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In the early part of the 20th century, smoking tobacco was not socially acceptable for women. Seeing a marketing opportunity, the tobacco industry targeted women of reproductive age. A 1950s advertisement promoted smoking to mothers with a picture of a baby and the caption, "Before you scold me, Mom … maybe you'd better light up a Marlboro."[1] The following decades saw advertising slogans like, "You've come a long way, baby," "It's a woman thing," "Light and luscious," and so on. The tobacco industry developed cigarettes for women (such as Virginia Slims, Eve, Kim, and Satin) and promoted smoking to women and girls as a sign of liberation, independence, self-confidence, glamour, adventure, exclusivity, and success.[2,3] Smoking rates among women peaked in the mid-1960s; they still remain substantial. The 2006 National Youth Tobacco Survey (United States) reported the then-current smoking prevalence of 21.3% (95% CI, 19.2%-23.4%) in high school girls, a rate that was not significantly changed from the 2004 survey.[4] A substantial proportion of women continue to enter their reproductive years tobacco dependent.

In utero tobacco smoke exposure has been repeatedly found to be associated with increased risk for premature birth, low birth weight, and sudden infant death syndrome.[5] Animal models suggest potential mechanisms for adverse effects of in utero tobacco smoke exposure on breathing, including alterations in ventilatory control and arousal responses,[6] reduction in lung volume,[7] and alterations in branching morphogenesis of the airways.[8]

Maternal smoking is associated with childhood asthma,[9] however the effects of prenatal vs postnatal smoking have been difficult to differentiate.[10] Previous studies demonstrating an impact of in utero tobacco smoke exposure on childhood wheezing have used retrospectively collected data.[11,12]

The results from the Generation R study by Duijts et al,[13] presented in this issue of *CHEST* (see page 876), help to document the role of in utero tobacco smoke exposure on childhood asthma. The researchers involved in this large, prospective cohort study performed assessments for smoking in each trimester of pregnancy and assessments for wheezing at 1, 2, 3, and 4 years after birth. There were sufficient cohorts with in utero only (n = 200), postnatal only (n = 323), and both pregnancy and postnatal parameters (n = 310) for independent effects to be discerned. They found in utero tobacco smoke exposure only, in utero and postnatal smoke exposure, and postnatal smoke exposure all associated with increased risk of wheezing in the offspring compared with those of nonsmoking mothers. The effect of first trimester smoking on wheezing was not statistically significant; the study was not powered to detect small effects.

These results document the impact of maternal tobacco smoking, both in the womb and outside of the womb, on wheezing in the offspring. As prospectively collected data, these results are not subject to recall bias. These findings are consistent with prior laboratory research demonstrating an effect of nicotine on lung development[7,8] and population studies demonstrating an effect of in utero tobacco smoke exposure on later wheezing using retrospective assessments of in utero tobacco smoke exposure.[11,12] These results also demonstrate that the adverse impact of maternal smoking for the offspring is not limited to the time in the womb; the adverse impact of maternal smoking on childhood wheezing continues after birth.

These results provide additional evidence of the importance of reducing smoking in women of reproductive age. Smoking cessation either before pregnancy or early in gestation among women who are tobacco dependent can minimize the harm to their offspring.

Tobacco is a highly addictive substance. Highly effective treatments for tobacco dependence are available.[14] Although not free of adverse effect, tobacco-dependence treatment medications are preferable to continued in utero smoke exposure, which has well-defined harms to the fetus. Health-care providers for women of reproductive age and women who are pregnant must be prepared to offer tobacco-dependence treatment.

Reduction of smoking in women of reproductive age needs to be a public health priority. Part of this must include reduction or elimination of tobacco promotion to children. Close to 90% of current smokers started smoking before 18 years of age. Exposure of children to smoking in the movies and to retail tobacco displays is associated with increased smoking rates in children. Public health campaigns focused on changing social norms related to tobacco and changing the images associated with tobacco use have had substantial success where they have been tried.[15] California has reduced smoking prevalence by 35% by focusing on four strategies, including reducing the marketing of tobacco products, restricting smoking in public places and workplaces, enforcement of laws prohibiting the sale of tobacco to minors, and support of tobacco-dependence treatment services.[16]

The results of the Generation R study add to the overwhelming evidence of harm of in utero tobacco smoke exposure and highlight the critical importance of effective tobacco-control programs. When girls and young women do not take up smoking, their future

offspring will be protected from these harms. When physicians offer tobacco-dependence treatment to women before they get pregnant or early in their pregnancy, harm can be reduced.

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Portopulmonary Hypertension

Light at the End of the Tunnel?

Portopulmonary hypertension (PoPH) is defined as pulmonary arterial hypertension (PAH) associated with portal hypertension, with or without liver disease.[1] As is the case for all forms of PAH, PoPH is a life-threatening disease characterized by a marked and sustained elevation of pulmonary vascular resistance, leading to increased pulmonary artery pressure, right ventricular failure, and ultimately death.[2] Prospective hemodynamic studies have shown that 2% to 6% of patients with portal hypertension develop significant pulmonary hypertension. Furthermore, this risk is independent of the presence or the severity of associated liver disease.[3,4] PoPH accounts for approximately 7% to 10% of the overall PAH population.[5] However, with increasing awareness of the disorder, this proportion is gradually increasing. Indeed, PoPH represents approximately 20% of all PAH cases in the recent French PAH registry and it is the most common form of nonidiopathic PAH.[6]

The presence of PAH in patients with liver disease contributes significantly to morbidity and mortality at the time of liver transplant.[1] However, there is considerable variation in published survival estimates for patients with PoPH who do not require a liver transplant.[7,8] In a small series reported by Kawut et al,[7] the overall transplant-free 3-year survival rates for patients with PoPH was 38%, worse than that observed in patients with idiopathic PAH (IPAH). In contrast, Le Pavec et al[8] found that 1-, 3- and 5-year survival rates were 88%, 75%, and 68%, respectively, among patients followed at the French national referral center, suggesting that these patients may, in fact, have less severe outcomes than those with IPAH.

In the current issue of *CHEST* (see page 906), Krowka et al[9] describe the demographics, medical history, and outcomes of patients with PoPH enrolled in the Registry to Evaluate Early and Long-term Pulmonary Arterial Hypertension Disease Management

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