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1. Beyond duty hour reform: redefining the learning environment. <http://www.im.org/AcademicAffairs/PSI/Documents/09-06-04%20Beyond%20Duty%20Hour%20Reform%20FINAL%20Report.pdf>. Accessed May 16, 2011.
2. Arora VM, Georgitis E, Siddique J, et al. Association of workload of on-call medical interns with on-call sleep duration, shift duration, and participation in educational activities. *JAMA*. 2008;300(10):1146-1153.
3. Osheroff JA, Forsythe DE, Buchanan BG, Bankowitz RA, Blumenfeld BH, Miller RA. Physicians' information needs: analysis of questions posed during clinical teaching. *Ann Intern Med*. 1991;114(7):576-581.
4. Zeng Q, Cimino JJ, Zou KH. Providing concept-oriented views for clinical data using a knowledge-based system: an evaluation. *J Am Med Inform Assoc*. 2002;9(3):294-305.
5. Verghese A. Culture shock: patient as icon, icon as patient. *N Engl J Med*. 2008;359(26):2748-2751.

EDITOR'S NOTE

Mobile Tablets: Benefits to Residents and Patients

I liked this Research Letter because I believe that mobile tablets may provide a number of useful benefits to the inpatient medicine service. As reported by the authors, the tablets seemed to increase the efficiency of residents, thereby potentially allowing for more time with patients and at educational conferences. Besides saving time by eliminating the search for open computers in physician medical charting areas, tablets can encourage residents to do more of their order writing and charting while sitting at the patient bedside. Order writing and charting can be incorporated into the physician-patient encounter, as when a resident discusses with a patient what tests he or she will have that day as the tests are ordered. Although there can be no substitute for focused attention to patients, being by the bedside using a tablet seems to me better than being in

a distant charting room. I also believe tablets may increase patient confidentiality by eliminating the possibility of common room computers being left signed on with patient data accessible when residents leave the room to respond to an inquiry or obtain a needed piece of information.

As the price of tablets declines and the power of information management and decision support grows, I believe that the value of tablets on the inpatient service will only increase—perhaps in ways we cannot currently even imagine.

Mitchell H. Katz, MD

RESEARCH LETTERS

Smoking Cessation, Weight Gain, and Risk of Type 2 Diabetes Mellitus Among Postmenopausal Women

Cigarette smoking is associated with an increased risk of type 2 diabetes mellitus.¹ However, smoking cessation is often accompanied by weight gain, which may explain the increased risk of diabetes that has been observed in several studies.²⁻⁶ 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.^{3,4} We used data from the Women's Health Initiative,⁷ 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.⁸ 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.

Cox proportional hazards regression models were used to estimate hazard ratios (HRs) and 95% confidence intervals (CIs) of diabetes risk by smoking status overall

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)

Smoking Status	Overall				Weight Gain <5kg		Weight Gain ≥5kg	
	No.	Cases	Multivariable-Adjusted, HR (95% CI) ^a	Multivariable-Adjusted, HR (95% CI) ^b	Cases	Multivariable-Adjusted, HR (95% CI) ^a	Cases	Multivariable-Adjusted, HR (95% CI) ^b
Never smokers	59 904	5735	1 [Reference]	1 [Reference]	4609	1 [Reference]	803	1 [Reference]
Former smokers (years since quitting)	47 799	4532	1.00 (0.96-1.04)	0.99 (0.95-1.03)	3528	1.00 (0.96-1.05)	714	0.96 (0.86-1.07)
<10	4949	598	1.16 (1.06-1.26)	1.14 (1.05-1.25)	431	1.15 (1.04-1.27)	125	1.12 (0.92-1.36)
10-<20	12 062	1224	1.02 (0.96-1.09)	1.02 (0.95-1.08)	955	1.05 (0.98-1.12)	183	0.88 (0.75-1.04)
20-<30	11 419	1022	0.95 (0.89-1.01)	0.94 (0.88-1.01)	791	0.94 (0.88-1.02)	171	0.95 (0.80-1.12)
≥30	16 363	1401	0.96 (0.90-1.02)	0.96 (0.90-1.02)	1122	0.96 (0.90-1.03)	195	0.98 (0.84-1.15)
<i>P</i> for trend	<.001	<.001	...	<.00175
Continuing smokers	5335	535	1.20 (1.09-1.31)	1.20 (1.10-1.32)	399	1.16 (1.04-1.29)	95	1.32 (1.06-1.64)
New quitters	2054	254	1.43 (1.26-1.63)	1.36 (1.19-1.54)	122	1.17 (0.98-1.41)	110	1.67 (1.36-2.05)

^aMultivariable 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).

^bMultivariable 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 gain ≥5 kg, weight loss 2.5-<5 kg, and weight loss ≥5 kg).

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 4 clinical trials).

Compared with never smokers, the HR for incident diabetes was 1.00 (95% CI, 0.96-1.04) for former smokers, 1.20 (95% CI, 1.09-1.31) for continuing smokers, and 1.43 (95% CI, 1.26-1.63) for new quitters after confounders were adjusted for. During the first 3 years of follow-up, a weight gain greater than 5 kg occurred in 10.2% of never smokers, 11.6% of former smokers, 12.3% of continuing smokers, and 30.5% of new quitters (median weight gain, 2.9 kg vs 0.3-0.5 kg in all other groups of smoking status). After weight gain was adjusted for (**Table**), the risk of diabetes remained elevated among continuing smokers (HR, 1.20; 95% CI, 1.10-1.32) and attenuated slightly but remained significantly elevated among new quitters (HR, 1.36; 95% CI, 1.19-1.54). Among former smokers, the diabetes risk decreased significantly as the time since quitting increased, and the risk was similar to that of never smokers after a cessation period of 10 years. We examined whether the increased risk of diabetes in new quitters was explained by weight gain in the analysis stratified by weight gain (**Table**). Among former smokers, the risk of diabetes was not greater compared with never smokers with similar weight gain. Among continuing smokers, the risk of diabetes was significantly increased compared with never smokers, both in women who gained 5 kg or more and in those who did not (*P* for interaction, 0.4). Among new quitters, the risk of diabetes among women who gained less than 5 kg was similar to the risk in continuing smokers, although it was not significantly elevated in this relatively small subgroup. However, among new quitters who gained

5 kg or more, there was a 67% excess risk of diabetes compared with nonsmokers (*P* for interaction, .02).

Our prospective analysis over 8½ years of follow-up shows that the increased risk of diabetes associated with quitting is confined to a subgroup that gains at least 5 kg. Our data in former smokers suggest that diabetes risk is likely to return to that in never smokers after about 10 years, independent of more recent weight gain. Since weight gain after smoking cessation can be prevented by regular moderate physical activity and dietary modification,⁹ smokers should not be deterred from quitting by concerns about an increase in the risk of diabetes.

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1. Willi C, Bodenmann P, Ghali WA, Faris PD, Cornuz J. Active smoking and the risk of type 2 diabetes: a systematic review and meta-analysis. *JAMA*. 2007; 298(22):2654-2664.
2. Hur NW, Kim HC, Nam CM, Jee SH, Lee HC, Suh I. Smoking cessation and risk of type 2 diabetes mellitus: Korea Medical Insurance Corporation Study. *Eur J Cardiovasc Prev Rehabil*. 2007;14(2):244-249.
3. Wannamethee SG, Shaper AG, Perry IJ; British Regional Heart Study. Smoking as a modifiable risk factor for type 2 diabetes in middle-aged men. *Diabetes Care*. 2001;24(9):1590-1595.
4. Yeh HC, Duncan BB, Schmidt MI, Wang NY, Brancati FL. Smoking, smoking cessation, and risk for type 2 diabetes mellitus: a cohort study. *Ann Intern Med*. 2010;152(1):10-17.
5. Will JC, Galuska DA, Ford ES, Mokdad A, Calle EE. Cigarette smoking and diabetes mellitus: evidence of a positive association from a large prospective cohort study. *Int J Epidemiol*. 2001;30(3):540-546.
6. Zhang LX, Curhan GC, Hu FB, Rimm EB, Forman JP. Association between passive and active smoking and incident type 2 diabetes in women. *Diabetes Care*. 2011;34(4):892-897.
7. The Women's Health Initiative Study Group. Design of the Women's Health Initiative clinical trial and observational study. *Control Clin Trials*. 1998; 19(1):61-109.
8. Margolis KL, Lihong Qi, Brzyski R, et al; Women Health Initiative Investigators. Validity of diabetes self-reports in the Women's Health Initiative: comparison with medication inventories and fasting glucose measurements. *Clin Trials*. 2008;5(3):240-247.
9. Spring B, Howe D, Berendsen M, et al. Behavioral intervention to promote smoking cessation and prevent weight gain: a systematic review and meta-analysis. *Addiction*. 2009;104(9):1472-1486.

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 reports^{1,2} but not documented by others.^{3,4} 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 ($\geq 40 \times 10^6$ spermatozoa per ejaculate), oligozoospermia ($< 40 \times 10^6$ but > 0 spermatozoa per ejaculate), and azoospermia (absence of spermatozoa), as specified in World Health Organization guidelines.⁵ 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 hyperestrogenic hypogonadism due to aromatization of steroids in estrogens in peripheral tissues⁶; (2) direct alterations of spermatogenesis and Sertoli cell function⁷; (3) hip, abdominal, and scrotal fat-tissue accumulation leading to the increase of scrotal temperature⁸; 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