EXHIBIT 54
Pesticide exposure as risk factor for non-Hodgkin lymphoma including histopathological subgroup analysis

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We report a population based case–control study of exposure to pesticides as risk factor for non-Hodgkin lymphoma (NHL). Male and female subjects aged 18–74 years living in Sweden were included during December 1, 1999, to April 30, 2002. Controls were selected from the national population registry. Exposure to different agents was assessed by questionnaire. In total 910 (91%) cases and 1016 (92%) controls participated. Exposure to herbicides gave odds ratio (OR) 1.72, 95% confidence interval (CI) 1.18–2.51. Regarding phenoxyacetic acids highest risk was calculated for MCPA; OR 2.81, 95% CI 1.27–6.22, all these cases had a latency period >10 years. Exposure to glyphosate gave OR 2.02, 95% CI 1.10–3.71 and with >10 years latency period OR 2.26, 95% CI 1.16–4.40. Insecticides overall gave OR 1.28, 95% CI 0.96–1.72 and impregnating agents OR 1.57, 95% CI 1.07–2.30. Results are also presented for different entities of NHL. In conclusion our study confirmed an association between exposure to phenoxyacetic acids and NHL and the association with glyphosate was considerably strengthened.

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Key words: phenoxyacetic acids; MCPA; glyphosate; insecticides; impregnating agents; non-Hodgkin lymphoma

Non-Hodgkin lymphoma (NHL) is a heterogeneous group of lymphoid malignancies, where new classification systems based on immunohistochemistry, cytogentics and evolving knowledge in clinical presentation and course has lead to modern classification systems.1 Today, it is therefore more adequate to discuss NHL as many different diseases, which share some features but also differ in several aspects.

Interest in the etiology of NHL has been strengthened by an observed substantial increase in the incidence of the disease from the 1960’s to the 1980’s as reported from most countries with reliable cancer registries. However, this increase has clearly leveled off in many countries since the early 1990’s, i.e., in Sweden, Denmark and the USA.2 The established risk factors for development of NHL include different immunosuppressive states, e.g., human immunodeficiency virus (HIV), autoimmune diseases as Sjögren’s syndrome and systemic lupus erythematosus (SLE), immunodepressants used after organ transplantation and some inherited conditions, for review see e.g., Ref. 3. However, these causes may only explain a minority of cases, with a possible exception for HIV-related increases among younger persons in certain areas.4

It has been shown that Epstein-Barr virus (EBV) plays an essential role in the pathogenesis of lymphomas after organ transplantation.5 A relation between lymphoma and elevated EBV-titters has been reported in a cohort.6 Normally, EBV-production is held back by active cellular and humoral immune mechanisms. In immunodeficiency states this balance is disrupted and EBV-infected B-cells begin to proliferate.7

During the last decades, research on the etiology of NHL has been directed towards other potential causes such as pesticides, which may explain the impressive increase in the incidence. Today, it is also reasonable to consider the leveling off in incidence as a probable consequence of a reduced carcinogenic influence related to NHL. Furthermore, our emerging knowledge concerning the spectrum of NHL subgroups makes it reasonable to investigate causative agents for these different types of disease.

In 1981, we published results from a case–control study from Sweden, indicating statistically significant increased odds ratios for NHL and Hodgkin lymphoma (HL) in persons who had been exposed to phenoxyacetic herbicides or impregnating chlorophenols.8 Our study was initiated by a case report.9 Some of these chemicals were contaminated by dioxins, of which 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) has been recognised as a complete carcinogen by IARC.10 Furthermore, these and several other related chemicals are immunotoxic.11–13 Our results have been confirmed in some other studies, regarding phenoxyacetic herbicides from e.g., Kansas14 and Nebraska.15

Furthermore, in 1999 we reported a new case–control study performed to evaluate more recent disease to pesticides and other chemicals, and we could thereby confirm our earlier findings regarding a relation with phenoxyacetic herbicides that was related to latency period.16

In that study, however, some newer compounds that are widely used today, such as the herbicide glyphosate, were still not very common. During the 1970’s certain chemicals, e.g., the phenoxy herbicide 2,4,5-trichlorophenoxyacetic acid (2,4,5-T), chlorophenols, and the insecticide dichlorodiphenyltrichloroethane (DDT), were prohibited due to health concerns. Later also the phenoxy herbicide 2,4-dichlorophenoxyacetic acid (2,4-D) was banned in Sweden. Reporting of these agents is therefore nowadays much less likely. It is also probable that the risk pattern has been influenced by protective measures during the last decades.

To further evaluate the relation between exposure to pesticides and other chemicals, focusing also on newer types of compounds, we have performed a new case–control study in Sweden. In our study we have also evaluated exposures in relation to different histopathological subtypes according to the most recent classification.7

Material and methods

The study covered 4 out of 7 health service regions in Sweden, associated with the University Hospitals in Lund, Linköping, Örebro and Umeå, and was approved by the ethics committees. Data were collected during December 1, 1999, to April 30, 2002, which was the time period for diagnosis of the cases. Regarding recruitment of cases and controls collaboration was established with another research group, which at the same time performed a parallel study on NHL in Sweden and Denmark.

Cases

All consecutive patients aged 18–74 years with newly diagnosed NHL, identified through physicians treating lymphoma and through pathologists diagnosing the disease, were approached if their physician did not judge this as less appropriate by ethical rea-
sons. This was done regardless of whether the person had accepted to participate in the parallel study with which we collaborated in the recruitment procedure. If they accepted to participate they were included as potential cases, and went through the data assessment procedure described below. No cases were excluded because of specific conditions potentially associated with NHL, but no case with e.g. HIV or posttransplantation NHL occurred. All the diagnostic pathological specimens were scrutinised by 1 out of 5 Swedish expert lymphoma reference pathologists, if they had not been initially judged by one of these 5. About 70% of all included cases were reviewed, whereas the remaining had been previously classified by one of the reference pathologists. If there was a disagreement from the original report the sample was reviewed by a panel of these pathologists. Therefore, some potential cases could later be excluded if a NHL diagnosis was not verified, and in those occasions all collected exposure information was disregarded. The pathologists also subdivided all NHL cases according to the WHO classification, to enable etiological analyses also for the different diagnostic NHL entities. Since all lymphoma treating clinics and all lymphoma pathologists in the involved regions were covered by the study, it may well be regarded as population based, although the possibility of some individuals not reported through the case ascertainment system used.

Controls
From the population registry covering whole Sweden, randomly chosen controls living in the same health service regions as the cases were recruited during several occasions within the study period. The controls were frequency-matched in 10 years age and sex groups to mirror the age and sex distribution of the included cases, and to increase efficacy in the adjusted analyses. If they accepted to participate, they were included as controls.

Assessment of exposure
All subjects who accepted to participate received a comprehensive questionnaire, which was sent out shortly after the subjects had been telephone interviewed by the other research group we had collaboration with as stated earlier. Their interview, however, did not focus on work environment or chemical exposure, but rather dealt with other life style factors and diseases. Our questionnaire included a total work history with in depth questions regarding exposure to pesticides, organic solvents and several other chemicals. For all pesticides not only numbers of years and numbers of days per year, but also approximate length of exposure per day were questioned. Since most work with pesticides was performed in an individualized manner, no job-exposure matrix was judged to be applicable. Furthermore, the questionnaire also included questions on e.g. smoking habits, medications, leisure time activities and proximity from home to certain industrial installations, but data on these factors are not included in this article.

Specially trained interviewers scrutinized the answers and collected additional exposure information by phone if important data were lacking, incomplete or unclear. These interviewers were blinded with regard to case/control status. All exposures during the same calendar year as the diagnosis and the year before were disregarded in the cases. Correspondingly, the year of enrolment and the year before were disregarded for the controls. As in our previous lymphoma studies we used a minimum criterion of one full day exposure to be categorized as exposed.8,18

Statistical methods
Unconditional logistic regression analysis (Stata/SE 8.2 for Windows; StatuCorp, College Station, TX) was used to calculate odds ratios (OR) and 95% confidence intervals (CI). Adjustment was made for age, sex and year of diagnosis (cases) or enrolment (controls). In the univariate analysis, different pesticides were analyzed separately and the unexposed category consisted of subjects that were unexposed to all included pesticides. When analyzing subgroups of NHL all controls were used in the separate analyses. In the dose-response calculations made for agents with at least 20 exposed subjects, median number of days of exposure among controls was used as cut-off. Latency period calculations and multivariate analyses included agents with statistically significant increased OR, or with an OR > 1.50 and at least 10 exposed subjects.

Results
In total, 1,163 cases were reported from the participating clinics. Of these, 46 could not participate because of medical conditions, 88 died before they could be interviewed. Since these were primarily excluded by the reporting physicians we had no information on e.g. final WHO categories on these cases. Three NHL cases were not diagnosed during the study period, 1 lived outside the study area and 30 were excluded not being NHL (HL 20, acute lymphoblastic leukaemia 1, other malignancy 7 and unclear diagnosis 2). Of the finally included 995 cases with NHL, 910 (91%) accepted to participate and answered the questionnaire. Of these, 819 were B-cell, 53 T-cell and 38 unspecified lymphomas, Table I.

Among the 1,108 initially enrolled controls 92 did not respond to the mail questionnaire, resulting in 1,016 (92%) controls to be included in the analyses.

The medium and median age in cases was 60 and 62 years, and in controls it was 58 and 60 years, respectively. Of the cases, 534 were males and 376 females, and of the controls the corresponding numbers were 592 and 424.

This report presents exposure data regarding different types of pesticides.

Herbicides
Exposure to herbicides gave for all NHL OR 1.72 (95% CI 1.18–2.51), Table II. Exposure to phenoxyacetic acids yielded OR 2.04 (95% CI 1.24–3.36). This group was further subdivided in 3 categories; (i) 4-chloro-2-methyl phenoxyacetic acid (MCPA), which is still on the market and not known to be contaminated by dioxins; (ii) 2,4,5-T and/or 2,4-D which often were used together and were potentially contaminated with different dioxin isomers; (iii) other types. MCPA seemed to give the most pronounced increase in OR. Exposure to other herbicides, regardless if they also had been exposed to phenoxyacetic acids or not, also gave a statistically significant OR 1.82 (95% CI 1.08–3.06). In this category the dominating agent was glyphosate, which was reported by 29 cases and 18 controls, which produced OR 2.02 (95% CI 1.10–3.71). If both phenoxyacetic acids and glyphosate were excluded, exposure to other herbicides (37 different agents reported, but no one by more than 6 subjects at most) gave a nonsignificant OR of 1.22 (95% CI 0.63–2.39).

Dose-response analyses regarding herbicides in total and glyphosate yielded an increased OR in the higher exposed group, Table II. For phenoxyacetic acids, however, no such association was demonstrated.

Regarding phenoxy herbicides and glyphosate an analysis was made taken the latency period for exposure into account. For the
When different NHL entities were analysed separately, the OR for the subtype small lymphocytic lymphoma/chronic lymphocytic leukaemia (SLL/CLL) was increased for both phenoxy herbicides and, especially, glyphosate, Table III. The entity diffuse large B-cell lymphoma (DLBCL) was significantly associated with exposure to phenoxyacetic acids, and an increased risk was also indicated for glyphosate. T-cell lymphomas seemed to be associated with all types of herbicides, but no statistically significant ORs were found due to relatively few exposed subjects. The least numerous categories (“unspecified NHL”) yielded high and statistically significant ORs for phenoxy herbicides and glyphosate.

**Insecticides**

In our study no overall increased OR was demonstrated for exposure to insecticides, OR 1.28 (95% CI 0.96–1.72), Table IV. The most reported insecticide DDT yielded OR 1.46 (95% CI 0.94–2.28). Increased risk was shown for mercurial seed dressing, OR 2.03 (95% CI 0.97–4.28).

In the dose-response analysis, OR 1.47 (95% CI 0.99–2.16) was found for the high category of insecticide exposure, Table IV. Similar trends were found for DDT and mercurial seed dressing. Different NHL entities were analysed separately, Table V. Hereby, certain exposures seemed to be associated with subtypes of NHL. Thus, the group follicular lymphoma was associated with DDT, OR 2.14 (95% CI 1.05–4.40) and mercurial seed dressing, OR 3.61 (95% CI 1.20–10.9) Furthermore, exposure to DDT increased the risk also for T-cell lymphoma, OR 2.88 (95% CI 1.05–7.95).

**Fungicides and rodenticides**

Exposure to fungicides was not a risk factor in our study, neither in total, OR 1.11 (95% CI 0.56–2.23), Table IV, nor for different subtypes of NHL, Table VI. Furthermore, there were no single substances among 24 reported that significantly differed between cases and controls. Also for rodenticides no increased risk was found, Table IV.

**Impregnating agents**

Exposure to impregnating agents yielded a statistically significant OR 1.57 (95% CI 1.07–2.30), Table IV. In a dose-response calculation OR increased further in the high exposure group. Creosote showed a statistically significant OR for high exposure, OR 3.35 (95% CI 1.20–9.27).

Table VI presents results for different NHL entities. An increased risk for SLL/CLL was associated with exposure to impregnating agents in total, and most pronounced for creosote,

**TABLE III – EXPOSURE TO VARIOUS HERBICIDES DIVIDED ACCORDING TO DIFFERENT LYMPHOMA ENTITIES**

<table>
<thead>
<tr>
<th>Lymphoma entities</th>
<th>Herbicides, total</th>
<th>Phenoxyacetic acids</th>
<th>MCPA</th>
<th>2,4,5-T and/or 2,4-D</th>
<th>Herbicides except ph</th>
<th>Glyphosate</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>B-cell lymphomas, total (n = 819)</td>
<td>1.68</td>
<td>1.99</td>
<td>2.59</td>
<td>1.69</td>
<td>1.72</td>
<td>1.87</td>
<td>1.14</td>
</tr>
<tr>
<td>B-cell lymphomas, (n = 195)</td>
<td>1.14–2.48</td>
<td>1.20–3.32</td>
<td>1.14–5.91</td>
<td>0.94–3.01</td>
<td>1.00–2.94</td>
<td>0.99–3.51</td>
<td>0.57–2.31</td>
</tr>
<tr>
<td>Follicular lymphoma/B-CLL (n = 195)</td>
<td>1.28–4.01</td>
<td>0.995–4.47</td>
<td>0.74–8.97</td>
<td>0.85–4.41</td>
<td>1.17–5.60</td>
<td>1.42–7.89</td>
<td>0.45–4.31</td>
</tr>
<tr>
<td>Diffuse large B-cell lymphoma (n = 239)</td>
<td>0.91–2.59</td>
<td>1.08–4.33</td>
<td>1.48–10.5</td>
<td>0.71–3.82</td>
<td>0.71–2.83</td>
<td>0.44–3.35</td>
<td>0.33–3.03</td>
</tr>
<tr>
<td>Other specified B-cell lymphoma (n = 131)</td>
<td>0.82–3.19</td>
<td>1.14–5.64</td>
<td>0.95–10.7</td>
<td>0.90–5.44</td>
<td>0.51–3.73</td>
<td>0.53–4.96</td>
<td>0.33–4.03</td>
</tr>
<tr>
<td>Unspecified B-cell lymphoma (n = 89)</td>
<td>1.09</td>
<td>1.14</td>
<td>1.35</td>
<td>0.88</td>
<td>1.52</td>
<td>1.47</td>
<td>0.71</td>
</tr>
<tr>
<td>T-cell lymphomas (n = 53)</td>
<td>1.64</td>
<td>1.62</td>
<td>2.40</td>
<td>1.02</td>
<td>1.57</td>
<td>2.29</td>
<td>2.24</td>
</tr>
<tr>
<td>Non-Hodgkin lymphoma (n = 38)</td>
<td>0.55–4.90</td>
<td>0.36–7.25</td>
<td>0.29–20.0</td>
<td>0.13–7.95</td>
<td>0.35–6.99</td>
<td>0.51–10.4</td>
<td>0.49–10.3</td>
</tr>
</tbody>
</table>

**Odds ratios (OR) and 95% confidence intervals (CI). Adjustment was made for age, sex and year of diagnosis or enrolment.**

1No exposed cases
Multivariate analysis

Since mixed exposure to several pesticides was more a rule than an exception, and all single agents were analyzed without adjusting for other exposure, a multivariate analysis was made to elucidate the relative importance of different pesticides. Criteria for agents to be included in this analysis are defined in Statistical Methods above. As seen in Table VII increased ORs were found but in general lower than in the univariate analysis.

Discussion

This was a population based case–control study on NHL, which is a strength of the investigation. Only living cases and controls were included, which was of advantage in comparison with interviewing next-of-kins. The study covered all new cases of NHL during a specified time. Pathologists in Sweden that were experts in lymphoma diagnosis confirmed all diagnoses. Thus, a main advantage compared with earlier studies was the possibility to study the different NHL entities, classified according to the recently developed WHO classification system. The histopathological subgroups may well be regarded as separate in etiology and pathogenesis, as well as they are known to be different regarding course, prognosis and best treatment.

The frequency matching on age groups, gender and health service regions increased the efficacy of the study and ensured exposure conditions for the controls representative for the population in the included geographical areas. We achieved a high response rate among cases and controls, which is another advantage. A motivating introduction letter that was sent out with the questionnaire and with reminders if needed may explain this.

Exposures were assessed by questionnaires with information supplemented over the phone. Thereby use of different pesticides could be checked by information in e.g., receipts and bookkeeping. However, no registries exist in Sweden on such individual use, which is a weakness in the assessment of exposure. Exposure to pesticides may be difficult to assess, and some misclassification regarding quantity of exposure has probably occurred, but such misclassification would most probably be nondependent of case/control status, and therefore only weaken any true risks. Use of protective equipment was not asked for which might have been a disadvantage of the study. However, such use would dilute the exposure and thus bias the result towards unity.

We have earlier published the results from 2 Swedish case–control studies on lymphomas, the first one on NHL and HL, and later on NHL. These studies showed an increased risk for lymphomas as a result of exposure to herbicides belonging to the class phenoxyacetic acids. In the first study we also found correlation with chlorophenols and organic solvents. Several other studies,
Unspecified B-cell lymphoma (types of insecticides, between NHL and other classes of pesticides, especially different
Other specified B-cell lymphoma (reflecting also later years of exposure.

strengthened protection instructions has prompted our new study, ing. This change of herbicide practice along with successively
other agents, among which glyphosate has been clearly dominat-
1990. MCPA, even if still used, has been largely substituted by
in Sweden 1977, and 2,4-D was withdrawn from the market in
ades. 2,4,5-T, which was contaminated by TCDD, was prohibited
wood in forestry, have substantially decreased during the last dec-
dominating both as weed killers in agriculture and against hard
may be of interest.

study also used a somewhat changed methodology, which also
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pesticides that have been related to NHL over time in different coun-
similar mechanism of action, which may explain the wide range of
chemically related and may exert their effects on humans through a

macroscopic aberrations and oxidative stress.34,35

portant to find etiological factors contributing to this shift in trend. Chlorinated compounds in the environment, which have been
regulated during the 1970’s and 1980’s, may at least partly explain this trend, as discussed by us.2 Phenoxycetic herbicides with
potential contaminating dioxins are examples of such substances. However, the prohibition of common environmental pollutants as
polychlorinated biphenyls (PCB) and the following decline in the environment is probably more important to explain the leveling
off of the incidence.2

In contrast to our 2 former case–control studies on NHL, this study included both genders and only consecutive living cases and
living controls. In our earlier studies we have only studied male lymphoma cases, making the results of this study more representa-
tive for the whole population. To facilitate comparisons with our earlier results we also made additional analyses of herbicide expo-
sure by gender. Only few women were exposed and separate analyses for both sexes still yielded an increased risk for NHL. Thus,
in the total material herbicide exposure gave OR = 1.72, 95% CI
1.18–2.51 (n = 74 cases, 51 controls), whereas for men only OR
= 1.71, 95% CI = 1.15–2.55 (n = 68 cases, 47 controls) and for
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In our study lymphocytic lymphoma/B-CLL was significantly associated with herbicides with highest OR for glyphosate but also
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with MCPA, and T-cell lymphoma with DDT and impregnating
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MCPA, glyphosate and mercurial seed dressing. It should be noted
that several ORs were increased for herbicides; insecticides and
impregnating agents but the calculations were hampered by low
numbers of exposed cases and controls.

Our earlier results of exposure to phenoxycetic herbicides as a
risk factor for NHL were confirmed in our study. As in our previous
lymphoma studies exposure to MCPA seemed to yield the highest
OR among the different phenoxycetic acids. This is of interest
because MCPA is known not to be contaminated by dioxins, as 2,4-
D and 2,4,5-T. At the same time MCPA is the only phenoxycetic
acid still in wider use in Sweden and many other countries.

Glyphosate is a broad-spectrum herbicide, which inhibits the
formation of amino acids in plants.29 The US Environmental
Protection Agency30 and the World Health Organization31 have con-
cluded that glyphosate is not mutagenic or carcinogenic. Since
then, however, some experimental studies indicate genotoxic, hor-
monal and enzymatic effect in mammals, as reviewed.32 Of partic-
lar interest is that glyphosate treatment of human lymphocytes
in vitro resulted in increased sister chromatid exchanges,33 chro-
mosomal aberrations and oxidative stress.34,35

| TABLE VII – MULTIVARIATE ANALYSES INCLUDING AGENTS ACCORDING TO DIFFERENT LYMPHOMA ENTITIES |
|-------------------------------------------------|------------------|------------------|------------------|------------------|
| Agents                                          | Univariate       | Multivariate     |                  |
|                                                 | OR CI            | OR CI            |                  |
| MCPA                                            | 2.81 1.27–6.22   | 1.88 0.77–4.63   |                  |
| 2,4,5-T and/or 2,4-D                           | 2.02 1.10–3.71   | 1.51 0.77–2.94   |                  |
| Glyphosate                                      | 2.03 0.97–4.28   | 1.58 0.74–3.40   |                  |
| Mercurial seed dressing                        | 1.63 0.51–5.20   | 1.17 0.34–4.02   |                  |
| Arsenic                                         | 2.30 0.96–4.58   | 1.70 0.73–3.98   |                  |
| Croesote                                        | 1.59 0.59–5.69   | 1.39 0.43–4.83   |                  |

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1 No exposed cases.

but not all, from different research groups have supported our
results, as reviewed,20 and also confirmed later, e.g., Ref. 21.

Furthermore, other groups have demonstrated associations
between NHL and other classes of pesticides, especially different
types of insecticides, e.g., organophosphates,26 carbamate,25 ind-
ane24 and chlorodane,22 but also other groups of herbicides as atra-
zine.26 Some case–control studies have found associations between
several classes of pesticides, e.g., Ref. 27 or merged groups of pesti-
cides as in one recent study,28 which demonstrate a significantly
increased risk for NHL associated with exposure to “nonarsenic pes-
ticides.” These authors discuss the fact that several pesticides are
chemically related and may exert their effects on humans through
a similar mechanism of action, which may explain the wide range
of pesticides that have been related to NHL over time in different
countries and with different exposure conditions.

Several factors urged for a third Swedish study on the relation
between pesticides, other chemicals and NHL, and the present
study also used a somewhat changed methodology, which also
may be of interest.

Thus, the use of phenoxycetic herbicides, which earlier were
dominating both as weed killers in agriculture and against hard
wood in forestry, have substantially decreased during the last dec-
ades. 2,4,5-T, which was contaminated by TCDD, was prohibited
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reflecting also later years of exposure.

Furthermore, the changing trend of the incidence of NHL in
many countries with reliable cancer registries, e.g., Sweden, with
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but a leveling off or even slight decrease after that, makes it im-
portant to find etiological factors contributing to this shift in trend. Chlorinated compounds in the environment, which have been
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in vitro resulted in increased sister chromatid exchanges,33 chro-
mosomal aberrations and oxidative stress.34,35
Glyphosate was associated with a statistically significant increased OR for lymphoma in our study, and the result was strengthened by a tendency to dose-response effect as shown in Table II. In our former study, very few subjects were exposed to glyphosate, but a nonsignificant OR of 2.3 was found. Furthermore, a meta-analysis combining that study with an investigation on hairy-cell leukemia, a rare NHL variant, showed an OR for glyphosate of 3.04 (95% CI 1.08–8.52). Recent findings from other groups also associate glyphosate with different B-cell malignancies such as lymphomas and myelomas.32,37,38

Glyphosate has succeeded MCPA as one of the most used herbicides in agriculture, and many individuals that used MCPA earlier are now also exposed to glyphosate. This probably explains why the multivariate analysis does not show any significant ORs for these compounds.

Exposure to insecticides was associated with a slightly increased OR, Table IV. In some other studies on the relation between pesticides and NHL, insecticides seem to be of some importance as causative agents.27,37,38 Especially, different organophosphates were indicated as risk factors in those studies, with a Canadian study showing statistical significant ORs for malathion and diazinon. In our study, only few subjects were exposed to different organophosphates, but we found a nonsignificant OR of 2.2 (95% CI 0.9–4.7) for malathion based on 5 exposed cases and 2 controls not shown in Table.

The organochlorine DDT has shown suggestive but rarely significant association with NHL in some studies.39–40 Our study showed a moderately but not significant increased OR for exposure to DDT.

**References**


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