ACUTE EFFECTS OF NICOTINE ON ENERGY BALANCE. Kenneth A. Perkins. Western Psychiatric Institute, University of Pittsburgh School of Medicine, Pittsburgh, PA.

Cigarette smoking is inversely related to body weight, demonstrating that smoking must cause an alteration in energy balance either by decreasing caloric intake, increasing caloric expenditure (especially metabolic rate), or both. Animal research strongly indicates that nicotine is the primary constituent of tobacco smoke which is responsible for smoking's effects on body weight. In order to more fully understand the role of nicotine in explaining smoking's effect in humans, our research effort has focused on the acute effects of nicotine on caloric intake and metabolic rate in male smokers using a measured-dose nasal spray method developed in our lab. We have found that nicotine intake typical of that of average smokers decreases caloric intake during an ad lib meal situation (without regard to sweet vs. nonsweet taste) and prolongs feelings of satiety following meal consumption. However, there is no effect of nicotine in altering taste preference or perception, apparently ruling this out as a possible mechanism to explain nicotine's appetite-suppressing effect. On the caloric expenditure side of the energy balance equation, we have found that nicotine produces a small but significant increase in metabolic rate at rest which is *enhanced* during light physical activity (typical of routine occupational or household tasks) but appears to be *reduced* in conjunction with the metabolic effects of meal consumption. Possible mechanisms responsible for these findings, an examination of chronic effects of smoking (i.e., smokers vs. nonsmokers) on energy balance, and results of newer research with female smokers will be discussed.

POSTCESSATION WEIGHT GAIN: CONTRIBUTIONS OF DIET AND LIPOPROTEIN LIPASE. Scott J. Leischow. Palo Alto Center for Pulmonary Disease Prevention; Maxine L. Stitzer, Andrew P. Goldberg and Ann Morrison. Johns Hopkins University Medical School, Baltimore, MD.

Weight gain is a common and usually unwanted result of smoking cessation, though the mechanisms contributing to the weight gain are unclear. The present study was designed to assess changes in caloric intake, metabolism, and physical activity as contributors to postcessation weight gain. Subjects were 16 healthy male smokers assigned in an inpatient setting to either smoke ad lib or quit smoking for a period of at least ten days after a four-day baseline period. Caloric intake and physical activity were assessed daily. Fasting levels of adipose tissue lipoprotein lipase (LPL), an enzyme that regulates the uptake and storage of triglycerides into fat cells, were assessed during the baseline period and at the end of the study. As expected, abstainers gained significantly more weight than smokers. Supporting earlier research, baseline LPL was positively correlated with the amount of postcessation weight gain in abstainers; however, the same relationship was found with smokers. Thus, the more adipose tissue LPL found during the baseline period, the greater the weight gain by the end of the study, regardless of whether a subject smoked or abstained. Caloric intake did not increase in smokers, but increased significantly over the course of the 10-day period in abstainers. On average, abstainers ate 578 more calories per day than smokers. When predicted daily caloric needs were compared with actual daily caloric intake, it was found that the excess caloric intake (above predicted daily needs) accounted for most of the weight gain. There was no difference between groups in physical activity. It is likely that increased caloric intake is the predominant contributor to postcessation weight gain, though a more fundamental metabolic process involving LPL may contribute to the increased caloric intake. The implications of this research on the mechanisms and prevention of postcessation weight gain will be discussed.

PHARMACOLOGIC INTERVENTION FOR POSTCESSATION WEIGHT GAIN: THE ROLE OF PHENYLPROPANOLAMINE. Robert C. Kleges. Memphis State University, Memphis, TN.

It has been well documented that smokers weigh less than nonsmokers, those who start smoking lose weight, and those who quit smoking gain weight. Most professional and lay people are aware of the weight-control properties of smoking. As such, the first purpose of the proposed presentation will be to overview the volitional use of smoking as a weight-control strategy. Recent empirical data, drawn from community surveys as well as prospective clinical trials, will demonstrate that the weight-control properties of smoking play an important role in both smoking maintenance as well as smoking relapse. Given the use of smoking as a weight-control strategy, it is important to develop effective interventions for reducing postcessation weight gain. One promising drug for the reduction of small amounts of weight is phenylpropanolamine (PPA). To evaluate the effect of PPA on postcessation weight gain, 57 smokers were randomly assigned, in a double-blind procedure, to chew gum with PPA, placebo gum, or no gum. After a baseline assessment, subjects were paid to quit smoking for a period of two weeks. Smoking cessation was verified bi-weekly, as well as random (spot), carbon monoxide assessment. Results indicated that 72% of smokers were successful in quitting smoking. Relative to the other two conditions, abstinent subjects receiving PPA gained significantly less ($p<0.05$) weight. Additionally, abstinence rates were significantly higher in subjects receiving PPA ($p<0.05$) relative to the other two conditions. Subjects in the PPA group also significantly reduced their dietary intake, relative to the other two groups. No changes in physical activity were observed. It is concluded that PPA may be an important method of reducing weight gain associated with smoking abstinence and may enhance smoking cessation efforts in certain individuals. The results of this investigation will be compared to other pharmacologic and nonpharmacologic approaches for reducing postcessation weight gain.

POSTSMOKING CESSATION WEIGHT GAIN: PREVENTIVE INTERVENTION WITH d-FENFLURAMINE. Bonnie Spring, Judith Wurtman, Ray Gleason, Richard Wurtman and Kenneth Kessler. The Chicago Medical School, North Chicago, IL.

Quitting smoking can result in weight gain, but the mechanisms responsible remain unclear. Several reports indicate that calorie intake increases after discontinuing the use of nicotine. Increases in carbohydrate and fat consumption have both been reported, but contradictory findings also exist. We directly measured calorie and macronutrient intakes before and after smoking cessation to test whether calorie and specifically carbohydrate (CHO) intake would increase and be associated with weight gain. In addition, we tested whether d-fenfluramine would suppress symptoms associated with withdrawal from smoking, including overeating and weight gain. Overweight, female chronic smokers ($n=31$), undergoing behavioral treatment to stop smoking were randomly assigned on a double-blind basis to receive d-fenfluramine (15 mg b.i.d.) or placebo for one week before quitting smoking and four weeks afterward. Direct measurements of calorie and nutrient consumption were made over three two-day periods prior to the initiation of drug treatment, 48 hours and 4 weeks after cessation. Weight was