be no substitute for focused attention to patients, being by
have that day as the tests are ordered. Although there can
corporated into the physician-patient encounter, as when
the patient bedside. Order writing and charting can be in-
by eliminating the search for open computers in physician
patients and at educational conferences. Besides saving time
in medical charting areas, tablets can encourage residents to
authors, the tablets seemed to increase the efficiency of resi-
during an average of 8½ years after the 3-year follow-up visit.
A total of 11,056 incident diabetes cases occurred during an average of 8½ years after the 3-year follow-up visit.
A total of 115,092 women without known diabetes were followed up from year 3 to diabetes diagnosis, date of death, loss to follow-up, or September 30, 2010, whichever occurred first. The definition of incident diabetes was a positive answer to questions regarding "newly prescribed treatment for diabetes with pills or insulin shots" or using "diet and/or exercise for diabetes" on any of the semiannual or annual follow-up questionnaires. Self-reported diabetes in the Women's Health Initiative has been validated by medication inventories and laboratory data as a reliable indicator of diagnosed diabetes. A total of 11,056 incident diabetes cases occurred during an average of 8½ years after the 3-year follow-up visit.

The main exposure included never smokers at both baseline and 3-year follow-up visit, former smokers at both baseline and follow-up visit, continuing smokers at both baseline and follow-up visit, and new quitters who smoked at baseline but were abstinent at the follow-up visit. A small proportion of women (0.6%) whose smoking status changed from never or former smokers at baseline to current smokers in year 3 were excluded. Weight was measured at both baseline and year 3 in 107,471 women. 10,380 of whom developed diabetes.

Cigarette smoking is associated with an increased risk of type 2 diabetes mellitus. However, smoking cessation is often accompanied by weight gain, which may explain the increased risk of diabetes that has been observed in several studies. Two studies with data on weight came to different conclusions about whether the increased risk of diabetes after smoking cessation is primarily attributable to postcessation weight gain. We used data from the Women's Health Initiative, a large prospective study with detailed information on smoking status, weight changes, and potential confounders, to assess the relationship between smoking cessation, weight gain, and subsequent diabetes risk. We examined diabetes risk by smoking status, including new quitters who smoked at baseline but no longer smoked at the 3-year follow-up visit. A total of 115,092 women without known diabetes were followed up from year 3 to diabetes diagnosis, date of death, loss to follow-up, or September 30, 2010, whichever occurred first. The definition of incident diabetes was a positive answer to questions regarding “newly prescribed treatment for diabetes with pills or insulin shots” or using “diet and/or exercise for diabetes” on any of the semiannual or annual follow-up questionnaires. Self-reported diabetes in the Women’s Health Initiative has been validated by medication inventories and laboratory data as a reliable indicator of diagnosed diabetes. A total of 11,056 incident diabetes cases occurred during an average of 8½ years after the 3-year follow-up visit.

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Cox proportional hazards regression models were used to estimate hazard ratios (HRs) and 95% confidence intervals (CIs) of diabetes risk by smoking status overall
and stratified by weight gain (categorized as <5 kg or ≥5 kg). In multivariable models, we adjusted for potential baseline confounders, including age at enrollment, race/ethnicity, education, body mass index, waist circumference, physical activity, alcohol intake, treatment for hypertension and high cholesterol levels, and participation in different Women’s Health Initiative study cohorts (observational study or clinical trials and different treatment assignments for all + clinical trials).

Table. Hazard Ratios (HRs) and 95% Confidence Intervals (CIs) for Incident Diabetes Since the 3-Year Follow-up Visit in Relation to Smoking Status at Baseline and Year 3 Visit (Overall and Stratified by Weight Gain)

<table>
<thead>
<tr>
<th>Smoking Status</th>
<th>No.</th>
<th>Cases</th>
<th>Multivariable-Adjusted, HR (95% CI)b</th>
<th>Multivariable-Adjusted, HR (95% CI)b</th>
<th>Weight Gain &lt;5kg</th>
<th>Cases</th>
<th>Multivariable-Adjusted, HR (95% CI)b</th>
<th>Weight Gain ≥5kg</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Never smokers</td>
<td>59,904</td>
<td>5735</td>
<td>1 [Reference]</td>
<td>1 [Reference]</td>
<td>4609</td>
<td>1 [Reference]</td>
<td>803 [Reference]</td>
<td>714</td>
<td>0.96 (0.86-1.07)</td>
</tr>
<tr>
<td>Former smokers (years since quitting)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;10</td>
<td>4949</td>
<td>598</td>
<td>1.16 (1.06-1.26)</td>
<td>1.14 (1.05-1.25)</td>
<td>431</td>
<td>1.15 (1.04-1.27)</td>
<td>125</td>
<td>1.12 (0.92-1.36)</td>
<td></td>
</tr>
<tr>
<td>10-20</td>
<td>12,062</td>
<td>1,224</td>
<td>1.02 (0.96-1.09)</td>
<td>1.02 (0.95-1.08)</td>
<td>955</td>
<td>1.05 (0.98-1.12)</td>
<td>183</td>
<td>0.88 (0.75-1.04)</td>
<td></td>
</tr>
<tr>
<td>≥30</td>
<td>11,419</td>
<td>1,022</td>
<td>0.95 (0.89-1.01)</td>
<td>0.94 (0.88-1.01)</td>
<td>791</td>
<td>0.94 (0.88-1.02)</td>
<td>171</td>
<td>0.95 (0.80-1.12)</td>
<td></td>
</tr>
<tr>
<td>P for trend</td>
<td>. . .</td>
<td>&lt;.001</td>
<td>. &lt;.001</td>
<td>. . .</td>
<td>. . .</td>
<td>. .</td>
<td>. .</td>
<td>. .</td>
<td></td>
</tr>
<tr>
<td>Continuing smokers</td>
<td>16,363</td>
<td>1,401</td>
<td>0.96 (0.90-1.02)</td>
<td>0.96 (0.90-1.02)</td>
<td>1122</td>
<td>0.96 (0.90-1.03)</td>
<td>195</td>
<td>0.98 (0.84-1.15)</td>
<td></td>
</tr>
<tr>
<td>New quitters</td>
<td>2,054</td>
<td>254</td>
<td>1.20 (1.09-1.31)</td>
<td>1.20 (1.10-1.32)</td>
<td>399</td>
<td>1.16 (1.04-1.29)</td>
<td>95</td>
<td>1.32 (1.04-1.64)</td>
<td></td>
</tr>
<tr>
<td>Former smokers (years since quitting)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td>95</td>
<td>1.32 (1.04-1.64)</td>
<td></td>
</tr>
</tbody>
</table>

a Multivariable model adjusted age at enrollment (<55, 55-59, 60-64, 65-69, 70-74, and ≥75 years); ethnicity (American Indian or Native Alaskan, Asian or Pacific Islander, black or African American, Hispanic/Latino, non-Hispanic white, and other); education (high school or less, some college/technical training, college or some postcollege, and master’s degree or higher); body mass index (calculated as weight in kilograms divided by height in meters squared) (<18.5, 18.5-24.9, 25.0-29.9, 30.0-34.9, 35.0-39.9, and ≥40); waist circumference (in continuous); physical activity as (<5, 5-<10, 10-<20, 20-<30, and ≥30 metabolic equivalent tasks per week); alcohol intake (nondrinker, past drinker, 1 drink/mo, 1 drink/mo—1 drink/wk, 1-<7 drinks/wk, and ≥7 drinks/wk); hypertension (yes, no); and high cholesterol level requiring pills (yes, no).

b Multivariable model adjusted for all factors in footnote 1 plus weight gain from baseline to year 3 (weight stayed within ±2.5 kg, weight gain 2.5-5 kg, weight loss 2.5-5 kg, and weight loss ≥5 kg).

Author Affiliations: Department of Community Medicine, Mary Babb Randolph Cancer Center, West Virginia University, Morgantown (Dr Luo); National Heart, Lung, and Blood Institute, National Institutes of Health, Department of Health and Human Services, Bethesda, Maryland (Dr Rossouw); Division of General Internal Medicine, University of California, Davis Medical Center, Sacramento (Dr Tong); Department of Community Health and Health Behavior, School of Public Health and Health Professions, University at Buffalo, and The State University of New York, Buffalo (Dr Giovinco); Department of Internal Medicine, Division of Geriatrics, David Geffen School of Medicine at UCLA, and Veterans Affairs Greater Los Angeles Healthcare System, Geriatrics Research Education and Clinical Center, Los Angeles, and the United States Department of Veterans Affairs, Washington, DC (Dr Geffen). Dr Rossouw is currently at Kaiser Permanente, Oakland, California, and Dr Luo is currently at Kaiser Permanente, Los Angeles, California.
California (Dr Lee); Division of Public Health Sciences, Fred Hutchinson Cancer Research Center, Seattle, Washington (Dr Chen); Preventive and Behavioral Medicine, University of Massachusetts Medical School, Worcester (Dr Ockene); Department of Public Health Sciences, University of California, Davis (Dr Qi); and HealthPartners Research Foundation, Minneapolis, Minnesota (Dr Margolis).

Correspondence: Dr Luo, Department of Community Medicine, Mary Babb Randolph Cancer Center, West Virginia University, PO Box 9190, Morgantown, WV 26506 (jluo@hs.csc.wvu.edu).

Author Contributions: Study concept and design: Margolis and Luo. Acquisition of data: Rossouw, Ockene, Margolis, and Luo. Analysis and interpretation of data: Rossouw, Tong, Giovino, Lee, Chen, Ockene, Qi, Margolis, and Luo. Drafting of the manuscript: Rossouw and Luo. Critical revision of the manuscript for important intellectual content: Rossouw, Tong, Giovino, Lee, Chen, Ockene, Qi, Margolis, and Luo. Statistical analysis: Qi and Luo. Obtained funding: Rossouw, Ockene, and Margolis. Study supervision: Rossouw and Margolis.

Financial Disclosure: None reported.


Obesity and Increased Risk for Oligozoospermia and Azoospermia

The global obesity epidemic parallels a decrease in male fertility. Yet, the association between body mass index (BMI) and sperm parameters remains controversial. A negative correlation between BMI and sperm concentration or total sperm count was shown by several reports1-3 but not documented by others.4-5 The purpose of this report was to update the level of evidence on the association between BMI and sperm count through a systematic review and meta-analysis.

Methods. A systematic review of available literature was conducted to investigate the impact of BMI on sperm count in men according to the Preferred Reporting Items for Systematic Reviews and Meta-analyses (PRISMA) statement. A PubMed and EMBASE search identified relevant studies published until October 2010. Authors of relevant studies were contacted by e-mail and asked to complete a standardized data form regarding total sperm counts according to BMI categories. Unpublished data obtained from patients followed at the Infertility Center of Jean Verdier Hospital, Bondy, France, between January 2007 and December 2010 were also included.

The following BMI categories were used for analyses: lower than 18.5, 18.5 to 24.9, 25.0 to 29.9, and 30.0 or higher (calculated as weight in kilograms divided by height in meters squared). Data were stratified according to total sperm count as having normozoospermia (≥40 × 10^6 spermatzoa per ejaculate), oligozoospermia (<40 × 10^6 but >0 spermatzoa per ejaculate), and azoospermia (absence of spermatzoa), as specified in World Health Organization guidelines.3 We performed random effects models to obtain summary estimates to account for interstudy variation. Studies were weighted according to an estimate of statistical size defined as the inverse of the variance of the log odds ratio (OR). Prevalent ORs and 95% confidence intervals are presented. We calculated the ORs of overweight and obese men presenting with oligozoospermia or azoospermia compared with normal-weight men.

Results. A total of 8873 articles were identified. In total, 31 articles were potentially appropriate to be included in the meta-analysis because they investigated the relationship between BMI and sperm parameters. A total of 14 eligible studies were included in the present meta-analysis, corresponding to a total study sample of 9779 individuals. Overweight men were at significantly increased odds of presenting with oligozoospermia (OR, 1.11; 95% CI, 1.01-1.20) or azoospermia (OR, 1.39; 95% CI, 0.98-1.97) compared with normal-weight men (Figure). Likewise, obese men were at increased risk of oligozoospermia (OR, 1.42; 95% CI, 1.12-1.79) or azoospermia (OR, 1.81; 95% CI, 1.23-2.66) compared with normal-weight men (Figure).

Comment. This meta-analysis based on 9779 men showed an inverse association between overweight or obesity and abnormal sperm count. This relationship may be explained by different pathophysiological hypotheses: (1) hypogonadotropic hypergonadotropic hypogonadism due to aromatization of steroids in estrogens in peripheral tissues; (2) direct alterations of spermatogenesis and Sertoli cell function; (3) hip, abdominal, and scrotal fat-tissue accumulation leading to the increase of scrotal temperature; and (4) accumulation of toxic substances and liposoluble endocrine disruptors in fatty tissue.2

Our strategy based on individual patient data and analysis of dichotomized sperm count made it possible to have a more homogeneous meta-analysis of the available evidence. Limitations of our study are the exclusion of 15 studies because of incomplete data or lack of response from authors and the variations in the study populations. Yet, this variability suggests that our findings may...