First, did these pregnant smokers fail to quit smoking despite proactive treatment for cessation by a trained multidisciplinary team? Smoking cessation may be the most important health intervention during pregnancy because smoking is the most preventable cause of preterm birth and all other complications during pregnancies except hypertensive pregnancy. Proactive treatment means motivational interview and psychological support plus nicotine replacement therapy. The former is a prerequisite for the effectiveness of the latter because smokers need reassurance first. Indeed, tobacco is the worst addictive product: all smokers have made serial attempts to quit, almost always failing with much pain, and further smoking increases the odds of depressive symptoms. The latter must combine patches with faster-acting forms (lozenge or spray) of nicotine replacement therapy. This “belt and braces” strategy has been shown to double the odds ratio of quitting during pregnancy. Sadly, this proactive treatment is poorly implemented despite being robustly evidence based.

Second, did these pregnant smokers receive at least nicotine replacement therapy during the trial, and at what dose? Indeed, it is less dangerous to smoke with patches than without, as it decreases not only the number of cigarettes but also the puff volumes. Although the precautionary principle and experimental findings pledge for avoiding nicotine per second during pregnancy (6), the issue for pregnant smokers and their unborn child is different, being about harm/benefit ratio. The main problems are carbon monoxide (which has strong affinity for fetal hemoglobin), tar, and hundreds of extremely toxic combustion byproducts. Moreover, the devastating effects of compensatory uptake (deeper puffs with higher temperature) when trying to reduce tobacco use without nicotine substitutes cannot be overlooked. Considering the very high urine cotinine level in the trial (1), this compensatory uptake seems most likely. Could McEvoy and colleagues provide data about the carbon monoxide breath test?

Vitamin C to Pregnant Smokers and Infant Airway Function: Missing the Forest for the Trees?

To the Editor:

The effect of maternal smoking during pregnancy on child lung function during the first months of life is an old concern (1). Serious consequences range from sudden infant death syndrome to asthma (2, 3). McEvoy and colleagues’ randomized controlled trial showing that adding daily supplemental vitamin C for pregnant smokers improves their infant’s newborn pulmonary function may be a step forward, but it deserved questions (4).

First, did these pregnant smokers fail to quit smoking despite proactive treatment for cessation by a trained multidisciplinary team? Smoking cessation may be the most important health intervention during pregnancy because smoking is the most preventable cause of preterm birth and all other complications during pregnancies except hypertensive pregnancy. Proactive treatment means motivational interview and psychological support plus nicotine replacement therapy. The former is a prerequisite for the effectiveness of the latter because smokers need reassurance first. Indeed, tobacco is the worst addictive product: all smokers have made serial attempts to quit, almost always failing with much pain, and further smoking increases the odds of depressive symptoms. The latter must combine patches with faster-acting forms (lozenge or spray) of nicotine replacement therapy. This “belt and braces” strategy has been shown to double the odds ratio of quitting during pregnancy. Sadly, this proactive treatment is poorly implemented despite being robustly evidence based (5).

Second, did these pregnant smokers receive at least nicotine replacement therapy during the trial, and at what dose? Indeed, it is less dangerous to smoke with patches than without, as it decreases not only the number of cigarettes but also the puff volumes. Although the precautionary principle and experimental findings pledge for avoiding nicotine per second during pregnancy (6), the issue for pregnant smokers and their unborn child is different, being about harm/benefit ratio. The main problems are carbon monoxide (which has strong affinity for fetal hemoglobin), tar, and hundreds of extremely toxic combustion byproducts. Moreover, the devastating effects of compensatory uptake (deeper puffs with higher temperature) when trying to reduce tobacco use without nicotine substitutes cannot be overlooked. Considering the very high urine cotinine level in the trial (1), this compensatory uptake seems most likely. Could McEvoy and colleagues provide data about the carbon monoxide breath test?

References


Copyright © 2019 by the American Thoracic Society

Vitamin C to Pregnant Smokers and Infant Airway Function: Missing the Forest for the Trees?

To the Editor:

The effect of maternal smoking during pregnancy on child lung function during the first months of life is an old concern (1). Serious consequences range from sudden infant death syndrome to asthma (2, 3). McEvoy and colleagues’ randomized controlled trial showing that adding daily supplemental vitamin C for pregnant smokers improves their infant’s newborn pulmonary function may be a step forward, but it deserved questions (4).

First, did these pregnant smokers fail to quit smoking despite proactive treatment for cessation by a trained multidisciplinary team?

Copyright © 2019 by the American Thoracic Society

**Reply to Braillon**

*From the Authors:*

We thank Dr. Braillon for his letter and comments in response to our recent publication (1). We agree that smoking cessation should be the foremost goal for pregnant smokers, as smoking during pregnancy is the largest preventable cause of perinatal morbidity and mortality (2). We share his commitment to improving the outcomes of both pregnant smokers and their offspring.

The letter’s initial concern revolves around the provision of proactive treatment for cessation, including a motivational interview and psychological support plus nicotine replacement therapy (NRT). This was a randomized trial to determine the ability of vitamin C supplementation in pregnant smokers to improve their offspring’s lung function, and not a smoking cessation trial. However, smoking cessation was encouraged and participants were educated about the negative effects of smoking at randomization and at each monthly prenatal visit under the guidance of Dr. David Gonzales of the Oregon Health & Science University Smoking Cessation Center (a co-author of this letter and a co-investigator on the study). The guidelines of the American College of Obstetrics and Gynecology (2) and the U.S. Public Health Service Clinical Practice Guidelines (3) for the management of smoking during pregnancy were followed and included the provision of the “5 A’s” for smoking intervention (ask, advise, assess, assist, and arrange), distribution of pregnancy-specific smoking cessation pamphlets, certification of research staff in smoking cessation, and completion of monthly smoking questionnaires with education.

We did not provide a motivational interviewing–specific intervention, and instead opted for a more standard behavioral intervention that also included health education regarding the risks of smoking during pregnancy. Recent data suggest that motivational interviewing has no incremental benefit over standard behavioral support for cessation during pregnancy (4). Furthermore, there are data that suggest that a health education intervention may be more efficacious than motivational interviewing for individuals with a lower willingness to quit smoking (5). NRT was not included in the study because it is not approved in the United States by the Food and Drug Administration, the American College of Obstetrics and Gynecology, or the U.S. Preventive Services Task Force for use in pregnancy (2, 3). Ultimately, the participants in the study received more smoking cessation counseling than would have normally been provided, and 10% of randomized smokers quit smoking during pregnancy as per monthly respiratory questionnaires and biochemical markers.

The second point in the letter is in regard to the detrimental effects of carbon monoxide on fetal development and concerns about increased compensatory uptake by randomized pregnant smokers not given NRT. Although we agree that carbon monoxide and other combustibles likely have deleterious effects, we have preclinical data demonstrating that nicotine is the primary mediator of the effects of *in utero* smoke on fetal lung development (6). Serial carbon monoxide levels in the randomized pregnant smokers decreased from a median of 11 ppm at randomization to a median of 10 ppm at midgestation and a median of 9 ppm during late gestation, mirroring the general decrease in the number of cigarettes smoked per day.

Although the primary goal should always be complete smoking cessation, progress in this area may be incremental given the large societal issues underlying smoking during pregnancy in the United States. We hope our findings regarding the potentially beneficial effects of vitamin C supplementation in pregnant smokers will help establish a simple, safe, and inexpensive way (in addition to continued smoking cessation interventions) to decrease the negative effects of *in utero* smoke on fetal lung development. Future studies may combine vitamin C, cessation counseling, and NRT products.

**Author disclosures** are available with the text of this letter at www.atsjournals.org.

Cindy T. McEvoy, M.D., M.C.R.*
Oregon Health & Science University
Portland, Oregon

Robert S. Tepper, M.D., Ph.D.
Indiana University School of Medicine
Indianapolis, Indiana

David Gonzales, Ph.D.
Oregon Health & Science University
Portland, Oregon

Eliot R. Spindel, M.D., Ph.D.
Oregon National Primate Research Center
Beaverton, Oregon

Cynthia D. Morris, Ph.D., M.P.H.
Oregon Health & Science University
Portland, Oregon

ORCID ID: 0000-0001-8501-8813 (C.T.M.).

*Corresponding author (e-mail: mcevoyc@ohsu.edu).

**References**


2. Committee on Underserved Women; Committee on Obstetric Practice. Committee Opinion No. 721: smoking cessation during pregnancy. *Obstet Gynecol* 2017;130:e200–e204.

