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Predictors of Serum Chlorinated Pesticide Concentrations among Prepubertal Russian Boys

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BACKGROUND: Few studies have evaluated predictors of childhood exposure to organochlorine pesticides (OCPs), a class of lipophilic persistent chemicals.

OBJECTIVES: Our goal was to identify predictors of serum OCP concentrations—hexachlorobenzene (HCB), β-hexachlorocyclohexane (β-HCH), and p,p’-dichlorodiphenyldichloroethylene (p,p’-DDE)—among boys in Chapaevsk, Russia.

METHODS: Between 2003 and 2005, 499 boys 8–9 years of age were recruited in a prospective cohort. The initial study visit included a physical examination; blood collection; health, lifestyle, and food-frequency questionnaires; and determination of residential distance from a local factory complex that produced HCB and β-HCH. Fasting serum samples were analyzed for OCPs at the U.S. Centers for Disease Control and Prevention. General linear regression models were used to identify predictors of the boys’ serum HCB, β-HCH, and p,p’-DDE concentrations.

RESULTS: Among 355 boys with OCP measurements, median serum HCB, β-HCH, and p,p’-DDE concentrations were 158, 167, and 284 ng/g lipid, respectively. Lower body mass index, longer breastfeeding duration, and local dairy consumption were associated with higher concentrations of OCPs. Boys who lived < 2 km from the factory complex had 64% (95% CI: 37, 96) and 57% (95% CI: 32, 87) higher mean HCB and β-HCH concentrations, respectively, than boys who lived ≥ 5 km away. Living > 3 years in Chapaevsk predicted higher β-HCH concentrations, and having parents who lacked a high school education predicted higher p,p’-DDE concentrations.

CONCLUSIONS: Among this cohort of prepubertal Russian boys, predictors of serum OCPs included consumption of local dairy products, longer local residence, and residential proximity to the local factory complex.


Introduction

Persistent, lipid-soluble organochlorine pesticides (OCPs) such as hexachlorobenzene (HCB), β-hexachlorocyclohexane (β-HCH), dichlorodiphenyldichloroethylene (DDT), and its primary metabolite p,p’-dichlorodiphenyl-dichloroethylene (p,p’-DDE) are ubiquitous in the environment. Although these insecticides and fungicides were banned in the 1970s (United States) and in the 1980s (Russia) (Barber et al. 2005; Breivik et al. 1999; Jaga and Dharmani 2003), DDT is still used in some countries to control malaria and yellow fever (Jaga and Dharmani 2003), and HCB and β-HCH are generated as by-products during the manufacture of other chlorinated chemicals (Courtney 1979; Jung et al. 1997). In addition to biomagnifying up the food chain, these compounds have long half-lives in both the environment and the body, ranging from years to decades, and persist long after use has ceased (Barber et al. 2005; Courtney 1979; Jaga and Dharmani 2003; Jung et al. 1997).

Human exposure typically occurs through diet (e.g., fatty fish, dairy products, meats, poultry) and less commonly through inhalation or dermal absorption (Darnerud et al. 2006; Gasull et al. 2011). OCPs are stored in adipose tissue, concentrated in breast milk (Rogan et al. 1986), and can be passed from mother to child via transplacental transfer or breastfeeding, a primary route of exposure for children (Rogan et al. 1986; Sala et al. 2001). Compared with adults, young children have disproportionately elevated exposures to pesticides because they have higher ventilatory rates and food consumption relative to body weight (National Research Council 1993) and greater hand-to-mouth transfer of contaminated soil and dust. Moreover, the ability to metabolize, detoxify, and excrete pesticides may be reduced in young children (Landrigan et al. 2003).

HCB, β-HCH, and p,p’-DDE have been associated with adverse health effects in animal studies, including neurodevelopmental toxicity (Courtney 1979; Ecobichon et al. 1990), cancer (Courtney 1979; International Agency for Research on Cancer (IARC))

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The opinions expressed in this article are those of the authors and do not necessarily reflect the official opinion of the Centers for Disease Control and Prevention or of the National Institute for Environmental Health Sciences.

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(for continuous variables) or relative to the reference category (for categorical variables) (10β, where β is the regression coefficient for a given predictor), holding all other variables constant. We also estimated adjusted mean concentrations of each OCP according to residential distance from the factory using least-square means, adjusted for all other predictors in the final model for each OCP. Additionally, we performed sensitivity analyses using log10-transformed whole-weight serum OCP concentrations adjusted for total lipids instead of direct lipid-adjusted OCPs in the final model. A p-value of < 0.05 was considered statistically significant. Tests for trend were performed by modeling categorical variables as an ordinal variable using integer values (0, 1, 2). All data analyses were performed using SAS version 9.2 (SAS Institute Inc., Cary, NC, USA).

Results

Study population. Characteristics of the 355 boys are shown in Table 1. At study entry, 84% of boys were 8 years old, 17% were overweight/obese, and 25% were underweight (de Onis et al. 2007). Among boys with and without serum OCP measurements (n = 355 vs. 144), there were no significant differences in height, weight, BMI z-scores, birth, or family characteristics, but there was a significant difference in household income (44% vs. 26% of families in the highest income category, respectively). Also, more boys with measured serum OCP concentrations had fathers who were employed at the factory complex compared with those without OCP measurements (14% vs. 9%).

Distribution of serum HCB, β-HCH, p,p′-DDE. The medians (25th, 75th percentiles) for serum HCB, β-HCH, and p,p′-DDE concentrations were 158 (107, 246), 167 (112, 270), and 284 (187, 492) ng/g lipid, respectively (Table 2). Median p,p′-DDE concentrations were about three times higher than concentrations previously reported for adolescents in the United States and Belgium, whereas HCB concentrations were about 7–12 times higher (Table 3) (Den Hond et al. 2011; Peterson et al. 2009). The median β-HCH concentration previously reported for 12- to 19-year-old U.S. adolescents (below the LOD of 7.8 ng/g lipid) (Peterson et al. 2009) was at least 20 times lower than the median concentration in our study population of Russian boys. Spearman correlations between the OCPs were r = 0.61 for β-HCH and p,p′-DDE, r = 0.54 for β-HCH and HCB, and r = 0.34 for HCB and p,p′-DDE.

Predictors of serum HCB, β-HCH, p,p′-DDE. BMI and residential distance from the factory complex were the strongest predictors, explaining 18% and 23% of the variability in the serum concentrations of HCB and β-HCH, respectively. All other covariates combined explained an additional 3% (HCB) and 13% (β-HCH) of the variability. For p,p′-DDE, BMI and breastfeeding duration combined explained 23% of the variability, all other model covariates explained an additional 7%.

Boys breastfed > 13 weeks had 16% (95% CI: −5, 41%), 63% (95% CI: 35, 96%), and 81% (95% CI: 43, 128%) higher predicted mean serum HCB, β-HCH, and p,p′-DDE concentrations, respectively, than non-breastfed boys, with a significant linear trend over increasing categories of breastfeeding for all three OCPs (Table 4). BMI was also a significant predictor of all three OCPs, with the highest mean concentrations among boys who were classified as underweight, and the lowest among boys who were overweight or obese. Living near the factory complex predicted increased serum concentrations of HCB (9.3%; 95% CI: −4.9, 25.6%) and HCB, β-HCH, and p,p′-DDE concentrations, respectively, than non-breastfed boys, with a significant linear trend over increasing categories of breastfeeding for all three OCPs (Table 4). BMI was also a significant predictor of all three OCPs, with the highest mean concentrations among boys who were classified as underweight, and the lowest among boys who were overweight or obese. Living near the factory complex predicted increased serum concentrations of HCB (9.3%; 95% CI: −4.9, 25.6%) and HCB, β-HCH, and p,p′-DDE concentrations, respectively, than non-breastfed boys, with a significant linear trend over increasing categories of breastfeeding for all three OCPs (Table 4).

Table 1. Characteristics of 8- to 9-year-old participants in the Russian Children’s Study at study entry (n = 355).

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Total boys</th>
</tr>
</thead>
<tbody>
<tr>
<td>Growth measurements</td>
<td></td>
</tr>
<tr>
<td>Height (cm)</td>
<td>129 ± 6.04</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>26.7 ± 5.53</td>
</tr>
<tr>
<td>BMI</td>
<td>15.9 ± 2.23</td>
</tr>
<tr>
<td>WHO height z-score</td>
<td>0.13 ± 1.01</td>
</tr>
<tr>
<td>WHO BMI z-score</td>
<td>−0.16 ± 1.31</td>
</tr>
<tr>
<td>Birth and neonatal history</td>
<td></td>
</tr>
<tr>
<td>Birth weight (kg)</td>
<td>3.3 ± 0.53</td>
</tr>
<tr>
<td>Gestational age (weeks)</td>
<td>39.0 ± 1.81</td>
</tr>
<tr>
<td>Duration of breastfeeding (weeks)</td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>46 (13)</td>
</tr>
<tr>
<td>1–13</td>
<td>143 (40)</td>
</tr>
<tr>
<td>&gt; 13</td>
<td>160 (45)</td>
</tr>
<tr>
<td>Boys’ dietary consumption of any local foods</td>
<td></td>
</tr>
<tr>
<td>Dairy</td>
<td>151 (42)</td>
</tr>
<tr>
<td>Poultry</td>
<td>29 (8)</td>
</tr>
<tr>
<td>Non-poultry meats</td>
<td>20 (6)</td>
</tr>
<tr>
<td>Fish</td>
<td>76 (21)</td>
</tr>
<tr>
<td>Eggs</td>
<td>54 (15)</td>
</tr>
<tr>
<td>Fruits and vegetables</td>
<td>341 (96)</td>
</tr>
<tr>
<td>Parental and residential characteristics</td>
<td></td>
</tr>
<tr>
<td>Duration of Chapaevsk residence (years)</td>
<td></td>
</tr>
<tr>
<td>&lt; 3</td>
<td>101 (28)</td>
</tr>
<tr>
<td>3 to &lt; 6</td>
<td>71 (20)</td>
</tr>
<tr>
<td>6 to &lt; 8</td>
<td>94 (26)</td>
</tr>
<tr>
<td>≥ 8</td>
<td>87 (25)</td>
</tr>
<tr>
<td>Any household smoking during pregnancy</td>
<td>55 (17)</td>
</tr>
<tr>
<td>Mother ≤ 25 years old at son’s birth</td>
<td>248 (70)</td>
</tr>
<tr>
<td>Minimum parental education</td>
<td></td>
</tr>
<tr>
<td>High school or less</td>
<td>30 (8)</td>
</tr>
<tr>
<td>Junior college/technical school</td>
<td>201 (57)</td>
</tr>
<tr>
<td>University/postgraduate training</td>
<td>122 (34)</td>
</tr>
<tr>
<td>Household income (RUB/month)</td>
<td></td>
</tr>
<tr>
<td>&lt; 175</td>
<td>110 (31)</td>
</tr>
<tr>
<td>175–250</td>
<td>88 (25)</td>
</tr>
<tr>
<td>&gt; 250</td>
<td>155 (44)</td>
</tr>
<tr>
<td>Father employed at factory complex</td>
<td>50 (14)</td>
</tr>
<tr>
<td>Residential distance to factory complex (km)</td>
<td></td>
</tr>
<tr>
<td>&lt; 2</td>
<td>65 (18)</td>
</tr>
<tr>
<td>2 to &lt; 5</td>
<td>159 (45)</td>
</tr>
<tr>
<td>≥ 5</td>
<td>131 (37)</td>
</tr>
</tbody>
</table>

Table 2. Distribution of measured OCPs (ng/g lipid) among 8- to 9-year-old boys enrolled in the Russian Children’s Study (n = 355). *

<table>
<thead>
<tr>
<th>OCP</th>
<th>n</th>
<th>Minimum</th>
<th>10th</th>
<th>25th</th>
<th>Median</th>
<th>50th</th>
<th>75th</th>
<th>90th</th>
<th>Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td>HCB</td>
<td>355</td>
<td>32</td>
<td>80</td>
<td>107</td>
<td>158</td>
<td>246</td>
<td>364</td>
<td>2,660</td>
<td></td>
</tr>
<tr>
<td>β-HCH</td>
<td>355</td>
<td>39</td>
<td>81</td>
<td>112</td>
<td>167</td>
<td>270</td>
<td>412</td>
<td>2,860</td>
<td></td>
</tr>
<tr>
<td>p,p′-DDE</td>
<td>355</td>
<td>49</td>
<td>122</td>
<td>187</td>
<td>284</td>
<td>492</td>
<td>835</td>
<td>9,370</td>
<td></td>
</tr>
</tbody>
</table>

*No values < LOD.

Table 3. Median OCP concentrations (ng/g lipid) in 8- to 9-year-old boys in the Russian Children's Study compared with other pediatric studies.

<table>
<thead>
<tr>
<th>Country</th>
<th>Year</th>
<th>n</th>
<th>Age range (years)</th>
<th>Population</th>
<th>HCB</th>
<th>β-HCH</th>
<th>p,p′-DDE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Russia</td>
<td>2003–2005</td>
<td>355</td>
<td>8–9</td>
<td>Boys</td>
<td>158</td>
<td>167</td>
<td>284</td>
</tr>
<tr>
<td>USDA (NHANES)</td>
<td>2003–2004</td>
<td>588</td>
<td>12–19</td>
<td>Boys and girls</td>
<td>13.4</td>
<td>&lt; LOD</td>
<td>93.6</td>
</tr>
<tr>
<td>Belgium</td>
<td>2003–2004</td>
<td>1,679</td>
<td>14–15</td>
<td>Boys</td>
<td>22.8</td>
<td>---</td>
<td>104</td>
</tr>
<tr>
<td>Faroe Islands</td>
<td>1986–1987</td>
<td>789</td>
<td>14</td>
<td>Boys and girls</td>
<td>13.4</td>
<td>&lt; LOD</td>
<td>467</td>
</tr>
<tr>
<td>Slovakia (contaminated)</td>
<td>2001</td>
<td>216</td>
<td>8–10</td>
<td>Boys and girls</td>
<td>79.6</td>
<td>---</td>
<td>344</td>
</tr>
</tbody>
</table>

Michalovce district)
these estimated increases correspond to mean HCB, β-HCH, and p,p’-DDE serum concentrations of 223 ng/g lipid (95% CI: 191, 260 ng/g lipid), 208 ng/g lipid (95% CI: 181, 240 ng/g lipid), and 284 ng/g lipid (95% CI: 233, 346 ng/g lipid), respectively, adjusted for the final model covariates for each OCP (Figure 1).

Any local dairy consumption (vs. none) predicted higher HCB, β-HCH, and p,p’-DDE serum concentrations of 14% (95% CI: 0.6, 30%), 21% (95% CI: 7, 36%), and 18% (95% CI: 1.37%), respectively, with adjustment for total dairy consumption (which was not a significant predictor of any of the OCPs) (Table 4). Although any local poultry consumption predicted higher HCB (β = 0.09) and total egg consumption predicted higher β-HCH (β = 0.09), only local dairy consumption was a significant predictor when poultry, egg, and dairy consumption were modeled simultaneously (data not shown).

Factors that predicted higher concentrations of only one of the OCPs were having parents who had a high school education or less (57% higher serum p,p’-DDE relative to boys whose parents had university/postgraduate training; 95% CI: 17, 111%), living in Chapaevsk for ≥ 3 years (higher β-HCH concentrations compared with those living < 3 years in Chapaevsk, trend p = 0.003) and having a father employed at the factory complex (higher β-HCH concentrations, p = 0.10).

The percent of variation (R²) explained by the predictors in each final OCP model ranged from 0.21 (for the model of HCB) to 0.36 (for β-HCH). Sensitivity analyses of predictors for whole weight of log_{10}-transformed serum OCPs adjusted for total lipids were consistent with predictions of lipid-adjusted serum OCPs (see Supplemental Material, Table S1).

Discussion
In the present study, we measured serum OCP concentrations and identified several demographic, lifestyle, and environmental predictors among boys living in Chapaevsk, Russia, a town contaminated by previous industrial activity. These results complement a publication describing predictors of dioxins and polychlorinated biphenyls (PCBs) among these boys (Burns et al. 2009). Despite the young age of our cohort (8–9 years), concentrations of OCPs were similar to or higher than concentrations reported for somewhat older pediatric populations (range, 8–19 years) in the United States and Europe (Barr et al. 2006; Den Hond et al. 2011; Patterson et al. 2009; Petrik et al. 2006).

Consistent with other studies of persistent organic pollutants in this cohort and other populations (Burns et al. 2009; Den Hond et al. 2009; Gallo et al. 2011; Humblet et al. 2010; Karmaus et al. 2001), lower BMI predicted higher serum OCP concentrations. This finding may be attributable to a smaller volume of distribution in boys with lower BMI, resulting in higher serum concentrations (Wolf et al. 2009).

Breastfeeding is a known route of early life exposure to lipophilic persistent compounds (Rogan et al. 1986). Consistent with other studies (Barr et al. 2006; Den Hond

Table 4. Final multivariable predictor models for serum concentrations of organochlorine pesticides based on linear regression models.

<table>
<thead>
<tr>
<th>WHO BMI z-score categories</th>
<th>Estimated % change in pesticide (95% CI)²</th>
<th>p-Value²</th>
<th>Estimated % change in pesticide (95% CI)²</th>
<th>p-Value²</th>
<th>Estimated % change in pesticide (95% CI)²</th>
<th>p-Value²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Underweight</td>
<td>28.3 (10.6, 48.8)</td>
<td>0.001</td>
<td>18.6 (3.1, 36.5)</td>
<td>0.02</td>
<td>9.0 (–8.5, 29.8)</td>
<td>0.34</td>
</tr>
<tr>
<td>Normal</td>
<td>Reference</td>
<td></td>
<td>Reference</td>
<td></td>
<td>Reference</td>
<td></td>
</tr>
<tr>
<td>Overweight/obese</td>
<td>−36.1 (–46.3, –23.9)</td>
<td>&lt; 0.001</td>
<td>−44.1 (–52.5, –34.2)</td>
<td>&lt; 0.001</td>
<td>−51.0 (–60.0, –39.9)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>p-Value for trend</td>
<td>&lt; 0.001</td>
<td></td>
<td>&lt; 0.001</td>
<td></td>
<td>&lt; 0.001</td>
<td></td>
</tr>
<tr>
<td>Breastfeeding duration (weeks)</td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>None</td>
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</tr>
<tr>
<td>1–13</td>
<td>2.0 (–18.3, 24.4)</td>
<td>0.84</td>
<td>13.2 (–6.4, 36.8)</td>
<td>0.20</td>
<td>8.0 (–14.4, 36.4)</td>
<td>0.52</td>
</tr>
<tr>
<td>&gt; 13</td>
<td>15.9 (–4.8, 41.0)</td>
<td>0.14</td>
<td>62.8 (35.0, 96.3)</td>
<td>&lt; 0.001</td>
<td>81.0 (43.3, 128.4)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>p-Value for trend</td>
<td>0.05</td>
<td></td>
<td>&lt; 0.001</td>
<td></td>
<td>&lt; 0.001</td>
<td></td>
</tr>
<tr>
<td>Residential distance from factory complex (km)</td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>&lt; 2</td>
<td>63.8 (37.0, 95.9)</td>
<td>&lt; 0.001</td>
<td>57.1 (31.8, 87.2)</td>
<td>&lt; 0.001</td>
<td>16.7 (–5.2, 43.7)</td>
<td>0.15</td>
</tr>
<tr>
<td>2 to &lt; 5</td>
<td>9.3 (–4.9, 25.6)</td>
<td>0.21</td>
<td>33.7 (17.1, 52.7)</td>
<td>&lt; 0.001</td>
<td>35.6 (15.1, 59.8)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>≥ 5</td>
<td>Reference</td>
<td></td>
<td>Reference</td>
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</tr>
<tr>
<td>p-Value for trend</td>
<td>&lt; 0.001</td>
<td></td>
<td>&lt; 0.001</td>
<td></td>
<td>&lt; 0.001</td>
<td></td>
</tr>
<tr>
<td>Local dairy consumption</td>
<td>14.4 (0.6, 31.0)</td>
<td>0.04</td>
<td>20.6 (6.9, 36.1)</td>
<td>0.003</td>
<td>17.5 (1.0, 36.7)</td>
<td>0.04</td>
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<tr>
<td>Total dairy consumption²</td>
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<td></td>
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<td></td>
</tr>
<tr>
<td>Low</td>
<td>−2.7 (–16.6, 13.5)</td>
<td>0.72</td>
<td>−2.9 (–16.1, 12.3)</td>
<td>0.69</td>
<td>−7.5 (–22.7, 10.8)</td>
<td>0.40</td>
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<tr>
<td>High</td>
<td>−0.7 (–15.0, 16.1)</td>
<td>0.93</td>
<td>−2.2 (–15.5, 13.2)</td>
<td>0.76</td>
<td>−10.7 (–25.8, 7.4)</td>
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<tr>
<td>p-Value for trend</td>
<td>0.93</td>
<td></td>
<td>0.76</td>
<td></td>
<td>0.23</td>
<td></td>
</tr>
<tr>
<td>Duration of Chapaevsk residence (years)</td>
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<td></td>
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</tr>
<tr>
<td>&lt; 3</td>
<td>Reference</td>
<td></td>
<td>Reference</td>
<td></td>
<td>Reference</td>
<td></td>
</tr>
<tr>
<td>3 to &lt; 6</td>
<td>−28.3 (7.9, 52.5)</td>
<td>0.005</td>
<td>−28.0 (9.0, 50.4)</td>
<td>0.003</td>
<td>−28.0 (9.0, 50.4)</td>
<td>0.003</td>
</tr>
<tr>
<td>≥ 8</td>
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</tr>
<tr>
<td>p-Value for trend</td>
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<td></td>
<td>0.003</td>
<td></td>
<td>0.003</td>
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</tr>
<tr>
<td>Father worked at factory complex</td>
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<tr>
<td>High school or less</td>
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<tr>
<td>Junior college/technical school</td>
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<tr>
<td>University/postgraduate</td>
<td>Reference</td>
<td></td>
<td>Reference</td>
<td></td>
<td>Reference</td>
<td></td>
</tr>
<tr>
<td>p-Value for trend</td>
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<td></td>
<td>0.003</td>
<td></td>
<td>0.003</td>
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<tr>
<td>Total model R²</td>
<td>0.21</td>
<td>0.36</td>
<td>0.30</td>
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</tr>
</tbody>
</table>

¹Variables were not retained in final models. Final models include predictors with p ≤ 0.10.
²Estimated change in pesticide concentration based on β parameter estimates for predicting log_{10} lipid-adjusted concentrations and then calculating 10^β. ³Wald statistic. ²Total dairy consumption is included in final models to reflect background levels.
et al. 2009; Gallo et al. 2011; Karmaus et al. 2001), longer breastfeeding duration (>13 weeks) predicted higher OCP concentrations. Although breastfeeding in our cohort ended years before OCP measurement, childhood concentrations of lipophilic compounds track closely with breastfeeding exposure in infancy (Patandin et al. 1999).

Residential distance from the primary factory may provide insight on chemical-specific pathways of exposure in this area. Specifically, living <2 km from the complex was associated with higher serum HCB and β-HCH concentrations, which is consistent with these compounds having been manufactured at the factory as a source of exposure. However, serum \( p,p' \)-DDE levels were highest for boys living 2 to <5 km from the factory and only moderately and nonsignificantly elevated among boys living within 2 km. DDT, the parent compound for \( p,p' \)-DDE, was not manufactured at the complex, and other exposure sources likely contributed to the boys’ \( p,p' \)-DDE levels.

Duration of residence in Chapaevsk and father’s prior employment at the factory were positive predictors of β-HCH. We hypothesized that longer residence in Chapaevsk would be associated with higher exposure to and bioaccumulation of both β-HCH and HCB, because both were produced locally. Therefore, it is unclear why duration of residence was a significant predictor of β-HCH but not HCB. Similarly, fathers’ prior occupation at the factory was not a significant predictor of HCB, despite the same potential for exposure from residues on the fathers’ work clothing, boots, tools, or skin (Lu et al. 2000). Mother’s employment at the factory did not predict any of the OCPs, but only 5% of mothers reported previous employment at the factory, limiting power to detect a statistically significant association.

Non-occupational exposure to HCB, β-HCH, and \( p,p' \)-DDE is primarily dietary (Darnerud et al. 2006; Gasull et al. 2011); therefore, we expected consumption of local foods high in fat, such as dairy and fish, to predict higher serum concentrations. However, local dairy consumption was the only significant predictor of all three OCPs. Studies of OCP exposure and local diet among children have been limited to two assessments concerned about local environmental contamination (Den Hond et al. 2009; Gallo et al. 2011), that did not find an association with local dairy. Dairy consumption has been associated with serum OCP concentrations in several adult populations, although none differentiated whether dairy foods were from local sources (Arrebola et al. 2009, 2012; Lee et al. 2007). Although previous studies among children reported associations between serum OCPs and consumption of local fish (Gallo et al. 2011) and consumption of fatty meats and vegetables (Den Hond et al. 2009), these foods were not significant predictors in our cohort.

We previously reported that consumption of most local foods predicted higher serum dioxins and PCBs in the same study cohort (Burns et al. 2009), consistent with findings reported for other study populations concerned about environmental contamination (Choi et al. 2006; Gallo et al. 2011; Schecter et al. 2003). It is unclear why local food consumption, apart from dairy, was associated with dioxin-like compounds but not serum OCP concentrations in our study cohort.

One limitation of our diet analyses is the inability to assess consumption of specific types of fish or fat content. We had only father’s reported employment history with no independent verification, and therefore can only speculate on the association observed with β-HCH. Although we attempted to evaluate many potential determinants of these exposures (e.g., parental education), there were probably other predictors that we could not assess.

A major strength of this study is the large sample size of young boys with serum HCB, β-HCH, and \( p,p' \)-DDE measurements. For all three OCPs, all serum concentrations were above the limit of detection with wide ranges of concentrations. Detailed dietary information, including local food consumption, as well as calculated residential distance from the factory complex, was also available. In this context, this study contributes to understanding of determinants of serum OCP levels among children, and in particular, highlights the potential importance of local risk factors for exposure.

Figure 1. Adjusted mean OCP serum concentrations of Russian prepubertal boys in relation to residential distance from factory complex. Adjusted means (95% CIs) calculated using least-square means adjusted for all other covariates in Table 4.

Conclusion

Our findings suggest that contamination from the local factory may be an important source of HCB and β-HCH exposure for boys in Chapaevsk. Residential distance from the primary factory was a significant predictor of serum HCB and β-HCH, both of which were manufactured at the complex. Father’s past employment at the factory and longer residence in Chapaevsk also predicted higher serum β-HCH, and local dairy consumption predicted higher serum concentrations of all three OCPs; these results add further support to local environmental contamination, at least partly from the factory, as a source of exposure. Consistent with other studies, longer breastfeeding duration and lower BMI predicted higher serum OCPs.

Our findings provide insight on determinants of OCP exposure, which may lead to local monitoring and continuation of remediation measures (e.g., soil removal) to reduce childhood and community exposure. Although our findings suggest that local dairy consumption and longer breastfeeding duration are primary determinants of OCP exposure, it would be premature to recommend reduced intake of local dairy foods or reduced breastfeeding without fully understanding the exposure pathway and the risk–benefit trade-offs from such a recommendation. It is important to keep in mind that these local food products were central to the children’s diet in this region, and that breastfeeding has well-established benefits. Recommendations to prevent childhood exposure include environmental cleanup of contaminated areas, regulatory enforcement of safe practices for industrial waste disposal and emissions control, and preferential consumption, when available, of foods produced in noncontaminated areas.

References


Predictors of chlorinated pesticides in Russian boys


