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Association Between Socioeconomic and Behavioral Characteristics with Urinary Neonicotinoid Levels in the United States, 2015-2016

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Chapter 1 – Introduction

Neonicotinoids are a class of pesticides used in agriculture and households[1]. They were developed in the 1990s – early 2000s and are said to be highly selective towards the targeted insects and harmless to untargeted organisms compared to organophosphates and carbamates[2]. Neonicotinoids include imidacloprid, acetamiprid, dinotefuran, thiamethoxam, and clothianidin[3]. Recently there have been rising concerns about the potential health impact of neonicotinoids because of their large use and their remains in the environment and food. Some countries have banned the use of neonicotinoids. In the European Union, neonicotinoids have been forbidden for outdoor use since 2018, and recently, all the exemptions in the use of these pesticides were removed[4]. In the United States, neonicotinoids are largely still in use, and they represent the most used pesticides in agriculture. Neonicotinoids have been used to treat about 90% of the corn and 50% of the soybeans that were planted in the USA[5]. Evidence has been brought out that they persist in food. For example, in 2012 they were detected in 72% of fruits and 45% of vegetables[6]. In this context the Centers for Disease Control and Prevention (CDC), conducted in the 2015-2016 National Health and Nutrition Examination Survey (NHANES) the dosage of neonicotinoids in urine samples. Considering the adequacy of urine analysis for neonicotinoids measurement in humans, the topic's importance, and the lack of studies, we decided to analyze the data from the NHANES 2015 – 2016 study on neonicotinoids.

The specific aims were:

- To examine the association between select sociodemographic variables (education, income, and race) with urinary neonicotinoids levels in the 2015-2016 NHANES sub-study.
- To examine the association between urinary neonicotinoids levels and the prevalence of cardiovascular disease in the 2015-2016 NHANES sub-study.

- To examine the association between behavioral characteristics (alcohol, smoking, and pesticide use) with urinary neonicotinoids levels in the 2015-2016 NHANES sub-study.

Chapter 2 – Background and Literature Review

• Description of the health problem

Neonicotinoids are a class of pesticides developed in the 1980s used in agriculture in the United States and other countries. The neonicotinoids pesticides have in common the nitromethylene heterocyclic root and include imidacloprid, acetamiprid, dinotefuran, thiamethoxam, and clothianidin[3]. They are used for their high affinity for insect neuronal nicotinic acetylcholine receptors (nAChRs)[7]. Once ingested by insects, neonicotinoids bind with nAChRs. Similar to how acetylcholine does, this bond causes nerve signaling in an irreversible manner. Neonicotinoids cannot be broken down by the enzyme that breaks down acetylcholine, acetylcholine esterase. As a result, neonicotinoids block the receptors and stimulate endlessly nerves causing paralysis at low concentrations and death at high concentrations [2]. These acetylcholine receptors are not only found in insects, but also in vertebrates. In mammals and humans, they are found in central and peripheral nervous system especially in neuromuscular and reproductive systems[8]. Due to the importance of acetylcholine receptors in mammals, including humans, there are concerns about the potential harm of neonicotinoids for untargeted animals and humans.

There is evidence of the adverse effects of neonicotinoids in animals other than those targeted for eradication. Stewart et al.[9] reported an association between neonicotinoids and bee colony collapse. Moffat et al.[10] reached a similar conclusion in their study on neonicotinoids receptors in bumblebees. A good affinity of bees' nAChRs for neonicotinoids was found, resulting in the collapse of the population of bees in agricultural areas where neonicotinoids were massively used. Hladik et al.[11] found similar results and emphasized the environmental persistence of neonicotinoids. Despite these concerns, neonicotinoids are still widely used. According to Douglas et al.[5], 90% of the corn and 50% of the soybeans grown in the US have been treated with neonicotinoids. In 2016, The Environmental Protection Agency (EPA) published a draft report on human health risk assessments intending to collect evidence on the concerns about the utilization of neonicotinoids. The conclusions emphasized the benefits of these chemicals in pest management both in agriculture and non-agriculture uses. The harmful effect admitted was only "the potential for on-field risk from some uses" [12].

The biological effects of neonicotinoids in mammals, as provided by experimental studies conducted in rats and in cell culture studies, are mostly through their metabolism in the liver, the release of metabolites, and excretion in the urine[13]. The toxicity combine interactions at several receptor locations, with some receptor types even having effects on the synapses that combine those of an agonist and an antagonist substance[3]. These are in two groups of receptors: nicotine low-affinity homomeric and high-affinity heteromeric receptors. It has been established that neonicotinoids are poor activators of mammalian neuronal nAChRs, by studies considering the two types of receptors. Imidacloprid, for example, is known as a partial agonist for nAChRs, and thiamethoxam is a poor agonist. On the contrary, acetamiprid and clothianidin are said to be super agonists[3].

The effect of neonicotinoids on human health may be acute and long-term. Acute toxicity is mainly due to accidental exposures during application of the pesticides, happening in agriculture and domestic use. Symptoms such as memory dysfunction, respiratory disorders, vomiting, nausea, hypotension, convulsions, muscular weakness, and hypothermia have been described in acute and subacute exposure to Acetamiprid[14]. There has not been much documentation about Thiacloprid intoxication in humans, health effects reported in mammals by experiments in rats showed toxicity to the liver and brain[15]. Thiamethoxam and Clothianidin are cytotoxic and harmful to the reproductive system[16].

The long-term effects have yet to be determined. Some studies have suggested neurological, hepatorenal, immunological, genotoxic, and reproductive effects[8]. Others suggested carcinogenicity for the ovary, the uterus, the thyroid, and the liver[17]. Li et al.[18] suggested that neonicotinoids promote breast cancer progression through G-protein-coupled estrogen receptors. In their review article, Zhang et al.[1] have presented a summary of the up-to-date, evidence-based potential health impacts of neonicotinoids. These are reduced sperm progressive motility in healthy men, increased levels of serum lipid molecules in the general population, urine steroid hormones in males of reproductive age, and urinary oxidative stress indicators in healthy people.

A study in Massachusetts reported that people's food contains neonicotinoids at a non-negligible level; 72% of fruits and 45% of vegetables[6]. Others have suggested that one of the most important sources of exposure to neonicotinoids in humans is through food and water intake[3]. Several specimen analyses have been proposed to measure neonicotinoids in the human body: urine, hair, serum and whole blood, milk, etc. Out of all, it has been proven that urine analysis is the most adequate[1,13].

Neonicotinoids have been a concern in Mead, Nebraska and originated legal proceedings against an ethanol plant, AltEn[19]. From 2015 to 2021 corn seeds treated with neonicotinoids were used to make ethanol. A lot of contaminated byproducts and millions of gallons of wastewater were produced. The wastewater was found to spill and run off the pile[19]. Inhabitants experienced various conditions suspected to be due to the neonicotinoids. People complained of nosebleeds, throat discomfort, and eye irritation. Then bee colonies began to die, birds and butterflies seemed confused, and domestic dogs were unwell and began to stumble around with dilated pupils[20]. An environmental risk perception survey conducted by the University of Nebraska Medical Center reported in 459 responders, respiratory disorders in 20% of the cases. Respectively, 41% and 17% reported feeling “some stress” and “a lot of stress” due to the environmental exposure to the neonicotinoids[21]. As a consequence of the environment scandal of exposure to neonicotinoids in the Mead, Nebraska community, the plant was shut down in February 2021 and AltEn has been cleaning up the site[19].

• Scientific background

Seven synthetic neonicotinoid pesticides have been developed and sold commercially since 1991. They are Imidacloprid, Nitenpyram, Acetamiprid, Thiamethoxan, Clothianidin, Thiacloprid, and Dinotefuran. We describe here those measured in the NHANES study.

Imidacloprid

Imidacloprid was the first neonicotinoid to be discovered in 1984 by addition of a 3-pyridylmethyl group to the nitromethylene heterocyclic parent molecule[3]. It was first registered for use in the United States in 1994[22]. It is a systemic pesticide that enters pests by ingestion or direct contact. Imidacloprid works by interfering with nicotinic acetylcholine receptors in the nervous system of the bug. Termites, earth insects, sucking insects, and some chewing insects are all controlled with imidacloprid. It is sprayed on seeds, soil, crops, buildings, and household pets as a topical flea control treatment[23]. Its anaerobic half-life is 27.1 days, while the aerobic half-life is 997 days. In the field, the dissipation half-life of imidacloprid ranges from 26 to 229 days, while in aqueous medium, the photolysis half-life at 24°C is less than an hour[23]. In the air, imidacloprid pesticide is not volatile[23]. A study conducted in California has shown that air samples collected following imidacloprid foliar spray applications do not contain residues of the pesticide [24].

In soil, Imidacloprid has a low tendency for adsorption to soil particles [24]. The half-life ranges from 27 to 229 days [25]. Imidacloprid has a short life in water when exposed to the sunlight, less than three hours [26] and has been rarely detected in surface water [27,28]. Studies have provided evidence that imidacloprid does not seep into groundwater from the soil profile [29]. When used to treat seeds, imidacloprid is absorbed and found in the pollen, leaves, and flowers. For example, 1.0 milligram seed treatment led to 13.0 parts per billion in pollen [30].

Imidacloprid has been found to have significant negative effects on beneficial invertebrates that were not its target [23]. For honeybees, imidacloprid has a Lethal Dose for half of the population, LD_{50} at 8 nanograms per bee. A chronic toxicity at smaller doses, as low as LD_{50} 0.01 to 1 ng/bee, was also observed affecting bee’s foraging and learning behavior [31]. Other invertebrates suffered imidacloprid side effects such as carabid beetle[32], lady beetle[33], aphid parasitoid[34], etc.

Imidacloprid’s toxicity is due to the formation of Nitric Oxide (NO) radicals, which level increases in the liver, the brain, and the plasma causing oxidative stress [35]. The increase in NO causes lipid peroxidation which can induce apoptosis causing death in the liver and brain [35].

In mammals, although the guanidine metabolite of imidacloprid lacks insecticidal characteristics, it is more toxic to mammals than the parent substance [36]. In humans, a study has suggested that exposure to high amounts in mothers was associated with smaller fetal head circumferences [37].

Acetamiprid

Acetamiprid is a pesticide used in agriculture to control of sucking-type insects on leafy vegetables, fruiting vegetables, Cole crops, citrus fruits, pome fruits, grapes, cotton, and ornamental plants and flowers [8]. It half-life in soil is 8 to 9 days and 34 days in water [38]. Aerobic soil respiration quickly breaks down acetamiprid. There aren't any significant problems with soil mobility because of the low use rate and quick degradation that limit off-site movement. It is anticipated that potable water will contain few environmental residues. Fish and silt will not bioaccumulate acetamiprid. Compared to the majority of other insecticides, acetamiprid has minimal environmental risks [38]. The LD_{50} in rats is 290 milligrams per cubic meter.

In the body acetamiprid enters several reactions such as demethylation, deacetylation, and hydrolysis of the cyano-imine linkage. Its metabolites are found mostly in blood, stomach, and liver[8]. The poisoning symptoms comprise memory dysfunction, respiratory disorders, digestives troubles, hypotension, convulsion, muscle weakness, and fever[8]. The effects of a chronic exposure to lower doses in rats were observed on the reproductive system, such as lower fertility, lower sperm count, etc. and neurological system: neural stem cell decrease, neocortex development disorder [39].

Acetamiprid has been classified as a “unlikely” human carcinogen [38].

Thiamethoxam

Thiamethoxan was first registered by the U.S. Environmental Protection Agency in 1999. It is used in farmland to eradicate insects that eat roots, leaves, and other plant tissues by sucking and chewing them. Agricultural uses include soil and seed treatments as well as leaf spraying for most row and vegetable crops like corn, soybeans, snap beans, and potatoes. It is also used to control insects in livestock pens, poultry houses, sod farms, golf courses, lawns, household plants, and tree nurseries. The half-life of thiamethoxan is 3 hours in aqueous conditions or 15-22 days in soil. The LD_{50} is 1,563 milligrams per rat.

When mice were exposed to thiamethoxam, endoplasmic reticulum stress and effects on ovarian function and oocyte development were observed [16]. It reduces estrogen production and lowers the development of the follicle [16]. Damages in DNA were also described with high doses [16].

Clothianidin

Clothianidin is a metabolite of another thiamethoxam. Clothianidin is registered for seed treatment use on corn and canola [40]. The half-life for its degradation in surface water is less than 1 day. In soil under the light, the half-life for clothianidin is 34 days. The aerobic soil metabolism ranges from 148 to 1155 days and the anaerobic aquatic metabolism is 27 days [40]. Clothianidin is stable to hydrolysis and has the ability to leach into ground water and be transported to surface water bodies via runoff [40].

In experimental exposure of animals to clothianidin, cytotoxicity and the reproductive system disorders were observed [18]. The literature provided evidence for the passage from mother to fetus via blood resulting in mild neurobehavioral effects in adult mice later on [41].

Thiacloprid

Thiacloprid is a pesticide used in agriculture for cotton and pome fruits. The pests it targets primarily are aphids and whiteflies for cotton; psylla, codling moth and plum curculio for pome fruits. In anaerobic water conditions, its half-life is 1 year; in aerobic water conditions, the half-life is 10 to 63 days. In soil, the half-life is 5 to 27 days [42]. The LD_{50} for acute toxicity by oral ingestion in mice is 621 mg/kg; by inhalation the LD_{50} is 0.48 mg/kg [43].

This substance poses a variety of health and harmful risks, including genotoxicity, reproductive and neurodevelopmental toxicity, and potential carcinogenesis trough an increase in cytokine proinflammation [15]. Thiacloprid also induces cell cycle slow down, chromosomal aberrations, and DNA damage [15].

• Limitations and gaps in existing literature

Despite the concern about neonicotinoids in human health, there still needs to be more research in the US on neonicotinoids and human health. In the 2015 and 2016 National Health and

Nutrition Examination Survey (NHANES), urinary levels of neonicotinoids were estimated and reported in a sub-group of the population.

• Rationale

Despite the concern about neonicotinoids in human health, there still needs to be more research in the US on the topic. Knowing how widely the neonicotinoids are used in agriculture in the US[5], and their persistence in fruits and vegetables[6], a legitimate question would be how exposed is the US population to the neonicotinoids? Are neonicotinoids present in the body of the general population? To answer these questions, the National Health and Nutrition Examination Survey (NHANES), in 2015 and 2016, measured and reported the urinary levels of neonicotinoids in a sub-group of the population.

Considering the adequacy of urine analysis for neonicotinoids measurement in humans, the topic's importance, and the lack of studies, we decided to analyze the data from the NHANES 2015 - 2016 study on neonicotinoids and evaluated the association of the level of neonicotinoids and some sociodemographic and behavioral characteristics.

Chapter 3 – Data and Methods

This exploratory cross-sectional study utilized the National Health and Nutrition Examination Survey (NHANES) 2015–2016 data to estimate covariate-adjusted associations between detectable neonicotinoids and demographic and behavioral variables. NHANES is a periodic study designed to evaluate health and nutrition of adults and children in the United States.

The 2015–2016 cycle sampled 15,327 people across 30 survey locations. Of these, 9,971 completed the interview, and 9,544 were examined. Neonicotinoids were only measured in a subset of 2,438 people of the study population within this cycle. Data on alcohol use is available on participants 18 years and older. Inclusion criteria are participants over the age of

18 years with urinary neonicotinoid measurements (among imidacloprid, acetamiprid, clothianidin, 5-hydroxy-imidacloprid, and N-desmethyl-acetamiprid) and with the planned, exploratory demographic, pesticide use, alcohol use, and smoking data. Participants with non-response to alcohol and smoking variables were eventually removed.

Data collection and measurement

The family and sample person demographics questionnaires were asked, in the home, by trained interviewers using the Computer-Assisted Personal Interview (CAPI) system. Persons 16 years and older and emancipated minors were interviewed directly. A proxy provided information for survey participants who were under 16 and for participants who could not answer the questions themselves.

Urine sample collection took place in the mobile examination center (MEC). This included the collecting, processing, storing, and shipping of urine. The controlled environment of the MEC allowed laboratory measurements to be performed under identical conditions at each survey location. The laboratory method uses 0.2 mL urine and is based on enzymatic hydrolysis of urinary conjugates of the target analytes, online solid phase extraction, reversed-phase high-performance liquid chromatography separation, and isotope dilution-electrospray ionization tandem mass spectrometry detection [44]

The analytical measurements were conducted following strict quality control/quality assurance (CLIA) guidelines. Along with the study samples, each analytical run included high- and low-concentration quality control materials (QCs) and reagent blanks to assure the accuracy and reliability of the data. The concentrations of the high-concentration QCs and the low-concentration QCs averaged to obtain one measurement of high-concentration QC, and low-concentration QC for each run were evaluated using standard statistical probability rules [45].

Outcome variables

For each neonicotinoid measured in urine samples, there is a limit of detection. Every analyte was categorized as ‘above or at detection limit’ and ‘below detection limit’. We defined the outcome variable as a binary variable named neonicotinoid, set to present if there is the presence of any of the neonicotinoids in the urine sample, and absent if no neonicotinoids were detected.

Exposures and Covariates

Demographics comprising age, gender, race/ethnicity, education level, household income. Age was grouped in three categories: 18 to less than 45 years old, 45 to less than 65 years old, and 65 years old and above [46]. Race and ethnicity were grouped in four categories Non-Hispanic White, Non-Hispanic Black, Hispanic, and other race including multiracial. Education level was divided into three categories, 11^{th} grade and less, high school graduate, and some college degree or college graduate. We categorized the household income into four categories, 0 to $19,999, $20,000 to $54,999, $55,000 to $99,999, and $100,000 and more. Pesticide-use variables were if the participant had used any chemical products to control pests in the household or in the yard within the previous seven days. There were several questions about smoking and alcohol use. We categorized the smoking status into never smoked, former smoker, and smokers. To enquire about the smoking status, it was asked if the participant have ever smoked 100 cigarettes in their lifetime. Those who reported never having smoked 100 cigarettes were asked about ever smoking a cigarette even once. Non-smokers were those who responded no to both questions. Participants who responded yes to any of these two questions were asked if they now smoke cigarettes. Current smokers were those who reported having smoked 100 cigarettes or having smoked at least once and who responded yes to the follow-up question. Former smokers are participants who reported having smoked cigarettes and responded no to the follow-up question, ‘do you now smoke’. Two categories of alcohol users were identified. Those who had at least 12 alcohol drinks per one year and those who ever had 4, 5, or more drinks every day.

Data analysis

The data analysis was conducted using SAS 9.4. Only the participants who participated in the neonicotinoids study were kept from the demographic dataset for the analysis. Logistic regression models were used to estimate prevalence odds ratio and corresponding 95% confidence intervals (CIs) for the association between the detection of neonicotinoids, and the covariates. Two multivariable models were performed to estimate the prevalence odds ratios, the first model included only the demographic variables, and the second model adjusted for demographics, pesticide use, smoking, and alcohol-use. To account for the complex survey design (including oversampling), survey non-response, and post-stratification adjustment to match total population counts from the Census Bureau, weights were created in NHANES. Following the recommendations of the CDC, we performed weighted analysis for the results to be representative of the US civilian noninstitutionalized resident population [47].

Chapter 4 – Results

• Study population

There were three databases that were used for this study. Demographics dataset, neonicotinoids from the laboratory dataset, and pesticide, alcohol, and smoking data from the questionnaire dataset. Merging these three datasets we obtained a sample size of 1510. When removing the non-respondents to smoking status, the sample size was 1387, and finally, after removing non-responders to alcohol use, we obtained a sample size of 901.

• Descriptive data

The mean age of the population was 48.4 years old (SD = 18.3) with a minimum at 18 and maximum at 80. The 25th percentile was 33 years old and the 75th percentile, 63 years old.

Table 1. Weighted Distribution of demographic and behavioral data NHANES 2015 – 2016, (n = 1510)

Modalities	Frequency	Percent
Age		
18 – 44	662	44.8
45 – 65	502	33.8
≥ 65	346	21.4
Gender		
Male	725	48.0
Female	785	52.0
Race/Ethnicity		
Hispanic	468	14.7
Non-Hispanic White	499	64.9
Non-Hispanic Black	316	10.9
Other Race - Including Multi-Racial	227	9.5
Education		
11th grade or less	328	12.5
High school graduate	322	20.9
College degree or graduate	797	66.6
Annual Household Income		
0 – 19,999	304	11.7
20,000 – 54,999	569	33.6
55,000 – 99,999	300	25.5
100,000 and over	254	29.1
Marital status		
Married or Living with partner	856	64.3
Widowed or Divorced or Separated	310	18.0
Single	283	17.7
Smoking status (n=1387)		
Current smokers	280	19.7
Former smokers	658	52.4
Never smoked	449	28.0
Pesticide-use (n=1387)		
Pesticide used in home		
Yes	146	10.0
No	1241	90.0
Pesticide used in lawn		
Yes	92	8.7
No	1295	91.3
Alcohol (n=901)		
Had at least 12 alcohol drinks/1 year	798	92.4
Never Had at least 12 alcohol drinks/1 year	103	7.6
Ever have 4/5 or more drinks every day	133	13.0
Never have 4/5 or more drinks every day	768	87.0

n = population; NHANES = National Health and Nutrition Examination Survey

Regarding pesticide use, 146 participants (10.5%) declared having used chemical products in the past 7 days in their home to control fleas, roaches, ants, termites, or other insects, and 92 (6.6%) used chemical products used in their lawn or garden to kill weeds in the past 7 days.

Alcohol use. The participants who provided information on alcohol use in our study were n=902. The average number of days for alcohol drink in the past 12 months was 4 days (SD = 11.8). The maximum number of days drunk alcohol in the past year was 250 days (75% = 4 days, 99% = 22 days). The average number of alcohol drinks per day was 2.7 drinks per day during the past 12 months (SD = 2.4). The maximum number of drinks per day was 15 drinks (3^{rd} percentile = 3). One hundred and thirty-three participants (14.5%) declared having 4 or 5 or more drinks per the past year. The average number of days they had 4 or more drinks per day was 2.3 days (15.2).

• Outcome data

Four neonicotinoids were measured, imidacloprid, acetamiprid, clothianidin, thiacloprid, and two metabolites, 5-hydroxyimidacloprid and N-desmethylacetamiprid.

Table 2. Weighted distribution of neonicotinoids per detection limit in urine samples, age 18 years and older, NHANES 2015 - 2016 (n = 1510)

Analytes	≥ detection limit (%)	< detection limit (%)
Imidacloprid	63 (4.2)	1426 (95.8)
Acetamiprid	9 (0.6)	1491 (99.4)
Clothianidin	113(7.6)	1383 (92.4)
Thiacloprid	1 (0.1)	1484 (99.9)
5-Hydroxyimidacloprid	275 (19.4)	1142 (80.6)
N-Desmethylacetamiprid	476 (31.8)	1019 (68.2)
Neonicotinoid*	**697 (46.8)**	**813 (53.2)**

* new defined variable that combines all neonicotinoids detected
n = population; NHANES = National Health and Nutrition Examination Survey

The metabolites were the most detected compounds, 5-Hydroxyimidacloprid and N-Desmethylacetamiprid respectively in 69% and 22% of the cases (Figure 1).

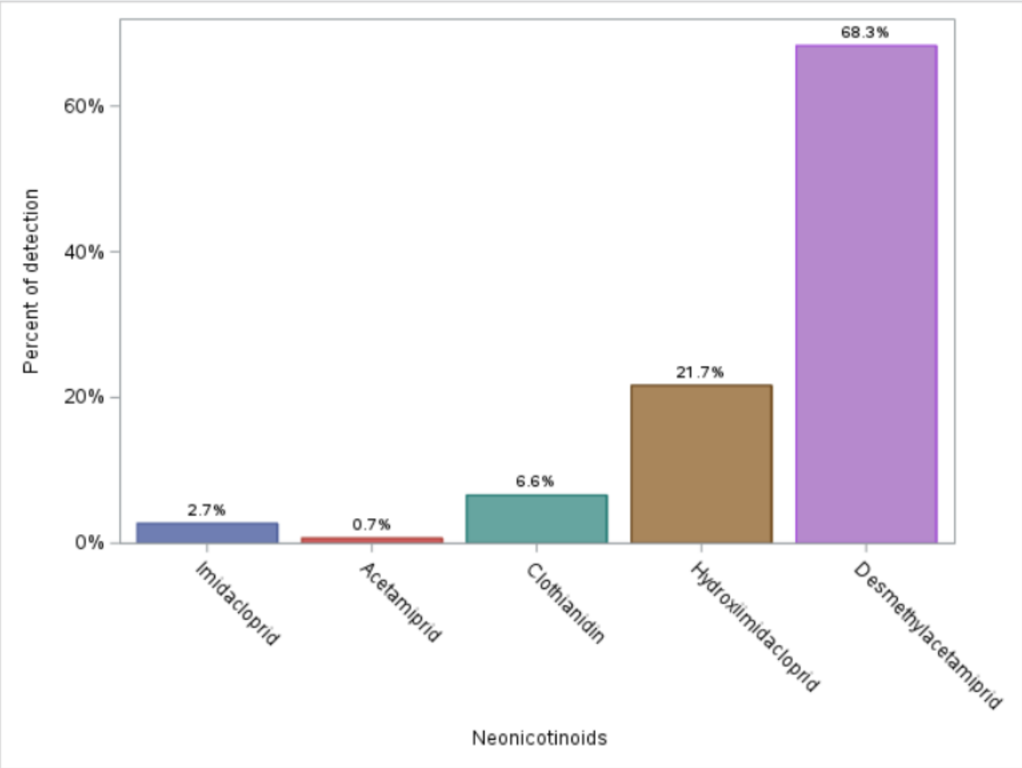


Figure 1. Percent of neonicotinoids detected in urine samples, NHANES 2015-2016

• Main results

We first performed a bivariate analysis to test the association between each covariate and the outcome. The results are presented in Table 3. Gender, race, education and smoking were associated with the detection of neonicotinoids.

When considering the full sample 2-year weight for the analysis, only marital status and smoking were associated with the neonicotinoids detection (Table 4).

Multivariate analyses were performed. The initial model included all the covariates unweighted (Table 5) and weighted (Table 6). The weighted multivariate analysis showed that only gender, education, and smoking were associated with urine neonicotinoids.

The prevalence odds ratio of neonicotinoids in participants aged 65 years old and above was 1.87 times higher than that of participants aged 18 years to 44 years, when adjusting for gender, marital status, race, education, income, alcohol use, and smoking. 95%CI = (1.04, 3.36) (Table 6).

The prevalence odds ratio of neonicotinoids in females was 1.67 times higher than that of males, after adjusting for age, marital status, race, education, income, alcohol use, and smoking. 95%CI = (1.12, 2.48) (Table 6).

The prevalence odds ratio of neonicotinoids in multiracial individuals was 1.71 times higher than that of non-Hispanic Whites, after adjusting for age, gender, marital status, education, income, alcohol use, and smoking. 95%CI = (1.08, 2.70) (Table 6).

Current smokers had 0.55 times lower prevalence ratio than that of non-smokers, after adjusting for age, gender, marital status, education, race, income, and alcohol use. 95%CI = (C0.35, 0.85) (Table 6).

Table 3. Prevalence ratios for the associations between neonicotinoids detection and socio-demographic and behavioral characteristics, NHANES 2015–2016.

	Prevalence Ratio	Confidence Interval
Socio-demographics (n = 1510)		
Age, years old		
18 – 44	1	
45 – 65	0.84	0.67, 1.06
≥ 65	0.83	0.63, 1.07
Gender		
Male	1	
Female	1.39	**1.13, 1.70***
Race		
Non-Hispanic Whites	1	
Non-Hispanic Black	1.25	0.94, 1.67
Hispanic	1.22	0.95, 1.57
Multiracial and other	1.81	**1.31, 2.48***
Marital status		
Married	1	
Divorced	0.77	0.59, 1.00
Single	1.01	0.77, 1.32
Education		
11th grade or less	1	
High school graduate	0.69	**0.51, 0.94**
College degree or graduate	1.02	0.79, 1.32
Income		
0 - 19,999	1	
20,000 - 54,999	1.03	0.77, 1.36
55,000 - 99,999	1.29	093, 1.77
100,000 and over	1.61	**1.15, 2.26***
Pesticide use (n=1387)		
Pesticide-use in household		
No	1	
Yes	1.10	0.78, 1.55
Pesticide-use in lawn		
No	1	
Yes	0.99	0.65, 1.52
Smoking (n=1387)		
Current smoker	0.47	**0.34, 0.64***
Former smoker	0.86	0.68, 1.10
Nonsmoker	1	
Alcohol (n=901)		
Had at least 12 alcohol drinks/1 year		
No	1	
Yes	1.11	0.74, 1.69
Ever have 4/5 or more drinks every day		
No	1	
Yes	0.70	0.48, 1.02

* significant association at level 0.05; n = population

Table 4. Weighted Prevalence ratios for the associations between neonicotinoids detection and socio-demographic and behavioral characteristics, NHANES 2015–2016.

	Prevalent Odds Ratio	95%CI
Socio-demographics (n = 1510)		
Age, years old		
18 – 44	1	
45 – 65	0.85	0.67, 1.06
≥ 65	1.01	0.71, 1.44
Gender		
Male	1	
Female	1.25	0.94, 1.66
Race		
Non-Hispanic Whites	1	
Non-Hispanic Black	1.07	0.77, 1.51
Hispanic	1.02	0.69, 1.50
Multiracial and other	**1.49**	**1.01, 2.18***
Marital status		
Married	1	
Divorced	0.68	0.49, 0.95
Single	1.02	0.71, 1.46
Education		
11th grade or less	1	
High school graduate	0.68	0.45, 1.03
College degree or graduate	1.02	0.63, 1.39
Income		
0 - 19,999	1	
20,000 - 54,999	0.86	0.58, 1.26
55,000 - 99,999	1.32	0.80, 2.18
100,000 and over	1.46	0.92, 2.33
Pesticide use (n=1387)		
Pesticide-use in household		
No	1	
Yes	0.88	0.60, 1.29
Pesticide-use in lawn		
No	1	
Yes	1.13	0.77, 1.66
Smoking (n=1387)		
Current smoker	0.59	**0.36, 0.96***
Former smoker	0.96	0.67, 1.39
Nonsmoker	1	
Alcohol (n=901)		
Had at least 12 alcohol drinks/1 year		
No	1	
Yes	1.11	0.75, 1.62
Ever have 4/5 or more drinks every day		
No	1	
Yes	0.67	0.38, 1.19

* significant association at level 0.05; n = population

Multivariate analysis

Table 5. Adjusted Prevalence ratios for the associations between neonicotinoids detection and socio-demographics, pesticide-use, smoking, and alcohol use NHANES 2015–2016 (n = 901).

	Prevalence Odds Ratio	Confidence Interval
Age, years old		
18 – 44	1	
45 – 65	0.96	0.68, 1.35
≥ 65	1.29	0.85, 1.97
Gender		
Male	1	
Female	1.60	**1.18, 2.16***
Marital status		
Married	1	
Divorced	0.81	0.55, 1.20
Single	1.05	0.71, 1.56
Race		
Non-Hispanic Whites	1	
Non-Hispanic Black	1.31	0.88, 1.95
Hispanic	1.27	0.87, 1.85
Multiracial and other	1.71	**1.08, 2.70***
Education		
11th grade or less	1	
High school graduate	0.62	0.38, 1.00
College degree or graduate	0.76	0.48, 1.21
Income		
0 - 19,999	1	
20,000 - 54,999	0.88	0.56, 1.37
55,000 - 99,999	1.22	0.74, 2.00
100,000 and over	1.26	0.74, 2.15
Pesticide use in household		
No	1	
Yes	1.20	0.74, 1.94
Pesticide use in lawn		
No	1	
Yes	0.79	0.45, 1.37
Smoking (n=1387)		
Nonsmoker	1	
Current smoker	0.55	**0.35, 0.85***
Former smoker	1.09	0.76, 1.58
Had at least 12 alcohol drinks/1 year		
No	1	
Yes	0.77	0.48, 1.22
Ever have 4/5 or more drinks every day		
No	1	
Yes	0.94	0.61, 1.44

* significant association at level 0.05; n = population

Table 6. Weighted and Adjusted Prevalence ratios for the associations between neonicotinoids detection and socio-demographics, pesticide-use, smoking, and alcohol use NHANES 2015–2016 (n = 901).

	Prevalence Ratio	Confidence Interval
Age, years old		
18 – 44	1	
45 – 65	0.99	0.63, 1.56
≥ 65	1.87	**1.04, 3.36***
Gender		
Male	1	
Female	1.67	**1.12, 2.48***
Marital status		
Married	1	
Divorced	0.88	0.51, 1.50
Single	1.15	0.71, 1.86
Race		
Non-Hispanic Whites	1	
Non-Hispanic Black	1.23	0.80, 1.90
Hispanic	1.15	0.74, 1.79
Multiracial and other	1.54	0.84, 2.84
Education		
11th grade or less	1	
High school graduate	0.50	**0.26, 0.96***
College degree or graduate	0.56	0.31, 1.02
Income		
0 - 19,999	1	
20,000 - 54,999	0.89	0.53, 1.49
55,000 - 99,999	1.55	0.74, 2.86
100,000 and over	1.39	0.73, 2.62
Pesticide use in household		
No	1	
Yes	1.08	0.58, 2.01
Pesticide use in lawn		
No	1	
Yes	0.61	0.28, 1.34
Smoking (n=1387)		
Nonsmoker	1	
Current smoker	0.61	0.34, 1.10
Former smoker	1.11	0.68, 1.79
Had at least 12 alcohol drinks/1 year		
No	1	
Yes	0.77	0.42, 1.40
Ever have 4/5 or more drinks every day		
No	1	
Yes	0.90	0.52, 1.55

* significant association at level 0.05; n = population

Table 7. Weighted and Adjusted Prevalence ratios for the associations between neonicotinoids detection and socio-demographics, NHANES 2015–2016 (n = 1510).		
	Prevalence Ratio	Confidence Interval
Age, years old		
18 – 44	1	
45 – 65	0.97	0.61, 1.51
≥ 65	1.86	**1.04, 3.32***
Gender		
Male	1	
Female	1.64	**1.11, 2.43***
Marital status		
Married	1	
Divorced	0.89	0.53, 1.49
Single	1.18	0.73, 1.90
Race		
Non-Hispanic Whites	1	
Non-Hispanic Black	1.21	0.79, 1.87
Hispanic	1.15	0.74, 1.77
Multiracial and other	1.51	0.79, 2.88
Education		
11th grade or less	1	
High school graduate	0.51	**0.27, 0.96***
College degree or graduate	0.57	0.31, 1.03
Income		
0 - 19,999	1	
20,000 - 54,999	0.89	0.53, 1.50
55,000 - 99,999	1.53	0.84, 2.78
100,000 and over	1.39	0.74, 2.61
Smoking		
Nonsmoker	1	
Current smoker	0.61	0.35, 1.06
Former smoker	1.08	0.68, 1.74
Had at least 12 alcohol drinks/1 year		
No	1	
Yes	0.78	0.42, 1.42

* significant association at level 0.05; n = population

Chapter 5 – Discussion

• Key results

Neonicotinoids were effectively detected in the urine of 46.8% of the sub-study sample in the participants in the NHAENES study. The N-Desmethyl-acetamiprid, the principal metabolite of acetamiprid, was the most detected, representing 68.3%, followed by the 5-Hydroxyimidacloprid, the principal metabolites of imidacloprid, 21.7%. Thiacloprid was the least detected neonicotinoid in only one case.

After adjusting for marital status, income, and alcohol use, the presence of neonicotinoids in urines was significantly associated with age, gender, and education.

After adjusting for marital status, income, and alcohol use, pesticide use, alcohol use, smoking status, the detection of neonicotinoids in urine was associated with age, gender, and education.

• Interpretation

The findings of this study are similar to the findings in the study by Harada et al. in Japan[13]. In their study, they administered by oral route deuterium-labeled neonicotinoid and measured their excretion in urine. They detected desmethyl-acetamiprid, dinotefuran, and imidacloprid in half of the sample. Thiacloprid was not detected[13]. In our study, the metabolite of imidacloprid, the 5-Hydroxyimidacloprid was detected more than the unmetabolized form detected in 2.7%. Harada et al. conducted the measurement within 24 hours after ingestion. In our case, the source of the exposure to the neonicotinoids is unknown. We can at least infer that the time between exposure and the measurement matters in detecting the level of neonicotinoids in human's organism.

Fruit and vegetable consumptions were found to be associated with neonicotinoids by Harada and al. In the US, Acetamiprid, the most detected neonicotinoid, is used in the growing of cotton, leafy vegetables, pome fruits, grapes. This finding suggests that fruit and vegetables are a potential source of exposure to neonicotinoids. Ueayama et al.[48] suggested fruit and vegetables but also drinking water as the sources of exposure to neonicotinoids.

Thiacloprid was detected in only one case in the sample. Casida[49] suggests that it is due to the fact that thiacloprid is easily metabolized in human organism. Another reason can be that this compound is growing cotton, therefore is less likely to get to human organism if food consumption is the route of exposure. Thompson et al.[2] suggested that the main source of exposure to thiacloprid was inhalation.

• Strengths and limitations

This study has the merit of showing the reality of the presence of neonicotinoids in humans' sample of the US general population. It suggests that the neonicotinoids most commonly detected are acetamiprid and imidacloprid.

This study also has some limitations. The unavailability of occupation data is the principal limitation. Because it is a cross-sectional study, there is a certain limitation in the measure of association that could be estimated. Whether the presence of neonicotinoids in people's body is associated with a specific health condition remains a question this study doesn't answer.

• Generalizability

The NHANES data in a nationwide study, and our results were weighted considering sampling biases. The results are therefore representative of the US civilian noninstitutionalized resident population aged 18 years old and above. It is not generalizable to the population of other countries.

Cited Literature Bibliography

1. Zhang D, Lu S. Human exposure to neonicotinoids and the associated health risks: A review. *Environ Int*. 2022;163:107201. doi:10.1016/j.envint.2022.107201

2. Thompson DA, Lehmler HJ, Kolpin DW, et al. A critical review on the potential impacts of neonicotinoid insecticide use: current knowledge of environmental fate, toxicity, and implications for human health. *Environ Sci: Processes Impacts*. 2020;22(6):1315-1346. doi:10.1039/C9EM00586B

3. Ensley SM. Chapter 40 - Neonicotinoids. In: Gupta RC, ed. *Veterinary Toxicology (Third Edition)*. Academic Press; 2018:521-524. doi:10.1016/B978-0-12-811410-0.00040-4

4. European Union high court nixes neonicotinoid exemptions. Chemical & Engineering News. Accessed March 9, 2023. https://cen.acs.org/environment/pesticides/European-Union-high-court-nixes/101/web/2023/01

5. Douglas MR, Tooker JF. Large-scale deployment of seed treatments has driven rapid increase in use of neonicotinoid insecticides and preemptive pest management in US field crops. *Environ Sci Technol*. 2015;49(8):5088-5097. doi:10.1021/es506141g

6. Chen M, Tao L, McLean J, Lu C. Quantitative Analysis of Neonicotinoid Insecticide Residues in Foods: Implication for Dietary Exposures. *J Agric Food Chem*. 2014;62(26):6082-6090. doi:10.1021/jf501397m

7. Houchat JN, Cartereau A, Le Mauff A, Taillebois E, Thany SH. An Overview on the Effect of Neonicotinoid Insecticides on Mammalian Cholinergic Functions through the Activation of Neuronal Nicotinic Acetylcholine Receptors. *International Journal of Environmental Research and Public Health*. 2020;17(9):3222. doi:10.3390/ijerph17093222

8. Phogat A, Singh J, Kumar V, Malik V. *Berberine Mitigates Acetamiprid Induced Hepatotoxicity and Inflammation via Regulating Endogenous Antioxidants and NF-KB/TNF-α Signaling in Rats*. In Review; 2022. doi:10.21203/rs.3.rs-2166138/v1

9. Stewart SD, Lorenz GM, Catchot AL, et al. Potential Exposure of Pollinators to Neonicotinoid Insecticides from the Use of Insecticide Seed Treatments in the Mid-Southern United States. *Environ Sci Technol*. 2014;48(16):9762-9769. doi:10.1021/es501657w

10. Moffat C, Pacheco JG, Sharp S, et al. Chronic exposure to neonicotinoids increases neuronal vulnerability to mitochondrial dysfunction in the bumblebee (Bombus terrestris). *FASEB J*. 2015;29(5):2112-2119. doi:10.1096/fj.14-267179

11. Hladik ML, Kolpin DW, Kuivila KM. Widespread occurrence of neonicotinoid insecticides in streams in a high corn and soybean producing region, USA. *Environmental Pollution*. 2014;193:189-196. doi:10.1016/j.envpol.2014.06.033

12. US EPA Search. Accessed March 31, 2023. https://search.epa.gov/epasearch/?querytext=neonicotinoids&areaname=&areacontacts=&areasearchurl=&typeofsearch=epa&result_template=#/

13. Harada KH, Tanaka K, Sakamoto H, et al. Biological Monitoring of Human Exposure to Neonicotinoids Using Urine Samples, and Neonicotinoid Excretion Kinetics. *PLOS ONE*. 2016;11(1):e0146335. doi:10.1371/journal.pone.0146335

14. Shamsi M, Soodi M, Shahbazi S, Omidi A. Effect of Acetamiprid on spatial memory and hippocampal glutamatergic system. *Environ Sci Pollut Res*. 2021;28(22):27933-27941. doi:10.1007/s11356-020-12314-6

15. Galdíková M, Holečková B, Šiviková K, Schwarzbacherová V, Koleničová S. Evaluating the genotoxic damage in bovine whole blood cells in vitro after exposure to thiacloprid. *Toxicology in Vitro*. 2019;61:104616. doi:10.1016/j.tiv.2019.104616

16. Liu Y, He QK, Xu ZR, et al. Thiamethoxam Exposure Induces Endoplasmic Reticulum Stress and Affects Ovarian Function and Oocyte Development in Mice. *J Agric Food Chem*. 2021;69(6):1942-1952. doi:10.1021/acs.jafc.0c06340

17. Öztaş E, Kara M, Boran T, et al. Cellular Stress Pathways Are Linked to Acetamiprid-Induced Apoptosis in SH-SY5Y Neural Cells. *Biology*. 2021;10(9):820. doi:10.3390/biology10090820

18. Li P, Ann J, Akk G. Activation and modulation of human α4β2 nicotinic acetylcholine receptors by the neonicotinoids clothianidin and imidacloprid. *Journal of Neuroscience Research*. 2011;89(8):1295-1301. doi:10.1002/jnr.22644

19. Rembert E. AltEn and Mead, One Year After the State Sued the Ethanol Plant. Nebraska Public Media. Published March 1, 2022. Accessed April 6, 2023. https://nebraskapublicmedia.org/en/news/news-articles/alten-and-mead-one-year-after-the-state-sued-the-ethanol-plant/

20. Gillam C. ‘There’s a red flag here’: how an ethanol plant is dangerously polluting a US village. *The Guardian*. https://www.theguardian.com/us-news/2021/jan/10/mead-nebraska-ethanol-plant-pollution-danger. Published January 10, 2021. Accessed April 6, 2023.

21. Rodriguez-Paar J. Health Effects of Neonicotinoids on an Agricultural Community. *Capstone Experience*. Published online December 1, 2022. https://digitalcommons.unmc.edu/coph_slce/230

22. Environmental Protection Agency (EPA). Imidacloprid Proposed Interim Registration Review Decision. Published online January 2020.

23. Fossen M. Environmental Fate of Imidacloprid. Published online April 2006.

24. Preliminary Monitoring Results of Imidacloprid and Carbaryl Applications for Glassy-winged Sharpshooter Control in Residential Areas of Imperial County. Published online 2002.

25. Romeh AA. Phytoremediation of Water and Soil Contaminated with Imidacloprid Pesticide by Plantago Major, L. *International Journal of Phytoremediation*. 2009;12(2):188-199. doi:10.1080/15226510903213936

26. Moza PN, Hustert K, Feicht E, Kettrup A. Photolysis of imidacloprid in aqueous solution. *Chemosphere*. 1998;36(3):497-502. doi:10.1016/S0045-6535(97)00359-7

27. Phillips PJ, Bode RW. *Concentrations of Pesticides and Pesticide Degradates in the Croton River Watershed in Southeastern New York, July-September 2000*. U.S. Geological Survey; 2002.

28. Pfeuffer RJ, Matson F. Pesticide surface water and sediment quality report. *South Florida Water Management District, West Palm Beach, FL*. Published online 2001.

29. Rouchaud J, Gustin F, Wauters A. Soil biodegradation and leaf transfer of insecticide imidacloprid applied in seed dressing in sugar beet crops. *Bulletin of environmental contamination and toxicology*. 1994;53:344-350.

30. Laurent FM, Rathahao E. Distribution of [14C] imidacloprid in sunflowers (Helianthus annuus L.) following seed treatment. *Journal of agricultural and food chemistry*. 2003;51(27):8005-8010.

31. Decourtye A, Lacassie E, Pham-Delègue MH. Learning performances of honeybees (Apis mellifera L) are differentially affected by imidacloprid according to the season. *Pest Management Science: formerly Pesticide Science*. 2003;59(3):269-278.

32. Kunkel BA, Held DW, Potter DA. Lethal and sublethal effects of bendiocarb, halofenozide, and imidacloprid on Harpalus pennsylvanicus (Coleoptera: Carabidae) following different modes of exposure in turfgrass. *Journal of Economic Entomology*. 2001;94(1):60-67.

33. Mullin CA, Saunders MCI, Leslie TW, Biddinger DJ, Fleischer SJ. Toxic and behavioral effects to Carabidae of seed treatments used on Cry3Bb1- and Cry1Ab/c-protected corn. *Environmental entomology*. Published online 2005. Accessed March 14, 2023. https://scholar.google.com/scholar_lookup?title=Toxic+and+behavioral+effects+to+Carabidae+of+seed+treatments+used+on+Cry3Bb1-+and+Cry1Ab%2Fc-protected+corn&author=Mullin%2C+C.A.&publication_year=2005

34. Kobori Y, Amano H. Effects of agrochemicals on life-history parameters of Aphidius gifuensis Ashmead (Hymenoptera: Braconidae). *Applied Entomology and Zoology*. 2004;39(2):255-261.

35. Duzguner V, Erdogan S. Acute oxidant and inflammatory effects of imidacloprid on the mammalian central nervous system and liver in rats. *Pesticide Biochemistry and Physiology*. 2010;97(1):13-18. doi:10.1016/j.pestbp.2009.11.008

36. Tomizawa M, Casida JE. Minor structural changes in nicotinoid insecticides confer differential subtype selectivity for mammalian nicotinic acetylcholine receptors. *British Journal of Pharmacology*. 1999;127(1):115-122.

37. Villar J, Gunier RB, Tshivuila-Matala COO, et al. Fetal cranial growth trajectories are associated with growth and neurodevelopment at 2 years of age: INTERBIO-21st Fetal Study. *Nat Med*. 2021;27(4):647-652. doi:10.1038/s41591-021-01280-2

38. US EPA - Pesticides - Fact Sheet for Acetamiprid. United States Environmental Protection Agency. Published March 15, 2002. Accessed March 15, 2023. https://www3.epa.gov/pesticides/chem_search/reg_actions/registration/fs_PC-099050_15-Mar-02.pdf

39. Arıcan EY, Gökçeoğlu Kayalı D, Ulus Karaca B, et al. Reproductive effects of subchronic exposure to acetamiprid in male rats. *Sci Rep*. 2020;10(1):8985. doi:10.1038/s41598-020-65887-0

40. US EPA - Pesticides - Fact Sheet for Clothianidin. United States Environmental Protection Agency. Published March 30, 2003. Accessed March 15, 2023. https://www3.epa.gov/pesticides/chem_search/reg_actions/registration/fs_PC-044309_30-May-03.pdf

41. Ohno S, Ikenaka Y, Onaru K, et al. Quantitative elucidation of maternal-to-fetal transfer of neonicotinoid pesticide clothianidin and its metabolites in mice. *Toxicology Letters*. 2020;322:32-38. doi:10.1016/j.toxlet.2020.01.003

42. Pang S, Lin Z, Zhang W, Mishra S, Bhatt P, Chen S. Insights Into the Microbial Degradation and Biochemical Mechanisms of Neonicotinoids. *Frontiers in Microbiology*. 2020;11. Accessed March 16, 2023. https://www.frontiersin.org/articles/10.3389/fmicb.2020.00868

43. US EPA - Pesticides - Fact Sheet for Thiacloprid. United States Environmental Protection Agency. Published September 26, 2003. Accessed March 15, 2023. https://www3.epa.gov/pesticides/chem_search/reg_actions/registration/fs_PC-014019_26-Sep-03.pdf

44. Baker SE, Serafim AB, Morales-Agudelo P, Vidal M, Calafat AM, Ospina M. Quantification of DEET and neonicotinoid pesticide biomarkers in human urine by online solid-phase extraction high-performance liquid chromatography-tandem mass spectrometry. *Anal Bioanal Chem*. 2019;411(3):669-678. doi:10.1007/s00216-018-1481-0

45. Caudill SP, Schleicher RL, Pirkle JL. Multi-rule quality control for the age-related eye disease study. *Statistics in Medicine*. 2008;27(20):4094-4106. doi:10.1002/sim.3222

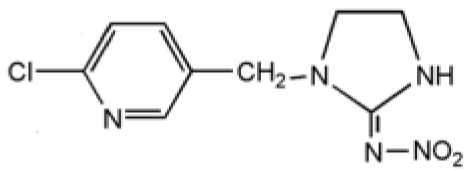
46. Chen J, Liang H, Tan Y, et al. Association of urinary iodine concentration with prediabetes/diabetes in adults: Analysis of the NHANES 2005–2016. *Journal of Trace Elements in Medicine and Biology*. 2023;77:127144. doi:10.1016/j.jtemb.2023.127144

47. NHANES Tutorials - Weighting Module. Accessed April 6, 2023. https://wwwn.cdc.gov/nchs/nhanes/tutorials/weighting.aspx

48. Ueyama J, Nomura H, Kondo T, et al. Biological monitoring method for urinary neonicotinoid insecticides using LC-MS/MS and its application to Japanese adults. *J Occup Health*. 2014;56(6):461-468. doi:10.1539/joh.14-0077-OA

49. Casida JE. Neonicotinoid Metabolism: Compounds, Substituents, Pathways, Enzymes, Organisms, and Relevance. *J Agric Food Chem*. 2011;59(7):2923-2931. doi:10.1021/jf102438c

Molecular Structure:



Chemical Formula: $C_9H_{10}ClN_5O_2$

Figure 2. Imidacloprid

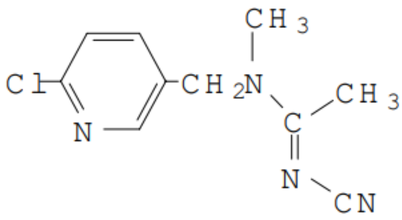


Figure 3. Acetamiprid

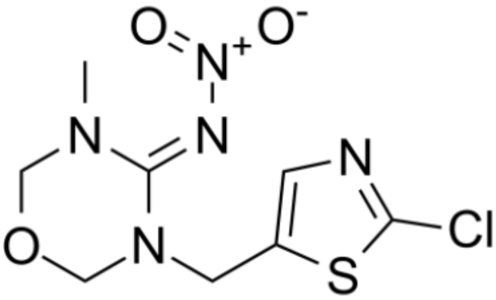


Figure 4. Thiamethoxan

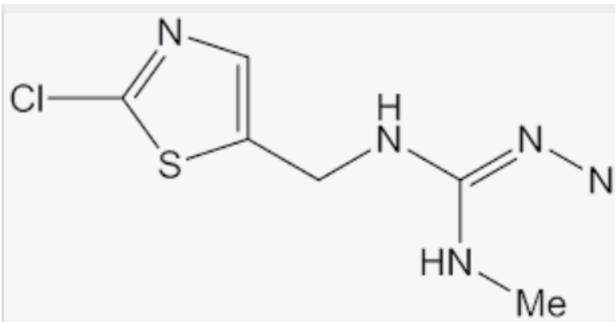


Figure 5. Clothianidin

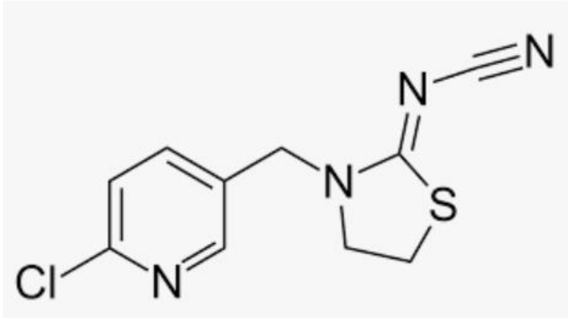


Figure 6. Thiacloprid

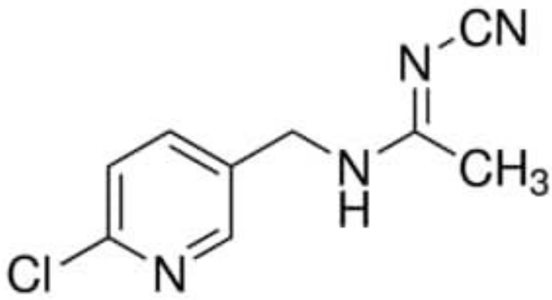


Figure 7. N-Desmethylacetamiprid

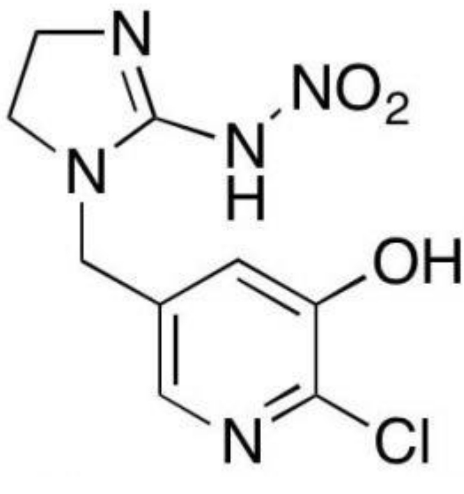


Figure 8. 5-Hydroxyimidacloprid

Biography & CV

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SUMMARY OF QUALIFICATIONS

- Over six years of data management and research experience.
- Five years in clinical care.
- Proficient in Microsoft Office, SPSS, SAS programming, and EPI Info, trained in GIS.
- Five years of experience in the district health department.

PROFESSIONAL EXPERIENCE

Epidemiology Educational Assistant **Fall 2022 to date**
UNMC COPH, Department of Epidemiology
Aided students in understanding epidemiology concepts and guided them to research through available resources to solve problems and better comprehend the materials.

Capstone experience, *UNMC COPH* **Ongoing**
Title: Association Between Socioeconomic and Behavioral Characteristics with Urinary Neonicotinoid Levels in the United States, 2015-2016
Working on NHANES data about neonicotinoids. Data cleaning, merging, and analysis

Public Health Intern **Fall 2022**
Central States Center for Agricultural Safety and Health (CS-CASH),
- Conducted a literature search on heat-related illnesses in Agricultural workers
- Produced evidence-based health-education materials on heat-related disease prevention for agricultural workers using the Feedyard-15 guidelines.

President and founder **Mar. 2020 – Present**
NGO DZIDZO, Lome, Togo
- Grant application for addressing various social health determinants of health
- Women empowerment by small businesses funding
- Water Sanitation and Hygiene project (WASH) implementation with a community lead approach

Keynote speaker, Manlius Pebble Hill Model United Nations Conference **Oct. 21, 2021**
Speech about the importance of fighting against poverty and its connection to health.

HIV/AIDS Medical officer **Jan. 2020 – Jul. 2021**
ACS Hospital, The Global Fund, Lome, Togo
- Managed HIV infection in children, adolescents & adults, and key populations & minorities;
- Performed planning, monitoring, and evaluation activities for the End AIDS in West Africa (#EAWA), a project of the President Emergency Program for AIDS Relief (PEPFAR) under the Family Health International (FHI 360) supervision
- Managed reproductive health issues (sexual and reproductive health rights, family planning, maternal & child, adolescent health, minorities' rights)

Chief Medical officer **Jun. 2017 – Jan. 2020**
Pagouda District Hospital and Ketao Social and Medical Center, Binah, Republic of Togo
- Implemented public health surveillance systems for reportable diseases according to the WHO's Integrated Disease Surveillance and Riposte (IDSR) guidelines.
- Trained and coordinated the health providers on reporting in the District Management Information System (DHIS2) software with advanced skills in extracting and analyzing reports
- Led monthly monitoring meetings at the district level and participated in quarterly regional health department monitoring meetings

EDUCATION

Fulbright Scholar, *US Department of State* **Current**
Master of Public Health in Epidemiology **Expected May 2023**
University of Nebraska Medical Center College of Public Health, Omaha, Nebraska
Relevant Coursework: Apply Epidemiology, Epidemiological Methods in Infectious Disease Outbreak Investigations, Epidemiologic Methods, Social Epidemiology, Biostatistics (I & II), SAS Programming, Public Health Leadership & Advocacy, Planning and Evaluation.

Medical Doctor **June 2016**
University of Lome Faculty of Health Sciences, Lome, Republic of Togo
Thesis title: Epidemiology, Diagnosis, and Treatment of Multidrug-Resistant Tuberculosis in Togo

VOLUNTEER EXPERIENCE

American Red Cross, Nebraska-Iowa Region **Since Aug. 2021**

- Trained in emergency response driving, response to disasters, and sheltering service/supervisor
- Responded to family fire: provided immediate support and enrollment of cases in the client intake system

NGO AIMES-AFRIQUE, Lome Togo **2014- 2016**

- Health education and awareness of non-transmitted diseases (Diabetes and Hypertension) in rural communities
- Worked with international multicultural teams with German NGOs AKTION-PITT and AERZTE Camp eV
- Medical consultation in the rural area

- UNMC Center for Global Health and Development member
- Secretary of Graduate and Professionals Representing Achievement, Diversity, and Service (GRADS)
- College liaison for the Student Alliance for Global Health (SAGH).

PRESENTATIONS AND PUBLICATIONS

- **Akliku, N.**, Samson, K. (2023, March 8). Mapping alcohol retail violations for Project Extra Mile [Oral Presentation]. Project Extra Mile's March Coalition Meeting, University of Nebraska Omaha, Omaha, NE, United States.
- Agbobli, Y.A., Konu, Y.R., Gbeasor-Komlanvi, F.A., Sadio, A.J., Tchankoni, M.K., Anani, J., **Akliku K.N.**, Bakoubayi A.W. & Ekouevi D.K. (2022). Prevalence and factors associated with burnout among healthcare workers during the COVID-19 pandemic in Togo, June 2021, *Archives of Environmental & Occupational Health*, https://doi.org.10.1080/19338244.2022.2042172
- **Akliku K.N.** (2021). Quality of life at workplace in workers of Campus Teaching Hospital of Lome. Faculty of Health Sciences, University of Abomey-Calavi
- **Akliku K.N.** (2016). Epidemiology, diagnosis, and treatment of multidrug-resistant tuberculosis in Togo. Faculty of Health Sciences, University of Lome

PROFESSIONAL AFFILIATIONS

- Member of the Council of State and Territorial Epidemiologists (CSTE)
- Member of the Public Health Association of Nebraska (PHAN)

SKILLS

- **Languages**: English, French, Ewe
- **Software**: SAS, GIS, SPSS, Epi Info, and Microsoft Office.