UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, DC 20460

OFFICE OF CHEMICAL SAFETY AND
POLLUTION PREVENTION

MEMORANDUM

DATE: March 25, 2016

SUBJECT: **Summary Reviews for Additional Epidemiological Literature Studies from Prospective Birth Cohort Studies.**

PC Code: See below
Decision Number: 514824
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EPA has conducted systematic reviews of the scientific literature on epidemiology studies on neurodevelopmental outcomes associated with OP exposure in 2012, 2014, and 2015. Although other studies exist, the most robust epidemiology studies are conducted through three major U.S. based prospective birth cohort studies: 1) Mothers and Newborn Study of North Manhattan and South Bronx conducted by Columbia University, referred to in this document as "CCCEH;" 2) Mount Sinai Inner-City Toxicants, Child Growth and Development Study, or the "Mount Sinai Study/Cohort;" and 3) Center for Health Assessment of Mothers and Children of Salinas Valley (CHAMACOS) conducted by the University of California Berkeley, or "CHAMACOS Study/ Cohort." In 2015, HED completed an updated epidemiological literature review of the organophosphates (D331251, A. Lowit et al, 09/15/2015). This document provides summaries of five additional studies which HED has become aware of since this 2015 review was completed. Two of these studies focus on organophosphate exposure and neurodevelopment (Rauh et al, 2015; Engel et al, 2015), with the remaining studies focusing on

organophosphate exposure and pediatric respiratory symptoms (Ranaan et al, 2015), prenatal permethrin and piperonyl butoxide exposure and neurodevelopment (Horton et al, 2011), and polybrominated diphenyl ether exposure and neurodevelopment (Eskenazi et al, 2013). These studies were reviewed due to the fact that the study populations assessed include the same three prospective birth cohort studies (CCCEH, CHAMACOS, and Mt. Sinai) that were the primary focus of the updated 2015 literature review, as well as the 2014 literature review associated with revised human health risk assessment for chlorpyrifos (USEPA, 2014).

This document supports the use of the 10X FQPA Safety Factor in the individual organophosphate human health risk assessments.

Chemical	PC Code	CAS No.
Dicrotophos	035201	141-66-2
Fosthiazate	129022	98886-44-3
Coumaphos	036501	56-72-4
Terbufos	105001	13071-79-9
Profenofos	111401	41198-08-7
Bensulide	009801	741-58-2
Diazinon	057801	333-41-5
Ethoprop	041101	13194-48-4
Dimethoate	035001	60-51-5
Malathion	057701	121-75-5
Phosmet	059201	732-11-6
Chlorethoxyfos	129006	54593-83-8
Acephate/ Methamidiphos	103301/ 101201	30560-19-1/ 10265-92-6
Pirimiphos-methyl	108102	29232-93-7
TCVP	083701	961-11-5
Tribufos	074801	78-48-8
Phorate	057201	298-02-2
Phostebupirim	129086	96182-53-5
DDVP	084001	62-73-7
Naled	034401	300-76-5
Trichlorfon	057901	52-68-6
Fenamiphos	100601	22224-92-6
AZM	058001	86-50-0
Methidathion	100301	950-37-8
Propetamphos	113601	31218-83-4
ODM	058702	301-12-2
Disulfoton	032501	298-04-4
Methyl parathion	053501	298-00-0
Temephos	059001	3383-96-8
Chlorpyrifos-methyl	059102	5598-13-0

Study 1. Engel et al (2015)

Engel SM, Bradman A, Wolff MS, Rauh V, Harley KG, Yang JH, Hoepner LA, Barr DB, Yolton K, Vedar MG, Xu Y, Hornung RW, Wetmur JG, Chen J, Holland NT, Perera FP, Whyatt R, Lanphear BP, Eskenazi B. Prenatal Organophosphorus Pesticide Exposure and Child Neurodevelopment at 24 Months: An Analysis of Four Birth Cohorts. Environmental Health Perspectives. 2015 (advance publication): http://dx.doi.org/10.1289/ehp.1409474.

Engel et al. (2015) reports on the results of a pooled analysis from four cohorts (N=936) to evaluate the association between prenatal urinary dialkyl phosphates (DAPs) and neurodevelopmental outcomes at 24 months only. Total DAPs is a non-specific measure of OP exposure and is the sum of six separate molecules — three dimethyl alkylphosphate (DMAP) molecules of dimethylphosphate, dimethylthiophosphate, and dimethyldithiophosphate (DMP, DMTP, and DMDTP), and three diethyl alkylphosphate (DEAP) molecules of diethylphosphate, diethylthiophosphate, and diethyldithiophosphate (DEP, DETP, and DEDTP). In addition, researchers in this study assessed the impact on these associations of the specific cohort, race/ethnicity, and the PON1 genotype of study participants. Researchers across the four children's health cohorts utilized the Bayley Scales of Infant Development II (BSID-II) to generate a Mental Development Index (MDI) and a Psychomotor Development Index (PDI) to assess neurodevelopment in early childhood. The four cohorts include CHAMACOS (N=377), HOME (N=265), Mt. Sinai (N=234), and CCCEH (N=60). The Cincinnati Children's HOME Study is a newer cohort which collected study data from mothers and infants in 2003 to 2006, and no separate studies have been published yet on OP exposure and associations with neurodevelopmental outcomes. It is noted that the CCCEH participants included in this analysis are from women enrolled in 2000 to 2001, a time period which is during the phase out of chlorpyrifos in residential settings.

The results of this pooled study are relatively consistent with those seen in the individual cohorts at 24 months. After controlling for race/ethnicity, smoking, and drug use during pregnancy, a statistically significant association was observed in the pooled population between total DAPs exposure and MDI decrements, but not with PDI decrements. Consistent with the results from Eskenazi et al. (2007), the strongest evidence of an association was observed for the CHAMACOS cohort, with statistically significant associations for both total DAPs and total DMAPs exposure and MDI decrements. No significant associations were seen within the Mt. Sinai and CCCEH cohorts, a result which is basically consistent with the previous observations at 24 months in these cohorts (Engel et al., 2011; Rauh et al., 2006). The study authors observed significant heterogeneity from combining the cohorts, especially with regards to race/ethnicity, and noted that impacts on specific subpopulations may be lost when looking at the pooled results.

Study 2. Eskenazi et al (2013)

Eskenazi B; Chevrier J; Rauch S; Kogut K; Harley KG; Johnson C; Trujillo C; Sjodin A; Bradman A. (2013). In Utero and Childhood Polybrominated Diphenyl Ether (PBDE) Exposures and Neurodevelopment in the CHAMACOS Study. ENVIRONMENTAL HEALTH PERSPECTIVES: 121(2), 257-262.

In this study of the CHAMCOS cohort, study authors assessed the relationship between prenatal and postnatal polybrominated diphenyl ethers (PBDE) exposure and neurodevelopmental outcomes (Eskenazi et al., 2013). They observed associations between PBDE exposure and neurodevelopmental outcomes, but concluded that these associations were independent of those previously observed in the CHAMACOS cohort between OP exposure and child neurobehavioral development (Bouchard et al., 2011; Eskenazi et al., 2007; Marks et al., 2010).

Specifically, PBDE exposure was assessed in blood samples collected prenatally in mothers either at ~26 weeks gestation (N=219) or at delivery (N=60), whereas child blood samples were collected postnatally at 7 years of age (N=272). In addition, maternal exposure to OPs was assessed by measuring DAPs in maternal urine at 13 and 26 weeks gestation, lead was measured in maternal prenatal and cord blood samples, PCBs were measured in maternal blood, and maternal thyroid hormone levels were assessed at 26 weeks gestation. Children's attention, motor functioning, and cognition were all assessed at 5 (N=310) and 7 years of age (N=323). Motor functioning and cognition were assessed by trained bilingual psychometricians using standardized approaches, including the McCarthy Scales of Children's Abilities for motor function and the Weschler Intelligence Scale for Children (4th edition) for cognitive functioning. In contrast, attention issues were assessed using mother-reported (5 year and 7 year assessments) and teacher-reported (7 year assessment) checklists on the behavior of a child, as well as a computerized vigilance test (Conner' Kiddie Continuous Performance Test or K-CPT) at the 5 year visit, with these results then being assessed by trained psychometricians. Checklists used were the Child Behavior Checklist (CBCL, version 1.5-5) at the 5 year visit and the Conners ADHD/DSM-IV Scales (CADS) at the 7 year visit.

Maternal prenatal PBDE exposure was significantly associated with child impaired attention at both 5 years and 7 years, with the 5 year old results being based on only the K-CPT results, and the 7 year old results being based solely on maternal reported attention problems. Furthermore, maternal PBDE exposure was associated with poorer fine motor coordination at both 5 and 7 years and intelligence decrements were observed at age 7 years. Postnatal child PBDE exposure at 7 years was marginally or significantly associated with some measures of teacher-reported child attention problems, but had no association with other measures of teacher-reported child attention problems. It is also noted that postnatal child PBDE exposure was not associated with any of the maternal-reported child attention problems at 7 years of age. After adjustment for potential confounding factors of birth weight, gestational age, and maternal thyroid hormone levels, these results remained unchanged. In addition, sensitivity analyses were conducted to determine whether exposure to PCBs, OPs (assessed as DAPs), and lead had confounded their evaluation, with study authors concluding that these exposures were independent of their PBDE exposure and neurodevelopmental outcome results.

Study 3. Horton et al (2011)

Horton MK, Rundle A, Camann DE, Barr DB, Rauh VA, Whyatt RM. (2011). Impact of Prenatal Exposure to Piperonyl Butoxide and Permethrin on 36-Month Neurodevelopment. Pediatrics: 127(3), e699-706.

The investigators examined the association between prenatal exposure to permethrin and piperonyl butoxide (PBO) and 36-month neurodevelopment from the Columbia Center for Children's Environmental Health (CCCEH) prospective cohort study. The CCCEH study consists of black and Dominican mothers and newborns living in upper Manhattan and South Bronx in New York City.

To evaluate cognitive and psychomotor development, Bayley Scales of Infant Development second edition (BSID II) were used to measure the mental development index (MDI) and the psychomotor developmental index (PDI). Permethrin and piperonyl butoxide levels were measured in personal air collected during pregnancy. Cis- and trans- permethrin and PBO levels were measured from the maternal umbilical cord blood at the time of delivery and from the maternal plasma collected within 48 hours of delivery. Permethrin in personal air was measured from 342 women, permethrin in plasma from 272 women and piperonyl butoxide (PBO) levels in personal air from 230 women. Mother's nonverbal intelligence was measured with the Test of Nonverbal Intelligence (TONI), and the quality of care at

home was measured by using the Home Observation for Measurement of the Environment (HOME) when the child was 2-3 years old.

In the statistical analysis, investigators considered the covariates (such as, ethnicity, education, age, prenatal environmental tobacco smoke exposure, gestational age, birth outcome data and gender), which were prognosticators of development and other factors that can influence the association between permethrin and/or PBO exposure and neurodevelopmental outcomes. Multivariate linear and logistic regression analyses were used to control covariates.

Investigators reported that, "there were no significant associations between cis- or trans- permethrin measured in personal air samples as well as maternal and/or umbilical cord plasma samples, and mental or motor development of a child at 36 months. However, there was a significant inverse association between prenatal PBO exposure and 36 month MDI. MDI scores reduced 1.2 points per log-unit increase in PBO exposure (95% confidence interval [CI]: -0.33 to -2.25; $p = 0.008$). The strongest adverse effect of PBO was at the highest quartile group when compared to the referent group (Odds ratio: 4.57; 95% CI: -0.3 to -8.84; $p = 0.04$). Children highly exposed to piperonyl butoxide in personal air samples (>4.34 ng/m^3) scored 3.9 points lower on the MDI than those with lower exposures (95% CI: −0.25 to −7.49; $p = 0.04$)" (Horton et al., 2011).

When comparing those individuals in the highest exposure group against all other individuals, the odds of delayed mental development due to prenatal PBO exposure was 3.11 (95% CI: 1.38 – 6.98; p = 0.006). If one compares those individuals in the highest exposure group against only those in the lowest exposure (reference) group, the odds of delayed mental development due to prenatal PBO exposure was 2.49 (95% CI: 0.95-6.54; p=0.06). In addition, for each log unit increase in PBO exposure, there was 1.3 fold increased odds of delayed mental development (95% CI: 1.06 – 1.66; p = 0.01).

The investigators concluded that prenatal exposure to piperonyl butoxide was negatively associated with 36-month neurodevelopment, whereas no association was observed between permethrin and 36-month neurodevelopment. The study authors also noted that these observations are independent of previous CCCEH evaluations where chlorpyrifos exposure was associated with neurodevelopmental outcomes (Young et al, 2005; Rauh et al, 2006). As part of this study, chlorpyrifos exposure was assessed, but was not associated with permethrin or PBO exposure.

Study 4. Raanan et al (2015)

Raanan R, Harley KG, Balmes JR, Bradman A, Lipsett M, and Eskenazi B. (2015) Early-life Exposure to Organophosphate Pesticides and Pediatric Respiratory Symptoms in the CHAMACOS Cohort. ENVIRONMENTAL HEALTH PERSPECTIVES: 123(2), 179-185.

The investigators evaluated the association between early-life exposure to organophosphates (OPs) and respiratory outcomes in children by using the cohort of mothers and children from the Center for the Health Assessment of Mothers and Children of Salinas (CHAMACOS) birth cohort. Exposure assessment was done by measuring six nonspecific Dialkyl Phosphate (DAP) metabolites [three diethyl phosphate (DEAP) and three dimethyl phosphate (DMAP) metabolites] in the urine of 359 pregnant women at (mean ± SD = 13.5 ± 4.8 and 26.4 ± 2.4 weeks) during pregnancy. For the children, OP metabolites in urine were measured five times at 0.5, 1, 2, 3.5, and 5 years of age. The investigators calculated the area under the curve (AUC) by summing the time-weighted averages from each time interval using the trapezoidal method. The time-weighted averages were calculated for each time interval by multiplying the time between measurements in years by the average of the two measured concentrations. The International Study of Asthma and Allergies in Childhood (ISAAC) questionnaire was used to obtain the

children's respiratory symptoms from their mothers at 5 and 7 years of age. Also the information regarding whether or not medications for asthma or wheezing or tightness of chest were prescribed by their physicians, was collected. Homes were inspected by trained personnel when the children were 6 and 12 months old. According to the investigators, generalized estimating equations (GEE) were used to examine associations of prenatal and childhood DAP concentrations with respiratory symptoms and exercise-induced coughing at 5 and 7 years of age, adjusting for child's sex and age, maternal smoking during pregnancy, secondhand tobacco smoke, season of birth, $PM_{2.5}$ concentration, breastfeeding, mold and cockroaches in home, and the distance from highway.

The investigators reported that *prenatal* total DAP urinary concentrations were not significantly associated with reported respiratory symptoms and exercise-induced coughing in children. But total DAPs and DEAP metabolites in urine from the *second half of pregnancy* were significantly associated with increased odds of respiratory symptoms in children (adjusted OR (aOR) for a 10 fold increase in concentration is 1.77; 95%CI: 1.06, 2.95, $p = 0.03$; aOR = 1.61; 95% CI: 1.08, 2.39, $p = 0.02$, respectively). But DMAP metabolites in urine from the second half of pregnancy were not significantly associated with respiratory symptoms. In addition, the investigators reported that respiratory symptoms and exercise-induced coughing at 5 and 7 years of age were significantly associated with total DAPs, DEAPs and DMAPs in children's urine collected between the ages of 6 months and 5 years (AUC). For a 10 fold increase in total DAPs concentration the OR is 2.53 (95%CI: 1.32, 4.86, $p = 0.005$) for respiratory symptoms, and the aOR = 5.40 (95%CI: 2.10, 13.91, p =<0.001) for exercise-induced coughing. They concluded that early-life exposure to OP pesticides was associated with respiratory symptoms consistent with possible asthma in childhood.

Investigators stated that, "the limitation in this study is using DAP metabolites as a marker of OP exposure since these may reflect exposure both to the parent pesticide compounds as well as to preformed DAPs in food or dust". In addition, varying exposure to OPs and fluctuated DAP metabolite levels might cause nondifferential exposure misclassification resulting in bias towards the null. The investigators noted that their cohort entailed Mexican mother and children living in an agricultural community in California, and more research is needed to determine if their findings are generalizable to other populations.

According to the investigators the strengths in this study were the repeated measuring of DAPs in urine (two times from mothers during pregnancy and five times from children during early childhood); the longitudinal study design with large sample size; well established and validated ISAAC questionnaire was used to obtain children's respiratory symptoms; and important covariates (exposure to other environmental agents and socioeconomic factors in the first year of life) were considered and adjusted during the statistical analysis.

The investigators discussed the pathophysiological causes of childhood asthma as follows: OP pesticides can easily pass through the placenta (Rauh, 2006; Whyatt, 2009) and may have adversarial effects on the surfactant synthesis in the fetus lungs during the second half of pregnancy. This may lead to development of asthma in childhood (Hameed, 2013; Wright, 2000). Also previous study in this same cohort found that maternal work in agriculture during the child's first year of life was associated with increased levels of T helper 2 (Th2) cytokines which can cause airway hyper-responsiveness and initiate asthma development.

The investigators concluded that their findings from this study, that "early life exposure to OPs is associated with asthma like respiratory symptoms in children," are consistent with the findings of previous studies.

Study 5. Rauh et al (2015)

Rauh VA.; Garcia WE.; Whyatt RM.; et al. (2015) Prenatal exposure to the organophosphate pesticide chlorpyrifos and childhood tremor. NEUROTOXICOLOGY: 51, 80-86.

CCCEH study authors (Rauh et al., 2015) evaluated the relationship between prenatal chlorpyrifos exposure and motor development/movement among 271 of the cohort participants who had reached the age of approximately 11 years and had a complete set of data including prenatal maternal interview data, prenatal chlorpyrifos marker levels from maternal and/or cord blood samples at delivery, postnatal covariates, and motor development/movement outcome data. It is noted that the study abstract does not appear to match the text of the article itself in terms of the number of study participants (N), with the N being cited as 263 in the abstract, whereas the N is listed as being 271 in all other places in the article.

The 271 children study participants were assessed at approximately 11 years old (mean age 10.9 ± 0.85 years; range 9.0-13.9 years). In order to obtain maternal and child sociodemographic information and medical information, maternal interviews were conducted prenatally and every one year after birth of the child. A full battery of child neurodevelopmental tests were conducted by a trained neuropsychological tester. This manuscript reports the results of one specific measure, the drawing of Archimedes spirals, assessed during this battery of tests. Spirals were drawn by each child using both the dominant and the non-dominant arm. The degree to which tremor was present based on these spiral drawings was assessed by a senior neurologist that specializes in movement disorders and this specialist was blinded from all study participant clinical information including chlorpyrifos exposure status.

When comparing children in the upper tertile of exposure (>6.17 pg/g; N=43) to those in the lower tertiles (N=228), they observed statistically significant associations between prenatal chlorpyrifos exposure and mild to moderate tremor in the dominant arm, both arms, either arm, and a marginally statistically significant association in the non-dominant arm. The specific OR calculated associated with these elevated risks of arm tremor are as follows: dominant arm (OR=3.2; 95% CI=1.3-8.1; p=0.015); both arms (OR=3.3; 95% CI=1.1-9.4; p=0.027); either arm (OR=2.2; 95% CI=1.1-4.6; p=0.028); and non-dominant arm (OR=2.1; 95% CI=0.99-4.3; p=0.055). These associations were observed even after controlling for potential confounding factors such as medication, sex, and ethnicity.

Another potential discrepancy worth noting is that in the abstract and in Table 4 of this article, quartiles of exposure are discussed, whereas the remainder of the article discusses tertiles of exposure. Regardless of whether quartiles or tertiles of exposure were used, for the purposes of conducting this assessment the study authors grouped all individuals in the highest exposure group (N=43) and compared them against all other study participants (N=228). Identical N were cited in both the abstract and the remainder of the article when discussing the "upper quartile" and "upper tertile" group. Therefore, this noted discrepancy is not anticipated to have impacted the study results.

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