A MULTIMODAL ASSESSMENT OF THE EFFECT OF CHEWING GUM ON NICOTINE WITHDRAWAL

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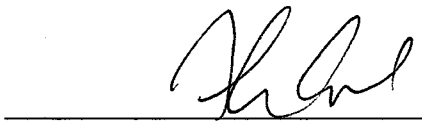

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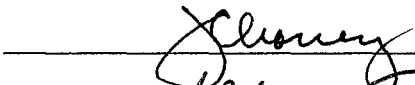

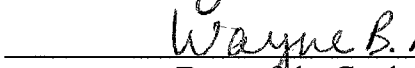

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TABLE OF CONTENTS

Chapter Page

LIST OF TABLES

LIST OF FIGURES

Abstract

The relationship between cigarette smoking and gum chewing is important to psychological research. Wrigley's has long believed that when smokers are in situations where smoking is prohibited, chewing gum will lessen their craving to smoke. This belief, however, was not backed up by scientific evidence until recently. In a previous study conducted in our laboratory (Cohen, Collins, & Britt, 1997), we found that based solely on self-report data, dependent cigarette smokers who were not permitted to smoke during a 4-hour time period, but were asked to chew gum, experienced significantly less total withdrawal symptoms when compared to a group of smokers who were not permitted chew gum during this time. In addition to reporting less overall withdrawal, those smokers that were permitted to chew gum reported less "craving" for a cigarette than their counterparts who did not have gum. These findings empirically validate Wrigley Chewing Gum commercials that assert "When you cannot smoke, chew gum." The present study was designed to examine the usefulness of the substitution of gum for cigarettes when a smoker is unable to smoke using both self-report and physiological (i.e., salivary cortisol) measures.

Twenty male smokers who reported smoking at least 16 cigarettes per day served as subjects. Potential subjects were called and asked to come to the lab for a brief orientation session where subjects were exposed to the laboratory setting. During this initial session, informed consent was obtained and subjects were given an idea of what to expect during the two experimental sessions. For each of the two experimental sessions, subjects were asked to relax for approximately 20 minutes upon arrival, provide a small sample of their saliva (sample 1) and smoke a cigarette. Upon completion of the

cigarette, subjects were again asked to relax for approximately 20 minutes and were asked to rate their current withdrawal symptoms by completing the WSC (Time 1). From this point forward, there was no access to cigarettes until the completion of the protocol. Next, subjects were asked to provide a small sample of their saliva (sample 2) and to watch a movie selected from a list. When the movie was over, subjects were asked to fill out the WSC (Time 2) and provide another sample of their saliva (sample 3). Subjects were then asked to remain in the lab and read magazines for one hour. During this hour, the research assistant came in once, half way through the period, where the subject was asked to fill out the WSC (Time 3) and provide another sample of their saliva (sample 4). At the end of the hour period the subject was again asked to fill out the WSC (Time 4) and provide a sample of their saliva (sample 5). During one of the experimental sessions, subjects had access to chewing gum from the start of the movie until the termination of the protocol, while on the other occasion they did not.

Results from this study are consistent with previous studies which show that chewing gum helps to reduce nicotine withdrawal when a nicotine dependent person cannot smoke. That is, when smokers were asked to chew gum they reported significantly less withdrawal as compared to sessions where they were not permitted to chew gum. In fact, as the withdrawal period lengthened, differences between the two experimental sessions (Gum Day and No-Gum Day) become more pronounced. When subjects were asked to chew gum, however, they did not report less "craving" for a cigarette compared to the sessions in which they were not permitted to chew gum. These findings suggest that chewing gum can produce a significant reduction in one's nicotine withdrawal but does little to influence craving for nicotine. Finally, this study examined

salivary cortisol as a potential physiological marker in the study of nicotine withdrawal. The results of these analyses were not very strong but did lend optimism for the use of salivary cortisol to be used as a physiological marker for nicotine withdrawal in future studies. In sum, considering both the self-report and the cortisol data, it appears that chewing gum helps with nicotine withdrawal at both a psychological and physiological level.

A Multimodal Assessment of the Effect of Chewing Gum on Nicotine Withdrawal

It is estimated that 32% of the American public smoked cigarettes in the past year, and nearly 27% have smoked cigarettes in the past month (National Institute on Drug Abuse [NIDA], 1994). Chronic cigarette smoking is associated with a number of serious medical illness including, cancer, coronary heart disease, and stroke. Given the high rate of cigarette consumption and the health problems related to their continued use, it should come as no surprise that cigarette smoking is the number one preventable cause of death in our society. It is directly responsible for approximately 390,000 deaths each year in the United States alone, which accounts for more than one out of every six deaths in our country (U.S. Department of Health & Human Services [DHHS], 1990). Despite the well-known health hazards, more than 50 million Americans continue to use tobacco products (US DHHS, 1988). Thus, smoking cessation could prevent a large number of deaths each year and defer the onset of a large number of these terminal illnesses. Many smokers find it difficult to stop using cigarettes and this is confirmed by the staggering rate that ex-smokers relapse. Of the seventeen million smokers that try to quit each year, fewer than 1 out of 10 actually succeed (Kessler, 1994).

During the past two decades, smoking cessation research has advanced notably in many different areas. For example, current designs and evaluations of treatments have become more theory driven, improved therapy process measures are used, and a variety of practical problems that were once a problem for researchers (i.e. subject attrition), have been reduced (US DHHS, 1988). Such improvements are recent however, and identify only a few published studies. Taking this information into account, it is

important to note that there are still aspects of cessation programs that have remained fairly stable over time, the most prominent being the low success rate (Lichtenstein & Glasgow, 1992). It is believed that smokers relapse for a variety of reasons, the main one being to relieve the withdrawal symptoms associated with smoking cessation (Gross & Stitzer, 1989).

It is now widely acknowledged that the cluster of symptoms observed following the cessation of smoking is largely due to the effects of nicotine withdrawal (US DHHS, 1988). There have been a number of studies that have defined the characteristics of nicotine withdrawal by examining the symptoms reported by smokers who initiated abstinence while being closely monitored on a research ward (Hatsukami et al., 1984) or in their natural environment (Hughes & Hatsukami, 1986; West & Russell, 1988; Shiffman & Jarvik, 1976). The symptoms that have been consistently identified are cravings for nicotine, irritability, restlessness, anxiety, difficulty concentrating, increased appetite or weight gain, decreased heart rate, and depressed mood (Stitzer & Gross, 1988). These withdrawal symptoms are thought to be an important factor contributing to the high rates of early relapse characteristic of people who attempt to quit smoking (Stitzer & Gross, 1988). At present, there is no comprehensive understanding of the natural time course and duration of nicotine withdrawal (Gross & Stitzer, 1989). What is known is that the subjective and physiologic symptoms of discomfort begin within 24 hours after smoking cessation (Gilbert & Pope, 1982), most subjective symptoms appear to peak within 48 hours (Hatsukami et al., 1984), and these symptoms then show steady decline during the first 3-4 weeks post cessation (West, Hajek & Belcher, 1987).

Another issue that continues to complicate the picture of nicotine withdrawal is the lack of consistency in the use of craving as a diagnostic criteria. Although the Diagnostic and Statistical Manual of Mental Disorders Third Edition-Revised (DSM-III-R; American Psychiatric Association [APA], 1987) included this symptom for nicotine withdrawal, the DSM-IV (APA, 1994) has excluded this symptom as a diagnostic criteria. The inclusion of craving for nicotine withdrawal but not for most other withdrawal syndromes in the DSM-III-R was taken to imply that craving is more closely tied to withdrawal from nicotine than for other drugs, however there is no data to support this idea (West & Kranzler, 1992). In addition, it is debatable whether craving during smoking cessation is actually influenced by nicotine administration (Hughes & Hatsukami, 1985), and craving appears to be controlled by nonpharmacological factors such as the environment (West & Schneider, 1987). For these reasons, craving was dropped from the DSM-IV.

Other data, however, suggest that craving for nicotine should not be dropped as a diagnostic criteria for nicotine withdrawal and that it is essential for researchers to examine this phenomena more closely. First, there are a number of studies that have found that craving is one of the more common and reliable effects of nicotine abstinence (Hughes, Gust & Skoog, 1991; Hughes & Hatsukami, 1986; Kozlowski & Wilkinson, 1987). Second, the construct of craving, if phrased correctly (e.g., thinking about smoking), has been shown to be relieved by nicotine (Schneider & Jarvik, 1985). Third, several studies have indicated that craving prospectively predicts relapse (Gritz, Carr & Marcus, 1991), and finally, given the prevalence of environmental cues for smoking

compared with cues for other drugs of abuse, it is indeed possible that this construct may be more present in the case of nicotine.

It makes sense therefore, that any empirically validated treatment program needs to focus on the withdrawal syndrome noted in the DSM-IV, as well as the construct of craving for a cigarette, since such a program could potentially be the most beneficial to the greatest number of people. One element that may prove useful in the treatment of nicotine withdrawal and craving for a cigarette is the use of chewing gum (Cohen, Collins & Britt, 1997). Recently, marketing promotions have suggested a link between cigarette smoking and chewing gum. William Wrigley, Jr., Co., one of the largest chewing gum manufacturers in the world, has spent a great deal of effort targeting smokers in their advertisements suggesting, "When you can't smoke chew gum". In our laboratory, we have data to suggest that William Wrigley, Jr., Co. may be on to something. We found that chewing gum not only helps with withdrawal, but reduces craving for a cigarette (Cohen et al., 1997). One criticism of the aforementioned study is that the data collected was entirely self-report data. The proposed study attempts to remedy this weakness by examining a physiological marker, in addition to the self-report measures used before, in order to add strength to the findings. Finding a physiological marker that taps into nicotine withdrawal, however, is a difficult task, given the imprecise nature of the construct. Given that increased levels of stress/distress is associated with nicotine withdrawal, it makes sense that we would choose to examine a physiological marker that has been shown to be sensitive to changes in a person's subjective levels of stress. One such marker is cortisol, the primary HPA peripheral hormone secreted by the adrenal cortex. It has been demonstrated that cortisol secretion rises predictably in

response to a number of stressors, and cortisol levels generally increase in proportion to the intensity of the stimulus (Kuhn, 1989). It would be expected, therefore, that a person experiencing nicotine withdrawal (and the associated stress/distress related to it) would have greater levels of cortisol when compared to a person that is not experiencing withdrawal. Along the same lines, if chewing gum helps with physiological withdrawal, one would expect to see lower levels of cortisol in abstinent smokers who chew gum when compared to abstinent smokers who do not chew gum.

The following review will first define and discuss the stress response, and will differentiate between the psychological and physiological components that make up this construct. The section on the psychological components will focus on the psychological measurement of stress, whereas the section devoted to the physiological components will address the body's response to stress, including health problems and the activation of the HPA axis, which leads to the release of cortisol, a hormone believed to be involved with the stress response. Second, a detailed description of the physiology of smoking will be presented, which includes a discussion of nicotine and its role in the maintenance of smoking behavior, as well as the behavioral and pharmacologic process that involves the maintenance of desired levels of nicotine in the body. This section will also discuss the relationship between smoking and cortisol and will define drug dependence and withdrawal. Third, concepts from behavioral economic theory will be presented in order to illustrate how this theory can give a better understanding of drug taking behavior. In addition, a number of studies which have applied behavioral economic theory to examine drug-taking behavior will be reviewed. Fourth, the psychological and physiological

factors involved in gum chewing will be addressed. Finally, the goals and hypotheses of the proposed study will be addressed.

The Stress Response

Definitions of Stress

The concept of stress is not unfamiliar to the general public, and is observed throughout society. Whether it is the businessman who is under constant pressure from his/her job, the distance runner who desperately wants to win a race, or the parent who worries where the next meal for his/her children will come from, it is clear that stress is being experienced. Due to the pervasive nature of stress, many disciplines have studied one aspect or another of it, and not surprisingly, the definitions of the term vary from field to field.

The term "stress" is such a commonly used word that, at first glance, it seems straightforward and in little need of definition. It is a construct, however, that has been examined a great deal, with little agreement on how it should be defined. The reason for the observed disagreement stems from the fact that various conditions (i.e., effort, fatigue, pain, fear, or the need for concentration) can produce stress, yet not one of these conditions can be singled out as being "the cause". In order to make sense of the confusion that existed, and continues to exist, in defining the term "stress", Mason (1975) identified three definitions of the term that have been used in stress research. Stress can refer to (1) an external event (stimulus-oriented), (2) an internal state of an organism (response-oriented), or (3) an experience that arises from a transaction between a person and the environment (interaction-oriented). More recently, Aldwin (1994), presented a comprehensive definition that refers to stress as the quality of experience, produced

through a person-environment transaction, that, through either overarousal or underarousal, results in psychological or physiological distress. This comprehensive definition incorporates the three elements pointed out by Mason, and it is for this reason, that a brief review of these elements are needed.

Stimulus-Oriented Theories

The first definition of stress places emphasis on the stimulus. This approach conceptualizes stress as an event, and researchers adhering to this definition have attempted to identify potential stressors or life events. Rahe, Meyer, Smith, Kjaer, and Holmes (1964), were among the first to establish that a cluster of social events requiring change in ongoing life adjustment is significantly associated with the time of illness onset. Similarly, a number of other studies (Graham & Stevenson, 1963; Rahe & Holmes, 1965; Weiss, Dlin, Rollin, Fischer, & Bepler, 1957) have established the relationship between "life stress" and illness onset. It has been deduced, therefore, that the clustering of social or life events plays some role in the etiology of various diseases (i.e., time of onset), but is not sufficient in itself to be the cause of the illness.

The first large-scale attempt at identifying such life stressors was conducted by Holmes and Rahe (1967). In this study, Holmes and Rahe used their Social Readjustment Rating Scale (SRRS) to bring greater precision to this area of research as well as determine which events, if any, were most closely associated with illness onset. The SRRS is made up of 43 life events, ranging from events that were considered to be "quite stressful" (i.e., death of a spouse, divorce, marital separation) to items that were termed "least stressful" (i.e., vacations, minor violations of the law, change in eating habits). In this original study, the event of marriage acted as the anchor point for the rating of each

other event. Thus, subjects were asked to rate if the adjustment required for a particular event was more or less intense and prolonged than that required for marriage, which was assigned an arbitrary value of 500. After completing the SRRS, subjects were asked to indicate which of the stressful life events they themselves had experienced during the past year. When this was completed, weights were assigned to those items that were endorsed, the items were totaled, and a risk of illness score was assigned.

Response-Oriented Theories

In contrast to the previous definition which focuses on the stimulus, a second definition of stress conceptualizes it as a response, and identifies patterns of physiological and psychological responses elicited by different situations. This definition comes from the work of Hans Selye (1973), one of the most influential researchers in the stress field. In developing his definition, Selye examined animal's responses to unpleasant stimuli (stressors) and found that a wide range of unpleasant situations tended to result in a more or less consistent group of physiological responses which he termed the General Adaptation Syndrome. From this work, stress came to be defined as the non-specific response of the body to any demand made upon it (Selye, 1973). Given this formulation, Selye believed that complete freedom from stress was not possible until death, and for that reason, people should stop trying to avoid stress, and learn to meet it more efficiently.

As mentioned previously, Selye suggested that a stressor elicits a series of responses called the General Adaptation Syndrome (GAS). The GAS is characterized by three distinct phases, the acute alarm phase, a more prolonged resistance phase, and an exhaustion phase. The acute alarm phase is the initial response the body experiences

when exposed to a stressor, and represents the physical expression of a generalized "call to arms" of the body's defensive forces. If an individual is exposed to a stressor for an extended period of time, a stage of more prolonged resistance follows, where essentially the opposite of what is seen in the alarm reaction takes place. This phase is seen as being an acquired adaptation phase, where the individual becomes accustomed to the stressor. Interestingly, if exposure to the stressor is continued past this point, the acquired adaptation is lost again, and the individual enters into a stage of exhaustion. This phase inevitably follows the resistance phase as long as the stressor is severe enough and applied for a sufficient amount of time.

Hence, Selye believed stress to be a state within the organism. Taking into consideration that this approach suggests that stress is inferred from physiologic changes, it is not the stimulus itself that is stressful, but rather the response to the stimuli within the organism that identifies it as stressful.

Interaction-Oriented Theories

A third definition views stress as an interaction between the environment and an individual that causes a mismatch between the resources of the individual and the demands of the environment. Lazarus and Folkman (1984) posited that depicting stress merely as an external event ignores individual differences in the perception or appraisal of stress. That is, what is stressful for one individual at one point in time may not be stressful for another individual or the same individual at another point in time. For instance, if a teenager loses a job at a department store, it is very likely that he/she would be able to find another job at a different store, making the same salary. In this case, the teenager does not have to worry about feeding his/her family and works only to have

extra spending cash. On the other hand, a middle-aged factory worker may have a much more difficult time finding a replacement job. In this case, the factory worker needs to find a job that will pay a similar salary to what he/she was making before so that he/she can afford to feed the family and pay bills. This example shows how the same life event, job loss, may be more or less stressful depending upon its individual and social context.

According to Lazarus and Folkman (1984), the perception or appraisal of stress, depends upon the extent of the environmental demand and the amount of resources that an individual has to cope with that demand. In theory, a person first recognizes that there is a problem and then determines what resources are required to meet the problem. Stress, therefore, results from an imbalance between the requirements of the environmental situation and one's ability to cope with it. Staying with the previous example, if both the teenager working in the department store and the middle-aged factory worker lost their jobs, it is likely that they would both experience stress, given that the event in itself is an unpleasant occurrence. It is much more likely, however, that the factory worker may see his/her unemployment as an insurmountable problem creating extreme stress due to the obligations he/she has (i.e., providing for family), whereas the teenager may see his/her unemployment as a minor setback (i.e., not being able to afford a movie).

As mentioned previously, there is little agreement about the various components of the stress process. The differences that are observed, however, among researchers who study the concept of stress arise, for the most part, because of the various degrees of emphasis placed on the individual components involved in stress, as well as the causal ordering of the components. Today, it is much more accepted to take a integrative

approach in studying the concept of stress, due to the fact that this type of approach acknowledges the importance of all the components mentioned previously. Within this integrative framework, stress is partially a function of the environment, partially a function of the internal characteristics of the individual, and partially a function of the interaction between the environment and the individual in question.

Taking into consideration that the integrative approach to the study of stress acknowledges that there is a component that is a function of internal characteristics of the individual, it seems the next logical step is to question whether this component is due to physiological factors, psychological factors or a combination of the two. Studies and discussions of stress, however, emphasize the importance of the integration of both psychological and biological perspectives for the measurement, mechanisms of action, and consequences of stress (Baum, Grunberg, & Singer, 1982; Jenkins, 1979). In general, these studies typically consider psychological activation of physiological processes that occur during and after stress.

Psychological Stress and Its Measurement

Making a clear distinction between psychological and physiological aspects of stress is plagued by as much confusion as the definition of stress itself. Given the high degree of disagreement among researchers and the complexity of this construct, it should come as no surprise that the amount of research conducted in this area over the last 25 years has been extraordinary. A review by Vingerhoets and Marcelissen (1988) counted nearly 10,000 articles published between 1976 and 1985 alone, therefore, a comprehensive review of the literature in this field is beyond the scope of this section.

As a result, this section will present a general overview of the concept of psychological stress and the ways in which researchers measure it.

The key component of psychological stress that distinguishes it from physiological stress components are the cognitive activities, such as evaluative perceptions, thoughts, and inferences, that are used by an individual to interpret and guide every adaptational interchange with the environment (Lazarus, Cohen, Folkman, Kanner, & Schaefer, 1980). That is, a person is said to appraise each situation he/she has with the environment relative to its significance for that person's well being. Thus, a situation that is appraised by one person as "threatening", may be appraised as "challenging" by another depending on how well that person feels he/she is equipped for handling the demand. Hence, the cognitive appraisal processes involved at the human level are complex, allowing individuals to recognize and distinguish between harm, threat, and challenge, in addition to making numerous other cognitive distinctions that give rise to the highly complex emotional qualities that humans experience.

Given the variation found in the literature regarding the theories of the construct of stress, it should come as no surprise that an abundant and confusing array of psychological stress measures exists. It was noted earlier that the theories of stress can be broken down into one of three categories: the stimulus-oriented theories, the response-oriented theories, and the interaction-oriented theories. In order to keep this section consistent with the last, the instruments available for the measurement of stress will be broken down in a similar fashion.

Stimulus-Oriented Measures

As mentioned previously, the stimulus-oriented theories focus on the innate potential for stress residing in the environment. It makes sense, therefore, that the measurement instruments that have arisen from this orientation are designed to address the significant characteristics of the environment that impinge upon the individual and include scaling methods that assign value to the stressful environmental stimuli. Although numerous aspects of the environment can be shown to be stress inducing, there are only a few that have given rise to consistent psychological measurement devices, most notably, the area of life events research.

Modern research in this area can be dated to the publication of the Schedule of Recent Experience (SRE; Hawkins & Holmes, 1957) and its revision (Rahe, Meyer, Smith, Kjaer & Holmes, 1964) which contained 42 items and was conceptualized as a life events incidence measure. Although there have been a number of revisions since the SRE was developed, the 42 original items have remained for the most part unchanged. The developers of the SRE were also responsible for coming up with a measure that was designed to tap into life change scaling, called the Social Readjustment Rating Questionnaire (SRRQ). The SRRQ was designed to measure the magnitude of adjustment associated with each of the 42 items on the SRE, and the mean values that were derived from this measure became labeled as Life Change Units (LCUS; Rahe, McKean & Arthur, 1967). When the life events included in these studies are rank ordered by mean LCU score, the resulting scale is the Social Readjustment Rating Scale (SRRS; Holmes, 1979), which was discussed in the previous section. Finally, it is only accurate to note that there are numerous other scales beyond the SRE and the SRRS that

measure life stress, namely, the Life Expectancies Survey (LES; Sarason, Johnson & Siegal, 1979), and a number of new life stress measures proposed by Horowitz, Schaefer, Hiroto, Wilner, & Levin (1977).

Despite the limitations inherent to a stimulus oriented definition of stress, life events scales show great promise as sensitive predictive measures of the construct (Derogatis & Coons, 1993). In general, they tend to be less affected by response biases and memory distortions than many of the other measures available. In addition, since stimulus oriented measures conceptualize stress as a cumulative phenomenon, it is possible to achieve a total stress score by finding the sum of all the events that are contributing to an individual's level of stress. These scores are helpful when researchers wish to compare a subject's current status with his/her previous status in order to evaluate the relationships of stress to disease, job performance, psychiatric symptoms, or numerous other variables.

In addition to major life events, daily hassles have also proven useful in the psychological measurement of stress. The Daily Hassles Scale (DHS; Kanner, Coyne, Schaefer, & Lazarus, 1981), which contains 76 self-report items, reflects the irritating, frustrating, and distressing demands of everyday life. Example items include worries about owing money, losing things, and feeling a shortage of time for family activities.

Response-Oriented Measures

Unlike stimulus-oriented life events measurement, which arose from a theoretical basis in stress research, response-oriented measurement stems from clinical research in the field of psychopathology. That is, hallmarks of psychological disorders such as, cognitive distortions, altered mood states, and disorganized interpersonal/social

relationships, have come to be adopted by these researchers as evidence of the presence of stress. In this area of stress measurement, hundreds of self-report measures have been developed to address various domains of psychopathology (i.e., mood, psychological adjustment, social competence) that could be classified as response-oriented stress measures. A review in this area, however, (Piotrowski & Lubin, 1990) revealed that seven out of ten of the most frequently used scales in health psychology were psychological symptom inventories and scales that reflect mood and affect, hence this section will limit its scope to instruments of these types. For a more complete review of the measurements available in this area one may consult Piotrowski and Lubin (1990), Monroe (1989), and Lamping (1985).

Most of the response oriented instruments have been multidimensional, measuring the multiple symptoms that define stress. The best known measures in this category include the Minnesota Multiphasic Personality Inventory (MMPI) and its revision (MMPI-2). A large amount of research has been done on these instruments in a range of clinical settings and with a broad spectrum of samples. The MMPI and the MMPI-2 have been central to personality research for over 50 years and has provided enormous scientific value (Graham, 1993). Although the MMPI and MMPI-2 are often criticized as operational definitions of stress, they are still used as outcome measures in stress studies. Examples of studies include a report by Davis and Wedseth (1978) that concluded that the scale was sensitive to stress among male college students and Pancheri et al. (1978) that found the MMPI to discriminate clearly between improved and nonimproved patients who had suffered a severe myocardial infarction. In the last decade, a subset of items on the MMPI known as the Cook-Medley Hostility Scale (Cook

& Medley, 1954) has been used repeatedly to successfully predict stress-related cardiovascular morbidity and mortality (Greenglass & Julkunen, 1989; Williams & Barefoot, 1988).

The SCL-90-R is another multidimensional self-report inventory that is designed to measure symptomatic psychological distress. It reflects psychological distress in terms of nine primary symptom dimensions and three global indexes of distress (Derogatis, Yevzeroff & Whittelsberger, 1975). Use of the SCL-90-R specifically in the area of stress research has been reported by Carrington, et al. (1980), where they showed the instrument to be highly sensitive to differences in the efficacies of various meditation interventions in reducing stress. Also, the SCL-90-R was used in several life events stress studies (Dohrenwend, Dohrenwend, Dodson & Shrout, 1984; Roth & Holmes, 1987) that showed the efficacy of this measure in the area of stress.

In addition to the multidimensional measures mentioned above, unidimensional psychological symptom measures, particularly those that have become synonymous with definitions of stress (i.e., anxiety, depression) are also used. The numbers of unidimensional psychological measures that exist are too numerous to document in this section, hence, three of the most popular measures in the area of stress research will be addressed here. For this section, discussion will focus on their relevance to stress research since each measure mentioned below is further described in the methods section of this paper.

The Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock & Erlbaugh, 1961) is a unidimensional symptom inventory focused on the measurement of depression. Within the area of stress research, this measure is broadly used to measure

the stress and distress associated with psychological disorders (Beck & Beamesderfer, 1974). In addition, the BDI has been shown to be sensitive to the stress associated with medical illness among in-patient (Schwab, Bialow, Brown & Holzer, 1967) and out-patient (Nielsen & Williams, 1980) populations.

The State-Trait Anxiety Inventory (STAI; Speilberger, Gorsuch, Lusshene, Vagg & Jacobs, 1983) is a self-report symptom mood inventory designed to provide an operational distinction between situational anxiety (state anxiety) and enduring personality characteristics (trait anxiety). Frequent examples of the use of the STAI in stress-related research are found in the literature (Johnson & Sarason, 1978; Sarason, Johnson & Siegal, 1979; Arena, Blanchard & Andrasik, 1984). Due to the STAI's brevity, ease of use, and its distinction between current emotional states versus characteristic personality traits, it continues to be an attractive instrument in stress research.

The Profile of Mood States (POMS; McNair, Lorr & Droppleman, 1971) is a sixty-five item adjective checklist that reflects measurement in terms of six primary mood states, and has proven itself to be a sensitive response-oriented measure of stress in a wide variety of contexts (Derogatis & Coons, 1993). It has shown predictive validity in a broad spectrum of clinical change studies (Imber, 1975; McNair, 1974; Haskell, Pugatch & McNair, 1969). In addition, the POMS has been used to assess the stress associated with pain (Shacham, Reinhardt, Raubertas & Cleeland, 1983), breast cancer (Taylor, Lichtman & Wood, 1984) and emotional attitudes of individuals during smoking cessation (Hall, Rugg, Tunstall & Jones, 1984).

Interaction-Oriented Measures

Finally, the interactional theorists emphasize that it is the characteristics of the individual that are the major mediating mechanisms between the stimulus characteristics of the environment and the responses they invoke. Interactionist theorists are critical of the stimulus and response theorists because both of these theories dismiss the importance of the person in the stress equation, and with it the extensive number of major mediating characteristics that form the basis for individual differences (Derogatis & Coons, 1993). Many theorists with this orientation go one step further, insisting that their approach is actually transactional. The transactional approach states that it is not only the individual that mediates the impact of the environmental stimulus upon responses, but in addition, the perceptual, cognitive, and physiological characteristics of the individual affect and become significant components of the environment as well (Lazarus, 1976). It makes sense therefore, that the measurement devices derived from this orientation would take into consideration the individual's functions on the one hand, and the characteristics of the external environment on the other. Some of the instruments that have been developed from the interactionist models include the Jenkins Activity Survey (JAS; Jenkins, Rosenman & Friedman, 1967) and the Derogatis Stress Profile (DSP; Derogatis, 1987).

The JAS is a self-report screening instrument to measure a specific pattern of behavior thought to have a high association with proneness to coronary disease, namely Type A behavior. The JAS has been used extensively in stress research and a number of reviews have been written (Glass, 1977; Goldband, Katkin & Morell, 1979; Jenkins & Zyzanski, 1980). The JAS has proven to be an extremely productive research instrument and has facilitated a large body of research on the relationship between physical disease

and psychosocial factors (Derogatis & Coons, 1993). Although the JAS has been used in a limited area of stress research (i.e, coronary heart disease), it represents a measure that will probably remain quite useful.

The DSP a unique instrument because it is one of very few stress instruments whose constructs were derived directly from stress theory, and incorporates stimulus, response, and interactional elements. The DSP has three stimulus scales which provide an indication of the level of environmental stress the individual is subjected to, five mediating behavior scales thought of as capable of magnifying or reducing the impact of stressors, and three response measures that indicate the level of conscious emotional distress that the individual is experiencing as a result of the stressor-mediator interaction. Each of these scales has an equal opportunity to contribute to the overall interactional stress score. A comprehensive monograph on the DSP describing both psychometric properties and validation studies has been published (Derogatis, 1987).

Overall, there has been a great deal of variation found in the literature concerning the psychological components of stress. Given the existing variation, it makes sense that there are volumes of measurement instruments that have been developed. In general, these measures can be placed into one of three categories, the stimulus-oriented measures, the response-oriented measures, and the interaction-oriented measures, each of which relate to a corresponding theory of stress. Stress, however, not only effects individuals at a psychological level, but has also been shown to have a deleterious effect on individuals' physical well being.

Physiological Responses to Stress

As mentioned previously, a universal definition of stress has not yet been agreed upon by the academic community, however, it is clear that aversive stimuli can produce more than negative emotional responses. Stress can lead to disease conditions such as, peptic ulcers, and can aggravate other disorders that occur in the absence of stress such as, heart attacks, strokes, asthma, menstrual problems, headaches, and skin rashes. Since many of the harmful effects of stress are produced not by the stimuli themselves but by an individual's reaction to the stressors, it should not come as a surprise that physiological changes occur within the individual. Specifically, when a person is confronted with stress-provoking stimuli, that person exhibits sympathetic arousal (the "fight or flight" response) which is characterized by increases in heart rate, blood pressure, respiration rate, muscle tension, skin conductance, and other systemic changes (Lester, Nebel & Baum, 1994).

Of all the changes that occur in response to stress, however, the secretions of the Hypothalamic-Pituitary-Adrenal (HPA) axis has been used most extensively as an index of the presence of stress (Vernikos-Danellis & Heybach, 1980). The HPA axis is one of the central endocrine systems involved in the body's response to stress (Axelrod & Reisine, 1984). Physical and psychological stress trigger a neuroendocrine response that begins with the release of corticotropin releasing factor (CRF) from the hypothalamus (Jaeckle & Lopez, 1986). CRF then triggers the release of adrenocorticotropic hormone (ACTH) from the anterior pituitary, which in turn, stimulates the adrenal cortex to secrete glucocorticoids (cortisol in humans, corticosterone in lower animals) (Lopez, Young, Herman, Akil & Watson, 1991).

The HPA axis works via a closed-loop feedback system that tightly regulates plasma cortisol levels (Lopez et al, 1991). Simply put, circulating cortisol interacts with various structures in the brain (i.e., the hippocampus, hypothalamus, and pituitary gland) by binding to various receptors, which negatively regulates the HPA axis. This negative regulation inhibits further secretion of ACTH and CRF, which comes to inhibit cortisol secretion as well (Keller-Wood & Dallman, 1985).

The regulation of cortisol secretion during stress is essential for the survival of the individual. Having too much or too little circulating cortisol leads to a variety of problems. Specifically, the absence of cortisol leads to the inability of the individual to cope with stress and eventually to death, whereas having too much cortisol in the system has been shown to lead to Cushing's disease and depression (Lopez et al., 1991). Thus, it can be seen that the HPA axis is designed to maintain adequate cortisol levels, much like a thermostat in a home is designed to regulate a comfortable living environment.

Cortisol

Cortisol, the primary HPA peripheral hormone secreted by the adrenal cortex, plays a primary role in maintaining nearly every physiological function that takes place in the human body (Kuhn, 1989). To name just a few of its many functions, cortisol helps to maintain blood glucose, lipid, protein, and nucleic acid synthesis. In addition, cortisol helps to regulate immune function, controls growth and development of many tissues, and it regulates behavior through actions on various neuronal systems in the brain (Kuhn, 1989).

Cortisol is not secreted at a constant rate, rather it is secreted in bursts of about 7 to 13 per day which are related to one's circadian rhythm (Kuhn, 1989). Cortisol

secretion reaches its peak in the early morning hours and its lowest point at the beginning of sleep. During a cortisol burst, levels can rise significantly in a short period of time (i.e., 10 to 30 minutes), and these levels begin to dissipate somewhat rapidly, given that cortisol has a short half-life of approximately 60 to 90 minutes. At low or normal levels of secretion, cortisol binds to proteins at a fairly constant rate, however, at high levels of secretion, cortisol in the circulation exceeds the capacity of the binding sites and the amount of free floating hormone increases (Kuhn, 1989).

Cortisol secretion rises predictably in response to a number of stressors, and cortisol levels generally increase in proportion to the intensity of the stimulus (Kuhn, 1989). It would be expected, therefore, that the more intense the stressor, the greater the level of cortisol. It is important to note, however, that the cortisol response is slower to see when compared to heart rate or blood pressure increases. The reason for this is that there is a 5 to 15 minute lag caused by the secretion of ACTH, its diffusion to the adrenal cortex, and finally the resulting cortisol secretion (Orth et al., 1983).

Physiology of Smoking & Nicotine

The Pharmacology of Smoking

Cigarettes, whether they are "tall," "slim," "filtered," or "unfiltered," are delivery systems for approximately 10 milligrams of nicotine, of which, about 1 to 2 milligrams reaches the lungs (Gold, 1995). While nicotine has a half-life of only 2 to 4 hours, it remains active in the user for 6 to 8 hours, especially with the regular intake of the drug that occurs with every additional cigarette smoked (Gold, 1995). In essence, nicotine stimulates the user continuously, not just with each new dose.

After absorption in the lungs, nicotine enters the bloodstream and binds to plasma proteins, which are distributed extensively to various body tissues, including the brain and the liver (Benowitz, 1986). Any unmetabolized nicotine remains in the system stimulates neurotransmitters, which play a role in the addictive effects of the drug (Benowitz & Fredericks, 1995). Specifically, nicotine's effect on the brain is believed to play a role in addiction because it enhances the release of a variety of neurotransmitters by brain cells (Gold, 1995). For instance, nicotine enhances the release of dopamine, which may produce pleasure, norepinephrine, which may suppress appetite, acetylcholine, which produces arousal, serotonin, which may reduce anxiety, and beta endorphin, which may reduce pain (Benowitz & Fredericks, 1995). In high doses, nicotine produces dizziness, nausea, convulsions, vomiting, muscle paralysis, cessation of breathing, coma and circulatory collapse, however in lower doses (i.e., doses that are seen in those who consume tobacco products) the effects are very different (Benowitz & Fredericks, 1995). Some of these effects include, an increase in heart rate and blood pressure, increased force of contraction of the heart, constriction of blood vessels in the skin and heart, relaxation of the skeletal muscles, increased body metabolism, and the release of a variety of hormones (i.e., epinephrine, cortisol) into the bloodstream (Benowitz & Fredericks, 1995).

Smoking and Cortisol

Nicotine has been shown to increase circulating levels of cortisol in male chronic smokers and there have been a number of studies that have documented the effects of cigarette smoking on the HPA axis (Wilkins et al., 1982; Winternitz & Quillen, 1977). In these studies, subjects smoked 2 or more high nicotine cigarettes of at least 2.0 mg

nicotine each in a 10 minute time frame. Such experimental conditions are rather artificial, however, since the high nicotine cigarettes used are not commercially available and few smokers smoke 2 or more cigarettes in such a short amount of time. The effects of smoking typical commercial cigarettes in a normal fashion are relatively small, somewhat unreliable, and probably state and trait dependent (Gilbert, 1995). Smoking nicotine cigarettes, however, has been shown to result in slight elevations in cortisol levels relative to nicotine-free cigarette smoking (Kirschbaum, Wust, & Strasburger, 1992).

The answer to the question of whether cigarette smokers have higher levels of cortisol compared to nonsmokers is still being debated. One study showed that the plasma cortisol levels were significantly higher among smokers as compared with nonsmokers (Gossain, Sherma, Srivastava, Michelakis, & Rovner, 1986), whereas another showed that there were no differences observed in cortisol levels between habitual smokers and nonsmokers in their 24-hour urine samples (Yeh & Barbieri, 1989). In addition, Pomerleau et al. (1987), found that smoking a single cigarette had no effect on cortisol either after a period of inactivity or immediately after a period of extreme exercise. What has been found, however, is that smoking may enhance one's ability to cope with stress by increasing cortisol output in those individuals who have inadequate responses to stress (Rubin & Warner 1975).

Nicotine Dependence and Withdrawal

A large body of research has shown that smoking cigarettes is addicting and that nicotine is the agent in cigarettes that leads to addiction (US DHHS, 1988). In the scientific community, the terms "drug addiction" and "drug dependence" are

synonymous in that both terms refer to the behavior of repeatedly ingesting mood-altering substances by individuals. The World Health Organization and the American Psychiatric Association have developed a set of criteria to determine whether tobacco-delivered nicotine is addicting. This criteria for drug dependence includes primary and additional indicators. The three primary criteria are sufficient to define drug dependence. First, highly controlled or compulsive use indicates that drug-seeking and drug-taking behavior is driven by strong and often irresistible urges. It can continue despite a true desire to quit or even repeated attempts to quit. Second, the drug has psychoactive or mood-altering effects. Last, the drug reinforces behaviors related to obtaining and consuming the drug itself. Therefore, the psychoactive chemical must be capable of functioning as a reinforcer that can directly strengthen behavior leading to further drug ingestion.

Additional criteria are often used to help characterize drug dependence. Some of these criteria are associated with the drug-taking behavior itself. These include: (a) the behavior may develop into regular stereotypic patterns of use, (b) the use of the drug despite its harmful effects, (c) relapse following abstinence, and (d) recurrent drug cravings. The other additional criteria are associated with the control that they have over the behaviors that increase the likelihood of harm to the individual by contributing to the regularity and overall level of the drug intake. These include, tolerance, physical dependence, and pleasant or euphoriant effects.

Tobacco use involves several biobehavioral processes of drug dependence, including nicotine reinforcement, however the initiation and maintenance of this dependence may be supported by other actions of nicotine. For example, some cigarette

smokers report that smoking helps them to think better, to cope with stress, and to keep body weight under control (US DHHS, 1988). The belief that tobacco use has these effects may contribute to initiation, maintenance, and relapse.

Cigarette smoking is an orderly behavioral and pharmacologic process that involves maintenance of the desired levels of nicotine in the body. Thus, the role of nicotine in controlling tobacco self-administration is similar to other addictive drugs (i.e., ethanol) in the use of their respective products (i.e., alcoholic beverages). It is less clear however, if the behavior-controlling pharmacologic properties of nicotine share critical dependence-producing properties with these other drugs. Standardized testing procedures have been used in both animal and human studies to determine if a drug is dependence producing (US DHHS, 1988). On the basis of these testing procedures, four general kinds of behavior-modifying drug effects seem to be distinct. These effects include: (a) drugs produce interoceptive stimulus effects, which means they produce effects that a person or animal can distinguish from the non-drug state; (b) drugs serve as rewards, where the presentation of the drug itself produces a strengthening of the behaviors which originally led to its presentation; (c) drugs serve as unconditioned stimuli, where they can directly elicit various responses, and in the ensuing period, these responses can be elicited by stimuli that are associated with the drug, including the presence of environmental or internal cues; and (d) drug administration or abstinence can also serve as punishers or aversive stimuli.

Each of these four behavior-modifying drug effects can be classified as a reinforcement model. The first three can be though of in terms of positive reinforcement models, and the last as a negative reinforcement model. The primary biobehavioral

mechanism by which drugs maintain drug seeking is by functioning as a positive reinforcer. More simply, a drug, such as nicotine, can serve as a stimulus that strengthens the behavior that leads to its own delivery. Even dependence-producing drugs however, do not have uniform positive reinforcing effects and may even be aversive under some conditions. Negative reinforcement is a mechanism by which drugs modify behavior and may be important in increasing the amount of control put forth by the drug over the individual. For example, if a person reduces his/her nicotine intake it is likely that he/she will experience one or more withdrawal symptoms which include, depressed mood, insomnia, irritability, anxiety, difficulty concentrating, restlessness, decreased heart rate, and weight gain. Thus, many individuals who use nicotine take it in order to avoid or relieve withdrawal symptoms, for instance, when they wake up in the morning or have been in a situation where the use of nicotine has been restricted (i.e., at the movie theater).

Behavioral Economics

Behavioral economics is the application of economic theory to the analysis of behavior (DeGrandpre, Bickel, Hughes, & Higgins, 1992). Since the early 1970's behavioral economic theory has provided a useful conceptualization for analyzing behavior (Hursh, 1984). One such conceptualization has been borrowed from the area of microeconomics called consumer demand theory which looks at the relationship between the price of a consumer good and the demand for that consumer good. It is important to note that in behavioral economics, the economic terms "purchased", "consumer good", and "price" are synonymous with the behavioral terms "self-administered", "reinforcer", and "response requirement".

One of the most fundamental principles of behavioral economics is the demand law. This law states that, "all else being equal, total consumption decreases as price increases" (Allison, 1979). This law holds true with regard to the effects of response requirement, or what a person must do to obtain a drug, on drug self-administration. More simply, drug consumption decreases as response requirement increases. Therefore, in behavioral terms, demand is defined as the amount of reinforcer that is self-administered versus the response requirement to obtain the reinforcer (DeGrandpre et al., 1992). The demand curve is related to the concept of demand. For this curve, the amount of the consumer good purchased is plotted on the Y-axis and the price of the consumer good is plotted on the X-axis.

Elasticity is a second important concept that is taken from the field of economics and is applied to behavioral theory. This term refers to the degree to which the consumption of a specific good decreases as response requirement, or price, increases (DeGrandpre et al., 1992). A consumer good can be considered either a luxury or a necessity based on that goods elasticity. That is, when the consumption of a reinforcer changes greatly with an increased cost, it is said to be a luxury or an elastic commodity. On the other hand, when the consumption of a reinforcer changes only a little with increased cost it is said to be a necessity or an inelastic commodity.

The third important concept that behavioral economics borrows from economics is cost, or unit price. Unit price can be thought of as the response requirement divided by the reinforcer size (Hursh, Raslear, Shurtleff, Bauman, & Simmons, 1988). Unit price can be increased by one of two ways, by increasing the response requirement, or by decreasing the size of the reinforcer. According to behavioral economic theory,

consumption should be the same if the unit price is the same, regardless of the components that make up that unit price. For example, a researcher could use several response requirements and several doses. Thus a unit price of 6 could be derived by various combinations of fixed-ratio (FR) response requirements and deliveries of the reinforcer. For example, when a person is permitted to smoke one cigarette for every six correct answers given (a fixed-ratio schedule of 6 [FR-6]) the unit price of the cigarette remains constant, despite the number of correct responses. More simply, the unit price of 6 remains unchanged regardless of the constituents that make up that unit price since there are many combinations that will yield 6 (i.e., 6/1, 12/2, 24/4).

For most reinforcers studied, as unit price increases, consumption of that reinforcer initially changes little and then at some unit price it falls rapidly (Bickel, DeGrandpre, Hughes, & Higgins, 1991). This has been shown recently in a study by Bickel et al. (1991) where he examined human cigarette smokers. For this study, the effects of various combinations of dose (1, 2, or 4 puffs) and response requirement (FR 200, 400, and 1600) on nicotine consumption were examined in 3 hour sessions. In general, the findings proved that self-administration remained stable until high unit prices were hit, and then decreased rapidly, and also that different combinations of dose and response requirement in which the end result was the same unit price produced similar amounts of responding and drug consumption.

One last concept that must be addressed in regard to behavioral economic theory revolves around the accompaniment of other reinforcers in the environment. The availability of alternative reinforcers (consumer goods) directly affects the consumption of a particular consumer good, and it is at this point that an understanding of substitute

and complement reinforcers comes in handy. Commodities are said to be substitutes when the change in the price of one commodity changes the consumption of another commodity oppositely (Bickel, Hughes, DeGrandpre, Higgins, & Rizzuto, 1992). For example, when a smoker goes to see a movie and is not permitted to smoke while in the theater, the price of smoking is great (i.e., removal from the theater), and the smoker may choose other less costly reinforcers found at the snack bar. Typically, the smoker stays away from sweets, but given the high cost of smoking he/she substitutes candy, a lower priced commodity, for cigarettes. Hence, an increase in the price of smoking, causes an increase in the consumption of candy. It has been suggested (Hursh & Bauman, 1987) that commodities are more likely to be substitutes when they share similar properties and effects. In the example above, both candy (sugar) and cigarettes share some common properties in that they are administered orally, they require some movement of the jaw muscles, and they offer stimulant effects to the consumer.

In contrast, a complementary relationship between reinforcers is said to exist when an increase or decrease in the consumption of one consumer good results in a similar change in the other reinforcers (Bickel et al., 1992). To better conceptualize this relationship, consider the association between the consumption of hot dogs and hot dog buns. If the price of hot dogs becomes too great; hot dog consumption should decrease, and presumably hot dog bun consumption would decrease as well. The converse is also true. If the price of hot dogs suddenly dropped to a point where people began to consume a greater number of hot dogs, it is also probable that the consumption of hot dog buns will increase as well. Hursh and Bauman (1987) noted that consumer goods are more likely to be complements the more that both are necessary to produce the desired state, or

effect. Expanding on the above example, imagine a hot dog barbecue with out hot dog buns. Eating hot dogs out doors becomes much more inconvenient without the buns.

Application of Behavioral Economics

Numerous researchers have applied behavioral economic theory to different types of consumer goods, including drugs, and have found this perspective to be useful in explaining the relationships between reinforcing stimuli. Studies done in laboratory settings indicate that both the use of coffee and alcohol increase the number of cigarettes that a smoker will smoke in a given time period (Epstein & Jennings, 1986). Keeping in line with behavioral economics, this finding suggests that these commodities have complementary relationships.

Marshall, Epstein, and Green (1980), randomly assigned coffee drinking smokers to one of four groups where they were given 0, 1, 2, or 3 cups of coffee during two one-hour sessions, during which time they were asked to work on crossword puzzles. Results showed that subjects who received coffee in any amount smoked more than the subjects who were not given access to coffee. Moderate and low rate smokers from the previous study were then examined further in a second study designed to assess the aspects of coffee that influence smoking behavior. In this study, subjects were randomly assigned to one of five groups in which they were provided with no drink, water, Potsum (a coffee substitute), caffeinated, or decaffeinated coffee. Results from this study showed that subjects who were given caffeinated or decaffeinated coffee smoked more than subjects in the Potsum, no drink or water control groups. These results provide experimental evidence of the role of coffee in setting the occasion for smoking, as well as ruling out

the presence of a liquid or caffeine as the important aspect of coffee in influencing smoking.

In a follow-up study (Marshall, Green, Epstein, Rogers, & McCoy, 1980), the relationship between cigarette smoking, coffee drinking, and urinary pH was examined. Previous research by Schacter et al. (1977), found that increased urine acidity causes increased excretion of nicotine in the urine. This urinary pH/nicotine excretion phenomenon is believed to be a physiological mechanism that could influence cigarette smoking behavior since the more nicotine one excretes through urine, the more that person will need to smoke to regulate his/her nicotine levels. It has been suggested that coffee has an acidifying effect on urine, and therefore may effect urinary pH (Marshall et al., 1980). Thus, urine acidity levels were manipulated to see if this level would directly effect cigarette smoking. The eight subjects in this study participated in each of the four conditions in which they received: water, coffee, coffee plus sodium bicarbonate, or coffee plus ascorbic acid. The results from this study were in line with the previous studies since it was found that subjects smoked more cigarettes in a one hour session when they were in one of the three coffee conditions. Coffee itself did not have an effect of increasing urine acidity, so increased urine acidity cannot account for the smoking increases observed in this study.

Results from the above studies show the importance of the repeated relationships between environmental stimuli (coffee) and smoking. Thus, if drinking coffee reliably influences smoking behavior, the regulation of one's coffee intake would be a necessary step in the regulation of one's smoking behavior. On a similar note, laboratory studies have examined the smoking-alcohol relationship and have provided comparable results.

Epstein and Jennings (1986), demonstrated that alcohol, like coffee, can set the occasion for increased smoking.

Griffiths, Bigelow, and Liebson (1976) looked at the effect of alcohol (ethanol) on the cigarette smoking of alcoholic subjects. In this study, cigarettes were obtained either by request or by operation of a lever (FR 5 or 10) during daily 6 hour sessions. The sessions were randomized so that on some days the subjects drank orange juice alone and on other days they drank orange juice plus ethanol. During the sessions in which there was ethanol added to the orange juice, the rate of cigarette smoking was found to be significantly higher than the days in which there was no ethanol added to the orange juice. Results from this study suggest that smoking and ethanol serve as compliments to each other, which as stated earlier is when an increase in the consumption of one consumer good (ethanol) is associated with the increase in another consumer good (cigarette smoking).

In addition to smoking and alcohol having a complementary relationship, support has been found for the substitutability or these two drugs. Perkins, Epstein, Sexton, and Pastor (1990) examined the consumption of alcohol, coffee, soda, and sweets (sweet, high-fat foods) of seven young female smokers over a three week period. This study involved baseline smoking (week 1), complete smoking cessation (week 2), and resumption of smoking (week 3). Results showed that there was an increased intake of sweets, and to a lesser degree, alcohol after smoking cessation which was reversed upon resumption of smoking. No significant changes across weeks were found with regard to the other substances.

The findings from the above study shows that smoking cessation, a behavior change that promotes health, may lead to changes in the consumption of other substances (e.g., sweets, alcohol), that may themselves have negative effects on one's health. Therefore, sweets and alcohol appear to be substitutes for smoking, that is, the change in the consumption of cigarette smoking changes the consumption of sweets & alcohol in an opposite way. When smoking was not available to the subjects (the unit price of smoking became too great), alternative consumer goods were used to replace cigarettes. Alternative reinforcers other than sweets and alcohol, such as soda and TV viewing, were also available to the subjects however, were not shown to act as substitutes. This implies that the effect of smoking cessation on alternative reinforcers is specific, not general in nature. So, if the findings of all the studies that examined the relationship between alcohol and cigarette smoking are taken into account, one can clearly see that alcohol can serve as both a substitute and a compliment to cigarette smoking.

More recently, the effect of chewing gum on nicotine withdrawal was examined (Cohen et al., 1997). Chewing gum was shown to influence urges to smoke as well as nicotine withdrawal. Cohen et al. (1997) provided smokers with access to chewing gum in a situation where smoking was prohibited. Subjects who had access to chewing gum showed significant decreases in craving for a cigarette and in the severity of the total withdrawal symptoms reported as compared to smokers who did not have access to chewing gum. This study suggests that chewing gum may actually be a viable alternative to cigarette smoking when individuals cannot smoke.

Chewing Gum

There are many theories as to why humans chew gum and other nonfood items, however no theory has sufficient evidence to back up its claim. One panel of psychiatrists and psychologists suggest that the top three reasons people chew gum are: (a) to relieve feelings of loneliness and boredom, (b) relief from tension by discharging nervous energy, and (c) to provide a quick, socially acceptable outlet for anger and irritation (Hendrickson, 1976). In addition, various studies have shown that gum chewing alleviates thirst and hunger, helps workers concentrate, and keeps people alert (Hendrickson, 1976).

There has been a great deal of research that has examined the advantages of gum chewing. This research was inspired by the establishment of the Wrigley-Beech-Nut Fellowship at Northwestern University during the Great Depression, and this industry-sponsored grant was set up for the sole purpose of researching the physiological effects of gum chewing.

Dr. Robert H. Veitch, Director of the Deafness Clinic at the Massachusetts Osteopathic Hospital recommended that anyone experiencing deafness during common colds should try chewing gum several hours a day for relief. Medical authorities point out that the chewing of gum induces frequent swallowing, which opens the air passages, allowing air pressure to be equalized inside the ear (Hendrickson, 1976).

Recently, it has been suggested in advertisements that chewing gum may serve as an alternative to smoking, however empirical studies examining this notion have not been undertaken. Given that nicotine itself has been shown to be an adequate positive reinforcer for animals (Goldberg, Spealman, & Goldberg, 1981) and humans

(Henningfield, Miyasato, & Jasinski, 1983), it would make sense that in order for gum chewing to serve as a substitute for smoking the mere act of chewing gum must also serve as a positive reinforcer. Clearly, there is something reinforcing about chewing gum, as evidenced by the large number of people who chew gum on a daily basis, however it is not clear what aspect of gum chewing accounts for the reinforcing effects experienced by gum chewers.

One hypothesis that could account for why some people believe that gum chewing is an adequate substitute for cigarette smoking is that both of these actions are reinforcing due to the fact that they both stimulate the jaw muscles. It has been shown that facial muscles constitute an emotional output system and are closely related to the experience of emotion (Dimberg, 1988). Perhaps when a person chews, facial muscles are stimulated in a similar way as when one smokes, which would in turn elicit similar emotions. If the emotions that come with this chewing/smoking muscle activation are positive, it would make sense that this type of stimulation would be reinforcing. Chewing has also been described as a tension outlet that may serve as a technique of relaxation (Hollingworth, 1939). In this study, it was found that "the collateral motor automatism involved in the sustained use of the conventional masticatory muscles does result in a lowering of tension." It was mentioned previously that many people smoke in order to avoid or relieve withdrawal symptoms, two of which are closely tied to tension namely, anxiety and restlessness. If chewing serves as a means of reducing tension-related withdrawal symptoms, perhaps the smoker that is reinforced by the alleviation of these withdrawal symptoms would find chewing gum to be an adequate substitute.

There are many other theories as to why chewing gum may serve as an adequate substitute for cigarette smoking. For example, both actions provide social reinforcement, both are conditioned reinforcers, and both have been shown to curb appetites, which help people to maintain their weight. If both activities are reinforcing in similar ways, perhaps the substitution of one commodity for the other is a credible idea. Nevertheless, most gum chewers and cigarette smokers would refrain from giving any reasons for their habits other than the fact that both activities are highly pleasurable.

Goals of Present Study

The present study was designed to examine the usefulness of the substitution of gum for cigarettes when a dependent smoker is unable to smoke and "craving" a cigarette using multimodal assessment. As previously mentioned, nicotine can serve as an effective positive reinforcer, and nicotine deprivation can increase the reinforcing effectiveness of cigarettes (Henningfield & Griffiths, 1979). Extended periods of deprivation are associated with an uncomfortable and distressful withdrawal syndrome which makes up another mechanism by which the reinforcing capability of nicotine would be further increased. The drug effect that provides the means for this discomforting withdrawal is physical dependence and several of the symptoms of nicotine withdrawal correspond to the effects of nicotine that are either known or suspected to promote tobacco dependence (US DHHS, 1988). Symptoms reported by large numbers of ex-smokers included "craving" for tobacco, anxiety, impatience (Hughes, Gust, & Pechacek, 1987), restlessness , nervousness, or irritability (Trahir, 1967), difficulty concentrating, increased appetite (Wynder, Kaufman, & Lesser, 1967),

somatic or physical complaints (Pederson & Lefcoe, 1976), and weight gain (Mausner, 1970).

In comparing the diagnostic criteria for nicotine withdrawal in the DSM-III-R and DSM-IV it can be seen that there are relatively few changes. Six of the eight symptoms listed in the DSM-IV are the same as they were in the DSM-III-R [(1) irritability, frustration, or anger, (2) anxiety, (3) difficulty concentrating, (4) restlessness, (5) decreased heart rate, and (6) increased appetite or weight gain], with "dysphoric or depressed mood," and "insomnia" being added. The other difference is the exclusion of "craving for nicotine" from the DSM-IV, which has been debated. The inclusion of this symptom for nicotine withdrawal but not for most other withdrawal syndromes in the DSM-III-R was taken to imply that craving is more closely tied to withdrawal from nicotine than for other drugs, however there is no data to support this idea (West & Kranzler, 1992). In addition, it is debatable whether craving during smoking cessation is actually influenced by nicotine administration (Hughes & Hatsukami, 1985). For example, it is possible for a person to crave nicotine even while smoking and clearly not experiencing withdrawal.

Although these data suggest that craving was justly dropped as a criterion in the DSM-IV, other data suggest the opposite. Craving is one of the most common and reliable effects of tobacco abstinence (Hughes & Hatsukami, 1986), and it has been shown that craving can be a predictor in relapse (Covey, Glassman, & Stetner, 1990). Lastly, due to the larger variety of environmental cues for smoking compared with other substances of abuse, craving for tobacco may be more prevalent than it is for other drugs of abuse.

Several studies have demonstrated that the symptoms resulting from cigarette deprivation mentioned above are alleviated if the person resumes smoking (Murphee & Schultz, 1968; Weybrew & Stark, 1967; Henningfield, 1987). In the present study, it was anticipated that gum chewing would again serve as a means of alleviating the signs and symptoms of tobacco withdrawal, especially the "craving" for a cigarette. In a previous study (Cohen et al., 1997), we found that based on self-report data, dependent cigarette smokers who were not permitted to smoke during a 4-hour time period, but were asked to chew gum, experienced significantly less total withdrawal symptoms when compared to a group of smokers who were not permitted chew gum during this time. In addition to reporting less overall withdrawal, those smokers that were permitted to chew gum reported less "craving" for a cigarette than their counterparts who did not have gum. These findings empirically validate the Wrigley Chewing Gum commercials that assert "When you cannot smoke, chew gum."

Because everyone experiencing nicotine withdrawal does not exhibit all of the symptoms listed in the DSM-IV, the hypotheses regarding the self-report data will again address both specific and general withdrawal symptoms. One hypothesis closely examines the most common and reliable symptom of withdrawal (i.e., craving), whereas the second examines the total withdrawal symptoms experienced. Specifically, if a smoker's craving for a cigarette is decreased by the use of gum, it is also predicted that the use of gum would affect the other symptoms of nicotine withdrawal, thereby serving as a substitute for smoking. It is expected that we will replicate the findings from our previous study.

In order to further examine the effect of chewing gum on nicotine withdrawal, a physiological measure will be examined in addition to the self-report instruments. I will collect samples of salivary cortisol, a hormone associated with the "stress response," in order to determine if the positive effects seen by the use of chewing gum is solely a result of psychological factors, or if physiological factors play a role as well. Given that it is known that cortisol levels fluctuate with one's mood and current level of stress, instruments designed to measure these constructs will be implemented so as to rule out possible extraneous variables that may contaminate the cortisol data. With regards to the cortisol data, the hypotheses for this study address three areas. First, we plan to examine the acute effect of nicotine on cortisol levels. As mentioned previously, the findings in this area are unclear, so a well controlled study looking at the differences in cortisol levels between smokers and nonsmokers is important. Second, we plan to examine the acute effect of withdrawal on cortisol levels. Taking into consideration that nicotine withdrawal is often reported as stressful to a smoker, and since cortisol levels have been shown to increase due to stress, it is expected that smokers' cortisol levels should be significantly higher than nonsmokers' cortisol levels over the period of abstinence. Finally, the effect that chewing gum has on physiological withdrawal will be examined. For the 20 dependent smokers we expect to find that their cortisol levels are significantly lower during the period of abstinence when they have access to chewing gum as compared to the abstinence period when they do not have access to chewing gum.

Statement of Hypotheses

For this study, several hypotheses are made, each predicting a significant difference in the severity of the withdrawal symptoms reported or in the levels of cortisol observed associated with the abstinence of nicotine.

Hypothesis 1: In the session where subjects are given access to chewing gum during the time in which they are not permitted to smoke (gum condition), it is predicted that subjects will have significantly lower scores on the cigarette craving item on the Withdrawal Symptom Checklist (WSC) than when they are not given access to gum during that time (no-gum condition). The null hypothesis states that there will be no significant difference on the item that is designed to measure craving on the WSC between the gum and no-gum groups. The dependent variable for this hypothesis is the reported level of craving (on a 4-point Likert scale of 0 to 3), and the independent variable is whether or not the subject is given access to chewing gum.

Hypothesis 2: It is predicted that the total score obtained from the Withdrawal Symptom Checklist (WSC) will be significantly lower in the gum condition than in the no-gum condition. This will indicate that the total number of withdrawal symptoms experienced by subjects in the gum condition is significantly less than their withdrawal symptoms experienced in the no-gum condition. The null hypothesis states that there will be no significant difference found on the total score of the WSC among the gum and no-gum groups. The dependent variable for this hypothesis is the total score from the WSC, and the independent variable is whether or not the subject is given access to chewing gum. The total score from the WSC was obtained by taking the sum of all the items listed on the WSC except the craving item.

Hypothesis 3: It is predicted that the salivary cortisol concentration (μg/100) will be significantly higher after smoking a cigarette, when compared to their cortisol levels before the cigarette. This will indicate the acute effect of nicotine on one's cortisol levels. The null hypothesis states that there will be no significant difference in the salivary cortisol levels found when comparing a smoker's cortisol levels pre and post cigarette consumption. The dependent variable for this hypothesis is the cortisol concentration (μg/100) found in the saliva, and the independent variable is the time of the measurement (pre or post cigarette).

Hypothesis 4: It is predicted that the salivary cortisol concentration (μg/100) will be significantly higher in those subjects who are not given access to chewing gum during the time in which they are denied access to cigarettes when compared to those subjects who are asked to chew gum. This will indicate the physiological effect of chewing gum on nicotine withdrawal via one's cortisol levels. The null hypothesis states that there will be no significant difference in the salivary cortisol levels found between those subjects who were asked to chew gum during the period of abstinence when compared to subjects who were not given access to chewing gum during that time. The dependent variable for this hypothesis is the cortisol concentration (μg/100) found in the saliva, and the independent variable is whether or not the subjects are given access to chewing gum.

Method

Subjects

Subjects for this study were 20 male students, who were recruited from various undergraduate psychology courses offered at Oklahoma State University. Subjects were dependent cigarette smokers who reported smoking 16 or more cigarettes per day for at

least the past 6 months. Potential participants were excluded if they used chewing tobacco or snuff, made a serious attempt to quit smoking within the last 6 months, reported heart dysfunction or disease, or were under 18 years of age. In addition, participants were included if they had no major illnesses, used no medications at the time of their participation, and consumed fewer than 15 drinks of alcohol per week. Females were excluded from this study due to the fact that during the menstrual cycle, there is only a 10 day time period (the follicular phase) where female hormonal secretions are stable (Chattoraj & Watts, 1987). Given the documented unstable nature of hormone levels in females, and the fact that this preliminary study measured salivary hormones across a 4 hour period of time, on two separate occasions, it was critical that any factor (i.e., menses) that may have led to changes in hormonal level be avoided.

Materials

General Habit Information. The GHI is a self-report questionnaire designed specifically for this study which is designed to gather information regarding personal habits that might influence a subject's cortisol levels. This questionnaire examines sleep habits, smoking habits, drinking habits, caffeine consumption, and medical information.

Inventory to Diagnose Depression (IDD; Zimmerman, Coryell, Corenthal, & Wilson,

1986). The IDD is a 22 item self-report instrument that is designed to diagnose major depressive disorder. To quantify the severity of depression, the item scores are totaled with higher scores showing greater severity. A score of 0 or 1 in each item represents no disturbance (0) or subclinical severity (1), and a score of 2 or more is counted as a symptom. The IDD differs from other depression scales in 3 ways: (1) it covers the

entire range of symptoms for major depressive disorder used in the DSM-III, (2) it not only quantifies the severity of depression but it can also be used to decide the presence or absence of a symptom, and (3) it assesses symptom duration.

The Daily Hassles Scale (DHS; Kanner, Coyne, Schaeffer, & Lazarus, 1981). The DHS is a 118 item self-report measure that reflects irritating, frustrating, and distressing demands of everyday life. Frequency and intensity scores are included. The scale's response options (3 point scale) range from somewhat to extremely. Example items include worries about owing money, losing things, and feeling a shortage of time for family activities.

Tobacco Withdrawal Symptom Checklist (WSC; Hughes & Hatsukami, 1986). The WSC is a 12-item self-report measure that is designed to assess the presence of tobacco withdrawal symptoms and the severity of each symptom. The severity of each symptom is based on a 4-point Likert scale, ranging from 0 (not present) to 3 (severe). In addition to the 12 items, there is room for respondents to list somatic difficulties (i.e., sweating, nausea) and any changes in behavior (i.e., increase in gum chewing or exercise) since discontinuing their tobacco use.

Procedure

This study will serve as the creative component for the degree of Doctor of Philosophy at Oklahoma State University. Potential subjects were called and asked to come to the lab for a brief orientation session where subjects were exposed to the laboratory setting. During this initial session, informed consent was obtained and subjects were given an idea of what to expect during the two experimental sessions. In addition, subjects were asked to provide one sample of saliva, were given instructions to

follow (re: diet and when they should eat) for the days they participated in the experiment, and were asked to fill out a number of questionnaires designed to assess various personal habits (i.e., when they typically go to sleep, hours of sleep, and use of nicotine and caffeine), mood, and current level of stress. Finally, the two experimental sessions were scheduled and a list of referral sources were given in the event they became distressed from filing out the questionnaires. At the beginning of each experimental session, a trained research assistant greeted and escorted subjects into the experimental room of the Behavioral Pharmacology Lab, where they were seated in a comfortable chair, behind a desk. Subjects were asked to relax for approximately 20 minutes while filling out a questionnaire designed to assess their current level of stress. Subjects were then asked to provide a small sample of their saliva (sample 1) and smoke a cigarette. Upon completion of the cigarette, subjects were again asked to relax for approximately 20 minutes and were asked to rate their current withdrawal symptoms by completing the WSC (Time 1). From this point forward, there was no access to cigarettes until the completion of the protocol. Next, subjects were asked to provide a small sample of their saliva (sample 2) and to watch a movie selected from a list. When the movie was over, subjects were asked to fill out the WSC (Time 2) and provide another sample of their saliva (sample 3). Subjects were then asked to remain in the lab and read magazines of a neutral subject matter (i.e., Newsweek, Times, Sports Illustrated) for one hour. During this hour, the research assistant came in once, half way through the period, where the subject was asked to fill out the WSC (Time 3) and provide another sample of their saliva (sample 4). At the end of the hour period the subject was again asked to fill out the WSC (Time 4) and provide a sample of their saliva (sample 5).

Subjects participated in this protocol on two occasions separated by 2 to 14 days. On one occasion, subjects had access to chewing gum from the start of the movie until the termination of the protocol, while on the other occasion they did not. The session in which subjects had/didn't have access to chewing gum was counterbalanced across the two groups.

Saliva/Cortisol Collection

Taking into consideration that cortisol is sensitive to the diurnal cycle, all subjects were asked to start the experimental protocol at approximately the same time. Saliva cortisol was collected on 5 occasions during the experiment. Subjects were instructed when they were recruited to eat a light meal at least 2 hours before coming to the lab. At the start of the protocol, subjects were asked about their sleep habits (i.e., time they go to bed and the time they wake in the morning usually and in the previous night) and their mood for that day. Saliva samples were collected using a commercially available collection device (Salivette®, Sarstedt, FRG), which consisted of a prepared test tube, containing a cotton roll swab. Subjects were asked to chew on the cotton roll swab and the dampened roll was placed into a clean 5-cc plastic tube.

After each sample was collected, the research assistant stored the samples at -20° C in a freezer located within the Behavioral Pharmacology Laboratory, Department of Psychology, Oklahoma State University. On two occasions, the samples were transported to the Behavioral Sciences Laboratory, V.A. Medical Center, Oklahoma City, where the samples were centrifuged and stored at -70° C until assayed. Supernatant samples were assayed at a later date using a radioimmunoassay technique with a commercially available kit (Orion Diagnostica, Espoo, Finland) adapted to measure the

low cortisol concentrations observed in saliva. Saliva samples were mixed with a fixed amount of 125I-labled cortisol derivative and cortisol antiserum. The labeled and unlabeled antigens were then allowed to compete for the high affinity binding sites of the antibody during an incubation period. The separation of bound and unbound antigen was performed with polyethylene glycol. The amount of labeled antigen in the sample is inversely proportional to the concentration of the unlabeled antigen. The actual concentrations in the unknown samples were obtained by means of a standard curve based on known concentrations of the unlabeled antigen analyzed in parallel of the unknown. The saliva cortisol assays were performed by M. L'Hermite-Baleriaux, Universite Libre de Bruxelles, Belgium.

There were a number of quality assurance steps that were taken in order to insure the accuracy and the reliability of the saliva cortisol measurement. These included: (1) subjects were informed that they would not be able to have a drink once the protocol began in order to avoid any dilution of the saliva, and that they needed to make sure that there were no residuals of food or drink in his/her mouth when they began; (2) after collecting the samples, they were frozen until they were centrifuged; and (3) after the samples were centrifuged, they were stored in a freezer until they were sent to be assayed.

Results

Design

All analyses used a within subjects design where each subject served as his own control. For hypotheses 1 and 2 self-report measures were taken four times on both the Gum and No-Gum Days. The first self-report measure (Time 1) was taken after each

subject smoked a standard cigarette. This measure was taken prior to the movie, and was used to determine the subject's baseline level of withdrawal. The second self-report measure (Time 2) was taken upon termination of the movie, and the third and fourth self-report measures (Time 3 and Time 4) were taken at consecutive 30 minute intervals. For hypotheses 3 and 4, salivary cortisol samples were taken five times on both the Gum and No-Gum Days. The first saliva sample was obtained shortly after each subject arrived at the laboratory (Pre-Cigarette) with the second sample being collected 20 minutes after a standard cigarette (Post-Cigarette). Samples 3, 4, and 5 were taken immediately after the movie and at consecutive 30 minute intervals thereafter. Means per condition are presented in Figures 1 – 3.

Insert Figures 1 – 3 about here

Hypothesis 1

Means for the "craving" item on the Withdrawal Symptom Checklist (WSC) were analyzed using a 2 X 4 (Condition X Time) repeated measures analysis of variance (ANOVA). These results are summarized in Table 1. For this hypothesis, craving served as the dependent measure. Findings did not support the hypothesis as a significant Condition by Time interaction was not observed, $F(3,57)=.559$, n.s.

Insert Table 1 about here

Hypothesis 2

A similar ANOVA was conducted on the total score obtained on the Withdrawal Symptom Checklist (WSC). These results are summarized in Table 2. For this hypothesis, the total score (minus the craving item) obtained from the WSC served as the dependent measure. The Condition X Time interaction resulted in a F of 2.537 (df = 3, 57), with a p-value of <.07. While a p-value of less than .07 is not significant given the two-tailed alpha levels typically used, previous research conducted in our laboratory makes directional (one-tailed) predictions possible. Considering the ANOVA procedure performs a 2-tailed test, and the fact that mean total withdrawal scores during the No-Gum Day were noticeably higher, performing exploratory analyses (i.e., Simple Effects Tests) seemed reasonable. These analyses did not result in significant differences across Condition at Time 1 [$F(1,57)=0.337$, n.s.] or Time 2 [$F(1,57)=0.863$, n.s.], but significant differences were observed at Time 3 [$F(1,57)=5.391$, $p<.05$] and Time 4 [$F(1,57)=9.111$, $p<.01$].

Insert Table 2 about here

Hypothesis 3

Means for salivary cortisol levels pre and post cigarette on both experimental sessions (Day 1 and Day 2) were compared using a 2 X 2 (Condition X Time) repeated measures analysis of variance (ANOVA). These results are summarized in Table 3. For this hypothesis, salivary cortisol levels pre and post cigarette for both Day 1 and Day 2

served as the dependent measure. Findings did not support the hypothesis as a significant effect for Time was not observed, $F(1,19)=1.240$, n.s.

Insert Table 3 about here

Hypothesis 4

Means for salivary cortisol levels during the withdrawal phase of the protocol on both experimental sessions (Gum Day and No-Gum Day) were compared using a 2 X 3 (Condition X Time) repeated measures analysis of variance (ANOVA). These analyses are summarized in Table 4. For this hypothesis, salivary cortisol levels post movie and on two consecutive 30 minute intervals for both the Gum Day and the No-Gum Day served as the dependent measure. Once again, the findings did not support the hypothesis as a significant Condition by Time interaction was not observed, $F(2,38)=1.145$, n.s.

Insert Table 4 about here

Discussion

The results of this study are consistent with previous studies which show that chewing gum helps to reduce nicotine withdrawal when a nicotine dependent person cannot smoke. That is, when smokers were asked to chew gum they reported significantly less withdrawal as compared to sessions where they were not permitted to chew gum. In fact, as the withdrawal period lengthened, differences between the two experimental sessions (Gum Day and No-Gum Day) become more pronounced. This

finding is consistent with a previous study completed in our laboratory (Cohen et al., 1997), where abstinent smokers who were asked to chew gum did not differ in reported withdrawal symptoms as compared to abstinent smokers who were not given access to chewing gum after watching a movie but did show significant differences 30 minutes later. The present study replicated this finding and expanded on it by extending the withdrawal period an additional 30 minutes which appeared to further accentuate the differences across condition. Given this encouraging finding, it is important not to misinterpret its meaning. The results do not suggest that chewing gum will allow a person to avoid withdrawal altogether. Withdrawal was clearly seen in all the subjects who participated in this study, yet it was observed less acutely in subjects when they were asked to chew gum.

Unlike Cohen et al., when subjects were asked to chew gum in the present study, they did not report less "craving" for a cigarette compared to the sessions in which they were not permitted to chew gum. This finding is inconsistent with the hypothesis that chewing gum will help with the craving associated with nicotine withdrawal. The discrepant findings across studies are not surprising given that the concept of craving has led to a great deal of confusion among both the scientific and public domains for years. In fact, the inclusion of craving as one of the characteristic symptoms of nicotine withdrawal is still being heavily debated among the research community. This debate is fueled by the fact that there is no agreed upon definition of the concept and that individuals indiscriminately use the term to refer to different physiological, psychological, and behavioral states. Given the many different definitions of the concept of craving, it cannot be assumed that the way in which one individual conceptualizes the

phenomenon is consistent with how another conceptualizes it. Hence, we cannot expect to find consistency across studies examining this construct.

The two findings mentioned above suggest that a behavioral substitute can produce a significant reduction in one's total withdrawal but does little to influence craving for nicotine. This finding is consistent with another study conducted in our laboratory (McChargue, 1998), where a nicotine-free herbal mixture was shown to help with smokeless tobacco withdrawal but not with craving. Results from McChargue (1998) add support to the notion that withdrawal symptoms are made more manageable by the use of substitutable reinforcers. Overall, these studies provide some promising effects of substitute reinforcers and extend our understanding of nicotine dependence as it relates to withdrawal.

From an applied standpoint these studies suggest that clinicians should encourage the use of alternate reinforcers but should not be overly optimistic about their value. The reason for this is that craving continues to be one of the most common and reliable effects of tobacco abstinence (Hughes & Hatsukami, 1986), and has been shown to be a major predictor of relapse (Covey, Glassman, & Stetner, 1990). Given that it is possible for a person to crave nicotine even while smoking and clearly not experiencing withdrawal and if the administration of nicotine itself does not consistently affect one's reported level of craving for nicotine, we cannot expect alternative substances (i.e., chewing gum, herbal mixtures) to influence it either. In addition, there are a larger variety of environmental cues for nicotine use as compared with other substances of abuse, hence craving for tobacco may be more prevalent than it is for other drugs of abuse. Perhaps then, smoking cessation programs that focus on helping individuals cope with the positive reinforcing

aspects of nicotine dependence (i.e., the stimulant/relaxant effect of nicotine, social aspects related to being a smoker) in addition to nicotine's negative reinforcing aspects (i.e., withdrawal symptoms) would show the most significant success rates.

Thus far, this discussion has focused on chewing gum as a potential substitute reinforcer for nicotine. We have some strong evidence to suggest that chewing gum helps with nicotine withdrawal, however, we do not have evidence that gum is actually serving as a substitute for nicotine. In fact, research conducted in our laboratory supports the notion that chewing gum does not serve as a substitute for nicotine (Stott, 1998). According to behavioral economic theory, commodities are said to be substitutes when the change in the price of one commodity changes the consumption of another commodity oppositely (Bickel, Hughes, DeGrandpre, Higgins, & Rizzuto, 1992). Stott (1998) utilized a free operant design examining cigarette smoking under 3 levels of effort/cost (low, medium, and high) and 2 conditions (Gum and No-Gum). Significant differences in gum consumption were observed only in the low effort/cost scenario, where smokers smoked less when they were asked to chew gum as compared to when they were not. As the effort/cost increased, however, smoking did not decrease and use of chewing gum did not increase. If chewing gum was truly a substitute for cigarettes, as the effort or cost associated with obtaining a cigarette increased, the use of chewing gum should have increased as the use of cigarettes decreased. Given these results, chewing gum does not appear to serve as a substitute. Thus, it is possible that we do not find that chewing gum helps with craving for a cigarette on a consistent basis due to the fact that it is not a substitute. Perhaps finding a commodity that helps with craving lies in finding a true substitute for nicotine.

Finally, this study examined salivary cortisol as a potential physiological marker in the study of nicotine withdrawal. The results of these analyses were not very strong. A conservative interpretation of the analyses suggest that there was no significance change in smokers' salivary cortisol levels pre and post cigarette and there was no significant effect of chewing gum observed with respect to nicotine withdrawal. First it was anticipated that we would observe a significant increase in smokers' salivary cortisol levels when we compared the samples obtained pre and post cigarette as other studies have noted this observation (Wilkins et al., 1982; Kirschbaum, Wüst & Strasburger, 1992; Gilbert, Meliska, Williams & Jensen, 1992). After careful review, it appears that these studies had subjects abstain from smoking for a minimum of 1 hour and a maximum of 13 – 15 hours before coming in to participate. The present study did not have smokers change their pattern of smoking on their days of participation, as we were interested in studying individuals who were current smokers on a typical day. Perhaps, if the present study followed a similar methodology and had smokers abstain for at least one hour prior to their participation on both the experimental sessions similar results would have been observed. What is clear from this study is that salivary cortisol levels do not increase pre to post cigarette when subjects are not asked to abstain from smoking prior to participation.

The utility of salivary cortisol as a physiological marker for the effect of chewing gum on nicotine withdrawal was also examined. Although we did not find the significant differences between the Gum Day and the No-Gum Day, inspection of Figure 3 gives us optimism. It has been noted that cortisol levels decline as the day progresses (with a peak in the early morning hours and with the lowest point at the beginning of sleep) and that

cortisol secretion rises predictably in response to a number of stressors (Kuhn, 1989). Given that dependent smokers report abstaining from smoking is a stressful event (Shiffman, 1979; USDHHS, 1988) we would anticipate a change in the consistent decline of one's salivary cortisol levels, with increases in cortisol secretion being observed specifically during the occurrence of the stressor (i.e., during nicotine abstinence). As it can bee seen in Figure 3, on the Gum Day we see the expected decline whereas on the No-Gum Day we see a leveling out of the cortisol levels during the period of nicotine abstinence. This observation adds credibility to the notion that chewing gum helps with withdrawal at a physiological level. Specifically, chewing gum appears to help with the stress associated with nicotine abstinence as we see the usual decline of salivary cortisol levels as the day progresses. Without gum however, we see a leveling off of one's cortisol levels, suggesting an increase in stress/anxiety.

In sum, considering both the self-report and the cortisol data, some interesting hypotheses can be formed. It appears that chewing gum helps with nicotine withdrawal at both a psychological and physiological level. Although our working hypotheses centered around the notion that chewing gum helps with withdrawal due to the fact that it serves as a substitute for nicotine appears to be incorrect. Rather, it appears that chewing gum helps because it influences the negative affect related to withdrawal (i.e., anxiety). This is apparent by the fact that cortisol, a hormone associated with the stress response, appears to be influenced by chewing gum and by the notion that many of the symptoms associated with nicotine withdrawal are analogous to the stress response (i.e., irritability, anxiety, difficulty concentrating, restlessness). In addition, other research conducted in our laboratory has shown that after a dependent smoker is presented with a stressor (e.g.,

public speaking task), both smoking and chewing gum lower smokers' subjective levels anxiety when compared to a control group of smokers (Britt, 1998). In order to test this notion, future studies needs to examine the role of chewing gum and anxiety. If it is found that reported levels of anxiety are less in subjects who are asked to chew gum in a stressful situation as compared to subjects who are not given access to chewing gum in the same condition then we might have a clearer picture as to why chewing gum might help with withdrawal.

Limitations

Despite the encouraging findings that have been mentioned, this study is not without limitations. First, the smokers who participated in this study were not trying to quit smoking, rather they were asked to abstain for two 4-hour intervals. It is possible that the observed results apply only to smokers who believe that once they leave a particular situation they will be able to resume their normal smoking behaviors, and do not apply to smokers who wish to quit smoking. Future research must address this question to examine potential differences between those who are abstaining from those who wish to stop smoking permanently. Second, the present study does not examine the long-term effectiveness of chewing gum as an aid for reducing the withdrawal symptoms in a population of smokers. Therefore, the conclusions drawn from this study are limited to individuals experiencing acute withdrawal rather than the more long-term withdrawal that smokers often encounter. Future studies should address the efficacy of chewing gum in individuals who are abstaining from smoking for longer periods of time, or at the peak of their withdrawal symptoms.

In addition, the results of this study are based on a relatively young sample of smokers, all of whom were male college students. It is possible that the observed effect of chewing gum is only effective with a younger sample of smokers who find chewing gum pleasurable. It has been noted that University students may have very different smoking patterns compared to their non-college peers, as education has been linked to smoking behavior (NIDA, 1994). Fifteen percent of college students smoke daily compared to 27% of their non-college peers. Only 9% of college students smoke half-a-pack per day, whereas 20% of their same aged peers smoke at this rate (NIDA, 1994). The extent to which these findings can be generalized to heavier smokers, older smokers, or even female smokers has yet to be examined.

Finally, this study gives us good preliminary data on the use of salivary cortisol as a physiological marker for tracking nicotine withdrawal. However, as noted earlier, changes in cortisol levels are slow to observe so perhaps a study that extends the period of withdrawal would show stronger findings. Given the relative ease of taking salivary cortisol samples and the fact that subjects can take the samples on their own at their home, it is possible that subjects' cortisol levels could be monitored all day. This way, we can get a better idea as to how nicotine withdrawal effects the normal daily cycle of the secretion of cortisol.

References

Aldwin, C. M. (1994). Stress, Coping and Development. New York: Guilford Press.

Allison, J. (1979). Demand economics and experimental psychology. Behavioral Science, 24, 403-415.

Arena, J. G., Blanchard, E. B., & Andrasik, F. (1984). The role of affect in the etiology of chronic headache. Journal of Psychosomatic Research, 28, 79-86.

Axelrod, J. & Reisine, T. D. (1984). Stress hormones: Their interaction and regulation. Science, 224, 452-459.

Beck, A. T., & Beamesderfer, A. (1974). Assessment of depression: The depression inventory. In P. Pichot (Ed.), Modern problems in pharmacopsychiatry. (Vol. 7). Basel: Karger.

Beck, A. T., Ward, C. H., Mendelson, M., Mock, J., and Erlbaugh, J. (1961). An Inventory for Measuring Depression. Archives of General Psychiatry, 4, 53-63.

Benowitz, N. L. (1986). Clinical pharmacology of nicotine. Annual Reviews of Medicine, 37, 21-32.

Benowitz, N. L., & Fredericks, A. B. (1995). Nicotine. In J. H. Jaffe (Ed.), Encyclopedia of Drugs and Alcohol. (Vol. 2., pp. 734-741). New York: Simon & Schuster MacMillan.

Bickel, W. K., DeGrandpre, R. J., Hughes, J. R., & Higgins, S. T. (1991). Behavioral economics of drug self-administration: II. A unit-price analysis of cigarette smoking. Journal of the Experimental Analysis of Behavior, 55, 145-154.

Bickel, W. K., Hughes, J. R., DeGrandpre, R. J., Higgins, S. T., & Rizzuto, P. (1992). Behavioral economics of drug self-administration: IV. The effects of response requirement on the consumption of and interaction between concurrently available coffee and cigarettes. Psychopharmacology, 107, 211-216.

Britt, D. M. (1998). Cigarette Smoking and Chewing Gum: Response to a Laboratory Induced Stressor. Unpublished doctoral dissertation, Oklahoma State University.

Carrington, P., Collins, G. H., Benson, H., Robison, H., Wood, L. W., Lehrer, P. M., Woodfolk, R. L., & Cole, J. (1980). The use of meditation-relaxation techniques for the management of stress in working population. Journal of Occupational Medicine, 22, 221-231.

Cohen, L.M., Collins, F. L., & Britt, D. M. (1997). The effect of chewing gum on tobacco withdrawal. Addictive Behaviors, 22, 769-773.

Cook, W. W., & Medley, D. M. (1954). Proposed hostility pharisaic virtue scales for the MMPI. Journal of Applied Psychology, 38, 414-418.

Covey, L.S., Glassman, A. H., & Stetner, F. (1990). Depression and depressive symptoms in smoking cessation. Comparative Psychiatry, 31, 350-354.

Davis, D. A. & Wedseth, J. C. (1978). A Minnesota Multiphasic Personality Inventory indicator of psychological distress in male students. Journal of Counseling Psychology, 25, 469-472.

DeGrandpre, R.J., Bickel, W. K., Hughes, J. R., & Higgins, S. T. (1992). Behavioral economics of drug-self-administration: III. A reanalysis of the nicotine regulation hypothesis. Psychopharmacology, 108, 1-10.

Derogatis, L. R. (1987). The Derogatis Stress Profile (DSP): Quantification of psychological stress. In G. Fava & T. Wise (Eds.), Advances in psychosomatic medicine. Basel: Karger.

Derogatis, L. R., & Coons, H. L. (1993). Self-report measures of stress. In L. Goldberger and S. Breznitz (Eds.), Handbook of stress: Theoretical and clinical aspects. (Vol. 2, pp. 200-233). New York: The Free Press.

Derogatis, L. R., Yevzeroff, H., & Whittelsberger, B. (1975). Social class, psychological disorder, and the nature of the psychopathologic indicator. Journal of Consulting and Clinical Psychology, 43, 183-191.

Dimberg, U. (1988). Facial electromyography and the experience of emotion. Journal of Psychophysiology, 2, 277-282.

Dohrenwend, B. S., Dohrenwend, B. P., Dodson, M., & Shrout, P. E. (1984). Symptoms, hassles, social supports, and life events: Problem of confounded measures. Journal of Abnormal Psychology, 93, 222-230.

Epstein, L. H. & Jennings, J. R. (1986). Smoking, stress, cardiovascular reactivity, and coronary heart disease. In K. A. Matthews, S. M. Weiss, T. Detre, T. M. Dembroski, B. Falkner, S. B. Manuck, & R. B. Williams Jr. (Eds.), Handbook of Stress, Reactivity, and Cardiovascular Disease. New York: John Wiley & Sons.

Gilbert, D. G. (1995). Smoking: Individual Differences, Psychopathology, and Emotion. Washington, DC: Taylor and Francis.

Gilbert, D. G., Meliska, C. J., Williams, C. L., & Jensen, R. A. (1992). Subjective correlates of cigarette-smoking-induced elevations of peripheral beta-endorphin and cortisol. Psychopharmacology, 106, 275-281.

Gilbert, R. M., & Pope, M. A. (1982). Early effects of quitting smoking. Psychopharmacology, 78, 121-127.

Glass, D. C. (1977). Stress, behavior patterns and coronary disease. American Scientist, 65, 177-187.

Gold, M. S. (1995). Tobacco. New York: Plenum Medical Book Company.

Goldband, S., Katkin, E. S., & Morell, M. A. (1979). Personality and cardiovascular disorder: Steps toward demystification. In I. G. Sarason & C. D. Speilberger (Eds.), Stress and anxiety. (Vol. 6). New York: Wiley.

Gossain, V. V., Sherma, N. K., Srivastava, L., Michelakis, A. M., & Rovner, D. R. (1986). Hormonal effects of smoking II: Effects on plasma cortisol, growth hormone, and prolactin. The American Journal of the Medical Sciences, 291, 325-327.

Graham, D. T., & Stevenson, I. (1963). Disease as a response to life stress. In H. I. Lief, V. F. Lief, & N. R. Lief (Eds.), The Psychological Basis of Medical Practice. New York: Harper & Row.

Graham, J. R. (1993). MMPI-2: Assessing personality and psychopathology. New York: Oxford University Press.

Greenglass, E. R., & Julkunen, J. (1989). Construct validity and sex differences in Cook-Medley hostility. Personality and Individual Differences, 10, 209-218.

Griffiths, R. R., Bigelow, G. E., & Liebson, I. (1976). Facilitation of human tobacco self-administration by ethanol: A behavioral analysis. Journal of the Experimental Analysis of Behavior, 25, 279-292.

Gross, J., & Stitzer, M. L. (1989). Nicotine replacement: Ten-week effects on tobacco withdrawal symptoms. Psychopharmacology, 98, 334-341.

Hall, S. M., Rugg, D., Tunstall, C., & Jones, R. T. (1984). Preventing relapse to cigarette smoking by behavioral skill training. Journal of Consulting and Clinical Psychology, 52, 372-382.

Haskell, D., Pugatch, D., & McNair, D. M. (1969). Time-limited psychotherapy for whom? Archives of General Psychiatry, 21, 546-552.

Hatsukami, D. K., Hughes, J. R., Pickens, R. W., & Svikis, D. (1984). Tobacco withdrawal symptoms: An experimental analysis. Psychopharmacology, 84, 231-236.

Hendrickson, R. (1976). The Great American Chewing Gum Book. Radnor, PA: Chilton Book Company.

Henningfield, J. E., & Griffiths, R. R. (1979). A preparation for the experimental and analysis of human cigarette smoking behavior. Behavior Research Methods Instrumentation, 11, 538-544.

Henningfield, J. E. (1987). Annual Progress Report: Biology of Dependence and Abuse Potential Assessment Laboratory. In: Annual Report of the Addiction Research Center, Addiction Research Center, National Institute on Drug Abuse.

Hollingworth, H. L. (1939). Chewing as a technique of relaxation. Science, 90, 385-387.

Holmes, T. H. (1979). Development and application of a quantitative measure of life change magnitude. In J. E. Barrett (ed.), Stress and mental disorder. New York: Raven.

Holmes, T. H., & Rahe, R. H. (1967). The social readjustment rating scale. Journal of Psychosomatic Research, 11, 213-218.

Horowitz, M., Schaefer, C., Hiroto, D., Wilner, N., & Levin, B. (1977). Life event questionnaires for measuring presumptive stress. Psychosomatic Medicine, 39, 413-431.

Hughes, J. R., Gust, S. W., & Pechacek, T.F. (1987). Prevalence of tobacco dependence and withdrawal. The American Journal of Psychiatry, 144, 205-208.

Hughes, J. R., Gust, S. W., & Skoog, K. (1991). Symptoms of tobacco withdrawal: A replication and extension. Archives of General Psychiatry, 48, 52-59.

Hughes, J. R. & Hatsukami, D. K. (1985). Short-term effects of nicotine gum. In J. Grabowski & S. Hall (Eds.), Pharmacological Adjuncts in Smoking Cessation. (NIDA Research Monograph No 53) (DHHS Publication No. ADM-85-1333). Washington, DC: U.S. Government Printing Office.

Hughes, J. R. & Hatsukami, D. K. (1986). Signs and symptoms of tobacco withdrawal. Archives of General Psychiatry, 43, 289-294.

Hursh, S.R. (1984). Behavioral economics. Journal of the Experimental Analysis of Behavior, 42, 435-452.

Hursh, S. R., & Bauman, R. A. (1987). The behavioral analysis of demand. In L. Green & J. H. Kagel (Eds.), Advances in behavioral economics (Vol. 1, pp. 117-165). Norwood, NJ: Ablex.

Hursh, S. R., Raslear, D., Shurtleff, R., Bauman, R., & Simmons, L. (1988). A cost benefit analysis of demand for food. Journal of the Experimental Analysis of Behavior, 50, 419-440.

Imber, S. D. (1975). Patient direct self-report techniques. In I. E. Waskow & M. B. Parloff (Eds.), Psychotherapy change measures. Rockville, MD: National Institute of Mental Health.

Jaeckle, R. S., & Lopez, J. F. (1986). Corticotropin releasing hormone: Clinical endocrinology and implications for Cushing's disease and endogenous depression. Psychiatric Medicine, 3, 111-134.

Jenkins, C. D., Rosenman, R. H., & Friedman, M. (1967). Development of an objective psychological test for the determination of the coronary-prone behavior pattern in employed men. Journal of Chronic Diseases, 20, 371-379.

Jenkins, C. D., & Zyzanski, S. J. (1980). Behavioral risk factors and coronary heart disease. Psychotherapy and Psychosomatics, 34, 149-177.

Johnson, J. H., & Sarason, I. G. (1978). Life stress, depression, and anxiety: Internal-external control as a moderator variable. Journal of Psychosomatic Research, 22, 205-208.

Kanner, A. D., Coyne, J. C., Schaefer, R., & Lazarus, R. S. (1981). Comparison of two modes of stress measurement: Daily hassles and uplifts versus major life events. Journal of Behavioral Medicine, 4, 1-39.

Keller-Wood, M. E., & Dallman, M. F. (1985). Corticosteroid inhibition of ACTH secretion. Endocrine Reviews, 5, 1-24.

Kessler, D. A. (Commissioner of Food and Drugs). (1994, March 25). Statement on nicotine-containing cigarettes. Statement delivered to the House Subcommittee on Health and the Environment.

Kirschbaum, C., Wüst, S., & Strasburger, C. J. (1992). 'Normal' cigarette smoking increases free cortisol in habitual smokers. Life Sciences, 50, 435-442.

Kozlowski, L. T., Wilkinson, D. A. (1987). Use and misuse of the concept of craving by alcohol, tobacco, and drug researchers. British Journal of Addiction, 82, 31-45.

Kuhn, C. M. (1989). Adrenocortical and gonadal steroids in behavioral cardiovascular medicine. In N. Schneiderman, S. M. Weiss, & P. G. Kaufmann (Eds.), Handbook of Research Methods in Cardiovascular Behavioral Medicine. (pp. 185-204). New York: Plenum Press.

Lamping, D. L. (1985). Assessment in health psychology. Canadian Psychology, 26, 121-139.

Lazarus, R. S. (1976). Patterns of adjustment. New York: McGraw-Hill.

Lazarus, R. S., Cohen, J. B., Folkman, S., Kanner, A., & Schaefer, C. (1980). Psychological stress and adaptation: Some unresolved issues. In H. Selye (Ed.) Selye's Guide to Stress Research. (Vol. 1, pp. 90-117). New York: Van Nostrand Reinhold Company.

Lazarus, R. S., & Folkman, S. (1984). Stress, Appraisal, and Coping. New York: Springer.

Lester, N., Nebel, L. E., & Baum, A. (1994). Psychophysiological and Behavioral Measurement of Stress: Applications to Mental Health. In W. R. Avison & I. H. Gottlib (Eds.), Stress and mental Health: Contemporary Issues and Prospects for the Future. (pp. 291- 314). New York: Plenum Press.

Lichtenstein, E. & Glasgow, R. (1992). Smoking cessation: What have we learned over the past decade? Journal of Consulting and Clinical Psychology, 60, 518-527.

Lopez, J. F., Young, E. A., Herman, J. P., Akil, H., & Watson, S. J. (1991). Regulatory biology of the HPA axis: An integrative approach. In S. C. Risch (Ed.), Central Nervous System Peptide Mechanisms in Stress and Depression. (pp. 1-52). Washington, DC: American Psychiatric Press, Inc.

Marshall, W. R., Epstein, L. H., & Green, S. B. (1980). Coffee drinking and cigarette smoking: I. Coffee, caffeine, and cigarette smoking behavior. Addictive Behaviors, 5, 389-394.

Marshall, W. R., Green, S. B., Epstein, L. H., Rogers, C. M., & McCoy, J. F. (1980). Coffee drinking and cigarette smoking: II. Coffee, urinary pH and cigarette smoking behavior. Addictive Behaviors, 5, 395-400.

Mason, J.W. (1975). A historical view of the stress field. Journal of Human Stress, 1, 6-27.

Mausner, J. S. (1970). Cigarette smoking among patients with respiratory disease. American Review of Respiratory Disease, 102, 704-713.

McChargue, D. E. (1998). Alternative to Smokeless Tobacco: Documented Reductions in Withdrawal Patterns. Unpublished doctoral dissertation, Oklahoma State University.

McNair, D. M. (1974). Self-evaluations of antidepressants. Psychopharmacologia, 37, 281-302.

McNair, D. M., Lorr, M., & Droppleman, L. F. (1971). Manual: Profile of Mood States. San Diego, CA: Educational and Industrial Testing Service.

Monroe, S. M. (1989). Stress and support: Assessment issues. In N. Schneiderman, S. M. Weiss, & P. G. Kaufmann (Eds.), Handbook of research methods in cardiovascular behavioral medicine. New York: Plenum Press.

Murphee, H. B., & Schultz, R. E. (1968). Abstinence effects in smokers. (Abstract.) Federal Proceedings, 27, 220.

National Institute on Drug Abuse. (1994). National survey results on drug use from monitoring the future study, 1975-1993, Volume II: College students and young adults (NIH Publication No. 94-3810). Washington, DC: U.S. Government Printing Office.

Nielson, A. C., III, & Williams, T. A. (1980). Depression in ambulatory medical patients. Archives of General Psychiatry, 37, 999-1004.

Orth, D. N., Jackson, R. V., DeCherney, G. S., DeBold, C. R., Alexander, A. N., Island, D. P., Rivier, J., Rivier, C., Speiss, J., & Vale, W. (1983). Effects of ovine synthetic corticotropin releasing factor: Dose response of plasma adrenocorticotropin and cortisol. Journal of Clinical Investigation, 71, 587-595.

Pancheri, P., Bellaterra, M., Matteoli, S., Christofari, M., Polizzi, C., & Puletti, M. (1978). Infarct as a stress agent: Life history and personality characteristics in improved versus non-improved patients after severe heart attack. Journal of Human Stress, 5, 16-22.

Pederson, L. L., & Lefcoe, N. M. (1976). A psychological and behavioural comparison of ex-smokers and smokers. Journal of Chronic Diseases, 29, 431-434.

Perkins, K. A., Epstein, L. H., Sexton, J. E., & Pastor, S. (1990). Effects of smoking cessation on consumption of alcohol and sweet, high-fat foods. Journal of Substance Abuse, 2, 287-297.

Piotrowski, C. & Lubin, B. (1990). Assessment practices of health psychologist: Survey of APA division 38 clinicians. Professional Psychology, 21, 99-106.

Pomerleau, O. F., Scherzer, H. H., Grunberg, N. E., Pomerleau, C. S., Judge, J., Fertig, J. B., & Burleson, J. (1987). The effects of acute exercise on subsequent cigarette smoking. Journal of Behavioral Medicine, 10, 117-127.

Rahe, R. H., & Holmes, T. H. (1965). Social, psychologic and psychophysiologic aspects of inguinal hernia. Journal of Psychosomatic Research, 8, 487.

Rahe, R.H., Meyer, M., Smith, M., Kjaer, G., & Holmes, T. H. (1964). Social stress and illness onset. Journal of Psychosomatic Research, 8, 35-44.

Rahe, R. H., McKean, J. D., & Arthur, R. J. (1967). A longitudinal study of life change and illness patterns. Journal of Psychosomatic Research, 10, 355.

Roth, D. L., & Holmes, D. S. (1987). Influence of aerobic exercise training and relaxation training on physical and psychological health following stressful life events. Psychosomatic Medicine, 49, 355-365.

Rubin, R. P., & Warner, W. (1975). Nicotine-induced stimulation of steroidogenesis in adrenocortical cells of the cat. British Journal of Pharmacology, 53, 357-362.

Sarason, I. G., Johnson, J. H., & Siegal, J. M. (1979). Assessing the impact of life changes. In I. G. Sarason & C. D. Spielberger (eds.), Stress and anxiety, Volume 6. New York: Wiley.

Schacter, S., Silverstein, B., Kozlowski, L., Perlick, D., Herman, C., & Liebling, B. (1977). Studies of the interaction of psychological and pharmacological determinants of smoking. Journal of Experimental Psychology: General, 106, 3-40.

Schwab, J. J., Bialow, M., Brown, J. M., & Holzer, C. E. (1967). Diagnosing depression in medical inpatients. Annals of Internal Medicine, 67, 695-707.

Selye, H. (1973). The evolution of the stress concept. American Scientist, 61, 692-699.

Shacham, S., Reinhardt, L. C., Raubertas, R. F., & Cleeland, C. S. (1983). Emotional states and pain: Intraindividual and interindividual measures of association. Journal of Behavioral Medicine, 6, 405-409.

Shiffman, S. M. (1979). The tobacco withdrawal syndrome. In N. A. Krasnegor (Ed.), Cigarette Smoking as a Dependence Process (NIDA Research Monograph 23, pp. 158-184). Rockville, MD: U.S. Department of Health, Education, and Welfare.

Shiffman, S. M., & Jarvik, M. E. (1976). Smoking withdrawal symptoms in two weeks of abstinence. Psychopharmacology, 50, 35-39.

Speilberger, C. D., Gorsuch, R. L., Lusshene, R. E., Vagg, P. R., & Jacobs, G. A. (1983). Manual for the State-Trait Anxiety Inventory (STAI Form Y). Palo Alto, CA: Consulting Psychologists Press.

Stitzer, M. L., & Gross, J. (1988). Smoking relapse: The role of pharmacological and behavioral factors. In O. F. Pomerleau & C. S. Pomerleau (Eds.), Nicotine replacement: A clinical evaluation. (pp. 163-184). New York: Liss.

Stott, H. (1998). Evaluating the Value of Chewing Gum as a Substitute Reinforcer for Nicotine. Unpublished masters thesis, Oklahoma State University.

Taylor, S. E., Lichtman, R. R., & Wood, J. V. (1984). Attributions, beliefs about control, and adjustment to breast cancer. Journal of Personality and Social Psychology, 46, 489-502.

Trahir, R. C. S. (1967). Giving up cigarettes: 222 case studies. Medical Journal of Australia, 1, 929-932.

U.S. Department of Health and Human Services. (1990). Smoking and Health: A National Status Report (DHHS Publication No. CDC 87-8396). Washington, DC: Government Printing Office.

U.S. Department of Health and Human Services. (1988). The Health Consequences of Smoking: Nicotine Addiction (DHHS Publication No. CDC 88-8406). Washington DC: Government Printing Office.

Vernikos-Danellis, J., & Heybach, J. P. (1980). Psychophysiologic mechanisms regulating the hypothalamic-pituitary-adrenal response to stress. In H. Selye (Ed.) Selye's Guide to Stress Research. (Vol. 1, pp. 206-251). New York: Van Nostrand Reinhold Company.

Vingerhoets, A. J., & Marcelissen, F. G. (1988). Stress research: Its present status and issues for future developments. Social Science and Medicine, 6, 279-291.

Weiss, E., Dlin, B., Rollin, H. R., Fischer, H. K., & Bepler, C. R. (1957). Emotional factors in coronary occlusion. Archives of Internal Medicine, 99, 628.

West, R. J., & Kranzler, H. R. (1992). Craving for cigarettes and psychoactive drugs. In D. Warburton (Ed.), Addiction Controversies. New York: Harwood Academic Publishers.

West, R. J., & Russell, M. A. H. (1988). Loss of acute nicotine tolerance and severity of cigarette withdrawal. Psychopharmacology, 94, 563-565.

West, R. J., Hajek, P., & Belcher, M. (1987). Time course of cigarette withdrawal symptoms during four weeks of treatment with nicotine chewing gum. Addictive Behaviors, 12, 199-203.

Weybrew, B. B., & Stark, J. E. (1967). Psychological and Physiological Changes Associated with Deprivation from Smoking. U.S. Naval Submarine Medical Center Report No. 490. Bureau of Medicine and Surgery, Navy Department.

Wilkins, J. N., Carlson, H. E., Van Vunakis, H., Hill, M. A., Gritz, E., & Jarvik, M. E. (1982). Nicotine from cigarette smoking increases circulating levels of cortisol, growth hormone, and prolactin in male chronic smokers. Psychopharmacology, 78, 305-308.

Williams, R. B. & Barefoot, J. C. (1988). Coronary-prone behavior: The emerging role of the hostility complex. In B. K. Houston & C. R. Snyder (Eds.), Type A behavior pattern. Research, theory, and intervention. New York: Wiley.

Winternitz, W. W., & Quillen, D. (1977). Acute hormonal response to cigarette smoking. Journal of Clinical Pharmacology, 17, 389-397.

Wynder, E. L., Kaufman, P. L., & Lesser, R. L. (1967). A short-term follow up study on ex-cigarette smokers. American Review of Respiratory Disease, 96, 645-655.

Yeh, J., & Barbieri, R. L. (1989). Twenty-four-hour urinary-free-cortisol in premenopausal cigarette smokers and nonsmokers. Fertility and Sterility, 52, 1067-1069.

Zimmerman, M., Coryell, W., Corenthal, C., and Wilson, S. (1986). A self-report scale to diagnose major depressive disorder. Archives of General Psychiatry, 43, 1076-1081.

APPENDIX A

GENERAL HABIT INFORMATION

1) Sleep Habits:

Hours of sleep last night: _____

Average hours of sleep per night over the last week: _____

2) Smoking Habits

How many cigarettes per day do you smoke? _____

On an average week, how many cigarettes do you smoke? _____

Does it bother you to abstain from smoking for 12 hours (circle): YES NO

If YES, how bothered (please circle one):

EXTREMELY VERY MUCH MODERATELY SLIGHTLY

3) Drinking Habits

How much alcohol do you drink? (please circle one)

NEVER 2 OR LESS PER DAY GREATER THAN 2 PER DAY

4) Caffeine Consumption

Number of soda cans	_____/day	_____/week
Number of cups of tea	_____/day	_____/week
Number of cups of coffee	_____/day	_____/week

Caffeine Consumption Today: _____

Does it bother you to abstain from drinking coffee for 12 hours? (circle)

YES NO

If YES, how bothered (please circle one):

EXTREMELY VERY MUCH MODERATELY SLIGHTLY

5) Other Information

Are you currently taking any medication (circle): YES NO

If YES, what are you taking? _____

Quantity/Description	Time	Date	
Last time you had a snack:	_____	_____	_____
Last time you ate a full meal:	_____	_____	_____
Last time you consumed caffeine or alcohol:	_____	_____	_____
Last time you consumed a prescription or over the counter drug:	_____	_____	_____

In general, how would you rate your health today? (please circle one)

GOOD FAIR MODERATELY POOR POOR

Do either of your parents have high blood pressure? (circle): YES NO

If YES, which parent? (please circle one)

MOTHER FATHER BOTH

IDD

Directions: On this questionnaire are groups of 5 statements. Read each group of statements carefully. Then pick out the statement in each group that best describes the way you have been feeling the PAST WEEK. Circle the number next to the statement you picked. For every group in which you circled #1, 2, 3, or 4, answer the follow-up question as to whether you have been feeling that way for more or less than 2 weeks by circling either "more" or "less" as appropriate.

1.
0 I do not feel sad or depressed.
1 I occasionally feel sad or down.
2 I feel sad most of the time, but I can snap out of it.
3 I feel sad all the time, and I can't snap out of it.
4 I am so sad or unhappy that I can't stand it.

If you circled #1, 2, 3, or 4: Have you been feeling sad or down for more or less than 2 weeks? More Less

2.
0 My energy level is normal.
1 My energy level is occasionally a little lower than normal.
2 I get tired more easily or have less energy than usual.
3 I get tired from doing almost anything
4 I feel tired or exhausted almost all the time

If you circled #1, 2, 3, or 4: Has your energy level been lower than usual for more or less than 2 weeks? More Less

3.
0 I have not been feeling more restless and fidgety than usual.
1 I feel a little more restless or fidgety than usual.
2 I have been very fidgety, and I have some difficulty sitting still in a chair.
3 I have been extremely fidgety, and I have been pacing a little bit almost every day.
4 I have been pacing for more than an hour per day, and I can't sit still.

If you circled #1, 2, 3, or 4: Have you felt restless and fidgety for more or less than 2 weeks? More Less

4.
0 I have not been talking or moving more slowly than usual.
1 I am talking a little slower than usual.
2 I am speaking slower than usual, and it takes longer to respond to questions, but I can still carry on a normal conversation.
3 Normal conversations are difficult because it is hard to start talking.
4 I feel extremely slowed down physically, like I am stuck in mud.

If you circled #1, 2, 3, or 4: Have you felt slowed down for more or less than 2 weeks? More Less

5.
0 I have not lost interest in my usual activities.
1 I am a little less interested in 1 or 2 of my usual activities.
2 I am less interested in several of my usual activities.
3 I have lost most of my interest in almost all of my usual activities.
4 I have lost all interest in all of my usual activities.

If you circled #1, 2, 3, or 4: Has your interest in your usual activities been low for more or less than 2 weeks? More Less

6.
0 I get as much pleasure out of my usual activities as usual.
1 I get a little less pleasure from 1 or 2 or my usual activities
2 I get less pleasure from several of my usual activities.
3 I get almost no pleasure from most of the activities which I usually enjoy.
4 I get no pleasure from any of the activities which I usually enjoy.

If you circled #1, 2, 3, or 4: Has your enjoyment in your usual activities been low for more or less than 2 weeks? More Less

7.
0 I have not noticed any recent change in my interest in sex.
1 I am only slightly less interested in sex than usual.
2 There is a noticeable decrease in my interest in sex.
3 I am much less interested in sex now.
4 I have lost interest in sex.

If you circled #1, 2, 3, or 4: Has your interest in sex been low for more or less than 2 weeks? More Less

8.
0 I have not been feeling guilty.
1 I occasionally feel a little guilty.
2 I often feel guilty.
3 I feel quite guilty most of the time.
4 I feel extremely guilty most of the time.

If you circled #1, 2, 3, or 4: Have you had guilt feelings for more or less than 2 weeks? More Less

9.
0 I do not feel like a failure.
1 My opinion of myself is occasionally a little low.
2 I feel I am inferior to most people.
3 I feel like a failure.
4 I feel I am a totally worthless person.

If you circled #1, 2, 3, or 4: Have you been down on yourself for more or less than 2 weeks? More Less

10.
0 I haven't had any thoughts of death or suicide.
1 I occasionally think life is not worth living.
2 I frequently think of dying in passive ways (such as going to sleep and not waking up), or that I'd be better off dead.
3 I have frequent thoughts of killing myself, but I would not carry them out..
4 I would kill myself if I had the chance.

If you circled #1, 2, 3, or 4: Have you been thinking about dying or killing yourself for more or less than 2 weeks? More Less

11.
0 I can concentrate as well as usual.
1 My ability to concentrate is slightly worse than usual.
2 My attention span is not as good as usual and I am having difficulty.
3 My ability to read or hold a conversation is not as good as it usually is.
4 I cannot read, watch TV, or have a conversation without great difficulty.

If you circled #1, 2, 3, or 4: Have you had problems concentrating for more or less than 2 weeks? More Less

12.
0 I make decisions as well as I usually do.
1 Decision making is slightly more difficult than usual.
2 It is harder and takes longer to make decisions, but I do make them.
3 I am unable to make some decisions.
4 I can't make any decisions at all.

If you circled #1, 2, 3, or 4: Have you had problems making decisions for more or less than 2 weeks? More Less

13.
0 My appetite is not less than normal.
1 My appetite is slightly worse than usual.
2 My appetite is clearly not as good a usual.
3 My appetite is much worse now.
4 I have no appetite at all, and I have to force myself to eat even a little.

If you circled #1, 2, 3, or 4: Has your appetite been decreased for more or less than 2 weeks? More Less

14.
0 I haven't lost any weight.
1 I've lost less than 5 pounds.
2 I've lost between 5 and 10 pounds.
3 I've lost between 11 and 25 pounds.
4 I've lost more than 25 pounds.

If you circled #1, 2, 3, or 4: Have you been dieting and deliberately trying to lose weight? Yes No

If you circled #1, 2, 3, or 4: Have you been losing weight for more or less than 2 weeks? More Less

15.
0 My appetite is not greater than normal.
1 My appetite is slightly greater than usual.
2 My appetite is clearly greater than usual.
3 My appetite is much greater than usual.
4 I feel hungry all the time.

If you circled #1, 2, 3, or 4: Has you appetite been increased for more or less than 2 weeks? More Less

16.
0 I haven't gained any weight..
1 I've gained less than 5 pounds.
2 I've gained between 5 and 10 pounds.
3 I've gained between 11 and 25 pounds.
4 I've gained more than 25 pounds.

If you circled #1, 2, 3, or 4: Has your appetite been increased for more or less than 2 weeks? More Less

17.
0 I am not sleeping less than normal.
1 I occasionally have slight difficulty sleeping.
2 I clearly don't sleep as well as usual.
3 I sleep about half my normal amount of time..
4 I sleep less than 2 hours per night.

If you circled #1, 2, 3, or 4: Which of these sleep problems have you experienced? (Circle all which apply)

1 I have difficulty falling asleep.
2 My sleep is fitful and restless I the middle of the night.
3 I wake up earlier than usual and cannot fall back to sleep

If you circled #1, 2, 3, or 4: Have you been having sleep problems for more or less than 2 weeks?

18.
0 I am not sleeping more than normal.
1 I occasionally sleep more than usual.
2 I frequently sleep at least 1 hour more than usual.
3 I frequently sleep at least 2 hours more than usual..
4 I frequently sleep at least 3 hours more than usual.

If you circled #1, 2, 3, or 4: Have you been sleeping extra for more or less than 2 weeks? More Less

19.
0 I do not feel anxious, nervous, or tense..
1 I occasionally feel a little anxious.
2 I often feel anxious..
3 I feel very anxious most of the time.
4 I fee terrified and near panic.

If you circled #1, 2, 3, or 4: Have you been feeling anxious, nervous, or tense for more or less than 2 weeks? More Less

20.
0 I do not feel discouraged about the future.
1 I occasionally feel a little discouraged about the future.
2 I often feel discouraged about the future.
3 I feel very discouraged about the future most of the time.
4 I feel that the future is hopeless and that things will never improve.

If you circled #1, 2, 3, or 4: Have you been feeling discouraged than usual for more or less than 2 weeks? More Less

21.
0 I do not feel irritated or annoyed.
1 I occasionally get a little more irritated than usual.
2 I get irritated or annoyed by things that usually don't bother me.
3 I feel irritated or annoyed almost all of the time.
4 I feel so depressed that I don't get irritated at all by things that used to bother me.

If you circled #1, 2, 3, or 4: Have you been feeling more irritable than usual for more or less than 2 weeks? More Less

22.
0 I am not worried about my physical health.
1 I am occasionally concerned about bodily aches and pains.
2 I am worried about my physical health.
3 I am very worried about my physical health.
4 I am so worried about my physical health that I cannot think about anything else.

If you circled #1, 2, 3, or 4: Have you been worried about your physical health for or less than 2 weeks? More Less

The Daily Hassles Scale

Directions: Hassles are irritants that can range from minor annoyances to fairly major pressures, problems, or difficulties. They can occur few or many times.

Listed in the center of the following pages are a number of ways in which a person can feel hassled. First, circle the hassles that have happened to you in the past month. Then look at the numbers on the right of the items you circled. Indicate by circling a 1, 2, or 3 how SEVERE each of the circled hassles has been for you in the past month. If a hassle did not occur in the last month do NOT circle it.

Hassles	SEVERITY 1. Somewhat severe 2. Moderately severe 3. Extremely severe		
(1) Misplacing or losing things	1	2	3
(2) Troublesome neighbors	1	2	3
(3) Social obligations	1	2	3
(4) Inconsiderate smokers	1	2	3
(5) Troubling thoughts about your future	1	2	3
(6) Thoughts about death	1	2	3
(7) Health of a family member	1	2	3
(8) Not enough money for clothing	1	2	3
(9) Not enough money for housing	1	2	3
(10) Concerns about owing money	1	2	3
(11) Concerns about getting credit	1	2	3
(12) Concerns about money for emergencies	1	2	3
(13) Someone owes you money	1	2	3
(14) Financial responsibility for someone who doesn't live with you	1	2	3
(15) Cutting down on electricity, water, etc.	1	2	3
(16) Smoking too much	1	2	3
(17) Use of alcohol	1	2	3
(18) Personal use of drugs	1	2	3
(19) Too many responsibilities	1	2	3

(20) Decisions about having children 1 2 3

(21) Non-family members living in your house 1 2 3

(22) Care for pet 1 2 3

(23) Planning meals 1 2 3

(24) Concerned about the meaning of life 1 2 3

(25) Trouble relaxing 1 2 3

(26) Trouble making decisions 1 2 3

(27) Problems getting along with fellow workers 1 2 3

(28) Customers or clients give you a hard time 1 2 3

(29) Home maintenance (inside) 1 2 3

(30) Concerns about job security 1 2 3

(31) Concerns about retirement 1 2 3

(32) Laid-off or out of work 1 2 3

(33) Don't like current work duties 1 2 3

(34) Don't like fellow workers 1 2 3

(35) Not enough money for basic necessities 1 2 3

(36) Not enough money for food 1 2 3

(37) Too many interruptions 1 2 3

(38) Unexpected company 1 2 3

(39) Too much time on hands 1 2 3

(40) Having to wait 1 2 3

(41) Concerns about accidents 1 2 3

(42) Being lonely 1 2 3

(43) Not enough money for health care 1 2 3

(44) Fear of confrontation 1 2 3

(45) Financial security 1 2 3

(46) Silly practical mistakes 1 2 3

(47) Inability to express yourself 1 2 3

(48) Physical illness 1 2 3

(49) Side effects of medication 1 2 3

(50) Concerns about medical treatment 1 2 3

(51) Physical appearance 1 2 3

(52) Fear of rejection 1 2 3

(53) Difficulties with getting pregnant 1 2 3

(54) Sexual problems that result from physical problems 1 2 3

(55) Sexual problems other than those resulting from physical problems 1 2 3

(56) Concerns about health in general 1 2 3

(57) Not seeing enough people 1 2 3

(58) Friends or relatives too far away 1 2 3

(59) Preparing meals 1 2 3

(60) Wasting time 1 2 3

(61) Auto maintenance 1 2 3

(62) Filling out forms 1 2 3

(63) Neighborhood deterioration 1 2 3

(64) Financing children's education 1 2 3

(65) Problems with employees 1 2 3

(66) Problems on job due to being a woman or man 1 2 3

(67) Declining physical abilities 1 2 3

(68) Being exploited 1 2 3

(69) Concerns about bodily functions 1 2 3

(70) Rising prices of common goods 1 2 3

(71) Not getting enough rest 1 2 3

(72) Not getting enough sleep 1 2 3

(73) Problems with aging parents 1 2 3

(74) Problems with your children 1 2 3

(75) Problems with persons younger than yourself 1 2 3

(76) Problems with your lover 1 2 3

(77) Difficulties seeing or hearing 1 2 3

(78) Overloaded with family responsibilities 1 2 3

(79) Too many things to do 1 2 3

(80) Unchallenging work 1 2 3

(81) Concerns about meeting high standards 1 2 3

(82) Financial dealings wit friends or acquaintances 1 2 3

(83) Job dissatisfactions 1 2 3

(84) Worries about decisions to change jobs 1 2 3

(85) Trouble with reading, writing or spelling abilities 1 2 3

(86) Too many meetings 1 2 3

(87) Problems with divorce or separation 1 2 3

(88) Trouble with arithmetic skills 1 2 3

(89) Gossip 1 2 3

(90) Legal problems 1 2 3

(91) Concerns about weight 1 2 3

(92) Not enough time to do the things you need to do 1 2 3

(93) Television 1 2 3

(94) Not enough personal energy 1 2 3

(95) Concerns about inner conflicts 1 2 3

(96) Feel conflicted over what to do 1 2 3

(97) Regrets over past decisions 1 2 3

(98) Menstrual (period) problems 1 2 3

(99) The weather 1 2 3

(100) Nightmares 1 2 3

(101) Concerns about getting ahead 1 2 3

(102) Hassles from boss or supervisor 1 2 3

(103) Difficulties with friends 1 2 3

(104) Not enough time for family 1 2 3

(105) Transportation problems 1 2 3

(106) Not enough money for transportation 1 2 3

(107) Not enough money for entertainment and recreation 1 2 3

(108) Shopping 1 2 3

(109) Prejudice and discrimination from others 1 2 3

(110) Property, investments or taxes 1 2 3

(111) Not enough time for entertainment and recreation 1 2 3

(112) Yardwork or outside home maintenance 1 2 3

(113) Concerns about news events 1 2 3

(114) Noise 1 2 3

(115) Crime 1 2 3

(116) Traffic 1 2 3

(117) Pollution 1 2 3

Have we missed any of your hassles? If so, write them in below:

(118) _____ 1 2 3

ONE MORE THING: HAS THERE BEEN A CHANGE IN YOUR LIFE THAT AFFECTED HOW YOU ANSWERED THIS SCALE? IF SO, TELL US WHAT IT WAS:

_____

Tobacco Withdrawal Symptom Checklist

Directions: Please rate (circle) the level of your **current** withdrawal symptoms.

	NOT PRESENT	MILD	MODERATE	SEVERE
1. Craving	0	1	2	3
2. Irritability	0	1	2	3
3. Anxiety	0	1	2	3
4. Difficulty Concentrating	0	1	2	3
5. Restlessness	0	1	2	3
6. Headache	0	1	2	3
7. Drowsiness	0	1	2	3
8. Intestinal Disturbance	0	1	2	3
9. Fatigue	0	1	2	3
10. Impatience	0	1	2	3
11. Hunger	0	1	2	3
12. Insomnia	0	1	2	3

Please list any somatic (bodily) difficulties you are currently experiencing (i.e. sweating, dizziness, nausea).

1._____ 2._____

3._____ 4._____

Have you noticed any changes since your last cigarette? Yes No

If yes, what have you noticed?

_____

_____

APPENDIX B

OKLAHOMA STATE UNIVERSITY
INSTITUTIONAL REVIEW BOARD
HUMAN SUBJECTS REVIEW

Date: 07-01-97 **IRB#:** AS-97-074

Proposal Title: A MULTIMODAL ASSESSMENT OF THE EFFECT OF CHEWING GUM ON NICOTINE WITHDRAWAL

Principal Investigator(s): Frank L. Collins, Lee M. Cohen

Reviewed and Processed as: Expedited

Approval Status Recommended by Reviewer(s): Approved

ALL APPROVALS MAY BE SUBJECT TO REVIEW BY FULL INSTITUTIONAL REVIEW BOARD AT NEXT MEETING, AS WELL AS ARE SUBJECT TO MONITORING AT ANY TIME DURING THE APPROVAL PERIOD.
APPROVAL STATUS PERIOD VALID FOR DATA COLLECTION FOR A ONE CALENDAR YEAR PERIOD AFTER WHICH A CONTINUATION OR RENEWAL REQUEST IS REQUIRED TO BE SUBMITTED FOR BOARD APPROVAL.
ANY MODIFICATIONS TO APPROVED PROJECT MUST ALSO BE SUBMITTED FOR APPROVAL.

Comments, Modifications/Conditions for Approval or Disapproval are as follows:

Signature: Date: July 1, 1997

Chair of Institutional Review Board

cc: Lee M. Cohen

Table 1

Craving

Source	df	MS	F	p
Time	03	25.608	101.191	.000
Error (Time)	57	0.253		
Condition	01	0.100	0.156	.697
Error (Condition)	19	0.639		
Time X Condition	03	0.083	0.559	.644
Error (Time X Condition)	57	0.149		

Table 2

Total Withdrawal Symptoms

Initial Analyses

Source	df	MS	F	p
Time	03	124.573	18.978	.000
Error (Time)	57	6.564		
Condition	01	15.006	0.830	.374
Error (Condition)	19	18.085		
Time X Condition	03	4.706	2.537	.066
Error (Time X Condition)	57	1.855		

Post Hoc/Simple Effects Test

Source	df	MS	F	p
Total Withdrawal Symptoms at Time 1				
Condition	01	0.625	0.337	n.s.
Error	57	1.855		
Total Withdrawal Symptoms at Time 2				
Condition	01	1.600	0.863	n.s.
Error	57	1.855		
Total Withdrawal Symptoms at Time 3				
Condition	01	10.000	5.391	$p<.05$*
Error	57	1.855		
Total Withdrawal Symptoms at Time 4				
Condition	01	16.900	9.111	$p<.01$*
Error	57	1.855		

Table 3

Cortisol Levels Pre-Post Cigarette (Times 1 and 2)

Source	df	MS	F	p
Time	01	1.318	1.240	.279
Error (Time)	19	1.064		
Condition	01	0.118	0.038	.847
Error (Condition)	19	3.095		
Time X Condition	01	0.100	0.132	.721
Error (Time X Condition)	19	0.759		

Table 4

Cortisol Levels During Withdrawal (Times 3, 4, and 5)

Source	df	MS	F	p
Time	02	0.249	0.749	.480
Error (Time)	38	0.332		
Condition	01	0.016	0.024	.877
Error (Condition)	19	0.641		
Time X Condition	02	0.283	1.415	.255
Error (Time X Condition)	38	0.200		

Figure Captions

Figure 1. Mean Craving Score for Gum and No-Gum Day at Time 1, Time 2, Time 3, and Time 4.

Figure 2. Mean Total Withdrawal Score for Gum and No-Gum Day at Time 1, Time 2, Time 3, and Time 4.

Figure3. Mean Salivary Cortisol Levels for Gum and No-Gum Day at Time 1, Time 2, Time 3, and Time 4.

Figure 1

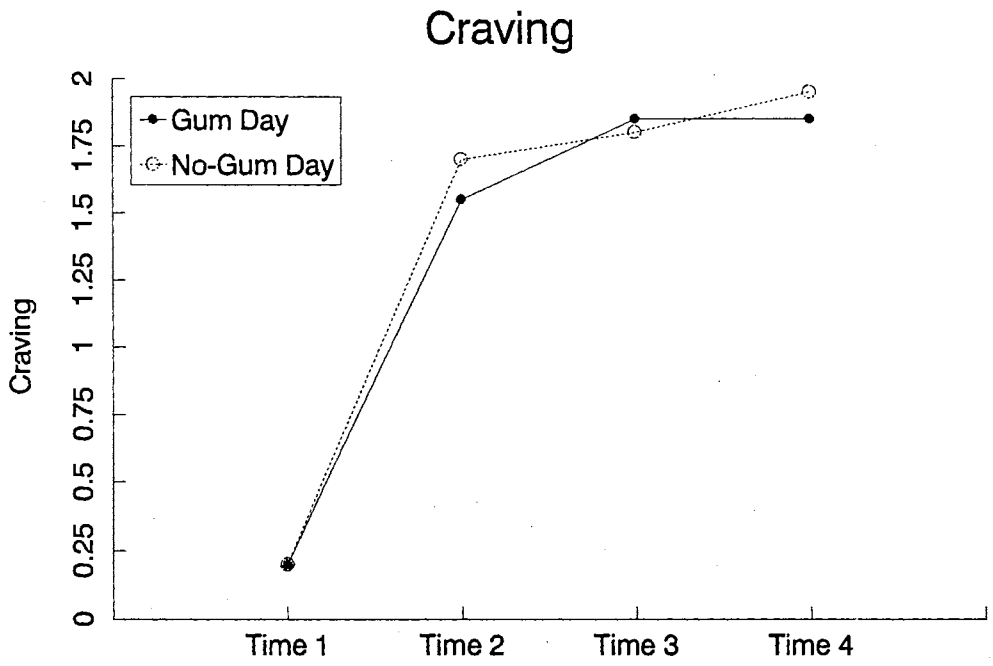


Figure 2

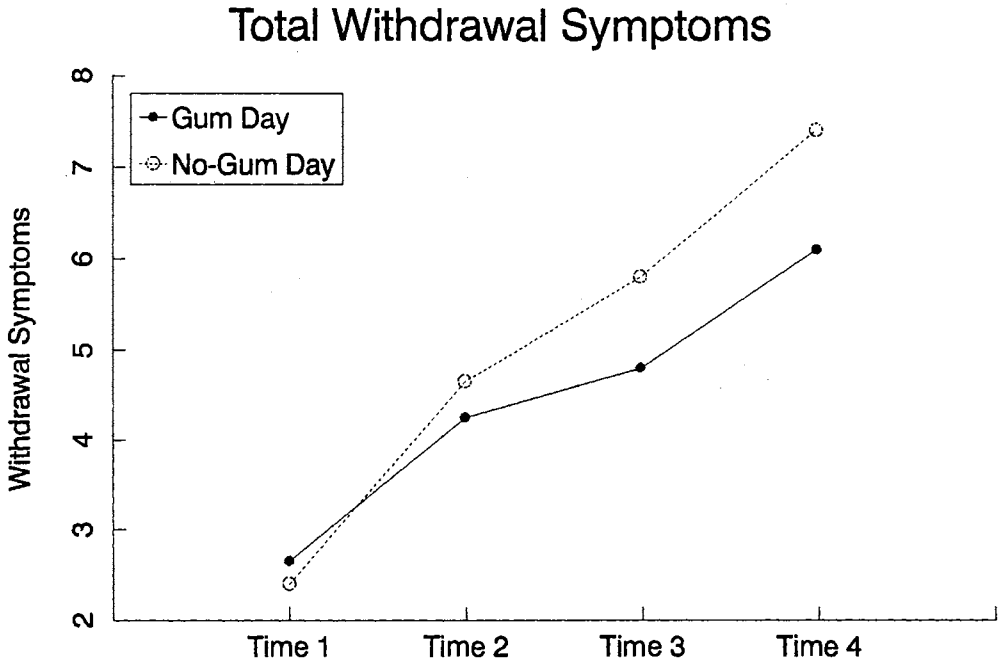
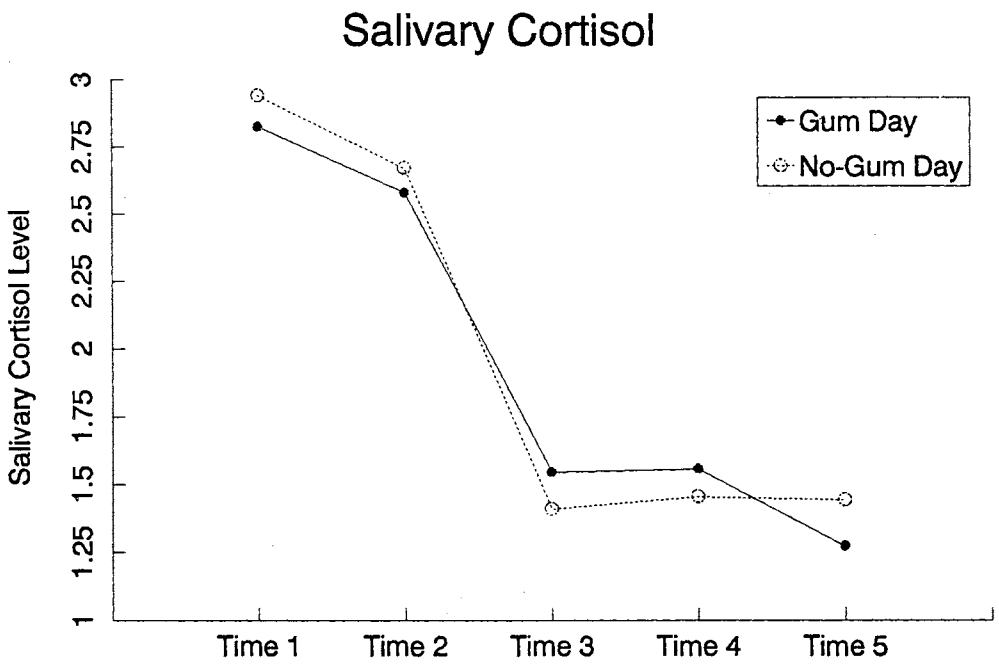


Figure 3


Salivary Cortisol
Salivary Cortisol Level
1
1.25
1.5
1.75
2
2.25
2.5
2.75
3
Time 1
Time 2
Time 3
Time 4
Time 5
Gum Day
No-Gum Day

v

VITA

Lee M. Cohen

Candidate for the Degree of

Doctor of Philosophy

Thesis: A MULTIMODAL ASSESSMENT OF THE EFFECT OF CHEWING GUM ON NICOTINE WITHDRAWAL

Major Field: Psychology

Biographical:

Personal Data: Born in Encino, California, July 2, 1972, the son of Fred and Hazel Cohen.

Education: Graduated from Canyon High School, Canyon Country, California in June 1990; received Bachelor of Arts degree in Psychology from the University of California, San Diego, La Jolla, California in June, 1994; received Master of Science degree in Psychology from Oklahoma State University, Stillwater, Oklahoma in May, 1996. Completed the requirements for the Doctor of Philosophy degree with a major in Psychology at Oklahoma State University in July, 1999.

Professional Memberships: American Psychological Association, Southwestern Psychological Association, Society of Behavioral Medicine, Association for the Advancement of Behavior Therapy, Society for Research on Nicotine and Tobacco.