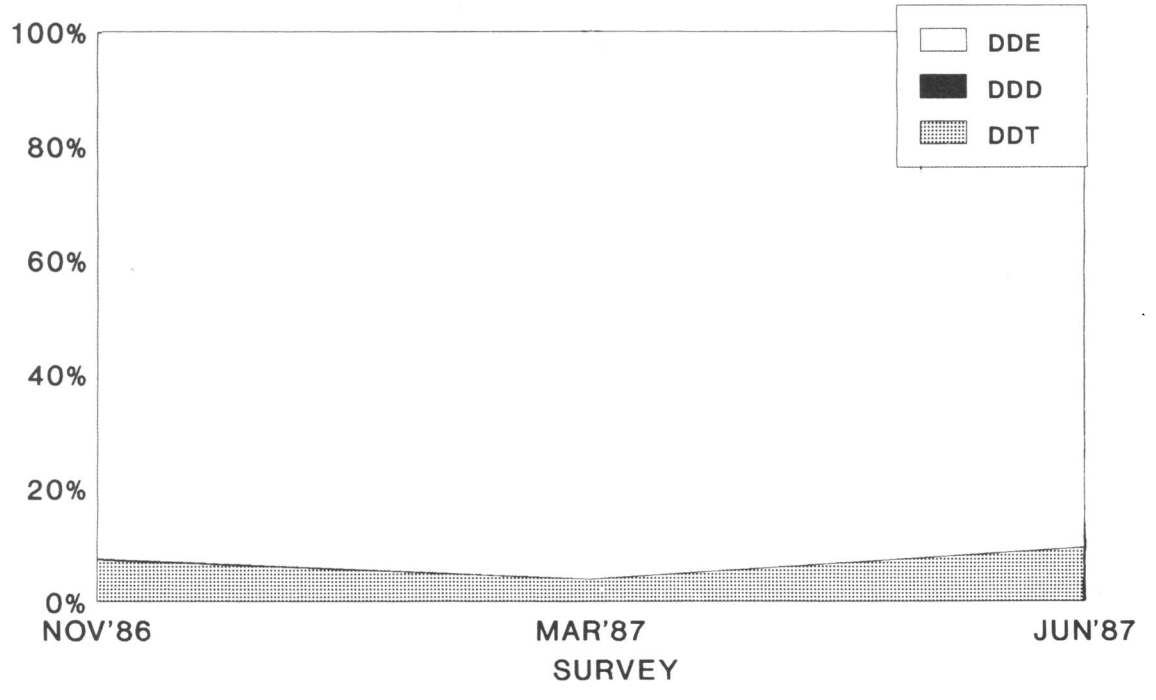


Fig. 2. **Changes in percentage composition of DDT and metabolites in the breast milk of mothers not exposed to DDT, except from background sources (control group).**



from eight different locations in Guatemala. The area with the highest level of DDT in breast milk was Livingston (mean, 864 μg/l for 30 whole-milk samples) where intra-domiciliary DDT was applied twice every year. Again, DDT might have been used in agriculture on a small scale around Livingston. Other areas had lower levels, apparently related to cultivation of cotton, without DDT being used for malaria control. The only comparable study from Africa was that of Kanja et al. (*19*) who analysed samples from mothers breast-feeding their first or second child. Nomads from Loitokitok, Ilbarma area (Kenya), had mean levels of 1.96 mg/l (milk fat). The highest value was from Rusinga Island at 18.73 mg/l (milk fat) for 25 milk samples. DDT was apparently used for malaria and tsetse-fly control on the island (*19*). If the data for the exposed group of the present study were recalculated to include only those mothers breast-feeding their first or second child ($n=65$), the mean ΣDDT level was 20.1 mg/kg. This represents the highest recorded value for Africa.

The most noticeable feature of the present serial data was not the significant differences of DDT levels between surveys, but the lack of it. Excluding DDD (see below), only three instances were determined where significant changes in levels were determined. For DDE and ΣDDT, no statistically significant changes were detected (Table 2), although ΣDDT did show a marked increase. The uptake of DDT after application was slower than might have been expected. There was a continued increase in levels up to Jun'87, when they were 37.2% higher than in Nov'86 and 29.1% higher than in Mar'87. This suggests that the uptake of DDT from the environment by the mother continues long after its once yearly application, and at a faster rate than its excretion via milk.

Breast-feeding mothers eliminate DDT, DDE and DDD much faster through milk production than any other mechanism present in the general population (*20*). With our present knowledge, therefore, it is a matter of conjecture whether or not a short-term, elevated output immediately following DDT application occurred. If it did, and was present over a longer period, then the results would have indicated this. This was further corroborated by the significant increase in %DDT (in breast milk) following DDT application. It is, however, a shortcoming of cross-sectional studies that events with longer duration of exposure have a higher proportion of cases than short-term incidents (*21*). The likelihood of detecting such an event would be increased by a longitudinal protocol with daily sampling before and after application.

No obvious factor was at hand to explain the difference in levels between the Nov'86 and Nov'87 surveys. An over- or under-representation (bias) of groups which could have higher levels of DDT in breast milk (such as primiparous mothers or the younger infants) for the Nov'87 and Nov'86 surveys was a possibility. The comparison of these variables did not show any marked difference in contribution of these groups. The standard deviations for the concentrations of DDT in breast milk for the two surveys (Nov'86 and Nov'87) were also quite large (2.47 and 4.80, respectively). More samples per survey could have reduced the influence of the variation.

The problem of interpretation of any change in levels of DDD (not discussed so far) was due to the very low quantities. A number of regression analyses and analyses of variance were performed, but significance was low. The apparent lack of toxicity and paucity of any substantial knowledge of this compound, other than as a drug (*22*), prevents any further meaningful discussion.

The percentage contribution of each of the three components of ΣDDT gave the best account of the dynamics in the exposed group (Fig. 1). It can be assumed that the percentage composition is independent of the absolute values. The increase in % DDT was significant from Nov'86 (42.57%) to Mar'87 (50.87%). A significant decrease was obtained from Mar'87 to Nov'87 (43.27%). The decrease in %DDE (Mar'87, Fig. 1) was more a result of the increased %DDT than a reduction in real DDE levels.

Finally, changes in levels of DDT, DDE and ΣDDT were not significantly related to surveys for the control group and no statistics were therefore presented. The small contribution of DDT to the total contamination is clear from Fig. 2.

The observed changes in levels of the organochlorines in the exposed group can be explained in the context of application of DDT uptake and elimination. Application of DDT resulted in an increase in exposure that gave rise to an elevated percentage DDT in the milk. During the following months, DDT was metabolized or excreted so that the percentage composition of DDT and DDE returned to the initial composition. The reduction in % DDT was corroborated by the difference in % DDT of the samples taken three months after spraying ceased (Jun'87, 45.85%), which was not significantly different from the preceding survey (Mar'87) or the following survey (Nov'87) (Table 2).

The estimated ADI for DDT is 0.02 mg/kg/day (*23*). Assuming the following parameters as standard, the daily intake of ΣDDT by infants via breast milk can be calculated for different situations:

—the mean weight of the infant at 5 kg (*1*);

—the mean daily milk intake at 800 ml (*1*);

—the mean fat content of the milk was 3.95% (this study);

—the mean ΣDDT level (exposed group) was 15.8 mg/kg milk fat;

—the maximum ΣDDT level was 59.3 mg/kg milk fat (this study);
—the mean daily ΣDDT intake for the infants of the whole group was 0.100 mg/kg/day; and
—the maximum daily ΣDDT intake observed during this study was 0.375 mg/kg/day.

The exposure of the children in the above two categories was 5 and 18.8 times the ADI. From the literature, these dosages were not expected to result in acute or obvious toxic effects in adults. The FAO/WHO report (*23*) mentioned that "neonates are not at increased risk" with regard to DDT. However, more recent information has indicated possible injury to infants at DDT levels in the breast milk of exposed mothers, as found in the present study.

The study of Rogan et al. (*3*) was the only one we could trace that determined the effects of DDT in breast milk on infants. DDE levels in breast milk were associated with hyporeflexia (using the Brazelton Neonatal Behavioral Assessment Scale) in infants just after birth (59% tested within one week after birth, the rest within 31 days), thereby also including prenatal exposure to DDE (*3*). Hyporeflexia, defined as the situation when more than four out of 20 reflexes were low or not elicited, increased significantly from 3.4% at 0–0.9 mg/kg DDE in milk (milk fat; $n=59$) to 14.1% at $\geqslant 6$ mg/kg DDE ($n=64$), the highest interval. In the present study, the measured levels represent the exposure to breast-feeding infants, some as old as two years (Table 1). Since the placenta acts as a barrier to DDT and DDE (*22, 24*), most of the effects of the organochlorines can be attributed to postpartum exposure, especially when the total lactation period is considered.

The dose–response relationship found by Rogan et al (*3*) indicates that an increase in exposure could result in more infants being classified as hyporeflexive. This in turn will mean that any significant increase over a certain level cannot be deemed as of no consequence.

The data of Rogan et al. (*3*) can be used to extrapolate the results obtained in the present study. It must, however, be stressed that this is very tentative since the extrapolation goes beyond the levels determined by Rogan et al. (*3*). The assumption is therefore made that the relationship between concentration in breast milk and its influence on reflexes remains constant over the range of concentrations to be tested. A multiplicative regression of percentage low reflexes on concentration of DDT, using the data of Rogan et al. (*3*) was significant ($P=0.02$) with a coefficient of determination of 69%. The percentage low reflexes was estimated for the ΣDDT levels in breast milk of the control group (0.69 mg/kg milk fat), the mean of the exposed group (15.83 mg/kg milk fat), the maximum level (59.3 mg/kg milk fat) and the mean ΣDDT levels for Nov'86, Mar'87, Jun'87 and Nov'87. The calculated percentage infants with low responses were calculated to be 4%, 22%, 43%, 19%, 20%, 24% and 23%, respectively. The confidence intervals were very wide, but could not be calculated reliably.

The levels of DDE determined for the exposed group in the present study were higher than those given by Rogan et al. (*3*). Together with the presence of the presumably more toxic DDT compound, this provides a well-founded basis for the assumption that the infants in the exposed group are at risk. The increase in levels after DDT application could, when compared with the results from Rogan et al. (*3*), present more risk to the infant, although the pitfalls associated with extrapolation should be taken into account. The long-term effects (behavioural, clinical or other) in infants, whether permanent or not, are not known. The determination of the actual risk to such infants must therefore be a priority.

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Résumé

Concentration du DDT et de ses métabolites dans le lait de femmes du Kwa-Zulu à la suite d'épandages dans le cadre de la lutte antipaludique

Une étude a été entreprise pour mesurer les concentrations de DDT, de DDE et de DDD dans le lait de femmes habitant deux régions différentes du Kwa-Zulu. Des épandages intradomiciliaires de DDT sont effectués annuellement dans l'une de ces régions, mais non dans l'autre, dans le cadre de la lutte antipaludique (les autres utilisations du DDT sont interdites dans le pays depuis 1976). Les femmes de la première région constituaient donc le groupe exposé et celles de la deuxième région le groupe témoin. Les échantillons de lait ont été recueillis dans deux hôpitaux au cours de quatre enquêtes transversales, en novembre 1986 et en mars, juin et novembre 1987.

Le DDE et le DDT ont été détectés dans tous les échantillons recueillis dans le groupe exposé lors des quatre enquêtes; trois échantillons de ce groupe se sont révélés exempts de DDD. En ce qui

concerne le groupe témoin, des échantillons n'ont été recueillis qu'au cours des trois premières enquêtes; tous contenaient du DDE, cinq contenaient du DDD et 19 seulement une quantité détectable de DDT. Lors de chaque enquête, des différences significatives ($P < 0,01$) ont été notées entre les deux groupes en ce qui concerne le DDT, le DDE, le DDD, la concentration totale de DDT et de ses métabolites (ΣDDT) et le pourcentage de DDT (%DDT). Aucune variation significative n'a été constatée au sein du groupe témoin. Une augmentation des concentrations de DDT et de DDE a été observée dans le groupe exposé après l'épandage de DDT et s'est maintenue pendant trois mois, mais le changement en ce qui concerne le DDE et ΣDDT n'était pas significatif. La concentration moyenne de DDE a varié entre 10,95 mg/kg de matière grasse du lait en juin 1987 et 6,86 mg/kg en novembre 1986. La concentration moyenne de DDT a augmenté de façon significative, passant de 4,89 mg/kg de matière grasse en novembre 1986 à 7,85 mg/kg en novembre 1987 ($P = 0,0038$), mais aucun autre changement significatif entre deux valeurs n'a été observé pour cette substance. Le pourcentage de DDT est passé de 42,57% (ΣDDT = 12,21 mg/kg de matière grasse) avant la campagne de pulvérisation (novembre 1986) à 50,87% (ΣDDT = 13,79 mg/kg de matière grasse) après celle-ci (mars 1987). Six mois et neuf mois après l'épandage (juin et novembre 1987), ce pourcentage était respectivement de 45,85 (ΣDDT = 19,49 mg/kg de matière grasse) et 43,27% (ΣDDT = 18,34 mg/kg de matière grasse).

Compte tenu des concentrations de DDE trouvées dans le lait du groupe exposé et de la présence de DDT, probablement plus toxique, il y a tout lieu de croire que cette situation présente un risque pour les nourrissons. L'évaluation du risque réel doit donc être considérée comme prioritaire.

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