EVIDENCE-BASED CARE SHEET

Abdominal Obesity and Smoking

What We Know

› Smoking and obesity are both serious public health concerns that are associated with cardiovascular diseases (CVD), cancer, metabolic abnormalities, and premature mortality. The combination of smoking and obesity further increases the risk of mortality, particularly from CVD. The life expectancy of an obese smoker is estimated to be 13 years fewer than that of a normal-weight smoker[2,5,7]

- Smoking initiation—especially among adolescents and women—is commonly related to weight concerns in individuals who view smoking as an effective means of weight control. Fear of gaining weight can be an impediment to smoking cessation. Nevertheless, the relationship between smoking and obesity is complex and incompletely understood[2,7]
  - Smokers tend to have a lower BMI—likely due at least in part to a combination of nicotine-induced acute increase in metabolism and reduction in appetite—and smoking cessation often leads to weight gain, but heavy smoking is associated with increased risk of obesity[7]
    - In addition, accumulating evidence suggests that smoking is associated with abdominal obesity, which reflects visceral fat deposition and is associated with an increased risk of CVD, diabetes mellitus, type 2 (DM2), hypertension, dyslipidemia, hyperglycemia, insulin resistance, and mortality compared to overall obesity. Indeed, even among individuals with a BMI in the normal range, higher waist circumference or waist-to-hip ratio is associated with increased risk for obesity-relatedcomplications and diseases[2,5,7]

› Authors of at least one large cohort study, however, found that current smokers in comparison to those who had never smoked had less abdominal obesity.[7] Research also points to a positive association between cigarette smoking and the prevalence of obesity in adolescent boys and girls[1]

› The underlying mechanism or mechanisms linking smoking and abdominal obesity have yet to be proven, but several possible mechanisms—including the direct effects of nicotine and the effects of smoking on sexhormone and other hormone levels—have been identified[4]

- Nicotine may lead to fat accumulation through a variety of mechanisms, including induction of insulin resistance and increased levels of stress hormones such as cortisol, which is associated with abdominal obesity and insulin resistance
- Adipocytokines, endocrine factors produced and secreted by adipocytes, have been linked to the pathophysiology of obesity. One adipocytokine is the hormone adiponectin. Adiponectin is believed to improve metabolism and to protect against heart disease since it is anti-inflammatory and anti-arteriosclerotic. Adiponectin has also been closely linked to smoking. Some studies have shown that in comparison to nonsmokers, smokers had lower levels of adiponectin. Yet other studies have found that adiponectin levels of former smokers were higher than smokers but lower than in individuals who had never smoked[4]
  - In a study appraising the changes in serum adiponectin levels one year after smoking cessation and the effect of adiponectin level changes on abdominal obesity, researchers

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found that adiponectin levels did not change after smoking cessation even though participants experienced weight gain and increased abdominal obesity. However, patients who had less abdominal obesity one year after smoking cessation experienced increased adiponectin levels

› Consequences of abdominal obesity in smokers include increased risk of DM2 and metabolic syndrome (MetS; i.e., a constellation of metabolic abnormalities—including abdominal obesity, hypertriglyceridemia, low levels of high-density lipoprotein [HDL] cholesterol, hypertension, and hyperinsulinemia—that increase a person's likelihood of developing CVD and DM2)[3,6,8]

- In a recent population-based study including 3,598 participants in Jiangsu, China, researchers found that the combination of current smoking and abdominal obesity was associated with a 2.8-foldincreased risk of developing DM2[6]
- In a study of 2,675 Japanese males with DM2, there was a positive correlation between heavy smoking and MetS among nondrinkers, due mainly to higher risk of abdominal obesity and dyslipidemia[8]
- Researchers who analyzed data from 11,559 participants in the Korean National Health and Nutrition Examination Surveys 2008–2010 found that MetS was significantly more common in smokers than in nonsmokers. Components of MetS that occurred more frequently in smokers than in nonsmokers included low HDL cholesterol and high triglycerides in men and abdominal obesity and high triglycerides in women[3]

› Although persons who quit smoking are likely to gain weight, and fear of gaining weight is a possible impediment to smoking cessation, a long period of smoking cessation may reduce abdominal obesity to a level similar to that of persons who never smoked[2]

What We Can Do

› Learn about the link between abdominal obesity and smoking so you can accurately assess your patients' personal characteristics and health education needs; share this information with your colleagues

› Identify patients who smoke, educate them about the hazards of smoking (including abdominal obesity) and the benefits of quitting smoking, and assist them with smoking cessation (e.g., by providing support, educating about nicotine replacement therapy, by requesting a referral to individual counseling or group therapy)

› Inform your patients that smoking is not an effective method of weight control and that, in the long term, smoking is associated with a higher risk for abdominal obesity and related health conditions (e.g., MetS, DM2, CVD)

Coding Matrix

References are rated using the following codes, listed in order of strength:

M	Published meta-analysis	**RV**	Published review of the literature	**PP**	Policies, procedures, protocols
SR	Published systematic or integrative literature review	**RU**	Published research utilization report	**X**	Practice exemplars, stories, opinions
RCT	Published research (randomized controlled trial)	**QI**	Published quality improvement report	**GI**	General or background information/texts/reports
R	Published research (not randomized controlled trial)	**L**	Legislation	**U**	Unpublished research, reviews, poster presentations or other such materials
C	Case histories, case studies	**PGR**	Published government report	**CP**	Conference proceedings, abstracts, presentation
G	Published guidelines	**PFR**	Published funded report		

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