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Intrauterine Tobacco Smoke Exposure and Congenital Heart Defects

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ABSTRACT

Tobacco use and second-hand smoke exposure during pregnancy are linked to a host of deleterious effects on the pregnancy, fetus, and infant. Health outcomes improve when women quit smoking at any time during the pregnancy. However, the developing heart is vulnerable to noxious stimuli in the early weeks of fetal development, a time when many women are not aware of being pregnant. Congenital heart defects are the most common birth defects. Research shows an association between maternal tobacco exposure, both active and passive, and congenital heart defects. This article presents recent evidence supporting the association between intrauterine cigarette smoke exposure in the periconceptional period and congenital heart defects and discusses clinical implications for practice for perinatal and neonatal nurses.

Key Words: congenital heart defect etiology, intrauterine tobacco smoke exposure, maternal smoking in pregnancy, second-hand smoke in pregnancy

Smoking during pregnancy is likely the most modifiable behavior associated with detrimental pregnancy and childhood health outcomes. Fetal exposure to second-hand smoke (SHS) is also harmful to the fetus. Among the deleterious effects of intrauterine tobacco smoke exposure is the development of congenital heart defects (CHDs). The incidence of CHDs is 6 to 8 per 1000 births,[1] and worldwide, 1.35 million newborns are born with a CHD each year.[2] Forty-six percent of deaths from congenital malformations and 3% of all infant deaths are attributed to CHDs.[3] Clinical research clearly shows an association between fetal cigarette smoke exposure and CHDs. This article presents an overview of recent studies demonstrating the association as well as clinical implications for practice for perinatal and neonatal nurses.

PREVALENCE AND EFFECTS OF SMOKING DURING PREGNANCY

The pregnancy risk assessment monitoring system (PRAMS) administered by the Centers for Disease Control and Prevention in collaboration with state health departments, is a state- and population-based surveillance system that monitors selected self-reported behaviors occurring during the 3 months before, during, and the 3 months after pregnancy. According to PRAMS data, the prevalence of maternal smoking before and during pregnancy is 24.7% and 12.3%, respectively.[4] Infants of women who smoke are nearly twice as likely to be preterm and are at higher risk of being small-for-gestation and low birth weight.[5,6] Other fetal and neonatal complications associated with smoking during pregnancy include intrauterine demise, neonatal death, and respiratory disease.[5] Musculoskeletal defects, limb reduction defects, missing or extra digit defects, clubfoot, craniosynostosis, facial defects, eye defects, gastrointestinal defects, oral clefts and many other birth defects are associated with maternal smoking in pregnancy.[7] Problems, including type I diabetes mellitus and visual problems, may develop after the neonatal period.[8,9] Pregnant nonsmokers exposed to SHS during the pregnancy may be affected. Adverse pregnancy and fetal and neonatal outcomes associated with maternal exposure to SHS include stillbirth, preterm birth, low birth weight, reduced gross motor function, and congenital malformations including CHDs.[10–14] Fetal and neonatal health outcomes improve when mothers quit smoking at any time during the

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pregnancy; unfortunately, the developing fetal heart is susceptible to the harmful effects of intrauterine cigarette smoke exposure early in pregnancy.

VULNERABILITY OF THE DEVELOPING HEART

The fetal heart begins to work toward full development by 16 days postconception. The earliest indication of heart development is the development of the cardiogenic cords. Cords of mesenchymal cells become canalized early to form 2 thin-walled endothelial tubes called "endocardial heart tubes." The tubes fuse to form a single heart tube. A series of constrictions and dilations develop distinct regions of the heart such as the sinus venosus, the primitive atrium, the primitive ventricle, the bulbus cordis, and the truncus arteriosus. The primitive heart tube grows rapidly and bends upon itself to form a U-shaped bulboventricular loop. As the development continues, the U-shaped heart loops into an S-shaped heart. By the fifth week of gestation, the cardiac separation is complete and the heart is divided into a 4-chambered heart. Partitioning of the bulbus cordis and the truncus arteriosus results in the development of the truncal ridges and bulbar ridges. The fused mesenchymal ridges form the aorticopulmonary septum that divides the truncus arteriosus and the bulbus cordis into the ascending aorta and the pulmonary trunk.[15,16] Since most CHDs occur between the second and ninth week of gestation, the fetal heart is susceptible to malformations due to teratogens, including tobacco smoke, before many women even suspect that they are pregnant.[17] Table 1 describes CHDs associated with fetal exposure to cigarette smoke.

Cardiac anomalies make up the largest category of birth defects. The exact mechanism by which smoking is related to CHD is not fully understood. Alterations and variations in genes contribute to the origin of CHDs.[19] More than 85% of CHDs are thought to be the result of a complex interaction between maternal exposures to environmental toxins and genetic susceptibilities.[20] An estimated 2% of CHDs are related to environmental toxins,[15] and as much as 1.4% of CHDs may be attributed to maternal smoking in pregnancy.[14]

Active and passive exposure to cigarette smoke during pregnancy exposes the fetus to more than 4000 chemicals, many of which are carcinogenic, mutagenic, or toxic.[21,22] Carbon monoxide (CO), cadmium, and nicotine are environmental toxins found in cigarettes and cigarette smoke that have adverse effects on the placenta and developing fetus.[23–25] Carbon monoxide inhibits the blood's ability to deliver oxygen to body tissues and vital organs by binding to hemoglobin and forming carboxyhemoglobin. Hemoglobin is subsequently unavailable for transporting oxygen, resulting in a hypoxic environment for the fetus. Atrial septal defects (ASDs), and conotruncal defects, particularly tetralogy of Fallot, are associated with CO exposure in utero.[23,26] Atrial septal defects occur between weeks 4 and 5 of gestation.[15] Conotruncal heart defects are also known as outflow tract defects. Common types of conotruncal heart defects are truncus arteriosus, transposition of the great arteries, double outlet of the right ventricle, and tetralogy of Fallot. Conotruncal heart defects occur between the 35th and 45th day of gestation.[3,15] Cadmium accumulates in the placenta and inhibits the metabolism of cortisol, which results in

Table 1. **Congenital heart defects associated with intrauterine tobacco smoke exposure**[15,18]

Congenital heart defect	Description of defect	Embryology	Gestational timing
Atrial septal defect	Abnormal opening in the septal wall between the right and left atria	Ostium secundum defect—excessive cell death and resorption of the septum primum or inadequate development of the septum secundum	4-5 wk
Truncus arteriosus	The pulmonary artery and the aorta are a single vessel in the heart	Lack of truncal separation into the pulmonary artery and the aorta	Approximately day 35
Transposition of the great arteries	The 2 main arteries carrying blood out of the heart are switched in position	Lack of rotation of the great vessels after separation	Approximately day 45
Tetralogy of Fallot	Combination of 4 defects, ventricular septal defect, pulmonary stenosis, right ventricular hypertrophy, and overriding aorta	Incomplete rotation of the great arteries, resulting in malalignment of vessels with rightward deviation and a narrow right ventricular outflow tract	Approximately day 45
Atrioventricular septal defect	Deficiency of the atrioventricular septum	Abnormal development of the endocardial cushions	Days 26-35

fetal growth restriction.[25] Fetal growth restriction may be an indication of poor perfusion and fetal hypoxia. Nicotine also contributes to hypoxia. Blood flow to the placenta is restricted secondary to the vasoconstrictive effects of catecholamines released from the adrenals and nerve cells after nicotine exposure.[24] Vasoconstriction alters delivery of oxygen and nutrients to the placenta, creating a hypoxic environment for the fetus.[25] Maternal smoking is a well-established risk factor for placental insufficiency and fetal hypoxia, both of which are noted to have some responsibility for abnormal cardiovascular development.[14]

The formation and maturation of the fetal cardiovascular system is a closely regulated process in which oxygen tension plays an important role.[27] Maternal smoking creates an environment of pathologic or chronic hypoxia for the fetus. Pathologic hypoxia exists when oxygen tension is lower than physiological levels or at lower levels than oxygen demands. Chronic hypoxia, defined as lower oxygen tension levels for an extended period of time, plays a key role in placenta insufficiency.[27,28] Hypoxic stress during perinatal development has been shown to suppress fetal cardiac function, alter gene expression, and increase cardiomyocyte apoptosis.[28] As a result, cardiomyocytes experience premature exit of the cell cycle and further growth is mainly via hypertrophy.[2,29,30]

ASSOCIATION BETWEEN INTRAUTERINE CIGARETTE SMOKE EXPOSURE AND CONGENITAL HEART DEFECTS

As early as the 1970s, research has shown the association of various CHDs and maternal smoking during pregnancy.[31] Table 2 summarizes recent evidence to support the assertion that periconceptional smoking and SHS exposure during pregnancy is associated with CHDs.

Sullivan et al[14] conducted a retrospective case-control study using Washington State birth certificates from 1989 to 2011 and linked hospital discharge codes to identify nonsyndromic CHD cases and maternal smoking status during pregnancy. The study objective was to determine whether there is an association between CHD and first trimester maternal smoking. Controls were randomly selected and matched by year of delivery. Study results showed a modest association between maternal smoking and CHD, particularly pulmonary valve anomalies, pulmonary artery anomalies, and ASDs, independent of other known risk factors for CHD. The prevalence of CHD increased with heavier reported cigarette use, and the relative odds of having a child born with a CHD was 27% greater for smoking mothers of 35 years of age or older than that for younger mothers. There were several limitations of this study, which include the following: the researchers were unable to confirm CHD diagnoses in the medical record, and using *International Classification of Diseases, Ninth Revision*, discharge codes may not have been reliable for accurately ascertaining and classifying CHD diagnoses; obtaining maternal smoking status through self-report from birth certificates may not be accurate; and the cases were limited to live births, excluding spontaneous or elective abortions.[14]

A prospective case-control study sought to determine whether maternal exposure to SHS from paternal smoking in the periconceptional period, defined as 3 months prepregnancy through the first trimester of pregnancy, is a risk factor for CHD in offspring.[32] The study was conducted in 4 tertiary maternal-child hospitals in 4 metropolitan areas of China. Congenital heart disease was diagnosed by prenatal echocardiogram. Mothers responded to a detailed questionnaire administered during pregnancy regarding environmental exposures for the periconceptional period. The authors concluded that paternal smoking during the periconceptional period was significantly associated with CHDs. Specific CHDs correlated with the degree of smoking: light paternal smoking (1-9 cigarettes per day) was associated with isolated conotruncal heart defects; medium smoking (10-19 cigarettes per day) was associated with septal defects and left ventricular outflow tract obstructions; and heavy smoking ($\geq$20 cigarettes per day) increased the risk of isolated conotruncal defects and left ventricular outflow tract obstructions. When fathers made no effort to maintain a distance from the mothers while smoking, the risk of CHD increased. Limitations of the study include possible selection bias since the sample population was assigned by referral or admission and may not truly represent the general population; information regarding paternal smoking during the periconceptional period was self-reported by the pregnant women; and the effect of other maternal SHS exposure was not controlled in this analysis. The study adds to the body of evidence that maternal SHS exposure is associated with CHDs.[32]

Using data from the National Birth Defects Prevention Study,[33] Patel et al[34] investigated the association between nongenetic risk factors and nonsyndromic atrioventricular septal defects (AVSD) using a case-control methodology. Maternal smoking status was obtained by telephone interview regarding behaviors during the periconceptional period, defined as 1 month before becoming pregnant through the first trimester. Active smoking during the periconceptional period was reported by 26% of mothers of all AVSD cases. Mothers who reported smoking cigarettes were more likely to have offspring with AVSDs, independent of other

Table 2. **Summary of studies demonstrating association between smoking and congenital heart defects**

Study	Method	Population/Sample	Data Collection	Results
Sullivan et al (2015)[14]	Retrospective case-control	Cases: Live-born nonsyndromic singletons diagnosed with CHD ($n = 14\,128$) Controls: Randomly selected matched on birth year ($n = 60\,938$)	CHD: Birth certificates and linked *ICD-9* codes Maternal smoking status: Self-reported cigarette smoking during pregnancy (Y/N; average number of cigarettes per day) from birth certificates	Offspring of mothers reporting cigarette use in 1st trimester more likely to have CHD (aOR, 1.16; 95% CI, 1.08-1.24) independent of other prenatal risk factors for CHDs. Maternal smoking most strongly associated with: • pulmonary valve anomalies (aOR 1.48; 95% CI, 1.15-1.90) • pulmonary artery anomalies (aOR, 1.71; 95% CI, 1.40-2.09) • Isolated atrial septal defects (aOR, 1.22; 95% CI, 1.08-1.38) Strong dose-dependence of the association and augmented risk in older mothers. Overall, 1.4% of all CHDs associated with maternal smoking during pregnancy.
Deng et al (2013)[32]	Prospective case-control	Cases: Singleton fetuses with nonsyndromic CHDs diagnosed prenatally ($n = 284$) Control fetuses with no birth defects ($n = 422$)	CHD: Prenatal echocardiogram Smoking status: Maternal interview/questionnaire • Smoked at any time during preconception period? (Y/N; number of cigarettes per day) • Father of fetus smoked during the period? (Y/N; number of cigarettes per day) • Father avoided mothers? (Y/N) Light: 1-9 cigarettes per day Medium: 10-19 cigarettes per day Heavy: ≥20 cigarettes per day Periconceptional period: 3 mo before pregnancy through the 1st trimester	Light paternal smoking increased risk of isolated conotruncal heart defects (aOR, 2.23; 95% CI, 1.05-4.73) Medium paternal smoking associated with septal defects (aOR, 2.04; 95% CI, 1.05-3.98) and left ventricular outflow tract obstructions (aOR, 2.48; 95% CI, 1.04-5.95) Heavy paternal smoking associated with isolated conotruncal defects (aOR, 8.16; 95% CI, 1.13-58.84) and left ventricular outflow tract obstructions (aOR, 13.12; 95% CI, 2.55-67.39) Paternal smoking with no avoidance behavior increased risk of CHD

(*continues*)

Table 2. **Summary of studies demonstrating association between smoking and congenital heart defects (*Continued*)**

Study	Method	Population/Sample	Data Collection	Results
Lee and Lupo (2013) [38]	Meta-analysis	33 studies published between 1971 and 2011	Medline database search and review of reference lists of articles	Positive association between maternal smoking during pregnancy and risk of CHDs as a group (RR, 1.11; 95% CI, 1.02-1.21; number of cases [*n*] = 18 282) Women who smoked during pregnancy associated with 12 of 17 CHD subtypes; highest risk was septal defects as a group (RR, 1.44; 95% CI, 1.16-1.79; *n* = 2977) Evidence of dose response observed for septal defects as a group, ASDs, and AVSDs
Patel et al (2012)[34]	Retrospective case-control	Cases: Mothers of infants with AVSD (*n* = 187) Control: Mothers of unaffected infants (*n* = 6703)	CHD: Subjects identified from the National Birth Defect Prevention Study Smoking status: Maternal telephone interview (self-report) related to behaviors during periconceptional period including • maternal active smoke exposure? (Y/N; number of cigarettes per day) • passive smoke exposure? (Y/N) Periconceptional period: 1 mo before pregnancy through the 1st trimester)	Mothers who smoked during periconceptional period more likely to have infant with AVSDs than nonsmokers (aOR, 1.5; 95% CI, 1.1-2.4). Association strongest in mothers who smoked more than 25 cigarettes per day Mothers exposed to passive smoke more likely to have infant with AVSD than unexposed mothers (aOR, 1.4; 95% CI, 1.0-2.0)
Alverson et al (2011)[35]	Case-control	Case: Live-born singleton infants with a CHD (*n* = 2525) Control: Random selection from live-birth logs of regional hospitals (*n* = 3435)	CHD diagnosed within 1 year birth by echocardiogram, cardiac catherization, surgery, or autopsy from birth defect registry Maternal smoking status: In-person postdelivery interview: • Number of cigarettes per day (0-10; 11-20; 21-39; ≥40)	Positive association between self-reported 1st trimester smoking and the following: • Secundum-type ASD (OR, 1.36; 95% CI, 1.04-1.78) • Right ventricular outflow tract defects (OR, 1.32; 95% CI, 1.06-1.65) • Pulmonary valve stenosis (OR, 1.35; 95% CI, 1.05-1.74) • Truncus arteriosus (OR, 1.90; 95% CI, 1.04-3.45) Levotransposition of the great arteries (OR, 1.79; 95% CI, 1.04-3.10)

(*continues*)

Table 2. **Summary of studies demonstrating association between smoking and congenital heart defects (*Continued*)**

Study	Method	Population/Sample	Data Collection	Results
Karatza et al. (2011)[36]	Case-control	Cases: Neonates (aged 1-28 d) diagnosed with CHD (n = 157) Controls: Neonates with proven normal cardiovascular anatomy/function (n = 208)	CHD: Diagnosed by echocardiogram Maternal smoking status: Periconceptional tobacco smoking by self-report during interview prior to echocardiography (Y/N; number of cigarettes per day (0-10 or ≥11) Periconceptional period: At least 1 mo before conception and continuously during 1st trimester Women who quit or started smoking during pregnancy excluded	Periconceptional smoking associated with increased risk of CHD (OR, 2.750; 95% CI, 1.659-4.476; P = .00001) Dose effect observed
Hackshaw et al (2011)[7]	Meta-analysis	172 observational studies published between 1959 and 2010	MEDLINE database literature search	Positive association between periconceptional maternal smoking and CHD (OR, 1.09; 95% CI, 1.02-1.17)

Abbreviations: aOR, adjusted odds ratio; ASD, atrial septal defect; AVSD, atrioventricular septal defect; CHD, congenital heart defect; CI, confidence interval; *ICD-9, International Classification of Diseases, Ninth Revision;* OR, odds ratio; RR, risk ratio.

risk factors, compared with mothers who did not smoke during the periconceptional period. A dose-dependence of the association was noted for mothers who smoked more than 25 cigarettes per day. Thirty-four percent of mothers of all AVSD cases reported periconceptional exposure to SHS. Mothers who had passive cigarette smoke exposure were more likely to have affected infants than unexposed mothers. The retrospective data collection method used in the study introduces recall bias, and nondisclosure of smoking status is a possibility with self-report. Potential miscoding of CHD could reduce the actual numbers of AVSDs. Still, the results of this study have important health information and consequences related to the dangers of both, active and passive smoking.[34]

Another case-control study investigated the association between first trimester maternal smoking and CHDs using data from the Baltimore-Washington Infant Study, which was the first population-based study to examine the etiology of CHDs. Maternal smoking status, including the amount of cigarette consumption, was assessed by in-person postpartum interview. Smoking behaviors were reported at 5 time intervals: 4 to 6 months before conception, 1 to 3 months before conception, and during the first, second, and third trimesters of pregnancy. Congenital heart defects were diagnosed by echocardiogram, cardiac catherization, surgery, or autopsy. The researchers demonstrated a positive association between first semester maternal smoking and secundum-type ASD, right ventricular outflow tract defects, pulmonary valve stenosis, truncus arteriosus, and levo-transposition of the great arteries.[35]

Karatza et al[36] demonstrated an association between periconceptional smoking and CHD in a case-control study in which neonates, aged 1 to 28 days, diagnosed with CHD were compared with neonates with proven normal cardiovascular anatomy and function. The periconceptional period was defined as at least 1 month before conception and continuously during the first trimester. The presence or absence of congenital heart disease was confirmed by echocardiography. Women who quit smoking or who started smoking during pregnancy were excluded. Maternal smoking status was by self-report using oral interviews prior to echocardiography. A dose effect was observed for women who smoked 11 or more cigarettes per day. As with other studies utilizing self-report for maternal smoking status, nondisclosure is a concern. Congenital heart disease in the study may have been underestimated if CHD was not suspected and detected in the neonatal period.[36]

A case-control methodology is often used to examine the association between intrauterine tobacco exposure and CHDs. Limitations utilizing case-control studies include recall errors, misclassification of exposure

status, and inaccurate CHD phenotype classification.[37] However, the use of randomized controlled trials to explore effects of antenatal tobacco exposure obviously presents ethical issues and is not practical.

The results of 2 meta-analyses show a positive association between periconceptional maternal smoking and CHD. Both studies utilized the MEDLINE database for the analysis. Lee and Lupo[38] identified 33 studies published between 1971 and 2011. The researchers found a positive association between maternal smoking during pregnancy and the risk of CHD as a group. Women smoking during pregnancy were associated with 12 of 17 CHD subtypes, the highest of which was septal defects as a group. Women who smoked during pregnancy were 44% more likely to have an infant with septal defects than women who did not smoke in pregnancy. There was evidence of a dose response for septal defects as a group as well as for ASD and AVSD subtypes. A strength of the study is the large population size (n = 18 282). The researchers conclude that mothers of offspring with CHD were 11% more likely to smoke during pregnancy than mothers of unaffected offspring.[38] Hackshaw et al[7] looked at observational studies dating from 1959 to 2010. Nonchromosomal birth defects among women who smoked during pregnancy were compared with nonsmokers. One hundred seventy-two articles were reviewed, and the study reported a significant association among smoking in pregnancy and several birth defects, including CHDs. The confirmed association regarding CHDs was not specific to any particular defect.[7,38]

Aside from the studies examining SHS effects on the fetus described previously,[32,34] others have also demonstrated an association between SHS, including paternal smoking around pregnant mothers, and CHD.[39] However, conclusions from research over the years vary. Malik et al[40] found an association between maternal smoking during pregnancy and CHD but did not observe an increased risk of CHD with maternal exposure to environmental tobacco smoke.

Smoking during pregnancy is associated with other untoward cardiovascular effects such as perinatal arterial ischemic stroke[41] and increased blood pressure in late adolescence.[42] Maternal smoking in pregnancy has adult consequences for the offspring of smokers as well. Smoking in pregnancy is related to adult atherosclerosis and elevated blood pressure.[43]

Several factors appear to interact with maternal smoking during pregnancy to increase the risk for CHDs. Smoking combined with genetic susceptibilities may increase the risk of having a CHD-affected pregnancy.[20,44] A substantial increase in CHDs was noted when mothers engaged in binge drinking while smoking during pregnancy.[45] Maternal obesity and smoking increases the risk of CHD,[46] and interactions between particular maternal genotypes and tobacco use by obese mothers may increase the risk of certain CHDs by as much as 2-fold.[47]

IMPLICATIONS FOR PRACTICE

The etiology of most of CHDs is beyond nurses' power to influence; however, unfavorable health effects related to smoking are often preventable. There are several ways perinatal and neonatal nurses can participate in tobacco prevention and cessation initiatives.

Mothers may have little understanding that smoking is associated with adverse children's health outcomes, including congenital anomalies.[48] The implication for practice is to increase the public's health awareness through education to emphasize the effects of smoking on children's health. Through population-based initiatives, advanced practice nurses can lead efforts to increase the community's knowledge about the dangers of smoking in the periconceptional period.

Maternal child nurses can also advocate for tobacco control policies to improve health outcomes both within and outside their practice. Research shows that increased cigarette taxes improve health outcomes, particularly for the highest-risk mothers and infants.[49] Within hospitals and obstetric practices, nurses can lead initiatives to transition to tobacco-free healthcare facilities, and supporting policy for tobacco-free workplaces in general protects nonsmoking women from SHS exposure.

Nurses can be extremely effective collaborating with other healthcare disciplines and agencies, such as The Family Nurse Partnership, March of Dimes, First Candle, and professional organizations, to build stronger coalitions for maternal child healthcare improvement. The Family Nurse Partnership is a program to help at-risk, first-time mothers have healthy pregnancies and improve child health and development.[50] March of Dimes works in local communities across the country to help mothers take charge of their health and to support families.[51] First Candle works with moms-to-be through educational programs that help foster healthy habits during pregnancy to improve healthy baby outcomes.[52] When nurses use their expert skills to team up with groups to implement change, they have a gratifying experience while making a difference in the lives of others.

The PRAMS data indicate that pregnancy may be a time when women are motivated to quit smoking, as smoking prevalence drops from 24.7% before pregnancy to 12.3% during pregnancy.[4] However, many women resume smoking after giving birth; relapse rates are as high are 50% or greater 6 months postpartum.[53–55]

Postpartum relapse puts infants at risk for SHS-related health problems. Infants who are exposed to SHS after birth are at greater risk for sudden infant death syndrome, decreased lung growth, allergic diseases, respiratory tract infections, asthma, and ear problems.[56–59] Postpartum relapse also increases the chance of fetal tobacco exposure during the early stages of subsequent pregnancies. Perinatal and neonatal nurses can implement tobacco prevention and cessation interventions in the pre- and periconceptional period and help prevent postpartum relapse for mothers who quit smoking.

The Public Health Service recommends that smoking cessation interventions be provided at each patient visit across settings. Five intervention steps, known as the 5As, are proven effective and recommended by The American Congress of Obstetricians and Gynecologists.[60] The 5 As are as follows: Ask about tobacco use at every patient encounter, Advise every tobacco user to quit, Assess tobacco users' willingness to quit, Assist willing patients to make a quit attempt, and Arrange for follow-up contact within the week after the quit date.[61] Quitlines, which are free and accessible, are effective for increasing cessation rates for specific and diverse populations.[62] When perinatal and neonatal nurses arrange quitline referrals during office visits or before postpartum hospital discharge, highly trained counselors provide ongoing counseling and support, reducing nurses' workload. Since second-hand tobacco smoke is associated with CHDs, including family or friends in tobacco prevention strategies may be beneficial. Studies demonstrate that parents are willing to accept help from nurses to help quit smoking.[63–65] Family and friends also have access to state quitlines by calling 1-800-QUIT NOW. Advanced practice perinatal and neonatal nurses have an opportunity to coordinate efforts and to educate staff on the 5As process.

The evidence demonstrates an association between intrauterine cigarette smoke exposure and several types of CHDs. The good news is that exposure to the environmental toxins of cigarette smoke is a modifiable health risk. Implementation of tobacco prevention and cessation strategies can help women quit smoking and remain abstinent. Depending on one's individual passion, skill set, and willingness to participate as agents for change, each of us can make a difference. Perinatal and neonatal nurse have an opportunity to develop and lead initiatives to reduce maternal smoking, thereby improving pregnancy and infant health outcomes.

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