

volume or puff number. These findings confirm the inference that smokers, at least when they are smoking cigarettes capable of delivering large amounts of nicotine, may be able to overcome the effects of mecamylamine through self-regulation of nicotine intake.

**PITUITARY HORMONE RESPONSE TO NICOTINE IN CIGARETTE SMOKERS.** Ovide F. Pomerleau, Cynthia S. Pomerleau and Mark J. Majchrzak. Dept. of Psychiatry, University of Michigan School of Medicine.

Integrating data from two experiments in our laboratory, we report the following pattern of pituitary hormone response to different levels of nicotine intake: At high plasma nicotine levels (mean peak nicotine in excess of 60 ng/ml) nausea occurred, and plasma levels of prolactin, adrenocorticotrophic hormone, beta-endorphin/beta-lipotropin, growth hormone, arginine vasopressin, and neurophysin I, increased significantly over pre-cigarette baselines without changes in thyroid stimulating hormone, luteinizing hormone, or follicle stimulating hormone. At intermediate plasma nicotine levels (mean peak nicotine between 30 and 40 ng/ml), no nausea occurred and a more selective pattern was observed, with significant elevations limited to beta-endorphin/beta-lipotropin, arginine vasopressin, and neurophysin I. At low plasma nicotine levels (mean peak nicotine less than 10 ng/ml), there were no significant hormonal elevations over baseline.

**New Fellows Address:** Conan Kornetsky, chair  
*Saturday, August 23, 11:00-11:50 a.m.*  
*Map Room, Washington Hilton*

**NEURAL MECHANISMS OF FEAR CONDITIONING MEASURED WITH THE ACOUSTIC STARTLE REFLEX.** Michael Davis. Dept. of Psychiatry, Yale University School of Medicine.

The acoustic startle reflex is an excellent model system to analyze how drugs alter both unconditioned and conditioned behavior. Startle is mediated by a simple neural circuit consisting of four central synapses. Startle is increased when elicited in the presence of cues previously paired with shocks. This effect is decreased by drugs such as diazepam, morphine, alcohol, clonidine, or buspirone. Small lesions of the central nucleus of the amygdala block fear-enhanced startle. Currently we are evaluating anatomical connections between the amygdala and the startle pathway and sites of action of drugs that are known to affect fear-enhanced startle.

**New Fellows Address:** Donald A. Overton, chair  
*Saturday, August 23, 1:00-1:50 p.m.*  
*Caucus Room, Washington Hilton*

**BEHAVIORAL PHARMACOLOGY OF NICOTINE DEPENDENCE.** Jack E. Henningfield, NIDA Addiction Research Center, Baltimore, MD.

An overview of tobacco and nicotine research that was conducted at The Johns Hopkins University School of Medicine and the National Institute on Drug Abuse, Addiction Research Center was presented. The primary focus of the Hopkins studies was to assess tobacco self-administration as any other form of drug self-administration would be studied, manipulating independent variables such as dose and reinforcement schedule value, while measuring dependent variables such as response rate and amount of substance obtained. The primary focus of the Addiction Research Center studies was to apply the methods used to assess the abuse liability and dependence potential of opioid like compounds in humans to evaluate nicotine. The main conclusion from the results of these studies was that tobacco dependence is an orderly, behavioral pharmacologic process, in which nicotine is critical.

**New Fellows Address:** Nancy A. Ator, chair  
*Monday, August 25, 3:00-3:50 p.m.*  
*Map Room, Washington Hilton*

**BEHAVIORAL PHARMACOLOGY OF OPIOID TOLERANCE.** Alice M. Young. Wayne State University.

My Fellows address will review psychological and pharmacological variables that modify the development and expression of tolerance to the behavioral actions of morphine and related opioids. Ongoing work from our laboratory will be used to illustrate how learning processes can modify tolerance development. Behavioral end-points discussed will include ongoing rates and patterns of schedule-controlled behavior, discriminative stimulus profiles, and analgesic effects. The implications of the impact of learning factors on tolerance development for our understanding of tolerance processes and the factors underlying opiate abuse will be considered.

**BEHAVIORAL TOLERANCE TO ALCOHOL: EXPECTANCIES AND INCENTIVES.** Muriel D. Vogel-Sprott. University of Waterloo.

Tolerance to alcohol refers to the observation that the intensity of response to a given dose diminishes after it is repeatedly administered. Such tolerance cannot be solely a function of drug exposures, for studies holding exposures constant indicate that environmental variables are also influential. This paper reviews one such research program which examines behavioral tolerance to a moderate dose of alcohol in social drinkers. These studies employ an instrumental learning paradigm to demonstrate that the outcomes of behavior under drug affect the acquisition, extinction and transfer of behavioral tolerance to alcohol. An interpretation in terms of contemporary cognitive learning theory is proposed, and practical implications for understanding and predicting behavioral tolerance or impairment in social drinkers is discussed.