dent Barack Obama's promise in his 2013 State of the Union address to enact gun-control legislation was stymied, and the composition of the current Congress augurs ill for such federal legislation.

Surveys show that a strong majority of Americans want better gun control, and the medical community is solidly behind them. So what stands in the way? One factor is gun advocates' opposition to systematic collection of data on gun-related injuries. Another is a fervent belief by gun advocates that any registration of guns would lead to confiscation of their weapons by the federal government. The greatest obstacle by far, however, is the reality that gun-control advocates are "outgunned" by the National Rifle Association (NRA), which mobilizes its members with scare tactics and pours industry-sourced money into blocking any efforts at gun control at the city, state, and national level. Many congressional leaders are NRA members, many more are beholden to the organization for financial campaign support, and still others are intimidated by the power of the NRA to strengthen their election opponents.

We have been unsuccessful in buffering the stress that violence in the media imposes on us. We are also unlikely to see a decline in the risk of firearm-related violence unless the public elects lawmakers at all levels of government with the courage to defy gun lobbyists. Without deep cultural and regulatory changes, the chances of reducing gun deaths in the United States are slim indeed.

Disclosure forms provided by the author are available with the full text of this article at NEJM.org.

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Preventing Peanut Allergy through Early Consumption — Ready for Prime Time?

Rebecca S. Gruchalla, M.D., Ph.D., and Hugh A. Sampson, M.D.

Kids can't take peanut butter to school. Some airlines no longer serve peanuts because of fear of anaphylaxis among passengers. These developments are just the tip of the iceberg as the prevalence of peanut allergy among children continues to increase worldwide, especially in westernized countries. In the United States alone, the prevalence has more than quadrupled in the past 13 years, growing from 0.4% in 1997 to 1.4% in 2008[1] to more than 2% in 2010.[2] Peanut allergy has become the leading cause of anaphylaxis and death related to food allergy in the United States.[3]

In 2000, largely in response to outcomes reported in infant feeding trials conducted in Europe and the United States, the American Academy of Pediatrics (AAP) recommended that parents refrain from feeding peanuts to infants at risk for the development of atopic disease until the children reached 3 years of age.[4] However, since the number of cases of peanut allergy continued to rise, many investigators and clinicians began questioning this advice. In 2008, after reviewing the published literature, the AAP retracted its recommendation, stating that there was insufficient evidence to call for early food avoidance.[5] Shortly thereafter, Du Toit et al.[6] noted that the prevalence of peanut allergy among Jewish children in London who were not given peanut-based products in the first year of life was 10 times as high as that among Jewish children in Israel who had consumed peanut-based products before their first birthday. In addition, subsequent studies that evaluated the early introduction of other allergenic foods, including egg[7] and cow's milk,[8] showed that earlier introduction of egg and milk into an infant's diet was associated with a decrease in the development of allergy.

But since these studies were observational, we needed data from controlled trials to provide reliable clinical guidance regarding the best time to introduce allergenic foods (e.g., milk, egg, peanuts, and tree nuts) to infants at high risk for the development of allergies (i.e., those from atopic families). Du Toit et al.[9] now address this question in the *Journal* in their landmark study, Learning Early about Peanut Allergy (LEAP). The investigators hypothesized that early introduction of peanut-based products (before 11 months of age) would lead to the prevention of peanut allergy in high-risk infants. More than 500 infants at high risk for peanut allergy were randomly assigned to receive peanut products (consumption group) or to avoid them (avoidance group). Approximately 10% of children, in whom a wheal measuring more than 4 mm developed after they received a peanut-specific skin-prick test, were excluded from the study because of concerns that they would have severe reactions. At 5 years of age, the children were given a peanut challenge to determine the prevalence of peanut allergy. The results are striking — overall, the prevalence of peanut allergy in the peanut-avoidance group was 17.2% as compared with 3.2% in the consumption group.

The trial was designed to examine two groups — children who had negative results on the peanut skin-prick test at enrollment (nonsensitized) and those who had "mild" sensitization at enrollment (wheals with mean diameters of 1 to 4 mm in response to the test). In these two groups the results on the prevalence of peanut allergy were equally striking. Among the children who initially had a negative result on the skin-prick test, the prevalence of peanut allergy was 13.7% in the avoidance group and 1.9% in the consumption group, and among those who had mild sensitization the prevalence was 35.3% in the avoidance group versus 10.6% in the consumption group. Thus, early consumption was effective not only in high-risk infants who showed no indication of peanut sensitivity at study entry (primary prevention) but also in infants who had slight peanut sensitivity (secondary prevention).

Du Toit et al. carefully defined their high-risk population, which included children with severe eczema, egg allergy, or both. Moreover, they determined whether these infants were sensitized to peanut at study entry and then challenged those in the peanut-consumption group to ensure that these children were unresponsive before sending them home to consume peanut-based products on a regular basis.

Given the results of this prospective, randomized trial, which clearly indicates that the early introduction of peanut dramatically decreases the risk of development of peanut allergy (approximately 70 to 80%), should the guidelines be changed? Should we recommend introducing peanuts to all infants before they reach 11 months of age? Unfortunately, the answer is not that simple, and many questions remain unanswered: Do infants need to ingest 2 g of peanut protein (approximately eight peanuts) three times a week on a regular basis for 5 years, or will it suffice to consume lesser amounts on a more intermittent basis for a shorter period of time? If regular peanut consumption is discontinued for a prolonged period, will tolerance persist? Can the findings of the LEAP study be applied to other foods, such as milk, eggs, and tree nuts?

These questions must be addressed, but we believe that because the results of this trial are so compelling, and the problem of the increasing prevalence of peanut allergy so alarming, new guidelines should be forthcoming very soon. In the meantime, we suggest that any infant between 4 months and 8 months of age believed to be at risk for peanut allergy should undergo skin-prick testing for peanut. If the test results are negative, the child should be started on a diet that includes 2 g of peanut protein three times a week for at least 3 years, and if the results are positive but show mild sensitivity (i.e., the wheal measures 4 mm or less), the child should undergo a food challenge in which peanut is administered and the child's response observed by a physician who has experience performing a food challenge. Children who are nonreactive should then be started on the peanut-containing diet. Although other studies are urgently needed to address the many questions that remain, especially with respect to other foods, the LEAP study makes it clear that we can do something now to reverse the increasing prevalence of peanut allergy.

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Bariatric Surgery before Pregnancy — Is This a Solution to a Big Problem?

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Obesity has reached epidemic levels in the United States. According to data from the Centers for Disease Control and Prevention (CDC), in 1990 no state in the United States had a prevalence of obesity of 15% or more, but by 2010, no state had a prevalence of obesity below 20%, and a third of states had a prevalence of 30% or higher.[1] This trend extends to pregnancy; almost a third of women entering pregnancy in the United States are obese.[2] Moreover, the prevalence of extreme obesity is also increasing. For example, in pregnant women, the subcategories of obesity that are increasingly being studied include morbid obesity (body-mass index [BMI, the weight in kilograms divided by the square of the height in meters], 40 to 50) and super obesity (BMI, >50).[3] Thus, to reduce the negative effects of obesity, we need to reduce not only the overall frequency but also the proportion of women with a BMI that exceeds 40.

Obesity in women of reproductive age is important both because of the risks of associated downstream diseases (including type 2 diabetes and cardiovascular disease) and because obesity is associated with poor outcomes for both the pregnant woman and the fetus. Such outcomes include fetal anomalies, gestational diabetes, preeclampsia, preterm birth, post-term birth, cesarean delivery, fetal growth restriction, and fetal macrosomia; longer-term risks for offspring include childhood obesity and the metabolic syndrome.[4]

Interventions to promote weight loss before pregnancy would be expected to improve these outcomes. Unfortunately, the usual approaches of counseling about weight loss, an improved diet, and increased exercise generally have a minimal effect. Bariatric surgery commonly leads to greater and more sustained weight loss than do lifestyle approaches. Evidence that bariatric surgery can prevent,[5] treat,[6] or even "reverse"[7] type 2 diabetes has increased enthusiasm for this approach. However, there are also concerns that potential malnutrition or malabsorption resulting from these surgeries may lead to pregnancy complications.

In this issue of the *Journal*, Johansson et al. examine pregnancy outcomes in women who underwent bariatric surgery as compared with matched controls (early-pregnancy BMI in the control cohort was matched to presurgery BMI in the bariatric-surgery cohort).[8] As compared with controls, women with a history of bariatric surgery had significantly lower incidences of gestational diabetes (1.9% vs. 6.8%) and large-for-gestational-age neonates (8.6% vs. 22.4%).