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Abstract

Second hand tobacco smoke exposure has been associated with neurobehavioral disorders among children. The objective of this study was to evaluate the relationship between second hand smoking and autism spectrum disorders (ASD) in children in the United States. Parent-reported postnatal tobacco exposure inside and outside the house and ASD diagnosis was examined in children younger than 12 years using the 2011-2012 National Survey on Children's Health. The physician diagnosis of current ASD as reported by the parents was used as the outcome variable. Univariate and multivariate logistic regression models adjusted for potential confounders were used to assess the associations. Of the 43,418 children, 23.6% had second hand smoke exposure, and 4.8% were exposed to tobacco smoke exposure inside the house (p-value <0.001). The mean age of children suffering from ASD in this study population was 6.5 years. In univariate logistic regression analysis, second hand tobacco exposure was associated with 36% greater odds of being diagnosed with ASD (p-value <0.001) and 39% greater odds of being diagnosed with ASD (p-value <0.001) if parents smoked inside the house. After adjusting for covariates, smoking (inside and outside the house) was not significantly associated with ASD risk in children. The prenatal, perinatal and other demographic factors are important predisposing factors for ASD in children and affect the relationship between second hand smoking and ASD.

Keywords: autism, pervasive developmental disorders, autism spectrum disorders, second hand smoking, risk factors

The Association between Autism Spectrum Disorders and Second Hand Tobacco Exposure

Autism spectrum disorder (ASD) is a range of complex neurodevelopmental disorders, characterized by social impairments, communication difficulties, and restricted, repetitive, and stereotyped patterns of behavior. Diagnostic and Statistical Manual of Mental Disorders 5th Edition (DSM 5) released in May 2013, redefined the autism spectrum to encompass the previous (DSM 4-TR) diagnoses of autism, Asperger's syndrome, pervasive developmental disorder not otherwise specified (PDD-NOS), childhood disintegrative disorder, and Rett's syndrome. Therefore, rather than categorizing the patients into distinct groups, the patients will be diagnosed in terms of severity of social and communication impairment, and restricted or stereotyped patterns of behavior.

ASD is diagnosed clinically based upon the deficits in reciprocal social interaction and communication. ASD is also associated with other co-morbid conditions like mental retardation, learning disabilities, epilepsy, tuberous sclerosis, anxiety disorders, depression, attention deficit hyperactivity disorder (ADHD), etc. The Autism Diagnostic Interview-Revised (ADI-R) and the Autism Diagnostic Observation Schedule (ADOS) are the diagnostic tools used for assessing suspected autistic children. The treatment of ASD is aimed at providing comprehensive behaviorally based educational intervention and treatment of associated co-morbid conditions. The exact cause of autism is not known but genes and the environment are thought to play an important role.

Purpose Statement

The purpose of this research project is to explore the relationship between the second hand tobacco exposure and the development of autism spectrum disorders (ASDs) in children. The relationship between tobacco exposure during fetal development and autism risk has been

studied and prenatal tobacco exposure is considered to be a risk factor for cognitive deficits in children. This study explored the effect of post natal second hand tobacco exposure on the development of autism and other autism spectrum disorders in children less than 12 years of age. Some related studies have reported that the apparent relationship between ASD and smoking could be due to the effect of confounding factors like socio-demographic characteristics. Hence, more extensive research is needed to explore the relationship between the two.

Literature Review

The ASDs are much more common than previously thought. The prevalence of ASDs has increased twenty-thirtyfold in recent years (Centers for Disease Control and Prevention (CDC), 2104). During 1960's and 1970's the prevalence of the disorder in Europe was around 1 in 2,500 children. According to CDC's Autism and Developmental Disabilities Monitoring (ADDM) Network report in April, 2014, 1 in 68 children suffer from ASDs, almost 30% higher than previous reported number (1 in 88) in 2012. The prevalence of ASDs on the whole has been estimated to be about 14.7 per 1000. Some regional differences have also been reported in the prevalence of ASD, the highest being in New Jersey (1 in 45 children) and lowest in Alabama (1 in 175 children) (CDC, n.d.). ASDs are prevalent in all racial, ethnic and socioeconomic groups (CDC, n.d.).

The ASDs are a huge public health concern. The lifespan of children suffering from ASD is reduced and most of these children die prematurely from core impairment or associated comorbidities. The costs associated with the care of such children are huge. According to CDC report in April, 2014, it has been estimated that it costs about $17,000 more per year to take care of a child with ASD ($21,000 more per year for those with more severe disease) than a child who does not have ASD (CDC, n.d.). Medical expenditures (including health care, education, ASD-

related therapy, family-coordinated services and caregiver time) for those with ASD are 4.1- 6.2 times greater than those without the ASD. Total societal costs of caring children with ASD were over 9 billion dollars in 2011. Additionally, intensive behavioral intervention costs are $40,000-$60,000 more per child per year in children with ASD (CDC, n.d.).

Many studies have been conducted to understand the etiopathogenesis of ASDs but studies have found inconsistent results. Data available on various demographic factors related to ASD like maternal age, social class, prenatal, perinatal and neonatal factors have shown variable results. The exact cause of autism is still unclear but genes and the environment are thought to play an important role (Schieve et al., 2012).

Genetics is one of the major risk factors for ASDs (Newschaffer, Croen, Daniels, Giarelli, & Grether, 2007; Hultman, Sparen, & Cnattingius, 2002; Zhang et al., 2010; Itzchak, Lahat, & Zachor, 2011). Autism has a high concordance rate in identical twins; if one sibling has ASD, the chance of developing ASD in the other siblings increases by 36-95%. ASDs are highly heritable disorders but there is only about 70% concordance in monozygotic twins which suggests that the non-genetic factors like environmental (prenatal and perinatal) factors might also play a major role in the development of ASDs (Itzchak et al., 2011). If parents have one child affected by ASD, there is 2%-18% chance of having a second child affected by ASD (CDC, n.d.). ASD is found more often in people with certain genetic or chromosomal conditions like Down syndrome, fragile X syndrome, tuberous sclerosis, etc. Numerous genes have been identified that are believed to be associated with impaired biologic mechanism in ASD.

Infection and immune dysfunction have been linked to the ASD etiology (Newschaffer et al., 2007). Studies are still ongoing to explore the association between the two. Dysregulation of both adaptive and innate immune systems are blamed as the autoantibodies to neural antigens,

immunoglobulins, inflammatory cytokines and other markers have been found in these children. Inconsistent results were obtained in studies related to prenatal exposure to viral infections like cytomegalovirus and rubella and development of ASD (Newschaffer et al., 2007). Vaccines have been blamed to lead to ASD but research in the area has not found any conclusive relationship between vaccines like MMR (Measles, Mumps and Rubella) and development of ASD in children.

Numerous neurotransmitters, peptides and growth factors have also been thought to play a causative role (Newschaffer et al., 2007). Abnormal levels of neuropeptides like Calcitonin Gene Related Peptide (CGRP) and Vasoactive Intestinal Peptide (VIP) and neurotrophins like Brain Derived Neurotrophic Factor (BDNF) and Neurotrophins 4/5 (NT4/5) have been reported in children with autism. Abnormal serotonin synthesis and abnormal levels of melatonin, oxytocin and vasopressin have also been seen in children suffering from ASD in some research studies (Newschaffer et al., 2007).

Endocrine factors have also been implicated in the etiology of ASD (Newschaffer et al., 2007). Abnormal levels of sex hormones especially testosterone and its precursors DHEA (dehydroepiandrosterone) and DHEA-S (dehydroepiandrosterone sulfate) have been reported. Maternal reproductive hormone dysregulation, hypothalamic/pituitary/adrenal (HPA) axis stress hormones and thyroid hormones are also an area of interest. Exposure to toxins (certain metals, other inorganic and organic compounds) has also been associated with the development of ASD. Higher rates of autism were reported in children whose mothers were exposed to drugs like thalidomide, valproic acid and misoprostol during pregnancy. Metals like lead and mercury are neurotoxic and have adverse neurodevelopmental outcomes in children and have adverse effects on children’s developing brain (Newschaffer et al., 2007).

Environment plays a very important role in the development of autism (Newschaffer et al., 2007; Hultman et al., 2002; Zhang et al., 2010; Williams, Helmer, Duncan, Peat, & Mellis, 2008; Croen, Grether, & Selvin, 2002; Kuzniewicz et al., 2014; Itzchak et al., 2011, Larsson et al., 2005). The environmental factors alter the genes and change their expression leading to the development of autism. Environmental exposures during prenatal, natal and post natal development can affect the growing brain of the baby and predispose to ASD. Maternal factors (maternal age, parity, maternal cigarette smoking during pregnancy, mother's country of birth, hypertensive diseases during pregnancy, uterine atony, pre-gestational and gestational diabetes, bleeding during pregnancy and cesarean delivery) and infantile factors (gestational age, birth weight, birth length, head circumference, multiple births, congenital malformations and Apgar scores [that determine how well the baby tolerated the birthing process]) have been seen to predispose a child to develop ASDs (Hultman et al., 2002). It has been postulated that the development of prenatal and neonatal asphyxia leading to intrauterine growth restriction may be the probable causative factors behind the development of neurodevelopmental disorders such as ASDs (Hultman et al., 2002).

Advanced parental ages have been seen to be associated with ASD (Newschaffer et al., 2007; Zhang et al., 2010; Sandin et al., 2012; Williams et al., 2008; Itzchak et al., 2011; Larsson et al., 2005). Studies have been conducted to find out the role played by the parental ages and results have been conflicting with some studies describing older maternal age as the risk factor while others reporting the role of advanced paternal age. In a study conducted by Itzchak et al. (2011), it was reported that the advanced parental age at birth of both genders was associated with ASD but the advanced maternal age was seen to have greater impact in the development of ASD. The studies supporting the role of advanced maternal age in ASD argue that with the

increasing maternal age, there is increase in the rate of chromosomal abnormalities, increase in obstetric complications (like uterine muscle dysfunction, reduced blood supply with age) and greater exposure to environmental toxins (Sandin et al., 2012).

The prevalence of ASDs has increased in recent years in the developing countries, coinciding with an increase in maternal age in these same countries, leading researchers to think about this association. It was postulated that with the increase in maternal age, the cumulated exposure to environmental toxins could result in DNA damage, germline mutations and global hypermethylation in germ cells (Sandin et al., 2012). These in turn, could lead to several types of developmental disorders in the offsprings. Also, older mothers are more likely to be highly educated or of higher socioeconomic status and hence have more access to the health care services; therefore more children (of these mothers) with ASD are diagnosed earlier (Bhasin & Schendel, 2007).

Socioeconomic status of the parents is also associated with the ASDs (Rai et al., 2012; Newschaffer et al., 2007; Bhasin & Schendel, 2007; Larsson et al., 2005). However, inconsistent results have been reported regarding the parental socioeconomic status in children suffering from ASDs. Many studies found that the rate of ASD is higher in families from higher socioeconomic status while few reported that the parental lower socioeconomic status was associated with the increased risk of ASD in the children. For a long time it was believed that autism was common in “sophisticated” families (Rai et al., 2012). Studies conducted in the United States found that the ASD was more common in higher socioeconomic status families, while studies in the countries with universal health care found the opposite to be true. In the United States, children from higher socioeconomic status have better access to medical services and hence are diagnosed and treated earlier. Rai et al. (2012), found out in their study conducted in Sweden, which has

universal health care, that the ASD was more common in children from families with lower household income and whose parents did manual jobs. Three measures of socioeconomic status were taken into consideration in this study; family income, occupation and education. Those children whose parents had manual and unclassified occupations, and had lower prenatal household income were found to have higher risk of ASD. Level of parental education was seen to have a minimum association with ASD in this study. It is believed that the social stress associated with lower socioeconomic status in the mother during prenatal and perinatal period plays a role in the development of ASD. Also, the exposure to environmental toxins like alcohol and tobacco might also play a role in the development of ASD in children of mothers belonging to lower socioeconomic status (Rai et al., 2012).

Gender of the child plays a great role in ASD (Newschaffer et al., 2007; Bhasin & Schendel, 2007; Croen et al., 2002). All the studies related to ASD have found the disorder to be more prevalent in boys than girls, with boys affected 5 times more than girls (CDC, n.d.). A recent report by the CDC (April, 2014) says that 1 in 42 boys and 1 in 189 girls are affected with this neurodevelopmental disorder in the United States (CDC, n.d.). The male to female ratio is not constant and changes with the presence or absence of mental retardation and dysmorphic features. In the absence of mental retardation, the ratio is even higher but in the presence of mental retardation the sex ratio (male to female) drops to 2:1. In the presence of dysmorphic features, the male to female ratio is lower (Newschaffer et al., 2007).

Mental status of the mother also plays a role in the development of ASD in children (Zhang et al., 2010; Larsson et al., 2005). A study conducted in China reported that the maternal unhappy emotional state during pregnancy is significantly associated with autism (Zhang et al., 2010). Emotional nervousness, anxiety and depression were more common during pregnancy in

mothers of children suffering from autism. Women with psychiatric disorders (especially schizophrenia) often have adverse pregnancy outcomes. Social and environmental stressors during pregnancy are associated with increased risk of ASD. The increase in levels of stress hormones (such as adrenalin) in the mother leads to vasoconstriction that in turn leads to placental vasoconstriction which may affect fetal cerebral blood flow or directly affect fetal hormone levels leading to abnormal brain development (Zhang et al., 2010). Parents of autistic children are more likely to have schizoid personality traits. Autistic children with parents suffering from one or more psychiatric disorder are more likely to be diagnosed than children of parents who do not have a psychiatric disorder.

Prematurity (birth before 37 weeks gestational age) is another risk factor for ASD (Leavey, Zwaigenbaum, Heavener, & Burstyn, 2013; Kuzniewicz et al., 2014). The extremely preterm infants (born before 28 weeks gestation) have higher rates of neurodevelopmental and physiological impairments than children who are born at term. The risk of ASD increases three-fold in infants born before 27 weeks (Kuzniewicz et al., 2014). The intracranial hemorrhage and high frequency ventilation, found more often in children who are born before 34 weeks are associated with a higher prevalence of ASD (Kuzniewicz et al., 2014). Hence, each additional week of gestation decreases the risk of ASD. Further, the small for gestational age is also associated with increased risk of ASD.

Low birth weight (less than 2500 grams) and very low birth weight (less than 1500 grams) is a risk factor for many neurodevelopmental disorders including ASD (Larsson et al., 2005; Itzchak et al., 2011). Low birth weight is usually an indicator of fetal growth problems that may arise from a wide variety of causes. Studies have documented that low birth weight

with or without prematurity is associated with a two-fold increased risk of ASD (Itzchak et al., 2011).

Racial differences have also been noted in the prevalence of ASDs (Newschaffer et al., 2007; Schieve et al., 2012; Pedersen et al., 2012). Research studies show variable results when the race of the child is concerned. Some studies found a higher prevalence in children with African-American mothers and lower in children with Mexican born mothers, with not much difference in the prevalence rates between white, Asian and US born Hispanic mothers (Newschaffer et al., 2007). Other studies found rates of autism to be comparable in white and African American children and lower in Hispanic children. Pedersen et al. (2012) reported that the prevalence of ASD in Hispanic children is much higher than previously reported. It was argued that the previous studies that showed lower prevalence in Hispanic population had small sample sizes. Also, the Hispanic community's access to health care facilities was very limited and many children from Hispanic community did not get properly diagnosed. Besides the language barrier, these children were more likely to receive care from under-resourced health care facilities, had lesser chances of having proper medical insurance and also, physicians were less likely to refer children from minority communities to specialists. The increasing prevalence of ASD in Hispanic community can also be attributed to the increasing awareness about ASD and an associated increase in community diagnosis (Pedersen et al., 2012)

On the other hand, Bhasin and Schendel (2007) reported that the prevalence of autism is significantly higher in African American children and these children tend to have more severe forms of the disease than white children. This was explained by the socioeconomic differences. African-American children are diagnosed at a later age, usually at school, because of the limited access of these children to health care services in the community. Also, the physicians might

underestimate the child's ability during testing because of the differences in the testing results (Bhasin & Schendel, 2007).

Immigrant status of the parents is also a potential risk factor for development of ASDs (Newschaffer et al., 2007; Schieve et al., 2012). Findings have been inconsistent when the immigrant status of the parents is concerned. Some studies report that the rate of autism increases if one of the parents is an immigrant. On the contrary, a study reported that the children of mothers who emigrated from Mexico were less likely to have autism than children born to US-born Hispanic mothers (Newschaffer et al., 2007). In a study conducted by Schieve et al. (2012), it was seen that as compared to non-Hispanic white children with US-born parents, the prevalence of ASD is lower in Hispanic children with 2 foreign-born parents and higher in Hispanic children with 2 US-born parents. This was explained by the fact that the Hispanic children with two foreign born parents are less likely to be diagnosed with ASD because they have language barriers, are less likely to seek medical care and rely more on folk-remedies and have stigma associated with mental health conditions. The physicians also lack understanding about Latino culture which also hinders provision of optimal health care services to Hispanic population.

Other factors like alcohol consumption by the mother and exposure to hazardous air pollutants also predispose the children to ASD (Newschaffer et al., 2007). Alcohol plays a role in autism risk by being a teratogen and also due to linked genetic predisposition to both autism and alcoholism. A number of studies have reported a higher incidence of alcoholism among family members of children with autism than among family members of controls (Newschaffer et al., 2007). Studies have also found an association between increased rates of ASD in children living in areas with higher levels of hazardous air pollutants (Newschaffer et al., 2007).

No study has been conducted to find an association between ASD and post natal second hand tobacco smoke exposure. But, the relationship between tobacco smoke exposure during fetal development and ASD risk has been studied. Prenatal tobacco smoke exposure is considered a risk factor for the cognitive deficits in children (Zhang et al., 2010; James, 2013; Kalkbrenner et al., 2012; Tran et al., 2013; Lee et al., 2012). Fetal hypoxia and modulation of neurotransmitter systems via nicotinic acetylcholine receptors due to tobacco exposure is thought to be the potential mechanism of development of autism and related disorders (Kalkbrenner et al., 2012). Second hand smoke includes polycyclic aromatic hydrocarbons, metals, and other chemicals with known adverse health effects, which may cause fetal hypoxia and affect fetal brain development (Zhang et al., 2010). The compounds in smoke serve as co-factors with genetic or other environmental hazards.

The relationship between the ASD and maternal smoking has been inconsistent. Tran et al. (2013) reported that if mother smoked during the entire duration of pregnancy, it was associated with increased risk of pervasive developmental disorders. The effect was not seen if the mother smoked only during the first trimester. The study reported that the cigarette smoking increases the risk of developing ASD by causing infarcts and calcification in the placenta and by causing utero-placental vasoconstriction (Tran et al, 2013). Some studies have found the relationship between higher functioning ASDs and maternal smoking but not between lower functioning (more severe) ones (Kalkbrenner et al., 2012).

James (2013) suggested that the increase in maternal serum levels of testosterone in response to stress could be related to ASD in children whose mothers smoked during pregnancy. Other studies have reported that the apparent relationship between ASD and smoking could be due to the effect of confounding factors like maternal education, maternal race, marital status,

socio-demographic characteristics, maternal mental health before childbirth (Lee et al., 2012; Klekbrenner et al., 2012, Tran et al., 2013).

National Survey of pregnant adult women reported that 20.4% women in the United States smoke tobacco products during pregnancy (Cornelius & Day, 2000). Compared with African-American and Hispanic women, white women have a greater tendency to smoke during pregnancy and also smoke more frequently than women of other races and ethnicities (Cornelius & Day, 2000). Women who smoke during pregnancy are usually single, have less education, lower socioeconomic status and are less likely to go for prenatal visits than women who do not smoke during pregnancy. Approximately two-thirds of women who smoke before pregnancy continue to smoke even during pregnancy and the amount of smoking does not decrease as the pregnancy progresses. Women who smoke during pregnancy continue to do so even after pregnancy (Cornelius & Day, 2000). Hence, the children born to such women are exposed to hazards of tobacco both, during and after delivery, and suffer from the developmental problems associated with cigarette smoking.

Approximately 7.6% children live in households where someone smokes inside the house exposing these children to many adverse health outcomes like respiratory problems, Sudden Infant Death syndrome, ear problems, reduced hearing and language problems, frequent and severe asthma attacks and cognitive, behavioral and developmental disorders, and reduced IQ scores (Kabir, Connolly, & Alpert, 2011; Cornelius & Day, 2000). A study exploring the relationship between second hand cigarette smoke and neurobehavioral disorders among children in the United States found out that children exposed to second hand cigarette smoke during childhood had two times higher risk of developing neurobehavioral disorders than children who did not have second hand smoke exposure (Kabir et al., 2011). The parent reported need of

treatment or counseling for emotional or behavioral problems was also much higher in children who were exposed to second hand cigarette smoke than children without second hand cigarette smoke exposure (Kabir et al., 2011).

Due to widespread implementation of laws that prohibit smoking in public places, the exposure to second hand smoke has declined tremendously. However, second hand smoke exposure decreased more for adults than children and adolescents. In case of children and adolescents, the exposure to second hand tobacco smoke occurs inside the house which indicates that prohibiting smoking in public places only has lesser impact on reducing the overall exposure of children to second hand tobacco exposure (Marano, Schober, Brody, & Zhang, 2009). Among children aged 3-19 years the prevalence of second hand tobacco smoke exposure is 17.8% (Marano et al., 2009). A serum cotinine level in non-smokers is a tool to determine the second hand smoke exposure in the previous 2-3 day period. Serum cotinine levels have been found to be greater in children who live in households with lower family income than in households with higher family income. Also, the cotinine levels are higher in children in households where the parents/guardians are less educated (high school education or less) than among children in households with higher education (Marano et al., 2009). The study also showed the levels of serum cotinine to be lower in Mexican-Americans as compared to non-Hispanic white and not significantly different between non-Hispanic whites and non-Hispanic blacks.

Due to lack of available data regarding the association between the second hand tobacco smoke exposure postnatally and ASD, the role of second hand tobacco exposure after birth as an etiologic factor for ASD is still uncertain. The association between second hand cigarette smoke exposure in children after birth and ASD could be due to the effect of confounding factors and hence, more extensive research is needed to explore the relationship between the two. The

present study aims to find out the association between second hand smoking and ASD in children controlling for confounders like age of the child, gender of the child, maternal education, maternal age, socioeconomic status and other potential confounders using data from the National Survey of Children's Health, 2011/2012.

Methods

The data for this study were taken from the 2011/12 National Survey of Children's Health (NSCH). This survey was conducted by the US Centers for Disease Control and Prevention, the Data Resource Center (DRC) (a project of the Child and Adolescent Health Measurement Initiative at Oregon Health and Science University) sponsored by the Maternal and Child Health Bureau, Health Resources, and Services and Administration. The purpose of the survey was to provide national and state specific prevalence estimates for a range of children's health and well-being indicators. Out of 95,677 children included in the survey, 43,418 (45.4%) were selected for this analysis for whom the data for ASD, second hand smoking and other demographic variables were complete.

Sampling Procedures

NSCH data was collected by randomly dialing telephones to identify households with one or more children less than 18 years of age. The information was gathered through a questionnaire. Among the household, the parent or guardian who was the most well informed about the child's or the children's health was asked questions. Multiple call back attempts were made to reach the family, if not available. Questions were asked about the demographic characteristics, child's health and functional status, health insurance coverage, health access and utilization, medical home, early childhood, middle childhood and adolescence, family

functioning, parental health, neighborhood and community characteristics and additional demographics.

Primary Outcome and Exposure Variables

Autism diagnosis (Yes/no) was selected as the outcome variable and was derived from the question, “Does the child currently have autism, Asperger’s disorder, childhood pervasive development disorder, or other autism spectrum disorder?” The exposure variable was tobacco exposure which was derived from the questions, “Does anyone living in your household use cigarettes, cigars or pipe tobacco”? and “Does anyone smoke inside child’s home”? The reply to this question was coded as 1) no (referent); 2) yes, but not inside the house; 3) yes, inside the house.

Covariates

Variables used as covariates were- mother’s mental health status (excellent(referent), good, fair/poor), gender (female (referent) and male), child’s age and age group (less than 2 years (referent), 3-5 years, 6-8 years, and 9-11 years), primary language (English (referent) or another language), birth weight (normal (referent) or less than 2500 grams), prematurity (no (referent) or yes), socioeconomic status (measured by how hard it was to meet basic needs of food and housing (very often or somewhat often (referent)/ not very often/ never), race and ethnicity (Hispanic (referent), white non-Hispanic, black non-Hispanic, others), mother’s educational level (high school or less and more than high school (referent), mother’s age (20 or younger (referent), 21-35 years, 36-58 years and 59 or older), family structure (two parent - biological or adopted (referent), two parent - step family, single mother - no father present, other family type) and generational status of household to determine the foreign status of mother

and/or father (first generation household (referent), second generation household and third or higher generation household).

Statistical Analyses

IBM SPSS version 22 was used for the analyses of the data. Descriptive analysis was performed on all the variables selected (both for the outcome (autism) and exposure (smoking). Logistic regression (unadjusted and adjusted) was performed to determine the association between exposure and outcome variable after adjusting for covariates. ASD diagnosis was the dependent variable. Second hand smoke categories were the independent variables. Model building began with (first model) running univariate logistic regression analysis for all covariates with ASD as outcome variable. In second multivariate model, after entering second hand smoking, age, was added. In the third model, in addition to age and second hand smoking, gender was included. In the fourth model, besides exposure variable (smoking), age and gender, race/ethnicity was included. The fifth model included the socioeconomic status in addition to the previous covariates. In the sixth model, birth weight and prematurity were entered in addition to smoking, age, gender, race/ethnicity and socioeconomic status. The seventh model incorporated the mother's characteristics like mother's age, mother's educational level and mother's mental status in addition to the variables in the earlier models. In the eighth model, all the variables were added. In the last model (ninth), only the variables that were significantly associated with the outcome variable were included (age, gender, race/ethnicity, socioeconomic status, mother's mental status, birth weight, and primary language)

Results

Out of those children who were diagnosed with ASD, 23.6 percent had second hand smoke exposure and around 4.8 percent of these children were exposed to second hand smoke

inside the house ($p<0.001$) (Appendix 1, Table 1). The mean age of children suffering from ASD in this study population was 6.5 years. As the age increased, the percentage of children diagnosed with ASD increased. The proportion of ASD diagnosis was highest in the age group 9-11 years (42.1% as compared to 23.7% for age group 3-5 years and 32.6% for age group 6-8 years, $p<0.001$). Males were found to have higher rate of ASD diagnosis than females (79.2% in males as compared to the 20.8% for females, $p<0.001$) and whites had higher rates of ASD than non-whites ($p<0.001$).

Among the parents who smoked, but not inside the house, 65.8% were whites and 7.3% were African-American and 13.5% were Hispanics ($p<0.001$) (Appendix 1, Table 2). Among the parents who smoked inside the house, around 60% were whites ($p<0.001$). Among the parents who smoked, 9.4% belonged to the lower socioeconomic status as compared to 3.9% who did not smoke; 15.2% of parents who smoked inside the house belonged to lower socioeconomic status. People who smoked or smoked inside the house had higher chances of having children with low birth weight and having premature babies. Parents/caregivers with English as a primary language were more likely to smoke than those who are non-English speakers ($p<0.001$). Mothers with less than high school education were more likely to smoke inside the house and mothers who did not smoke had higher levels of education ($p<0.001$).

Univariate Logistic Regression

In univariate logistic regression analysis, second hand tobacco exposure was associated with 36 percent greater odds of ASD ($p<0.001$) (Appendix 1, Table 3). In addition, if parents/caregivers smoked inside the house, there were 39% greater odds of ASD. As the age increased, the odds of developing ASD also increased. Children aged 3-5 years had about 4

times higher risk than 0-2 year olds, ages 6-8 years had around 6 times higher risk than 0-2 years olds, and children aged 9-11 years had about 7 times the risk than 0-2 years olds ($p<0.001$).

Male children had more than 3 times higher risk of ASD than female children ($p<0.001$). Children from the lower SES were twice as often affected with ASD than children from higher socioeconomic strata ($p<0.001$). Primary language other than English was found to have a protective role.

Children born prematurely or with low birth weight had 52% and 46% higher odds respectively, to develop ASD ($p<0.001$). As the age of the mother increased, the ASD risk in offspring increased. Mother's mental status was significant in ASD diagnosis. A three-fold higher risk of ASD diagnosis was noted in children of mothers with fair/poor mental status as compared to those who had excellent/very good mental status ($p<0.001$). Less than high school or high school educational status of mother was associated with greater odds of developing ASD but was not statistically significant. Also, having at least one step parent or single mother family was associated with greater odds of development of ASD in children. Children belonging to third generation household had twice the ASD risk than children belonging to first generation household.

Multivariate Logistic Regression

In multivariate models, the smoking variable was adjusted for potential confounders. Upon adjusting for age, smoking in general was significantly associated with ASD, but second hand smoke exposure inside the house was not significantly related with ASD diagnosis (Appendix 1, Table 4). When further adjusted for other covariates (including gender, race/ethnicity, socioeconomic status, birth weight, prematurity, mother's age, mother's education level, mother's mental status) smoking in general, was still significantly associated with ASD.

But upon further adjustment (primary language, family structure and generational status of household) the second hand smoking and ASD relationship lost its significance.

Discussion

In this study, it was found that 4.8% children were exposed to second hand tobacco smoke inside the house and 23.6% were exposed to second hand tobacco smoking overall (including in the car, in/outside public places, etc.). This percentage was lower than the percentage reported in the study by Kabir, Connolly, and Alpert (2011), who found that 6% children were exposed to second hand smoking inside the house. The percentage of second hand tobacco smoke exposure has decreased in the recent past because of many laws and regulations that have been passed in the past few years including ban of smoking inside public places and buildings. But children are still being exposed to tobacco smoke inside the house and efforts need to be made to change the smoking behavior in the parents and caregivers of children.

In the study, it was found that as the age increased the number of children diagnosed with ASD significantly increased. It has been seen in previous research studies related to ASDs that the latent period between the appearance of symptoms and diagnosis of such disorders in the kids could range between 20-60 months explaining the rising prevalence of the disorder as the age increases (Newschaffer et al., 2007). But recently, because of the increasing use of diagnostic questionnaires by the pediatricians and psychiatrists, these disorders are being diagnosed at an earlier age and fewer children go undiagnosed. Male children were found to have more than three times the risk of the disorder than the female children in this analysis, even though the prior research studies found the ratio to be even higher (Newschaffer et al., 2007). The risk was also found to be much higher in this study in white children as compared to the African-American and Hispanic children, which could also be explained by the better diagnostic and treatment

services available to the white children as compared to children belonging to other races and ethnicities.

The characteristics of the parents (especially the mother), as seen in this study, also played an important role in developing ASDs later on in children's lives. Children belonging to the lower sections of society were found to have higher risk of ASDs as seen in similar other research studies that proved that ASDs were more common in children whose mothers who had lower education and had lower socioeconomic status (Rai et al., 2012; Larsson et al., 2005). The stressors related to lower socioeconomic status of the mother during prenatal and perinatal period predispose the children to develop ASDs later on in life. Children whose mothers had lower education were found to have 7% higher odds of developing the disorder in this research study as compared to the children whose mothers had higher education but this finding was not found to be statistically significant. Also, as the mother's age increases the risk of getting the ASDs in the offspring increases. The poor mental status of the mother was also found to be a risk factor for development of ASDs which is in accordance with prior research studies that concluded that poor mental status of the mother is a predisposing factor for development of ASDs in children (Zhang et al., 2010; Larsson et al., 2005).

Children born prematurely and with low birth weight were found to have a higher risk of the disorder in this study. This is in consistence with earlier studies emphasizing that the prenatal, natal and perinatal factors play an important role in ASDs. English speaking families and children living in higher generational households had increased ASD risk than non-English speakers as seen in similar other studies that concluded that the recent immigrants have lower rates than the rest of the population (Newschaffer et al., 2007; Schieve et al., 2012). This could be because of the fact that children of recent immigrant families are not diagnosed earlier

because of language and other barriers including lack of insurance coverage, lack of understanding of different cultures by health care providers and stigma associated with mental health conditions in different cultures.

In this study we found that the relationship between the tobacco exposure inside the house and ASD lost its significance when adjusted for maternal factors (maternal mental status, maternal education and maternal age) and other factors (age of child, gender, birth weight, prematurity, race, socioeconomic status, etc.). However, effect of smoking overall on ASD was significant when adjusted for child's age, gender, race/ethnicity, socioeconomic status, birth weight, prematurity, mother's age, mother's education, mother's mental status. However, the association lost its significance when adjusted for immigration status and family structure.

The previous studies related to ASD only reported the effects of prenatal, perinatal and other demographic factors on the development of ASDs in children (Newschaffer et al., 2007; Schieve et al., 2012; Zhang et al., 2010; Rai et al., 2012; Bhasin & Schendel, 2007; Tran et al, 2013; Larsson et al., 2005; Itzchak et al., 2011 Kalkbrenner et al., 2012; Lee et al., 2012 Hultman et al., 2002; Sandin et al., 2012; Williams et al., 2008; Croen et al., 2002; Kuzniewicz et al., 2014; Leavey et al., 2013; Pedersen et al., 2012). Currently literature that discusses the effects of post-natal tobacco exposure on the development of ASDs in children is sparse, even though the adverse impact of post natal tobacco smoking on ADHD and other neurobehavioral disorders in children has been documented. This is the first study that explores the association between the post natal tobacco smoke exposure and autism and other related pervasive disorders in children.

Limitations of the study include the fact that the results could be impacted by recall bias since the data was taken from a survey. Also, even though many confounding variables were taken into consideration, there might have been some that were not addressed. The strengths of

the study are that the data was a nationally representative sample. Further research is needed to evaluate the association between post-natal smoking and ASD.

It has been seen in some countries, especially the developing countries that although prevalence of smoking declined in recent years, but the prevalence of ASD is increasing at an alarming rate. The better diagnostic interviewing tools and greater awareness among the health care providers and in the general population possibly leads to earlier and increased detection of such cases and could possibly explain to some extent the alarming increase in the prevalence of ASD in recent years but more extensive research is needed to find out the exact cause.

We conclude that second hand tobacco smoking is not significantly associated with ASDs. We also found that the natal and prenatal factors are the important etiologic factors for ASDs. As the impact of autism and the related disorders on the economy is huge, if efforts are made to change the smoking behavior among parents and care givers of children, that can tremendously reduce the impact of these pervasive developmental disorders on our economy.

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Appendix 1

Results

Table 1. ***Descriptives and Chi-square Values***

Variable	Autism diagnosis positive, n (%)	Autism diagnosis negative, n (%)	p-value
Smoking			
No	694 (71.5)	32864 (77.4)	<0.001
Yes, not inside	229 (23.6)	7983 (18.8)	
Yes, inside the house	47 (4.8)	1601 (3.8)	
Age of the child			
0-2 years	16 (1.6)	3633 (8.6)	<0.001
3-5 years	230 (23.7)	13542 (31.9)	
6-8 years	316 (32.6)	12641 (29.8)	
9-11 years	408 (42.1)	12632 (29.8)	
Gender of the child			
Female	202 (20.8)	21066 (49.6)	<0.001
Male	768 (79.2)	21383 (50.4)	
Race/ethnicity			
Hispanic	97 (10.0)	6168 (14.5)	<0.001
White, Non-Hispanic	664 (68.5)	27712 (65.3)	
Black, Non-Hispanic	79 (8.1)	3836 (9.0)	
Multiracial/others	130 (13.4)	4732 (11.1)	
Socioeconomic (how hard it is to get by on family income)			<0.001
Somewhat often/ not very often/never	865 (89.2)	40212 (94.7)	
Very often	105 (10.8)	2236 (5.3)	
Birth weight			
Normal birth weight	846 (87.2)	38568 (90.9)	<0.001
Low birth weight (<2500 Grams)	124 (12.8)	3880 (9.1)	
Prematurity			
No	805 (83.0)	37398 (88.1)	<0.001
Yes (before 37 weeks)	165 (17.0)	5050 (11.9)	
Primary language			
English	933 (96.2)	38820 (91.5)	<0.001
Other than English	37 (3.8)	3628 (8.5)	
Mother's mental status			<0.001
Excellent/very good	584 (60.2)	32007 (75.4)	
Good	246 (25.4)	7938 (18.7)	
Fair/poor	140 (14.4)	2503 (5.9)	
Mother's education			
More than HS	710 (73.2)	31648 (74.6)	0.336
Less than HS/HS	260 (26.8)	10800 (25.4)	
Mother's age			
20 or younger	1(0.1)	219 (0.5)	<0.001
21-35 years	355 (36.6)	18638 (43.9)	
36-58 years old	608 (62.7)	23481 (55.3)	
59 or older	6 (0.6)	110 (0.3)	

Variable	Autism diagnosis positive, n (%)	Autism diagnosis negative, n (%)	p-value
Generational status of household			
First generation	7 (0.7)	608 (1.4)	0.003
Second generation	133 (13.7)	7232 (17.0)	
Third generation	830 (85.6)	34608 (81.5)	
Family structure			
Two parent- biological/adopted	661 (68.1)	32610 (76.8)	
Two parent- step family	83 (8.6)	2519 (5.9)	<0.001
Single mother- no father present	224 (23.1)	7203 (17.0)	
Other family type	2 (0.2)	116 (0.3)	

Table 2. ***Percentage of Parents who Smoke in Various Demographic Categories***

Variable	Smoking			p-value
	No, n (%)	Yes, not inside, n (%)	Yes, inside the house, n (%)	
Autism diagnosis				<0.001
No	32864 (97.9)	7983 (97.2)	1601 (97.1)	
Yes	694 (2.1)	229 (2.8)	47 (2.9)	
Age of the child				<0.001
0-2 years	2787 (8.3)	772 (9.4)	90 (5.5)	
3-5 years	10601(31.6)	2730 (33.2)	441 (26.8)	
6-8 years	10087 (30.1)	2400 (29.2)	470 (28.5)	
9-11 years	10083 (30.0)	2310 (28.1)	647 (39.3)	
Gender of the child				0.318
Female	16424 (48.9)	4007 (48.8)	837 (50.8)	
Male	17134 (51.1)	4205 (51.2)	811 (49.2)	
Race/ethnicity				<0.001
Hispanic	5009 (14.9)	1110 (13.5)	146 (8.9)	
White, Non- Hispanic	21986 (65.5)	5405 (65.8)	985 (59.8)	
Black, Non-Hispanic	3000 (8.9)	600 (7.3)	315 (19.1)	
Multiracial/others	3563 (10.6)	1097 (13.4)	202 (12.3)	
Socioeconomic (how hard it is to get by on family income)				<0.001
Somewhat often/not very often/never	32243 (96.1)	7437 (90.6)	1397 (84.8)	
Very often	1315 (3.9)	775 (9.4)	251 (15.2)	
Birth weight				<0.000
Normal birth weight	30633 (91.3)	7352 (89.5)	1429 (86.7)	
Low birth weight (<2500)	2925 (8.7)	860 (10.5)	219 (13.3)	
Prematurity				<0.001
No	29661(88.4)	7151 (87.1)	1391 (84.4)	
Yes	3897 (11.6)	1061 (12.9)	257 (15.6)	
Primary language				<0.001
English	30391 (90.6)	7747 (94.3)	1429 (98.0)	
Other than English	3167 (9.4)	465 (5.7)	219 (2.0)	
Mother's mental status				<0.001
Excellent/very good	26278 (78.3)	5453 (66.4)	860 (52.2)	
Good	5761 (17.2)	1951 (23.8)	472 (28.6)	
Fair/poor	1519 (4.5)	808 (9.8)	316 (19.2)	

Variable	Smoking			p-value
	No, n (%)	Yes, not inside, n (%)	Yes, inside the house, n (%)	
Mother's education				<0.001
More than HS	26699 (79.6)	4902 (59.7)	757 (45.9)	
Less than HS/HS	6859 (20.4)	3310 (40.3)	891 (54.1)	
Mother's age				<0.001
20 or younger	123 (0.4)	80 (1.0)	17 (1.0)	
21-35	13326 (39.7)	4764 (58.0)	903 (54.8)	
36-58	20027 (59.7)	3342 (40.7)	720 (43.7)	
59 or older	82 (0.2)	26 (0.3)	8 (0.5)	
Generational status of household				<0.001
First generation	521 (1.6)	87 (1.1)	7 (0.4)	
Second generation	6332 (18.9)	954 (11.6)	79 (4.8)	
Third generation	26705 (79.6)	7171 (87.3)	1562 (94.8)	
Family structure				<0.001
Two-parent-biological/adopted	27039 (80.6)	5415 (65.9)	817 (49.6)	
Two-parent-step family	1467 (4.4)	914 (11.1)	221 (13.4)	
Single mother- no father present	4962 (14.8)	1860 (22.6)	605 (36.7)	
Other family type	90 (0.3)	23 (0.3)	5 (0.3)	

Table 3. ***Univariate Logistic Regression***

Variable	Odds ratios (95% CI)	p-values
Smoking		
No	Reference	<0.001
Yes, not inside the house	1.36 (1.17-1.58)	<0.001
Yes, inside the house	1.39 (1.03-1.88)	0.031
Age of the child		
0-2 years	Reference	<0.001
3-5 years	3.86 (2.32-6.41)	<0.001
6-8 years	5.68 (3.43-9.39)	<0.001
9-11 years	7.33 (4.44-12.10)	<0.001
Gender of the child		
Female	Reference	<0.001
Male	3.75 (3.20-4.38)	<0.001
Race/ethnicity		
Hispanic	Reference	<0.001
White, Non-Hispanic	1.52 (1.23-1.89)	<0.001
Black, Non-Hispanic	1.31 (.97-1.77)	0.078
Multiracial/others	1.75 (1.34-2.28)	<0.001
Socioeconomic		
Somewhat often/not very often/never	Reference	
Very often	2.18 (1.78-2.69)	<0.001
Primary language		
English	Reference	
Other than English	0.42 (.31-.59)	<0.001
Birth weight		
Normal birth weight	Reference	
Low birth weight	1.46 (1.20-1.76)	<0.001

Variable	Odds ratios (95% CI)	p-values
Prematurity		
No	Reference	
Yes	1.52 (1.28-1.80)	<0.001
Mother's mental status		
Excellent/very good	Reference	<0.001
Good	1.70 (1.46-1.98)	<0.001
Fair/poor	3.07 (2.54-3.70)	<0.001
Mother's education		
More than high school	Reference	
Less than high school/high school	1.07 (0.923-1.24)	0.336
Mother's age		
20 or younger	Reference	<0.001
21-35	4.17 (0.58-29.83)	.155
36-58	5.67 (0.80-40.50)	0.084
59 or older	11.95 (1.42-100.46)	0.022
Generational status of household		
First generation	Reference	0.004
Second generation	1.60 (0.74-3.43)	0.230
Third generation	2.08 (0.99-4.40)	0.055
Family structure		
Two parent-biological/adopted	Reference	<0.001
Two parent-step family	1.63 (1.29-2.05)	<0.001
Single mother- no father present	1.53 (1.32-1.79)	<0.001
Other	0.85 (0.21-3.45)	0.821

Table 4. ***Multivariate Logistic Regression***

Model	Odds ratio (95% CI)	p-values
Model 1- Unadjusted		
Smokes, no	Reference	<0.001
Smokes, not inside	1.36 (1.17, 1.58)	<0.001
Smokes, inside the house	1.39 (1.03, 1.88)	0.031
Model 2*		
Smokes, no		<0.001
Smokes, not inside	1.39 (1.2, 1.62)	<0.001
Smokes, inside the house	1.30 (0.96, 1.75)	0.089
Model 3**		
Smokes, no	Reference	<0.001
Smokes, not inside	1.39 (1.20, 1.62)	<0.001
Smokes, inside the house	1.33 (0.98, 1.80)	0.065
Model 4***		
Smokes, no	Reference	<0.001
Smokes, not inside	1.38 (1.184, 1.61)	<0.001
Smokes, inside the house	1.31 (0.97, 1.78)	0.077
Model 5****		
Smokes, no	Reference	0.003
Smokes, not inside	1.30 (1.11, 1.52)	0.001
Smokes, inside the house	1.17 (0.86, 1.59)	0.308

Model	Odds ratio (95% CI)	p-values
Model 6***		
Smokes, no	Reference	0.005
Smokes, not inside	1.29 (1.10, 1.50)	0.001
Smokes, inside the house	1.15 (0.84, 1.56)	0.381
Model 7****		
Smokes, no	Reference	0.048
Smokes, not inside the house	1.21 (1.03, 1.42)	0.019
Smokes, inside the house	0.951 (0.69, 1.30)	0.754
Model 8*****		
Smokes, no	Reference	0.126
Smokes, not inside	1.15 (0.97, 1.35)	0.104
Smokes, inside the house	0.87 (0.63, 1.19)	0.376
Model 9******		
Smokes, no	Reference	0.156
Smokes, not inside	1.14 (.98, 1.33)	0.102
Smokes, inside the house	0.89 (0.65, 1.21)	0.452

Notes: *Model 2 adjusted for age in addition to exposure variable; **Model 3 adjusted for age, gender in addition to exposure variable; ***Model 4 adjusted for age, gender, race/ethnicity in addition to exposure variable ****Model 5 adjusted for age, gender, race/ethnicity, socioeconomic status in addition to exposure variable *****Model 6 adjusted for age, gender, race/ethnicity, socioeconomic status, birth weight and prematurity in addition to exposure variable; ******Model 7 adjusted for age, gender, race/ethnicity, socioeconomic status, birth weight, prematurity, mother's age, mother's education and mother's mental status in addition to exposure variable *******Model 8 adjusted for age, gender, race/ethnicity, socioeconomic status, birth weight, prematurity, mother's age, mother's education, mental status, primary language, generational status of household and family structure ********Model 9 after adjusting for variables that were significantly associated with the outcome variable (age, gender, race/ethnicity, socioeconomic status, mother's mental status, birth weight, primary language)

Appendix 2

IRB Letter

WRIGHT STATE UNIVERSITY

Office of Research and Sponsored Programs
201J University Hall
3640 Col. Glenn Hwy.
Dayton, OH 45435-0001
(937) 775-2425
(937) 775-3781 (FAX)
e-mail: rsp@wright.edu

DATE: November 1, 2013

TO: Bhupinder Kaur, PI, Graduate Student
Community Health
Naila Khalil, Ph.D., Faculty Advisor

FROM: B. Laurel Elder, Chair
WSU Institutional Review Board

SUBJECT: SC# 5321
'The Association Between Autism Spectrum Disorders and Tobacco Exposure'

At the recommendation of the IRB Chair, your study referenced above has been recommended for exemption. Please note that any change in the protocol must be approved by the IRB; otherwise approval is terminated.

This action will be referred to the Full Institutional Review Board for ratification at their next scheduled meeting.

NOTE: This approval will automatically terminate two (2) years after the above date unless you submit a "continuing review" request (see http://www.wright.edu/rsp/IRB/CR_sc.doc) to RSP. You will not receive a notice from the IRB Office.

If you have any questions or require additional information, please call Robyn Wilks, IRB Coordinator at 775-4462.

Thank you!

Enclosure

RESEARCH INVOLVING HUMAN SUBJECTS

SC# 5321

ACTION OF THE WRIGHT STATE UNIVERSITY
EXPEDITED REVIEW
Assurance Number: FWA00002427

Title: 'The Association Between Autism Spectrum Disorders and Tobacco Exposure'

Principal Investigator: Bhupinder Kaur, PI, Graduate Student
Community Health
Naila Khalil, Ph.D., Faculty Advisor

The Institutional Review Board Chair has approved an exemption with regard to the use of human subjects on this proposed project.

REMINDER: Federal regulations require prompt reporting to the IRB of any changes in research activity [changes in approved research during the approval period may not be initiated without IRB review (submission of an amendment), except where necessary to eliminate apparent immediate hazards to subjects] and prompt reporting of any serious or on-going problems, including unanticipated adverse reactions to biologicals, drugs, radioisotope labeled drugs or medical devices.

B Lamal Ebb. Ph.D.

Signed Chair, WSU-IRB
Approval Date: November 01, 2013
IRB Mtg. Date: November 18, 2013

Appendix 3

Public Health Management Competencies Addressed by CE

Know Effective Communication Strategies used by Health Service Organizations:
While working on this CE project I learned how communication strategies by different health service organizations help to promote awareness about harmful effects of second hand smoking among the public and also help the policy makers make laws and regulations against smoking. Similarly, organizations are working hard to raise the awareness about autism among parents and health care providers so that the children can be diagnosed earlier and are provided timely intervention. These organizations organize fundraising and awareness-raising events and also get help of cable TV, news releases, press conferences, social media, and public media to achieve that.

Be Capable of Applying Decision-Making Processes:
Decision-making process is involved whenever a new project is started. When I started to work on my CE, I had to go through proper process like choosing a topic, consulting an expert in the field, choosing appropriate methods, review evidence, collect/find data, generate hypothesis, conduct analysis, interpret results, and generate conclusions and recommendations. Being in a managerial position, one has to go through similar steps to achieve the goal.

Have Knowledge of Systems Thinking Principles:
An understanding of a system by examining the linkages and interactions between components that comprise that system is required to succeed in any venture. In this CE project, I operated in various systems like IRB, while reviewing current scientific literature about ASD and second hand smoking, when acquiring data from data keepers, worked with the MPH faculty and staff, and also learned how the home environment affects the growing children (in this case, second hand smoking).

Have an Awareness of Strategies for working with Stakeholders to Determine Common and Key Values to Achieve Organizational and Community Goals:
Parents/grandparents/guardians, school teachers, public servants, public health and medical community and the affected children are all the stakeholders in this project. One has to work with each of these to help decrease the rising prevalence of ASDs.

Have an understanding of effective mentoring methods:
Providing guidance, counseling, teaching, modeling, motivation and inspiration, and providing valuable feedback are important aspects of mentoring process which I constantly achieved from my mentors throughout the process.

Be able to determine how public health challenges can be addressed by applying strategic principles and management-based solutions:
Challenges posed by rising prevalence of ASD can be addressed by raising awareness in the parents and caregivers by increasing knowledge about various factors (especially environmental)

that lead to development of ASD, implementing strategies to increase community outreach, diagnosing and treating children with ASD at an earlier stage so that they can be managed better.

Knowledge of ethical principles relative to data collection, usage, and reporting results: Going through the IRB procedure helped me to understand the ethical principles related to data collection, usage and reporting results.

Knowledge of ethical standards for program development
In every organization certain policies and procedures dictate expectations and requirements to its employees. While working on the CE, I followed certain set of procedures and protocols which are expected from the students working on their CEs.

Appendix 4

List of Competencies Met in CE

Tier 1 Core Public Health Competencies

Domain #1: Analytic/Assessment
Identify the health status of populations and their related determinants of health and illness (e.g., factors contributing to health promotion and disease prevention, the quality, availability and use of health services)
Describe the characteristics of a population-based health problem (e.g., equity, social determinants, environment)
Use variables that measure public health conditions
Identify sources of public health data and information
Recognize the integrity and comparability of data
Identify gaps in data sources
Adhere to ethical principles in the collection, maintenance, use, and dissemination of data and information
Use information technology to collect, store, and retrieve data
Describe how data are used to address scientific, political, ethical, and social public health issues
Domain #2: Policy Development and Program Planning
Gather information relevant to specific public health policy issues
Describe how policy options can influence public health programs
Explain the expected outcomes of policy options (e.g., health, fiscal, administrative, legal, ethical, social, political)
Domain #3: Communication
Participate in the development of demographic, statistical, programmatic and scientific presentations
Domain #4: Cultural Competency
Recognize the role of cultural, social, and behavioral factors in the accessibility, availability, acceptability and delivery of public health services
Domain #5: Community Dimensions of Practice
N/A
Domain #6:Public Health Sciences
Describe the scientific evidence related to a public health issue, concern, or, intervention
Retrieve scientific evidence from a variety of text and electronic sources
Discuss the limitations of research findings (e.g., limitations of data sources, importance of observations and interrelationships)
Domain #7: Financial Planning and Management
N/A
Domain #8: Leadership and Systems Thinking
Use individual, team, and organizational learning opportunities for personal and professional development

Concentration Competencies

Public Health Management:
Know effective communication strategies used by health service organizations
Be capable of applying decision-making processes
Have a knowledge of systems thinking principles
Have an awareness of strategies for working with stakeholders to determine common and key values to achieve organizational and community goals
Have an understanding of effective mentoring methods
Be able to determine how public health challenges can be addressed by applying strategic principles and management-based solutions
An understanding of marketing principles and strategies
A knowledge of ethical principles relative to data collection, usage, and reporting results
A knowledge of ethical standards for program development