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Executive Summary

Introduction

This report documents a research study undertaken by the Agricultural Health Unit, Moree District Hospital, in 1991 and 1992 into the potential pesticide exposure of cotton chippers in the Gwydir Valley in the North-West of NSW. The study was funded by the Cotton Research and Development Corporation Ltd.

"Cotton chipping" involves the manual removal of weeds from cotton fields. "Chipping" refers to the use of a short hoe to "chip" out certain types of weeds.

The study was undertaken in two parts:

- Part 1 estimated the degree of pesticide exposure experienced by cotton chippers in the course of a growing season, using depression of erythrocyte cholinesterase activity as a proxy measure for exposure to all classes of pesticides used on cotton and as a direct measure of exposure to organophosphate pesticides;

- Part 2 estimated the distribution of exposure to pesticides over different parts of the body by recovery of pesticide residues deposited on clothing in the course of chipping work.

In addition, the opportunity was taken to interview a gang of chippers who had been accidentally exposed to pesticide spray drift during the course of the study, in an effort to identify safety procedures which may have failed or could be improved.

Methods and Results

Demographic and occupational details and initial blood samples were collected from 417 cotton chippers who were enrolled into Part 1 of the study. Of these, 16 were later excluded on the basis of possible recent exposure to organophosphate pesticides. Follow-up blood samples were collected from 115 of the chippers enrolled initially. We were unable to identify any obvious selection biases in the initial recruitment or follow-up of chippers in the study.
Analysis of erythrocyte cholinesterase activity from the blood samples revealed a six percent decline in the mean enzyme activity in those chippers who were followed up. This is highly statistically significant. No decline would be expected in an unexposed population. A number of the chippers suffered reductions in cholinesterase activity of greater than 30 percent of their initial values. Such reduction of cholinesterase activity may cause symptoms in susceptible individuals, although none of the chippers reported any symptoms specifically attributable to pesticide exposure in the course of Part 1 of the study.

Part 2 of the study revealed that it is possible to recover significant amounts of endosulfan and profenofos pesticide residues from clothing worn while chipping. Cotton chippers have traditionally worn minimal clothing during their work, an observation confirmed by this study. Most of the pesticides used in cotton agriculture are readily absorbed through the skin. Therefore there appears to be considerable opportunity for dermal exposure of cotton chippers to pesticide residues on the cotton plants after aerial spraying.

**Recommendations**

i) That the cotton industry be made aware that:

   a) cotton chippers may be exposed to pesticides in the course of their work, either from dermal contact with pesticide residues on or contained in the cotton plants and weeds, or from accidental exposure to spray drift from aerial spraying operations;

   b) although there is no evidence to suggest that cotton chippers are at particular risk of acute intoxication by pesticides, doubt exists about the biological significance and possible long term effects of the levels of pesticide exposure which cotton chippers may experience in the course of their work.
ii) That further research be undertaken by appropriate bodies to:

a) re-evaluate the appropriateness of currently recommended minimum re-entry periods for pesticides used on cotton crops, in the light of current and future cotton chipping work practices.

b) establish the most efficient, cost-effective and practical form of protective clothing for cotton chipping, and the minimum laundering requirements for such clothing.

iii) That the cotton industry be encouraged to institute the following measures as soon as possible:

a) A minimum clothing requirement for cotton chippers consisting of shoes or boots, socks, long trousers, long sleeved shirt (sleeves to be worn down), gloves and hat.

b) Mechanisms to prevent chippers being sent to work in fields which are wet with dew (which may enhance dermal exposure to pesticide residues).

c) Provision of adequate hand washing facilities at chipping workplaces (ie in cotton fields) and encouragement of chippers to wash their hands prior to eating or smoking and after finishing work.

d) Improvements in communication procedures between aerial spray operators, property owners and chipping contractors and overseers in order to reduce the possibility of accidental exposure of chippers to aerial spray drift.

e) Provision of emergency shower or other wash-down and clothing change facilities at the work site in the event of accidental exposure of chippers to aerial spray-drift.

f) Development and promulgation of preventative measure such as those outlined above as an alternative to an extensive cholinesterase testing programme for all cotton chippers. The technical and administrative infrastructure currently available is inadequate to support widespread cholinesterase testing for
cotton chippers. The casual and itinerant nature of their employment means that adequate operation of a mass testing programme may be impossible or unduly expensive.

However, it is desirable that some form of monitoring programme be set up to check the adequacy of the changes to dress code and work practices recommended above. One solution might involve recruitment of at least 50 cotton chippers as a "sentinel" cohort. They would undergo monthly cholinesterase blood tests and who would be required to adhere to the industry's revised dress code and work practices. In order to increase the cost-effectiveness of such a monitoring programme and to make it logistically possible given existing resources, the "sentinel" chippers could be drawn from local residents who indicate that they will be available for follow-up throughout the season.
1 Introduction

This report documents a research study undertaken by the Agricultural Health Unit, Moree District Hospital, in 1991 and 1992 into the potential pesticide exposure of cotton chippers in the Gwydir Valley in the North-West of NSW. The study was funded by the Cotton Research and Development Corporation Ltd.

2 Background

"Cotton chipping" involves the manual removal of weeds from cotton fields. "Chipping" refers to the use of a short hoe to "chip" out certain types of weeds. Depending on the crop height and the mix of weeds in the cotton field, direct pulling of weeds by hand may also be carried out.

Cotton chippers are typically employed on a casual, seasonal basis, usually by a contractor who organises chippers into teams or "gangs" and contracts their services out to cotton growers and property owners. In the Gwydir Valley, chipping commences shortly after cotton planting in late October or early November, with the number of chippers employed peaking in December. The use of chipping services begins to tail off in late January as the cotton crop begins to "close in", making negotiation of the fields and access to the crop beds (where the weeds grow) more difficult.

The Gwydir Valley is located in the major cotton producing area in Eastern Australia. No official estimates of the number of cotton chippers employed in the Gwydir Valley each year are available from the Australian Bureau of Statistics or the Commonwealth Employment Service. However, based on the number of chippers enrolled in this study and on the number of known chipping contractors operating in the area, it is likely that between 1500 and 2000 cotton chippers may be employed in the Gwydir Valley each year. In addition, a small but growing amount of chipping work is being performed by family members (who are not formally employed as cotton chippers) on smaller, dry-land cotton farms.

In the second half of 1990, the Agricultural Health Unit at Moree district Hospital began to offer a voluntary cholinesterase testing service.
During the 1990/91 growing season approximately 100 self-selected agricultural workers from the surrounding district were tested. Because individual baseline cholinesterase activities were not available in most cases, results were compared to previously derived population means. Nearly half of the 43 cotton chippers who participated had plasma and erythrocyte cholinesterase activities greater than 30% below the population mean. This degree of cholinesterase inhibition was not observed in any of the other occupational groups tested, although the number of subjects was small.

These findings were surprising, because cotton chipping had not previously been considered an occupation with appreciable risk of exposure to cholinesterase-inhibiting pesticides. Cotton chipping does not involve the direct handling, mixing or spraying of pesticides.

In the absence of exposure to pesticides in other occupational or domestic settings (such as working as a general farm hand or while spraying a domestic garden), and barring accidents (such as accidental exposure of cotton chippers to aerial spray drift while working in the field, as reported later in this document), the only potential sources of pesticide exposure for cotton chippers are foliar residues from aerial spraying and plant sap containing systemic pesticides. The later are applied, usually in pellet form, in the pre-emergent phase and are taken up systemically by crop and weeds alike.

Consideration of the following factors suggested that it was possible that cotton chippers could be exposed to measurable levels of foliar pesticide residue in the course of their work:

- Modern cotton agriculture involves extensive use of pesticides. In 1984, of all agricultural industries, the market for insecticides used on cotton represented nearly one quarter of all insecticides sold worldwide¹.

- Cotton chipping involves physical contact with cotton plants and weeds.

- Due to the climate of the cotton growing districts during the growing season, cotton chippers have traditionally worn minimal clothing, allowing ample opportunity for dermal exposure to foliar pesticide residues.
Since the work of Wolfe et al.\textsuperscript{2} in 1967 and Durham et al.\textsuperscript{3} in 1972, it has been accepted that the dermal route of exposure is the most significant for most classes of pesticide in almost all occupational settings.

A search of the scientific literature revealed a number of references to potential pesticide exposure in workers entering cotton fields. In 1958, Quinby et al.\textsuperscript{4} first highlighted the potential for exposure in workers who did not directly handle pesticides but merely entered fields which had been sprayed from the air. In 1969, Finley and Rogillio\textsuperscript{5} demonstrated that it was possible to recover measurable (and potentially biologically significant) quantities of pesticide from the clothing of cotton chippers. In 1975, Burns and Parker\textsuperscript{6} reported significant cholinesterase depression in a group of 12 "cotton scouts" (known as "bug checkers" in NSW) entering fields which had been sprayed with methyl-parathion. Also in 1975, Ware et al. used whole garments of cotton field labourers as a collection device to estimate safe re-entry periods for cotton fields. In 1979, Wicker et al.\textsuperscript{7} found significant, although asymptomatic, plasma and erythrocyte cholinesterase depression in cotton scouts entering cotton fields sprayed with organophosphate pesticides. In 1985, Spittler and Bourke\textsuperscript{8} referred to the potential for pesticide exposure in cotton field labourers, although they were primarily concerned with exposure amongst pesticide spray applicators.

In addition to specific references to cotton field labourers, there is a sizeable literature on the potential for dermal exposure to foliar pesticide residues amongst field workers in a number of other agricultural industries, particularly in orchards. Spear et al.\textsuperscript{9} provides a good review.

Based on these considerations, a study of potential pesticide exposure which was based on a larger, more representative sample of cotton chippers was undertaken. In addition, an attempt was made to estimate the distribution of pesticide exposure over various parts of the body so that some initial recommendations for preventative strategies could be made, in the event that significant levels of pesticide exposure were found.
The reader is referred to a document published by the NSW Department of Agriculture\textsuperscript{10} for a full description of pesticide usage in the cotton industry. However, a summary of the recommended pesticide application schedule appears below:

<table>
<thead>
<tr>
<th>Stage 1</th>
<th>Stage 2</th>
<th>Stage 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>to 9 Jan</td>
<td>10 Jan - 13 Feb</td>
<td>from 13 Feb</td>
</tr>
<tr>
<td>(35 days)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Endosulfan</td>
<td>Endosulfan</td>
<td>Methomyl</td>
</tr>
<tr>
<td>Thiodicarb</td>
<td>Thiodicarb</td>
<td>Thiodicarb</td>
</tr>
<tr>
<td>Monocrotophos</td>
<td>Methomyl</td>
<td>Parathion</td>
</tr>
<tr>
<td>Sulprofos</td>
<td>Monocrotophos</td>
<td>Monocrotophos</td>
</tr>
<tr>
<td>Chlorpyrifos</td>
<td>Sulprofos</td>
<td>Sulprofos</td>
</tr>
<tr>
<td>Chlorfluazuron</td>
<td>Profenofos</td>
<td>Profenofos</td>
</tr>
<tr>
<td><em>Bacillus thuringiensis</em></td>
<td>Chlorpyrifos</td>
<td>Chlorpyrifos</td>
</tr>
<tr>
<td><em>No pyrethroids</em></td>
<td>Pyrethroid (+/- piper-</td>
<td><em>No pyrethroids</em></td>
</tr>
<tr>
<td></td>
<td>onyl butoxide)</td>
<td></td>
</tr>
</tbody>
</table>

Cholinesterase-inhibiting organophosphate pesticides are used throughout the cotton growing season.
3 Goals and Methods

The study was undertaken in two parts: the first to estimate the degree of cholinesterase depression experienced by cotton chippers in the course of a growing season; the second to estimate the distribution of exposure to pesticide residues over different parts of the body. In addition, the opportunity was taken to interview a gang of chippers who had been accidentally exposed to pesticide spray drift during the course of the study in an effort to identify safety procedures which may have failed or could be improved.

3.1 Part 1: Cholinesterase activity measurement

Goal

To estimate the prevalence and degree of occupational exposure to cholinesterase inhibiting pesticides amongst cotton chippers through the use of serial assays of erythrocyte cholinesterase activity.

Methods

Data Collection

It has long been recognised that there is considerable individual variation in cholinesterase activity and that, ideally, two or three "baseline" measures of cholinesterase activity should be obtained before there is any possibility of exposure to cholinesterase inhibiting compounds\textsuperscript{11}. The casual, seasonal and often itinerant employment patterns of cotton chippers, combined with the geographical spread of their workplaces (cotton farms), made meeting this ideal difficult to achieve. Following discussions with a panel of cotton chipper contractors and property owners, it was decided that the only recruitment strategy which was logistically feasible given the resources available to this study was to enrol cotton chippers at their place of work, as soon as possible after each chipper had commenced employment (as a cotton chipper). This meant that most of the chippers enrolled into the study were potentially exposed to pesticides prior to the initial assay for cholinesterase activity. The frequency distribution of the approximate number of working days engaged in chipping prior to enrolment in the study is shown in Figure 1. The mean numbers of working weeks
engaged in chipping prior to enrolment (ie the mean time lag between commencement of chipping work and enrolment into the study) by month of enrolment is shown in Figure 2.

Figure 1. Number of weeks engaged in chipping prior to enrolment in study.
The study team visited cotton fields on three days (Tuesday to Thursday) of each week from 5 November, 1991 to 12 March, 1992, with the exception of a two week period over the Christmas and New Year holidays. One or two cotton properties were visited on each day and all chippers present on those properties were invited to be enrolled into the study. Of 423 chippers who were asked to participate, 6 refused. Demographic characteristics of these 6 chippers were not recorded.

Only those chippers who were employed by six cotton chipping contractors who had agreed to assist with the study were enrolled. We were unable to obtain information about the demographic characteristics of chippers employed by contractors who did not assist with the study. However, there is no reason to believe that the chippers who did participate differ in any systematic way from other cotton chippers, either in their demographic characteristics or their work practices and employment pattern.

At enrolment, the questionnaire which appears in Appendix 1 was administered at the place of work by one of four interviewers. The nature of clothing worn by each chipper was observed and recorded by the interviewer.
The purpose and nature of the study was explained and each participant was asked to sign a consent form and a release form allowing the Agricultural Health Unit to contact each chipper's employer as well the chipper themselves in the event that potentially hazardous degrees of cholinesterase inhibition were found.

Two weeks after the commencement of the study, those chipping "gangs" who had been visited by the study team previously were followed up (the "gang" having moved on to a different property by this time). A follow-up questionnaire, reproduced in Appendix 2, was administered to those chippers who had previously been enrolled in the study, and a follow-up venous blood sample was drawn. Chipppers who had not previously been enrolled were also invited to join the study.

This pattern of fortnightly visits to each of the chipping "gangs" which contained chipppers who were participating in the study was maintained as far as possible until the conclusion of the study in the third week of March, 1992. This coincided with the cessation of most commercial chipping activity.

Twenty millilitres of venous blood was drawn at each encounter with a study subject. Magnotti et al. caution against inadvertent direct contamination of the blood sample by pesticide residues on the skin. Therefore considerable attention was paid to skin preparation prior to each venepuncture. Preparation consisted of washing the antecubital fossa with soapy water, followed by rinsing with clean water (supplied by the study team) and double swabbing the puncture site with isopropyl alcohol skin wipes. Blood was drawn into lithium heparin glass tubes and stored at between 2 and 6 degrees Celsius in a portable cool box prior to analysis.

Cholinesterase assays and a standard clinical battery of liver function tests were carried out on the initial blood samples (ie those drawn at the time of enrolment of each chipper into the study). All subsequent blood samples drawn from each chipper were subject only to cholinesterase assays. Liver function tests were carried out in order to detect significant liver dysfunction, which may complicate the interpretation of plasma cholinesterase activity assays.

The liver function tests and cholinesterase assays were carried out by the staff of the pathology laboratory at Moree District Hospital. The
mean delay in carrying out assays on blood samples was 26 hours, the median delay 25 hours and the maximum delay was 73 hours. Blood was stored in a refrigerator at 6 degrees Celsius prior to analysis.

Plasma and erythrocyte cholinesterase activity assays were carried out by the potentiometric method (also known as the Michel method). In this method, plasma is diluted with a buffer, acetylcholine is added as a substrate and the fall in pH caused by liberated acetic acid is measured over a fixed period of time. Erythrocyte cholinesterase activity is determined in the same way after centrifuging, washing and haemolyzing the red cells.

As a quality control measure, blood samples taken during the one venepuncture were divided on two separate occasions, one in December 1991 and one in early February 1992. The first sample was analysed by the Moree Hospital staff in the usual manner. The second was sent by air freight to an independent, commercial, NATA registered laboratory which routinely processed a large number of cholinesterase activity assays using the Ellman colorimetric method. The results of each assay were compared for each individual. The laboratory staff at Moree Hospital did not have prior knowledge of the days on which these quality control checks were to be carried out.

All study data were recorded in a computerised relational database constructed specially for this study.

The original study design called for collection of detailed information on the identity and location of every cotton field in which each enrolled chipper worked, and information on the timing and nature of every pesticide application to each of these fields. In this way, a profile of the potential for exposure to pesticide residues could be built up for each chipper. The resulting exposure gradient between chippers would remove the need for a large control group. Panels of chipping contractors and property owners were consulted prior to the commencement of the study regarding the feasibility of collecting this information retrospectively at the conclusion of the study.

In fact, this information proved impossible to collect, mainly due to doubt about in which fields and properties gangs of chippers had worked. Future studies of this type should employ prospective data collection using diaries to avoid this problem.
As a result, changes in cholinesterase activities amongst chippers have been compared with a putative control group, constructed using the upper range of well-established, published values for intrinsic intra-individual variation in erythrocyte cholinesterase activity in unexposed people, measured by the same laboratory method as used in this study. Although the use of such a putative control group is not ideal, we feel that it does not invalidate the results reported below.

Analysis

Chippers who reported potential occupational or domestic exposure to cholinesterase-inhibiting compounds in the 3 months prior to their commencement of cotton chipping work were excluded from the analysis. The reasons for exclusion are listed in the Results section. The one chipper in whom liver function tests revealed significant liver dysfunction was also excluded.

In order to keep the analysis relatively simple and the results easily understandable, it was decided to analyse only the erythrocyte cholinesterase results. Erythrocyte cholinesterase is generally regarded as a more reliable and less variable measure of the biological effects of cholinesterase inhibiting compounds than plasma cholinesterase

Statistical analysis was undertaken using Version 6.04 of the SAS statistical package for microcomputers.

Descriptive statistics and frequency distributions were calculated for demographic and work practice variables.

A compound clothing score was derived for each chipper by summing a weighted mean of the indicator variables for each of the clothing types (i.e., if the chipper was wearing a shirt then $1 \times$ the shirt weight is accumulated, otherwise 0 is added to the overall clothing score). Weights were formulated after examination of the relative rates of dermal deposition of pesticide on each part of the body, derived from Part 2 of the study (see below). The following weights were used:
<table>
<thead>
<tr>
<th>Item of clothing</th>
<th>Weight</th>
</tr>
</thead>
<tbody>
<tr>
<td>Absence of shoes</td>
<td>1.5</td>
</tr>
<tr>
<td>Thongs</td>
<td>0.9</td>
</tr>
<tr>
<td>Shorts</td>
<td>1.2</td>
</tr>
<tr>
<td>Gloves</td>
<td>-0.2</td>
</tr>
<tr>
<td>Shirt sleeves</td>
<td>0.5</td>
</tr>
<tr>
<td>Singlet</td>
<td>0.7</td>
</tr>
<tr>
<td>No shirt</td>
<td>1.2</td>
</tr>
</tbody>
</table>

The analysis of the variation in erythrocyte cholinesterase in those chippers from whom follow-up blood samples were obtained was carried out using two null hypotheses.

The first null hypothesis ignores the potential for exposure to cholinesterase-inhibiting pesticides prior to enrolment into the study. The mean change in erythrocyte cholinesterase levels observed in the cotton chippers is compared to a mean change of zero which is expected in the absence of an external influence.

The co-efficient of variation of erythrocyte cholinesterase activity in an unexposed population has been variously estimated to range between 8 and 12%\(^{16}\). We took the higher figure of 12%. From this we calculated the variance expected in an unexposed population.

Because the variances of the exposed and putative unexposed groups are unequal, a significance test based on the statistic

\[
d = \frac{x_1 - x_2}{\sqrt{\frac{s_1^2}{n_1} + \frac{s_2^2}{n_2}}}
\]

where \(d\) is approximately distributed as a standardized normal deviate\(^{17}\), was used.

A second, more conventional test of the same null hypothesis was also carried out by performing a paired t-test on the difference between the
first and last erythrocyte cholinesterase measure for each chipper who was followed up.

We believe that measures of location and tests of significance based on this first null hypothesis (ignoring potential for exposure before the first cholinesterase assay) to be inherently conservative due to the potential for pre-enrolment exposure to pesticide residues discussed above.

The alternative null hypothesis takes the potential for pre-exposure into account by ignoring the direction of change of cholinesterase between successive measures. This hypothesis is supportable if one accepts that some of the chippers may have been suffered maximal cholinesterase inhibition prior to entering the study (due to cotton field exposure) and that subsequent blood tests were measuring cholinesterase recovery, not inhibition.

The fact that the mean initial cholinesterase (ie measured at enrolment) declined gradually throughout the course of the season (see the Results section) adds weight to this view. In order to test the statistical significance of this change in erythrocyte cholinesterase measured at enrolment, the enrolled chippers were divided into two groups: those enrolled during the first part of the chipping season in October and November, and those enrolled during the balance of the season. Because the distribution of cholinesterase activities at enrolment was not normally distributed, a non-parametric test was used rather than an unpaired t test on the mean cholinesterase activities. Accordingly, ranks were assigned to the erythrocyte cholinesterase value for each chipper at enrolment, and an unpaired t test was performed on the mean of the ranks for the two groups described above. This can be shown to be equivalent to performing a Wilcoxon rank sum test\(^8\).

In order to construct a test based on this alternative hypothesis, the expected mean absolute variation in an unexposed population must be calculated. It can be shown that this is equal to 0.6745 times the standard deviation, where 0.6745 is the inverse of the standard normal cumulative distribution function for a one-tailed probability of 0.25. The standard deviation is estimated by multiplying the assumed co-efficient of variation, 0.12, by the mean cholinesterase for all chippers at enrolment (used as an estimate of the unexposed population mean.
cholinesterase if it were to be estimated by the laboratory methods used by this study).

The mean of the absolute value of the change in cholinesterase in chippers can then be compared to the mean of the absolute values of variation in cholinesterase in a putative unexposed population, using the test described above.

In order to evaluate the combined effects of various factors on the observed variation in chippers who were followed up, a generalised linear model was fitted to the data, with the maximum change in cholinesterase activity between successive measures for each chipper as the dependent variable, and the following as independent variables: age, sex, date enrolled into study, chipping gang, smoking status, hand washing before eating and weighed clothing score. A fully saturated first order model was fit and variables eliminated to examine their influence on the fit of the model based on changes in the total $R^2$. 
3.2 Part 2: Estimation of Dermal Exposure to Pesticide Residues

Goal

To estimate the degree and distribution of dermal and respiratory exposure to pesticide residues while chipping cotton.

Methods

The following protocol was repeated three times: twice in fields which had recently been sprayed with endosulfan, and once in a field which had been sprayed with profenofos.

Four volunteers were dressed in specially made calico suits covering the trunk and limbs. Calico "overboots", plain white cotton gloves, surgical theatre caps (made from tissue paper) and half-face respirators fitted with activated charcoal filters were also worn.

The volunteers entered a cotton field which had been subject to aerial spraying with a known substance a known time beforehand and undertook normal cotton chipping activities for a period of one hour.

The volunteers then left the field and the calico suits were cut off them in sections using scissors. Care was taken to avoid cross-contamination between sections and volunteers by using a different pair of scissors for each volunteer and by cleaning the blades of the scissors with fresh isopropyl alcohol wipes after cutting each section. Each clothing section, together with the gloves, overboots, caps and respirator cartridges was then sealed in individual glass jars and stored in a coolbox prior to being transported by air for assay at the NSW Health Department Division of Analytical Laboratories (DAL) in Lidcombe, Sydney.

Identical, unexposed sections of calico, gloves, caps and respirator cartridges were also dispatched as negative controls.

A series of six positive controls was also dispatched to DAL for each of the two target pesticides. The positive controls were created by pouring known quantities of a set of serial dilutions of the target pesticide onto otherwise unexposed samples of calico which had been placed in
glass jars. Technical grade acetone was used as the diluent. The serial dilutions were carried out using volumetric flasks and disposable micropipettes. For the first set of positive controls, using endosulfan, the acetone solvent was not allowed to evaporate before the glass jars were sealed. The acetone was allowed to evaporate for the second set of controls using for profenofos.

At DAL, pesticide residues were eluted from the contents of each jar with "nanograde" quality cyclohexane. The elute was then analysed with a high pressure gas chromatograph fitted with an electron capture device in order to estimate the quantity of the target pesticide present on each of the sections of clothing and respirator cartridges. The surface area of each clothing section was also measured or, in the case of the gloves and caps, estimated.

Results are presented in Table 7 as absolute quantities of pesticide recovered and as relative quantities per unit surface area of each clothing section.

In addition, the quantity of pesticide recovered per unit surface area has been multiplied by the standard body surface areas according to Berkow's "rule of nines" to give an approximate indication of the maximum potential dermal deposition of pesticide assuming uniform distribution over that particular part of the body. Finally, weighting factors published by Wester and Maibach have been applied in order to adjust for differences in dermal absorption rates in different areas of the body. The final column of figures in Table 7 gives a relative indication of the potential contribution of dermal exposure to pesticide residues on each part of the body to the overall exposure via the dermal route.

4 Results

4.1 Part 1: Cholinesterase Activity Measurement

Enrolment, Exclusions and Follow-up

417 chippers were enrolled into the study. Of these, 16 were excluded from the analysis because of self-reported potential exposure to cholinesterase-inhibiting pesticides either in the 3 months prior to
enrolment or during the course of the study. Chippers were specifically asked at each encounter about occupational and non-occupational exposure to or handling of pesticides. A list of the reported prior exposures of the chippers who were excluded from analysis appears in Appendix 3. A sensitivity analysis was performed for each of the results and statistical tests reported below by including the 16 "excluded" chippers. There was no appreciable effect on any of the results or statistical tests.

52% of enrolled chippers indicated that they lived in the Moree district.

One or more follow-up cholinesterase measures were collected from 115 of the enrolled chippers. The age and sex distributions of all chippers enrolled in the study and of chippers for whom follow-up information is available are shown in Figures 3 and 4 respectively. They are comparable.

![Figure 3 - Age/Sex Distribution of all Chippers enrolled in the study.](image-url)
Figure 4 - Age/Sex Distribution of Chippers who were followed-up
Clothing Practices, Smoking and Hand-Washing Habits

Table 1 below indicates the proportion of chippers wearing the indicated items of clothing at enrolment. The column headed "Concordance at follow-up" indicates the proportion of those chippers who were followed up who were wearing the same items of clothing at follow-up.

<table>
<thead>
<tr>
<th>Item of Clothing</th>
<th>Percentage of Chippers wearing Item at Enrolment</th>
<th>Concordance at Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bare Feet</td>
<td>0.2</td>
<td>-</td>
</tr>
<tr>
<td>Thongs</td>
<td>9.6</td>
<td>88</td>
</tr>
<tr>
<td>Shoes or boots</td>
<td>90.2</td>
<td>92</td>
</tr>
<tr>
<td>Shorts</td>
<td>59.5</td>
<td>77</td>
</tr>
<tr>
<td>Long Pants</td>
<td>40.5</td>
<td>77</td>
</tr>
<tr>
<td>Singlet</td>
<td>7.0</td>
<td>65</td>
</tr>
<tr>
<td>Short Sleeved Shirt</td>
<td>58.8</td>
<td>86</td>
</tr>
<tr>
<td>Long Sleeved Shirt (with sleeves rolled down)</td>
<td>34.2</td>
<td>79</td>
</tr>
<tr>
<td>Gloves</td>
<td>21.3</td>
<td>67</td>
</tr>
<tr>
<td>Hat</td>
<td>53.6</td>
<td>89</td>
</tr>
</tbody>
</table>

51% of the enrolled chippers were smokers. 73% indicated that they usually smoked while chipping.

37% of chippers indicated that they did not wash their hands before eating while at work. Of those who usually washed their hands, 73% used their own drinking water for this purpose, 12% used water supplied by their employer and 15% used irrigation water.
Reported Symptoms

None of the cotton chippers reported symptoms which were consistent with acute intoxication by cholinesterase-inhibiting compounds during the course of the study.

43% of chippers reported problems with sunburn, predominantly on the arms, legs and face.

19.2% of chippers reported problems with skins rashes on the forearms and lower legs.

15.2% of chippers reported problems with cuts and abrasions from the cotton plants and weeds, again predominantly on the forearms and lower legs.

Cholinesterase Assay Quality Control

The Pearson correlation co-efficients for the correlation between the reference laboratory erythrocyte cholinesterase assays and the Moree laboratory assays was 0.95, indicating excellent agreement. The correlation co-efficient for plasma cholinesterases was 0.97.

Intra-individual change in Erythrocyte Cholinesterase

The mean initial erythrocyte cholinesterase for all enrolled chippers was 0.84 units.

The mean and variance of the intra-individual change in erythrocyte cholinesterase for the 115 chippers for whom follow-up cholinesterases were available were -0.0498 and 0.0095313.

The expected mean intra-individual change in cholinesterase in an unexposed population is 0 and the expected variance, using the mean initial erythrocyte cholinesterase as an estimate of the unexposed population mean (a conservative assumption) and assuming a co-efficient of variation of 0.12, is \((0.12 \times 0.84)^2 = 0.0101606\).

Substituting these values in the formula for the d statistic given in the Methods section, using values of 115 for both \(n_1\) and \(n_2\), gives a value of 3.8. Referring d to a table of critical values for a standardised
normal deviate\textsuperscript{20} gives a probability value (p-value) of 0.00007. This indicates that it is highly unlikely that the mean change in erythrocyte cholinesterase observed in the chippers who were followed up would be observed by chance.

A more traditional paired t-test, in which the mean of the difference between the initial and final erythrocyte cholinesterase activities for each chipper who was followed up is compared with zero, gives a value of t of 5.318 with a probability (p-value) of 0.00013, also a highly significant result.

It should be noted that the mean decrease is just 6% of the mean initial cholinesterase. However, the mean decrease obscures the fact that a substantial proportion of chippers experienced much greater decrements in their cholinesterase activity.

Figure 5 illustrates the frequency distributions of cholinesterase change of the chippers who were followed-up and an unexposed population. The skewness to the left in the frequency distribution of changes for the chippers, shown to statistically significant, can be appreciated.
The frequency distribution for the study subjects is clearly bimodal. Visual inspection of scatter plots of change in cholinesterase versus chipping gang, age group, sex and date of enrolment revealed that by clustering the chippers into two groups of 57 and 58 each, based on the identity of the "gang" in which they worked, much of the bimodality could be removed. Such clustering of chippers has an a priori logical basis because chippers working in one gang tend to work in the one field or on the same property, and will therefore experience similar exposures to pesticide residues. If a gang works predominantly in fields which have been sprayed with cholinesterase-inhibiting pesticides, the overall exposure for the chippers in that gang is likely to be much greater than for chippers in a gang which rarely works in such fields.

Figure 6 illustrates the frequency distributions of cholinesterase change after the chippers were clustered into 2 groups on the basis of the gang in which they worked.

![Graph of Proportion of Chippers vs Intra-Individual Change in Erythrocyte Cholinesterase](image)

The d statistic for the cluster of chippers in the left hand peak of the bimodal distribution is 5.8, which is also highly statistically significant. The mean decrease in cholinesterase in this cluster is 0.103, which represents a mean decrement of 12.3%.
Regression Analysis

Regression analysis revealed that only two variables, the gang identity and the date of enrolment, were statistically significant predictors of the intra-individual change in cholinesterase.

Decline in initial Cholinesterase during the course of the Season

Figure 7 shows the distribution of erythrocyte cholinesterase activities at enrolment for two groups: those enrolled in October or November and those enrolled subsequently.

An unpaired t-test on the ranks of the two groups (equivalent to a Wilcoxon rank sum test), assuming equal variances, gives a value for the t statistic of 6.7506 with 400 degrees of freedom, which is significant at the 0.0001 level. The t statistic is also significant at this level if unequal variances are assumed.

4.2 Part 2: Estimation of Dermal Exposure to Pesticide Residues

Three recovery trials were carried out, each using four volunteers.
The first two were in fields which had been subject to aerial spraying with endosulfan 240g per litre in an ultra-low volume formulation at 3.0 litres per hectare.

The first field had been sprayed just 7 hours earlier. The mean crop height was approximately 30 centimetres.

The second field had been sprayed 24 hours earlier. The approximate mean crop height was 50 centimetres.

The third recovery trial was conducted in a field which had been sprayed with profenofos 250g per litre at 3.0 litres per hectare 30 hours earlier. The mean crop height was approximately 70 centimetres.

The results of the recovery trials, used to validate the pesticide assays, are shown below in Figure 8.

![Graph](image)

The lower limit of detection of the assays lies between 0 and 0.5 µg for both endosulfan and profenofos. This is 2 or more orders of magnitude less than the amounts actually recovered during the recovery trials.

The results of individual assays are presented in Appendix 4. A summary of the results appears below in Table 7. The absolute amounts of
pesticide recovered from the respirator cartridges ranged between less than 0.1 μg to 1.6 μg. These quantities are 2 to 3 orders of magnitude less than the quantities of pesticide recovered from the sections of clothing and can be considered negligible. Actual amounts appear in Appendix 4.
### Trial No. 1 - Endosulfan (Crop Height 30 cm)

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<tr>
<th>Body Part</th>
<th>Mean Deposition Rate (μg/sq cm)</th>
<th>Relative surface area (%)</th>
<th>Relative absorption rate</th>
<th>Percentage contribution to total dermal absorption.</th>
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<td>Relative surface area (%)</td>
<td>Relative absorption rate</td>
<td>Percentage contribution to total dermal absorption.</td>
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Figure 9 - Relative contribution of body parts to total dermal absorption
Accidental Exposure of Chippers to Pesticide Spray Drift

5.1 Background

On December 9, 1991, one of the cotton chipper contractors who was co-operating with the study informed the Agricultural Health Unit that a gang of chippers, some of whom were participating in the study, had been accidentally exposed to spray drift from nearby aerial spraying operations. The pesticide was thought to be an endosulfan preparation (later confirmed by the property owner). Although it was not possible to attend the site of the incident immediately, it was decided to interview the chippers involved in this incident the following morning, in order to gain some insight into the circumstances surrounding such accidents.

5.2 Methods

A questionnaire was prepared (see Appendix 5) to collect information about the circumstances surrounding the accidental exposure and symptoms experienced by each chipper. The questionnaire included both open-ended questions and specific questions about a range of symptoms which might be attributable to pesticide exposure (not just specifically endosulfan exposure) were included. A number of symptoms which were not likely to be attributable to pesticide exposure were also included. Collection of blood samples from the exposed chippers was not possible for logistical reasons.

Questionnaires were administered to the chippers involved in the incident by a team of four Agricultural Health Unit staff members, between 7:30am and 9:00am on December 10 (the day after the incident). Chippers were interviewed individually, generally out of earshot of the other chippers. The chippers had no knowledge that they were to be interviewed prior to the arrival of the Agricultural Health Unit staff, and had minimal opportunity for discussion amongst themselves before each was interviewed. The only person who appeared to have any knowledge of the substance to which the chippers were exposed was the "ganger" (overseer), although a specific question on this point was not included in the questionnaire.
5.3 Results

Sixteen chippers were interviewed (including the "ganger"). Two chippers who had been exposed at the time of the incident on the previous day were absent from work on December 10 and were not interviewed. Otherwise, all chippers who had been at work in the field in which the incident occurred were present and agreed to be interviewed.

The age and sex distribution of the chippers interviewed is shown below in Figure 10.

![Age/sex distribution of chippers involved in overspray incident](image)

Following are summaries of the chippers answers to the open-ended questions.

Description of Incident

At about 7:00 am on December 9, the majority of the chippers noticed that an aeroplane had commenced spraying in a field immediately adjacent to where they were working. At approximately 7:30 am, the wind apparently changed direction suddenly and began to blow the spray towards where the chippers were working.
Chippers reported immediately noticing a strong smell of "chemicals". Four chippers reported that droplets were deposited on their skin, and two reported that their clothes were wet by the droplets.

The chippers were instructed by their ganger that they had the choice of sitting in the bus used to convey them to the field for a period or to continue working. Ten of the chippers reported that they sat in the bus for 30 to 60 minutes, then carried on working. None of the chippers were advised to wash or change their clothes or take any other action.

The frequencies of symptoms reported by the interviewed chippers are shown in Figure 11 below.

![Figure 11 - Symptoms reported by chippers involved in overspray incident](image)

Apart from headache, symptoms attributable to acute irritation of mucous epithelial surfaces (eyes, naso-pharynx, trachea) appear to predominate.

These symptoms are consistent with the acute symptoms described for endosulfan in a number of manufacturers’ Material Safety Data Sheets.
The ICI Safety Data Sheet for Endosan ULV \(^2\) (endosulfan 240 grams per litre in an ultra-low volume formulation) states:

"Health Effects
Short term exposure by all routes is considered to be toxic. SKIN: Will have a degreasing action on the skin. Can be absorbed through the skin with resultant toxic effects. EYES: A moderate to severe eye irritant. INHALATION: Inhalation of vapour can result in headaches, dizziness and possible nausea. INGESTION: Ingestion can result in vomiting, diarrhoea, convulsions and loss of consciousness."

The Incitec Material Safety Data Sheet for "Crop King Endosulfan ULV" (endosulfan 240 grams per litre)\(^2\), manufactured by Consolidated Fertilizers Ltd of Murarrie, Queensland states:

"HEALTH EFFECTS
Highly toxic by ingestion, inhalation or by skin absorption. Toxic symptoms in humans include headache, disorientation and cramps..."

It should be noted that endosulfan is usually formulated with an alkyl-benzene such as toluene or xylene as the solvent. It is unclear whether the health effects described in material safety data sheets for various endosulfan formulations relate to this solvent carrier or to endosulfan itself. Irritation of mucosal surfaces and headaches have been described for acute exposure to low concentrations of alkylbenzenes\(^2\), such as might occur in a cotton field during aerial spraying.

**Discussion**

Based on the reports of the cotton chippers involved, it appears that a number of chippers were exposed to varying and unquantified amounts of spray drift (in the form of vapour or droplets) from aerial spraying of endosulfan in a field adjacent to where they were working. When interviewed separately without prior notice, and without knowledge of what they had been exposed to, the chippers reported a remarkably similar set of symptoms in the 24 hours after the incident which were generally consistent with previously described effects of endosulfan formulations.
This incident can be viewed as a sentinel event for a number of issues which the cotton industry as a whole must address:

- there was a failure of communication between the aerial spray operator, the owner or manager of the property being sprayed, the owner or manager of the property on which the chippers were working and the chipping contractor and the "ganger" (overseer), which resulted in the failure to prevent simultaneous aerial spraying and chipping in close proximity to each other.

- there was no plan in place to deal with an incident such as accidental overspraying of chippers with pesticide. In particular, there were no emergency wash down and clothes changing facilities to minimise exposure in the event of an accident. There was no apparent mechanism established by which the chipper ganger could determine the nature of the pesticide involved, and no advice was offered to the chippers regarding medical follow-up or where they or health professionals could seek further information later that day should serious effects develop.

- in this particular instance, no serious acute or long-term effects occurred or are likely, based on current knowledge of the toxicology of endosulfan preparations. However, the pesticide being sprayed might just as easily have been an organophosphate or a carbamate with a much higher potential for acute health effects at low and medium doses.

The cotton industry must accept that incidents such as this, although they may happen rarely, may have serious short- and long-term effects on the health of those involved and that efforts must be made to both prevent and plan for such events.
6 Discussion and Conclusions

There are limitations to the design of Part 1 of the study due to the lack of true baseline pre-exposure cholinesterase values, a situation imposed by the seasonal, casually-employed and largely itinerant nature of the cotton chipper workforce.

Nevertheless, we consider that there is enough evidence to conclude that a proportion of cotton chippers are exposed to sufficient pesticide residues in the course of their work to cause detectable cholinesterase inhibition.

Cholinesterase depression was used in this study as a biological marker of exposure because it is a well established and relatively inexpensive technique. However, there is no reason to believe that exposure to pesticide residues amongst cotton chippers is limited only to the cholinesterase-inhibiting pesticides. Other pesticides, particularly endosulfan and the semi-synthetic pyrethroids, are also extensively used in the cotton industry. These pesticides are applied in an identical fashion and in similar concentrations to the organophosphates and carbamates. There is no reason to believe that their physical characteristics prevent dermal deposition and absorption.

The results of Part 2 of the study lend further weight to this proposition. The deposition rates per square centimetre of endosulfan are comparable to those for profenofos. The pattern of deposition appears to be consistent with direct contact with the cotton plants, and the differences in the pattern of deposition reflect the different crop heights and working practices (e.g. amount of "hand-pulling" of weeds) at the time of the three field trials.

Given this evidence for measurable exposure, what can be said about the health implications?

There was no evidence of acute toxicity amongst the cotton chippers who participated in this study (apart from the symptoms reported by the group of chippers who were accidentally oversprayed with endosulfan, as described in Section 5). This is consistent with the observed absolute levels of cholinesterase inhibition and the probable rate of change of the cholinesterase activity. Ordinarily clinical symptoms do not occur
until erythrocyte cholinesterase activity is inhibited by at least 50% of baseline. The development of signs and symptoms, however, is related more to the rate of decline in enzyme activity than to the absolute level of inhibition. Symptoms are rarely observed when the decline in activity is less than about 4% of baseline per day. If we assume that the largest shifts in an individual's cholinesterase activity observed in this study occurred gradually, then the maximum rate of change was of the order of 2% per day. We cannot rule out the possibility that the observed changes in cholinesterase occurred in a stepwise fashion over a much shorter periods of time, perhaps in response to working in fields with particularly high levels of pesticide residue.

In such a circumstance, the possibility of cotton chippers experiencing a sufficiently sudden decline in cholinesterase activity to cause symptoms cannot be ruled out. For this reason, and because accidental exposure may occur, it may be advisable for the cotton industry to sponsor the development of reporting mechanisms for suspected "pesticide reactions" amongst cotton chippers and other potentially exposed workers. The reporting mechanism might include rapid access to cholinesterase testing facilities in order to obtain serial cholinesterase activity measures from anyone suspected of suffering from acute organophosphate toxicity. Such post hoc testing has been shown to be valuable even in the absence of pre-exposure baseline data. Suitable training in the recognition of pesticide toxicity for cotton chippers and their supervisors ("gangers") would also be desirable.

There is increasing evidence that episodes of acute toxicity from organophosphate and carbamate pesticides, aside from causing immediate distress and disruption, have subtle but measurable long-term neuropsychological sequelae.

The neuropsychological effects of chronic, sub-clinical exposure to cholinesterase are more controversial. Although epidemiological data are limited, a number of investigators have hinted that persistent neurological effects can follow both acute and chronic organophosphate exposure. A report in 1961 of persistent central nervous system effects in 16 workers exposed for up to 10 years to organophosphate pesticides was greeted with some scepticism at the time, despite the evidence for long-term memory and concentration difficulties and increased rates of psychiatric illness in the workers. Persistent electroencephalographic disturbances following sub-acute exposure to
organophosphate pesticides have also been noted in human and animal studies\textsuperscript{30,31,32}. Karczmar, in a review of the subject area, concluded that the evidence for chronic effects from chronic exposure was inconclusive\textsuperscript{33}.

Other potential effects of the pesticides commonly used on cotton also need to be considered.

Rupa et al.\textsuperscript{34} examined reproductive outcomes including fertility, and the rates of spontaneous abortions and congenital defects in a group of 1016 males exposed to pesticides in cotton fields and their spouses, comparing them to a similar number of unexposed control couples. They found significant decreases in fertility and increases in the rate of spontaneous abortions amongst exposed couples. The workers were exposed to unquantified amounts of DDT, BHC, endosulfan and a range of organophosphates and synthetic pyrethroids. Despite severe methodological problems with the study, such as indeterminant nature of the pesticide exposures and the failure to account for important confounders such as age and socio-economic status, the results are disturbing and warrant further investigation.

A considerable number of the cotton chippers enrolled in our study were women of reproductive age. Three of the enrolled subjects contacted the investigators after the conclusion of the study in order to inquire about possible teratogenic effects of pesticide exposure. Each revealed that they had been pregnant in their first trimester while working as a cotton chipper. Follow-up cholinesterase levels were obtained from two of these subjects during the study (prior to them contacting us). One of these workers showed a 32\% decline in cholinesterase over a 4 week period, the other a 9\% decline over 6 weeks, indicating substantial organophosphate exposure.

The cotton industry needs to carefully consider what level of potential occupational exposure to pesticides is acceptable for such workers. Restriction of entry of women of reproductive age into the cotton chipping workforce is not available as a solution, both on moral and social grounds and because of legal impediments to such restriction embodied in various State and Federal anti-discrimination laws.
Possible carcinogenic effects also need to be considered, although there is only weak evidence from animal bio-assays that any of the pesticides currently used on cotton have carcinogenic potential.

**Intervention Strategies**

The trend in community and workplace expectations is that exposure to hazardous substances be reduced at least to the point where any biochemical changes caused by the substances are known not to pose long-term risks to health. Based on the findings of this study, such an assurance could not currently be given to cotton chippers, despite the absence of evidence of observable acute health effects due to pesticide exposure amongst cotton chippers.

For this reason, a range of strategies to reduce the risk of exposure to cotton field pesticides should be considered.

These might include:

a) re-evaluation of recommended or mandatory re-entry periods to cotton fields after pesticide application, and consideration of restricting entry to any previously sprayed field which is wet for any reason, including dew.

b) reduction of dermal exposure through improved clothing requirements for cotton chipping.

The results of Part 2 of this study provides some guidance in the development of clothing recommendations. For example, if the feet, legs and hands of cotton chippers were to be protected through shoes, trousers and gloves, a reduction of pesticide exposure of between 70 and 85% could be expected, depending on the crop height.

c) Provision of hand-washing and emergency wash-down facilities at the work site.
7 Recommendations

i) That the cotton industry be made aware that:

a) cotton chippers may be exposed to pesticides in the course of their work, either from dermal contact with pesticide residues on or contained in the cotton plants and weeds, or from accidental exposure to spray drift from aerial spraying operations.

b) although there is no evidence to suggest that cotton chippers are at particular risk of acute intoxication by pesticides, doubt exists about the biological significance and possible long term effects of the levels of pesticide exposure which cotton chippers may experience.

ii) That further research be undertaken by appropriate bodies to:

a) re-evaluate the appropriateness of currently recommended minimum re-entry periods for pesticides used on cotton crops, in the light of current and future cotton chipping work practices.

b) establish the most efficient, cost-effective and practical form of protective clothing for cotton chipping, and the minimum laundering requirements for such clothing.

iii) That the cotton industry be encouraged to institute the following measures as soon as possible:

a) A minimum clothing requirement for cotton chippers consisting of shoes or boots, socks, long trousers, long sleeved shirt (sleeves to be worn down), gloves and hat.

b) Mechanisms to prevent chippers being sent to work in fields which are wet with dew.

c) Provision of adequate hand washing facilities at chipping workplaces (ie in cotton fields) and encouragement of chippers to wash their hands prior to eating or smoking and after finishing work.
d) Improvements in communication procedures between aerial spray operators, property owners and chipping contractors and overseers in order to reduce the possibility of accidental exposure of chippers to aerial spray drift.

e) Provision of emergency shower or other wash-down and clothing change facilities at the work site in the event of accidental exposure of chippers to aerial spray-drift.

f) Development and promulgation of preventative measure such as those outlined above as an alternative to an extensive cholinesterase testing programme for all cotton chippers. The technical infrastructure is not currently available to support widespread cholinesterase testing for cotton chippers, and the casual and itinerant nature of their employment means that adequate operation of a mass testing programme may be impossible or unduly expensive.

However, it is desirable that some form of monitoring programme be set up to check the adequacy of the changes to dress code and work practices recommended above. Such a programme might involve recruitment of at least 50 sentinel cotton chippers who would undergo monthly cholinesterase blood and who would be required to adhere to the industry’s revised dress code and work practices. In order to increase the cost-effectiveness of such a monitoring programme and to make it logistically possible given existing resources, the sentinel chippers could be drawn from local residents who indicate that they will be available for follow-up throughout the season.
Appendix 1

Agricultural Health Unit
Moree District Hospital

Cotton Chipper Survey
Initial Questionnaire

These questions are part of a survey to measure pesticide exposure in cotton chippers. None of the personal information which you give will be given to anyone else without your permission.

Surname: ___________________________ First Name: ___________________________

Date of Birth: _____/_____/_______ Sex: Male ☐ Female ☐

Postal Address: ___________________________________________________________

Do you usually live in the Moree area? Yes ☐ No ☐

Employer: ________________________________________________________________

Ganger: __________________________________________________________________

How many weeks ago did you start cotton chipping THIS SEASON? _____________

How many days per week do you plan to work as a cotton chipper? _____________

How long do you plan to work as a cotton chipper this season?
   Less than 1 month ☐ 3 months ☐
   1 month ☐ 4 months ☐
   2 months ☐ 5 months ☐

Will this be in the Moree area only? Yes ☐

No ☐

In the last 3 months, what other types of jobs have you had? Please list them:
________________________________________________________________________
________________________________________________________________________
________________________________________________________________________

Have you worked with chemicals in any of these jobs? Yes ☐ No ☐

If Yes, which chemicals and when? __________________________________________

-42-
Have you used insect sprays or powders at home or anywhere else in the last 3 months? Yes ☐ No ☐
If Yes, please provide details: ____________________________________________________________

Have you had any blood tests for pesticides? Yes ☐ No ☐
If Yes, where and when? ____________________________________________________________

Do you smoke? Yes ☐ No ☐
If Yes, do you smoke: while chipping ☐ during breaks ☐ only after work ☐

When you are chipping, do you usually wash your hands before eating at lunch time or after work? Yes ☐ No ☐
If Yes, where? __________________________________________________________

Have you experienced any of the following health problems during chipping:
Tick the problems which you have had

Skin rashes ☐ Which parts of the body? ____________________________________________
Cuts and scratches ☐ Which parts of the body? ____________________________________________
Sunburn ☐ Which parts of the body? ____________________________________________
Other problems __________________________________________________________

To be completed with Agricultural Health Unit staff

I, ___________________________, understand the purpose of this research programme and the arrangements for blood testing for pesticide exposure. I understand that the results of any significantly abnormal blood test will be given to me. I agree to take part in this survey.

Signature: ___________________________ Date: / /199
Witness: ___________________________ Date: / /199

I also consent/do not consent for significantly abnormal blood test results to be given to my employer.

Signature: ___________________________ Date: / /199
Witness: ___________________________ Date: / /199
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1) No Shirt  
2) Singlet  
3) Short Sleeved Shirt  
4) Long Sleeved Shirt  
5) Shorts  
6) Long Pants  
7) Gloves  
8) Hat  
9) Bare Feet  
10) Thongs  
11) Shoes or Boots  
12) Sun Screen  
13) Sun Glasses  
14) Other

Estimated percentage of time spent hand-pulling weeds: ________
Appendix 2

Agricultural Health Unit
Moree District Hospital

Cotton Chipper Survey

These questions are part of a survey to measure pesticide exposure in cotton chippers. None of the personal information which you give will be given to anyone else without your permission.

Surname: ___________________________________ First Name: ________________________________

Date of Birth: ______/_____/______

Employer: ______________________________________________________
Ganger: ______________________________________________________

Have you changed gangs in the last 2 weeks? Yes [ ] No [ ]

If Yes, please write the name of the new ganger and the date that you changed gangs: ______________________________________________________

How many days per week did you work as a cotton chipper in the last 2 weeks? _____

Have you worked with chemicals in the last 2 weeks? Yes [ ] No [ ]

If Yes, which chemicals and when? ______________________________________________________

Have you used insect sprays or powders at home or anywhere else in the last 2 weeks? Yes [ ] No [ ]

If Yes, please provide details: ______________________________________________________

Have you experienced any of the following health problems while chipping in the last 2 weeks:

Tick the problems which you have had in the last 2 weeks

Skin rashes [ ] Which parts of the body? ________________________________________________

Cuts and scratches [ ] Which parts of the body? __________________________________________

Sunburn [ ] Which parts of the body? __________________________________________________

Other problems ____________________________________________________________
<table>
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<tr>
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<td>Facial Sunburn:</td>
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<td>2</td>
</tr>
<tr>
<td>Forearm Dermatitis:</td>
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<td>Forearm Sunburn:</td>
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</table>

1) No Shirt
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4) Long Sleeved Shirt
5) Shorts
6) Long Pants
7) Gloves
8) Hat
9) Bare Feet
10) Thongs
11) Shoes or Boots
12) Sun Screen
13) Sun Glasses
14) Other

Estimated percentage of time spent hand-pulling weeds: _____
Appendix 3

Reported potential prior exposure to cholinesterase inhibiting compounds which resulted in exclusion from the analysis stage of the study.

1. Repeated use of Nuclolol 200 (diazinon) dog wash (1 person)
2. Recent handling of Lorsban (chlorpyrifos) (3 persons)
3. Recent use of diazinon (2 persons)
4. Recent handling of cotton chemicals (unspecified) (8 persons)
5. Recent handling of organophosphate pesticides (type unspecified) (3 persons)
<table>
<thead>
<tr>
<th>OBS</th>
<th>Pesticide</th>
<th>Trial No.</th>
<th>Volunteer no.</th>
<th>Sample no.</th>
<th>Description</th>
<th>ug of endosulfan</th>
<th>Sample area (cm^2)</th>
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</tbody>
</table>

**Crop height approx. 30 cm, 7 hours after spraying with endosulfan 240g/L ULV @ 2.0L/Ha**
References


