

CAFFEINE: PSYCHOLOGICAL EFFECTS, USE AND ABUSE

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ABSTRACT

Caffeine, probably the most widely used drug, affects the psychological state of those who consume it. Abuse results in symptoms of caffeinism which include agitation, disorientation and a syndrome which may be mistaken for anxiety/neurosis. It is a habit-forming drug in which tolerance develops. It affects sleep in a dose related manner which is dependent on the daily caffeine intake, i.e., high users have less effect. Its central nervous system stimulation can cause pleasant effects with improved attention and concentration at lower doses. At high doses, the reverse may occur. Used judiciously, it may be a useful therapy in the treatment of hyperkinetic children. These and other effects of caffeine are discussed in this review article.

INTRODUCTION

Caffeine is among the most widely used drugs because of its ubiquitous occurrence in commonly consumed beverages such as coffee, tea and cola. Many drugs contain caffeine and are readily accessible to the public in the form of OTC stimulants and combination analgesics. Clearly caffeine is an important drug-food substance in our society which deserves attention.

To begin to have a new consciousness about caffeine so that we can become aware of how this drug can affect our physiology and psychology is a problem. The reasons for this are certainly complicated, but we can start by considering a factor dominating all of our lives, our "habits." When we become aware of and take responsibility to change habits, we are taking a first step in the process of awakening. The result must be not only an improvement in the quality of our lives but the world itself will be changed for the better.

The use and abuse of caffeine is a major public "habit" and may be as important a factor as heredity and environment in the etiology of physiological and psychological disorders. To recognize this, we must know that we are creatures of habit. Most people are caffeine consumers because from birth this food-drug is set before us, if not offered directly, along with orange juice, cereal, dessert and cigarettes.

This paper reviews the literature relating to the psychological effects of caffeine. Caffeine is a potent central nervous system stimulant and much of its "psychological" activity may be related to this action of the drug. Its effects on the nervous system are obviously adverse at high doses. It may not be obvious that at lower doses when used in moderation, it may have beneficial effects. For example, its possible therapeutic use in hyperkinetic children certainly would seem advantageous when compared to the current treatment with more powerful stimulants which have concomitant adverse reactions. Also, with the intense day to day pressures imposed on and accepted by many of us, is there any harm in "relaxing" with a hot cup of coffee? On the other hand, caffeine is a drug which is subject to abuse. The fact that it is a drug with a potentially powerful physiological effect escapes most of us who think of coffee as a relatively harmless beverage. Recently published studies and reports of personal observations have shown without doubt that caffeine abuse (caffeinism) may result in a syndrome which resembles and may be confused or confounded with true psychotic states. This may lead to misdiagnosis and mistreatment. A question arises from the varied reports of caffeine consumption in psychiatric populations: Does caffeine stimulate psychosis or does psychosis stimulate caffeine consumption?

These are not trivial findings because of the ready availability of caffeine and the epidemic of psychological problems which we are experiencing in this era. This report reviews some of the knowledge of caffeine's effects with the hope that we will all be more educated and more careful in the use of this commonly ingested drug.

The physiological action of caffeine is briefly reviewed, as psychological and physiological effects must go hand-in-hand. In addition to its central nervous system effects, caffeine has significant effects on the cardiovascular system, gastric acid secretion and catecholamine (adrenaline) release. In large doses, it has been shown to be a mutagen in animals, plants and bacteria, and has been shown to exhibit teratogenic properties in various animal species.

PHYSIOLOGICAL AND PHARMACOLOGICAL EFFECTS

J. Murdoch Ritchie, in Goodman and Gilman's Pharmacology Text (Ritchie, 1975) described the pharmacological effects of caffeine. The largest sources of caffeine are from the plants used to make coffee, tea, cocoa and kola (the basis of cola beverages), although it is also found in Latin America as mate' and guarana. Caffeine particularly has a profound effect on the central nervous system, but it also affects, to a lesser degree the heart muscle, gastric

secretion and diuresis. Interestingly, caffeine is ingested daily by a vast number of people and is unique in that it is a potent drug, considered to be part of our normal diet.

Caffeine stimulates the central nervous system first at the higher levels, the cortex and medulla, and finally the spinal cord at higher doses. Mild cortex stimulation appears to be beneficial resulting in more clear thinking and less fatigue. Caffeine has been shown to improve attention in a study which simulated night driving (Leinart, 1966). The onset of the effect of caffeine occurs within one hour and lasts for three to four hours (Baker, 1972).

The equivalent of one or two cups of coffee (150 to 250 mg of caffeine) is sufficient to induce adverse effects. The occurrence of hyperesthesia, an unpleasant sensory sensation, can be stimulated by large doses of caffeine.

The medullary, respiratory, vasomotor and vagal centers are stimulated by caffeine. This effect is due to an increased sensitization to carbon dioxide but needs large doses to elicit this effect, 150 to 250 mg, parenterally. The spinal cord is stimulated at higher doses and convulsions and death may result. More than 10 g are needed for such toxicity to occur in man (Ritchie, 1975).

Stimulation of the CNS is followed by depression (Klein and Salzman, 1975), although the effect is small at low doses e.g. a single cup of coffee. After two hours, Klein reported that males (but not females) showed a lower CNS stimulation compared to placebo. The post stimulation "let down" with caffeine results in fatigue and lethargy and the constant stimulation caused by chronic caffeine dosing could be disastrous (Abrams, 1977; Dowell, 1965).

Children, because of their smaller size, are more susceptible to caffeine. One report noted that hyperactivity and insomnia observed in children could be attributed to excess caffeine intake from cola drinks (Consumer Research, 1973). According to Dr. Page, "There is no doubt that children should be kept from using coffee and the popular caffeine containing soft drinks." (Abrams, 1977).

Caffeine's effect on the cardiovascular system is less profound than its central nervous system action. Its direct stimulatory effect on the heart may be neutralized by its central vagus stimulation. The direct effect predominates at very large doses with tachycardia and, eventually, arrhythmias resulting. Caffeine's ability to potentiate cyclic AMP can explain its

ability to potentiate ionotropic responses to B-adrenergic agonists and glucagon (Ritchie et al, 1975).

Although caffeine dilates blood vessels by a direct action, its central effect is one of constriction. At higher doses, the dilating effect is apparent (Peach, 1972; Poisner, 1973).

Similarly, because its direct and central effects are antagonistic, the resultant effect of caffeine on blood pressure is unpredictable. The net effect is usually of less than 10 mm of Hg in blood pressure (Ritchie et al., 1975). Caffeine's purported efficacy in hypertensive headaches may be due to a decrease in blood flow as a result of the increased cerebral resistance (Ritchie et al., 1975).

Caffeine also stimulates releases of catecholamines from the adrenal medulla and norepinephrine is released from nerve endings in the isolated heart (Bellett et al., 1971)..

It has been shown that prolonged augmentation of gastric secretion results from caffeine administration and that ulcer patients have sustained elevation of acid as opposed to normals (Ritchie et al., 1975).

Although a dose of approximately 10 g or more taken orally can be fatal, an oral (3.2 g IV) one gram dose will cause adverse effects (Gleason et al., 1969). The toxic effects are due to CNS and circulatory system stimulation and include some well recognized prominent symptoms in addition to those which can result at high doses or in hypersensitive persons: insomnia, restlessness, excitement, tinnitus, flashes of light, quivering muscles, tachycardia, extra systoles, and even low grade fever and mild delirium have been observed.

Harrie (1970) described a patient whose constant headaches were due to excessive caffeine consumption. He states, "I suspect that the condition is much more common than supposed and could well be one of the more frequent causes of chronic recurrent headache." Headaches can also be precipitated by caffeine withdrawal especially by those who have the "habit".

Although caffeine is well absorbed when taken orally, its absorption may be erratic because of its low solubility and because it may cause gastric irritation. Caffeine is principally metabolized with only 10 percent excreted in the urine unchanged (Ritchie et al., 1975).

Caffeine has a physiological half-life of three and a half hours (Parsons and Neims, 1978) to six hours (Aranda et al., 1979). Its physiological effects are observed in less than one hour (Parsons and Neims, 1978). Infants do not metabolize caffeine as well as adults and thus have a half-life of about four days (Aranda et al., 1975). Certainly, continuous ingestion of caffeine by infants can be dangerous. If a cup of coffee is consumed by an adult six or seven times a day it would result in a high steady concentration of caffeine in the blood. As little as four cups a day can result in appreciable omnipresent amounts of caffeine in the body.

Caffeine can accumulate in severe liver disease (Stratland, 1976) when its half-life can increase to 96 hours. If these patients drink coffee they should be closely monitored.

Caffeine is known to interact with other drugs resulting in a modified effect. For example, caffeine administered with nardil (an MAO inhibitor) caused headaches and high blood pressure (Pakes, 1979). This potentially dangerous interaction was first noted by Berkowitz et al., (1971) and implicated serotonin in the mechanism.

Caffeine and barbitol are antagonistic, with caffeine (in coffee) reducing the sleeping time induced by barbitol. Decaffeinated coffee had no effect (Aeschbacher et al., 1975). In another study, caffeine resulted in reduced sleeping time which was counteracted by pentobarbitol in hospitalized patients (Forrest et al., 1972).

PSYCHOLOGICAL EFFECTS OF CAFFEINE

Because of the wide spread use of caffeine and its known potent physiological effects, caffeine has been the subject of research in psychological related studies. This work has been stimulated by personal experiences and observations as well as by efforts to understand its action and mechanism.

Habituation and Tolerance: Caffeine ingestion and coffee drinking have been investigated with regard to the degree that this habit results in tolerance and withdrawal effects. These studies look beyond the obvious social implications and psychic dependence (Ritchie et al., 1975) of coffee consumption which may be related to the "first cup of coffee to wake me up" or "the coffee break" or to its association with smoking. In the latter case, it is of interest that coffee drinkers were shown to take more nicotine when deprived of coffee (Kozlowski, 1976).

Caffeine has not only been considered habit forming, but also addicting. Crothers considered morphinism and caffeinism to be similar, with caffeine causing loss of self-control, spells of agitation and depression as well as psychotic behavior (Stephenson, 1977). Ritchie mentions a report by Colton that tolerance can develop for the diuretic, salivary stimulation and sleep disturbance effects of caffeine.

Cola consumed in amounts of 48 to 111 ounces per day (144 to 333 mg of caffeine per day) was reported to have caused physical effects on withdrawal (Diamond and Pfifferling, 1974). The resultant effects were depression, nervousness, decreased alertness, sleeping difficulty, frequent mood changes, and various other behavioral difficulties which were reattributed to caffeine withdrawal.

The dependence of coffee drinkers on caffeine was illustrated in a study by Kozlowski (1976) in which coffee drinkers drank more coffee if the caffeine content was lowered.

Abrams (1977) says "There is no doubt that a certain degree of psychic dependence, that is habituation, develops from the use of xanthine beverages".

A questionnaire completed by more than 200 young housewives showed that the perceived effects of caffeine depended on previous use (Goldstein et al., 1969). The heavy coffee drinkers had few sleep disturbances and less evidence of nervousness after their morning coffee as compared to nondrinkers. If the morning coffee was stopped, the habitual coffee drinkers experienced nervousness, headache and irritation. The non-coffee drinkers reacted negatively to coffee, experiencing effects opposite to the coffee drinkers. An experiment was devised to verify the results of the questionnaire involving 18 housewives, non-coffee drinkers, and 38 who drank five or more cups per day. The results confirmed those obtained from the questionnaire previously administered (Goldstein et al., 1969). This experiment was double-blind and placebo controlled and caffeine was administered in coffee at 0, 150 and 300 mg. Coffee drinkers showed a dose-response effect whereas non-coffee drinkers showed signs such as nervousness, jitters and upset stomachs at all doses of caffeine but not on placebo.

Ritchie (1975) says that tolerance and psychological dependence to caffeine beverages does occur to some extent but he feels that this does not present a problem. He says that

coffee or tea drinking are socially acceptable and are apparently not harmful when practiced in moderation.

However, it does appear that at least in some persons excess consumption of caffeine can result in severe psychological dependence and withdrawal effects and is a problem to be reckoned with.

Behavioral Effects: Caffeine's stimulating activity on the central nervous system as well as other body organs results in certain physiological effects which may be considered to be behavior oriented. Caffeine produces more rapid, clearer flow of thought, allays drowsiness and fatigue, increases the capability of a greater sustained intellectual effort and more perfect association of ideas. It also causes a keener appreciation of sensory stimuli, and reaction time is diminished. Motor activity is increased; typists, for example, work faster with fewer errors. Tasks requiring delicate muscular coordination and accurate timing may, however, be adversely affected. All of this occurs at doses of 150 to 250 mg of caffeine (approximately two cups of coffee) according to Ritchie (1975).

In 1912, Hollingsworth who was a psychologist reported caffeine's effect on mental and motor efficiency in a study sponsored by Coca-Cola. In nine double-blind tests, he found beneficial effects for both mental and motor performance at doses of 65 to 130 mg of caffeine. At a dose of 300 mg, caffeine caused tremors, poor motor performance and insomnia. These results have withstood the test of time (Stephenson, 1977).

Goldstein (1965) showed no effect of caffeine on objective measures of performance although most subjects "felt" more alert and physically active. However, some subjects felt nervous.

Mitchell, Ross and Hurst showed caffeine to prevent attention lapses in a visual monitoring test which simulated night driving. The effect persisted for the two to three hour experiment (Stephenson, 1977).

A 200 mg dose of caffeine resulted in decreased decision time scores and improved motor time scores in volunteers (Smith et al., 1977). Hand steadiness, however, was impaired. After a caffeine intake of 200 mg, introverts performed less well on a verbal ability test as compared to extroverts when time pressure was applied (Ritchie et al., 1975).

Wayner et al. (1976) reported on the effects of caffeine on dependent and induced behavior in mice. Caffeine, (3.125, 6.25, 12.5, 25, 50 and 100 mg/kg) was tested on lever pressing, schedule induced licking and water consumption of mice. The effect on mice at 80 percent of body weight was different than when mice were allowed to recover the lost weight. At the lower weight, caffeine had little effect except at the highest dose (equivalent to 100 cups of coffee given at once). At their ordinary weight, the mice were more sensitive to caffeine, with all measures enhanced, even at the lowest dose (equivalent to approximately three cups of coffee). At high doses, all measures decreased; the mice became tolerant.

Castellano (1976) studied mice behavior under two sets of conditions. One involved a natural preference (swimming towards a light-"L") and the other involved an acquired behavior pattern (swimming toward the dark-"D"). A facilitation of learning and consolidation after caffeine dosing was noted in naive mice after the "D" procedure. Natural tendencies were also enhanced by caffeine as noted by improved performance in the "L" procedure. Animals pre-trained in the "D" procedure exhibited behavioral disruption after treatment. Animals pre-trained in the natural -U procedure needed very high doses to cause disruption. Caffeine decreases fiveHT turnover in rat brain. Amphetamines do not show the results as demonstrated in this paper, whereas other drugs such as hallucinogens show a similar effect. The implication is that the mechanism of caffeine's action may be similar to hallucinogenic drugs.

Effect on Sleep: Caffeine is known to cause insomnia because of its central nervous system stimulating activity. In fact, its major therapeutic use is to allay sleep and drowsiness, being the only OTC stimulant approved by the FDA. Several studies investigating this action in some detail have been published.

Karacan (1976) found that caffeine given half an hour before sleep adversely affected the sleeping process in normal subjects. The effect is dose related. Caffeine's effect simulates clinical insomnia and gave the same response as coffee containing an equivalent amount of caffeine. Decaffeinated coffee showed no effect on sleep.

Dorfman and Jarvick (1970) showed a dose-response effect of caffeine on the self estimation of sleep latency (which was increased) and quality (which was decreased). This was a double-blind study in which 0, 60, 120, and 250 mg of caffeine was administered one hour before bedtime.

Mikkelsen (1978) notes that caffeine seems to inhibit deeper stages of sleep as opposed to disturbances of the REM stage. Other studies show contradictory evidence, REM being affected by caffeine, leaving the situation to be resolved.

The tolerance developed to caffeine's effect on sleep by coffee drinkers has been documented by Colton (Stephenson, 1977). Non-coffee drinkers were more sensitive to coffee's insomniac effect whereas coffee drinkers were relatively insensitive in this regard. Non-coffee drinkers experienced disturbed sleep patterns and delayed onset of sleep.

Mueller-Limmroth (Stephenson, 1977) showed that the quality of the first three hours of sleep was impaired by the ingestion of coffee before retiring. This is approximately equal to the half-life of caffeine in the body.

Goldstein did extensive work on the effect of coffee and showed that coffee drinkers slept more soundly when they took placebo as opposed to caffeine in coffee. If 150 to 200mg of caffeine was taken before bedtime, there was an increased sleep latency which was less pronounced in persons who were heavy ingestors of caffeine (Goldstein et al., 1965).

These studies show that caffeine has a profound effect on sleep. Heavy and continued use of caffeine results in tolerance so that heavy users have less sleep disturbance or need more to obtain its stimulating effect.

Treatment of Hyperkinetic Children: Hyperkinetic children have been shown to respond to central nervous system stimulants, resulting in improved attention, concentration, –and decreased activity. Side effects are usually disturbing with the more powerful drugs and include insomnia, anorexia, nervousness, weight loss and abdominal pain.

A study by Schnackenberg (1975) showed that 200 to 300 mg of caffeine was similar in effect to methylpheniclate in treating hyperkinetic impulse disorder secondary to minimal brain dysfunction syndrome. Some hyperkinetic children, he observed, drank coffee to calm down. Sixteen children who had shown improvement on methylphenidate but who had annoying side effects were given one cup of coffee at breakfast and lunch. Test scores showed a similar improvement with coffee as compared to methylpheniclate and the annoying side effects disappeared when the children were on caffeine. Schnackenberg recommends 200 to 300 mg of caffeine in a time-release form.

In 1977, Reichard and Elder published an article on caffeine's effect on reaction time in hyperkinetic children. They tested the effect on a choice reaction time task and simple reaction time as compared to normal children. Caffeine increased the accuracy of stimulus identification and processing and decreased lapse of attention in the hyperkinetic group. This is what might be expected based on caffeine's known effects on such tasks in normals. Hyperkinetic children have a slower reaction time, are less able to maintain attention and have a lower rate of correct responses on a vigilance performance task as compared to normal children. In this study, six normal and six hyperkinetic children were compared in a double-blind design. Caffeine significantly raised the rate of correct responses on simple reaction time in the hyperkinetic group. The reaction time was reduced with caffeine but was not significantly less than the control period or placebo. Similar results were found with choice reaction time. The response is a function of the initial state of the children, i.e., the more severely afflicted had a larger response. The authors note that other studies have shown methylpheniclate was more effective than caffeine in controlling certain aspects of clinical behavior (impulsivity and hyperactivity). This result does not contradict those obtained in this study; they are compatible.

Garfinkel was unable to confirm the results of caffeine's effectiveness in controlling the behavior of children with minimal brain damage (Stephenson, 1977). Children responding to methylpheniclate did not necessarily respond to caffeine.

Firestone and associates in a study funded by the Ontario Mental Health Foundation(1978) showed a significant improvement with methylphenidate as rated by mothers and teachers on tests of impulsivity and motor control. No significant improvement was noted with caffeine although some children showed a slight improvement. Side effects with both drugs were minimal. Each of 21 hyperactive children received 500 mg of caffeine, 300 mg of caffeine, and 20 mg methylpheniclate. This was a carefully controlled study consisting of 17 boys and four girls. In 1978, Firestone did a study comparing 300 mg of caffeine with placebo in a double-blind crossover design. In this study, subjective ratings by teachers and parents as well as a reaction time task showed caffeine to be better than placebo although the difference was not statistically significant. Firestone concludes on the basis of the most recent study that caffeine is not a meaningful alternative as a treatment for hyperkinetic children.

The use of caffeine in the treatment of hyperkinetic children remains unresolved at this time. Further work seems warranted to ensure that if caffeine is useful in this prevalent condition that it be available as a viable alternate treatment in lieu of more powerful CNS stimulants.

"Restless Legs, Anxiety and Caffeinism" (Lutz, 1978)

Restless legs is a syndrome which may be associated with anxious – depressed as well as other clinical states. Dr. Lutz, in an article titled as above, suggest that this syndrome is primarily caused by caffeine. Anxiety is not a causative factor. Caffeine stimulates the nervous system and has a direct contractile effect on striated muscle. This is reflected in anxiety, depression, insomnia: and the heightened proprioceptive awareness may result in restless legs. This manifestation consists. of nervousness and movement of legs as a result of a distressing creeping sensation. Its symptoms are most obvious at night when the patient is trying to be still, and results in insomnia. Dr. Lutz describes cases of this disorder in detail and cites examples, all of which were alleviated when caffeine was removed from the diet. This condition has been attributed to many causes including psychiatric disturbances, e.g. restless legs is a frequent symptom of hysteria, anxiety, depression. In periods of stress, "normal" persons are also afflicted. All of these states are associated with high central nervous system arousal. Also, rest. less legs syndrome, was first described in England at the time when coffee and tea first were introduced in the country. Thus, diagnosis of the restless legs syndrome, as has also been observed in certain psychological disorders, may simply be the result of overdosage of ubiquitous caffeine.

Psychological Disorders: Dr. John Greden, a professor of psychiatry at the University of Michigan, says . caffeinism can be found among those who have psychiatric problems". Symptoms of excessive caffeine consumption are similar to anxiety neurosis(Avery, 1980) and include nervousness, irritability, recurrent headache. twitching, and gastrointestinal disturbance among other symptoms (Greden, 1974). This is a known effect of caffeine and Greden adds "...all medications including caffeine have a potential for abuse and many individuals clearly ingest symptom-producing doses daily".

Other studies support the relationship indicated above. For example, a prisoner v6thsevere anxiety symptoms admitted to drinking 50 cups of coffee per day (Niolde, 1975). The symptoms remitted after the coffee drinking stopped. Excess drinking of coffee by

prisoners is not uncommon and may initiate a vicious cycle: a bored person drinking more coffee resulting in caffeinism which may result in more consumption.

The intake of caffeine (coffee, etc.) has been correlated with the degree of mental illness in psychiatric patients. It is not clear if the caffeine intake intensifies the psychiatric disorder or whether those with more severe problems tend to drink more coffee. In any event, in another study by Dr. Greden and associates (Greden, 1978) 83 hospitalized psychiatric patients were interviewed and showed an association to symptoms with high caffeine intake. This may provide an explanation of some problems which have been experienced in diagnosing outpatient disorders. Eighteen of the 83 patients (22 percent) were high caffeine consumers (7~0 mg or more). They scored significantly higher on the State-Trait anxiety index and the Beck Depression Scale than lower caffeine consumers. The high consumers had more clinical symptoms: their physical health was worse; they used more sedatives, hypnotics, and minor tranquilizers. These patients showed a tolerance to sleep effects which could be due to a change in body kinetics or metabolism. Catecholamines contribute to the anxiety profile and patients may drink more coffee in response to stress, accentuating a neurotransmitter response cycle. Since caffeine affects catecholamine levels and inhibits phosphodiesterase breakdown of C-AMP, sensitizing receptor sites, the association of caffeine with anxiety and depressive symptoms is indeed a possibility.

Dr. Greden considers caffeine to be a psychotropic drug and 25 percent of the population may take more than 500 mg per day, a large physiologically active dose. He describes three cases in which caffeinism may be misdiagnosed as an anxiety syndrome.

Dr. Greden concludes that caffeine is found among a fairly large percentage of hospitalized patients with psychiatric symptoms. Caffeine should not be used as part of psychiatric treatment routines, e.g., to reduce drowsiness from psychotropic medications as has been occasionally suggested.

Dr. John Neil and associates (1978) reported on the possible complication of caffeinism in diagnosing psychiatric patients. He suggests that self-medication may confound behaviors of patients. Caffeine has been considered the most popular "psychotropic" drug in North America and coffee and tea drinking are not usually in the records of psychiatric patients. In this experiment, hypersomniac patients with various diagnoses and caffeine consumption participated. The authors conclude that "self medication with large doses of caffeine is a likely response to the anergia and hypersomnia experienced during certain

types of depression". This may lead to diagnostic confusion and a complicated course of therapy. Mixed depressive states may be caused by excess caffeine consumption and they suggest, also, that unipolar 11 depressives may use more caffeine as they become depressed.

Caffeine, in these patients, provides only transitory relief as it is not a true antidepressant. Caffeine also may render anxiolytic and anti-psychotic medications less effective.

Mikkelsen (1978) noted caffeine's involvement in schizophrenic-like states similar to that observed by Greden in anxiety/neurosis symptoms of patients who consumed large quantities of caffeine (coffee). One case cited was of a white male in a catatonic state who threatened his mother after having gone on a coffee jag over injustices caused to him by his mother. He developed paranoid delusions which he felt were, at least in part, due to the coffee. A 30-year-old white single female exhibited paranoid and auditory hallucinations. An anxiety state had resulted in increased coffee consumption. In the hospital she noted the correlation of these strange feelings with coffee consumption. Other examples of psychotic behavior as noted in the literature are described in this paper. Forty years ago a case of psychosis was reported in which a 24-year-old female took 60 gr (about four g) of caffeine. Manic symptoms developed. He theorizes that adenylyclase which is increased by caffeine may be a receptor for dopamine. If this system is abnormal in schizophrenics, caffeine may further sensitize the patient. Certainly, coffee should be considered as a factor in this disease.

Reimann (1967) noted that symptoms of a psychoneurotic woman disappeared when coffee was reduced. She presented with an irregular fever, insomnia, anorexia and irritability, having consumed large amounts of coffee.

Clearly, as recommended by Drs. Greden, Mikkelsen and Neil, caffeine intake should be considered as a factor in diagnosing and treating psychiatric patients.

SUMMARY

A review of the literature reveals that caffeine is an important factor in modifying the psychological state of its consumers under the present condition of usage. Caffeine is probably the most widely used drug and those who drink coffee, tea, cola or take OTC caffeine containing drugs are all potential and susceptible candidates. Those of us who're "normal" can expect manifestations which may be subtle at low doses, overt at high doses,

with the possibility of being the victims of a habit which results intolerance and possible severe withdrawal symptoms. The pleasant stimulant feeling which often occurs at low doses may be replaced by psychological symptoms which resemble anxiety and depressive neuroses at high doses. Those with more severe psychological problems may have their symptoms exaggerated with excessive caffeine usage, or such symptoms can actually be caused by excess. Diagnosis of such conditions must take caffeine usage into account.

As a result of its potent physiological activity, caffeine can alter our behavior. it affects our sleeping habits generally resulting in insomnia and hyperactivity. Task oriented performance, attention, and concentrations may be modified by caffeine. At lower doses, these effects appear to be beneficial. At higher doses, we can expect the reverse, including toxic and rebound effects.

The common "Restless Legs Syndrome" which has often been related to psychological disturbances may, in fact, be primarily a symptom of caffeinism according to Lutz.

Caffeine has been investigated as a possible treatment for hyperkinetic children since central nervous system stimulants have been shown to be effective in this condition. Results of caffeine treatment are controversial, some studies showing a beneficial effect with little adverse reactions and other studies showing little or no benefit.

Caffeine's effect on our body, our nervous system, our mind, our psychology is no illusion. It is a potent drug. That it may cause symptoms of mental illness as recently published is no small concern. With these findings we see that caffeine abuse is more prevalent than we may imagine. These facts should be brought to the attention of the medical community as well as the public in order that we may have the opportunity of being aware of the possible interactions between ourselves and our environment.

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ABSTRACTCaffeine, probably the most widely used drug, affects the psychological state of those who consume it. Abuse results in symptoms of caffeinism which include agitation, disorientation and a syndrome which may be mistaken for anxiety/neurosis. It is a habit-forming drug in which tolerance develops. It affects sleep in a dose related manner which is dependent on the daily caffeine intake, i.e., high users have less effect. Its central nervous system stimulation can cause pleasant effects with improved attention and concentration at lower doses. At high doses, the reverse may occur. Used judiciously, it may be a useful therapy in the treatment of hyperkinetic children. These and other effects of caffeine are discussed in this review article.

INTRODUCTIONCaffeine is among the most widely used drugs because of its ubiquitous occurrence in commonly consumed beverages such as coffee, tea and cola. Many drugs contain caffeine and are readily accessible to the public in the form of OTC stimulants and combination analgesics. Clearly caffeine is an important drug-food substance in our society which deserves attention.

To begin to have a new consciousness about caffeine so that we can become aware of how this drug can affect our physiology and psychology is a problem. The reasons for this are certainly complicated, but we can start by considering a factor dominating all of our lives, our "habits." When we become aware of and take responsibility to change habits, we are taking a first step in the process of awakening. The result must be not only an improvement in the quality of our lives but the world itself will be changed for the better. The use and abuse of caffeine is a major public "habit" and may be as important a factor as heredity and environment in the etiology of physiological and psychological disorders. To recognize this, we must know that we are creatures of habit. Most people are caffeine consumers because from birth this food-drug is set before us, if not offered directly, along with orange juice, cereal, dessert and cigarettes.

This paper reviews the literature relating to the psychological effects of caffeine. Caffeine is a potent central nervous system stimulant and much of its "psychological" activity may be related to this action of the drug. Its effects on the nervous system are obviously adverse at

high doses. It may not be obvious that at lower doses when used in moderation, it may have beneficial effects. For example, its possible therapeutic use in hyperkinetic children certainly would seem advantageous when compared to the current treatment with more powerful stimulants which have concomitant adverse reactions. Also, with the intense day to day pressures imposed on and accepted by many of us, is there any harm in "relaxing" with a hot cup of coffee? On the other hand, caffeine is a drug which is subject to abuse. The fact that it is a drug with a potentially powerful physiological effect escapes most of us who think of coffee as a relatively harmless beverage. Recently published studies and reports of personal observations have shown without doubt that caffeine abuse (caffeinism) may result in a syndrome which resembles and may be confused or confounded with true psychotic states. This may lead to misdiagnosis and mistreatment. A question arises from the varied reports of caffeine consumption in psychiatric populations: Does caffeine stimulate psychosis or does psychosis stimulate caffeine consumption?

These are not trivial findings because of the ready availability of caffeine and the epidemic of psychological problems which we are experiencing in this era. This report reviews some of the knowledge of caffeine's effects with the hope that we will all be more educated and more careful in the use of this commonly ingested drug.

The physiological action of caffeine is briefly reviewed, as psychological and physiological effects must go hand-in-hand. In addition to its central nervous system effects, caffeine has significant effects on the cardiovascular system, gastric acid secretion and catecholamine (adrenaline) release. In large doses, it has been shown to be a mutagen in animals, plants and bacteria, and has been shown to exhibit teratogenic properties in various animal species.

PHYSIOLOGICAL AND PHARMACOLOGICAL EFFECTS. J. Murdoch Ritchie, in Goodman and Gilman's Pharmacology Text (Ritchie, 1975) described the pharmacological effects of caffeine. The largest sources of caffeine are from the plants used to make coffee, tea, cocoa and kola (the basis of cola beverages), although it is also found in Latin America as 'mate' and guarana. Caffeine particularly has a profound effect on the central nervous system, but it also affects, to a lesser degree the heart muscle, gastric secretion and diuresis. Interestingly, caffeine is ingested daily by a vast number of people and is unique in that it is a potent drug, considered to be part of our normal diet.

Caffeine stimulates the central nervous system first at the higher levels, the cortex and medulla, and finally the spinal cord at higher doses. Mild cortex stimulation appears to be beneficial resulting in more clear thinking and less fatigue. Caffeine has been shown to

improve attention in a study which simulated night driving (Leinart, 1966). The onset of the effect of caffeine occurs within one hour and lasts for three to four hours (Baker, 1972).

The equivalent of one or two cups of coffee (150 to 250 mg of caffeine) is sufficient to induce adverse effects. The occurrence of hyperesthesia, an unpleasant sensory sensation, can be stimulated by large doses of caffeine.

The medullary, respiratory, vasomotor and vagal centers are stimulated by caffeine. This effect is due to an increased sensitization to carbon dioxide but needs large doses to elicit this effect, 150 to 250 mg, parenterally. The spinal cord is stimulated at higher doses and convulsions and death may result. More than 10 g are needed for such toxicity to occur in man (Ritchie, 1975).

Stimulation of the CNS is followed by depression (Klein and Salzman, 1975), although the effect is small at low doses e.g. a single cup of coffee. After two hours, Klein reported that males (but not females) showed a lower CNS stimulation compared to placebo. The post stimulation "let down" with caffeine results in fatigue and lethargy and the constant stimulation caused by chronic caffeine dosing could be disastrous (Abrams, 1977; Dowell, 1965).

Children, because of their smaller size, are more susceptible to caffeine. One report noted that hyperactivity and insomnia observed in children could be attributed to excess caffeine intake from cola drinks (Consumer Research, 1973). According to Dr. Page, "There is no doubt that children should be kept from using coffee and the popular caffeine containing soft drinks." (Abrams, 1977).

Caffeine's effect on the cardiovascular system is less profound than its central nervous system action. Its direct stimulatory effect on the heart may be neutralized by its central vagus stimulation. The direct effect predominates at very large doses with tachycardia and, eventually, arrhythmias resulting. Caffeine's ability to potentiate cyclic AMP can explain its ability to potentiate ionotropic responses to B-adrenergic agonists and glucagon (Ritchie et al, 1975).

Although caffeine dilates blood vessels by a direct action, its central effect is one of constriction. At higher doses, the dilating effect is apparent (Peach, 1972; Poisner, 1973). Similarly, because its direct and central effects are antagonistic, the resultant effect of caffeine on blood pressure is unpredictable. The net effect is usually of less than 10 mm of Hg in blood pressure (Ritchie et al., 1975). Caffeine's purported efficacy in hypertensive headaches may be due to a decrease in blood flow as a result of the increased cerebral resistance (Ritchie et al., 1975).

Caffeine also stimulates releases of catecholamines from the adrenal medulla and norepinephrine is released from nerve endings in the isolated heart (Bellett et al., 1971).. It has been shown that prolonged augmentation of gastric secretion results from caffeine administration and that ulcer patients have sustained elevation of acid as opposed to normals (Ritchie et al., 1975).

Although a dose of approximately 10 g or more taken orally can be fatal, an oral (3.2 g IV) one gram dose will cause adverse effects (Gleason et al., 1969). The toxic effects are due to CNS and circulatory system stimulation and include some well recognized prominent symptoms in addition to those which can result at high doses or in hypersensitive persons: insomnia, restlessness, excitement, tinnitus, flashes of light, quivering muscles, tachycardia, extra systoles, and even low grade fever and mild delirium have been observed.

Harrie (1970) described a patient whose constant headaches were due to excessive caffeine consumption. He states, "I suspect that the condition is much more common than supposed and could well be one of the more frequent causes of chronic recurrent headache." Headaches can also be precipitated by caffeine withdrawal especially by those who have the "habit".

Although caffeine is well absorbed when taken orally, its absorption may be erratic because of its low solubility and because it may cause gastric irritation. Caffeine is principally metabolized with only 10 percent excreted in the urine unchanged (Ritchie et al., 1975). Caffeine has a physiological half-life of three and a half hours (Parsons and Neims, 1978) to six hours (Aranda et al., 1979). Its physiological effects are observed in less than one hour (Parsons and Neims, 1978). Infants do not metabolize caffeine as well as adults and thus have a half-life of about four days (Aranda et al., 1975). Certainly, continuous ingestion of caffeine by infants can be dangerous. If a cup of coffee is consumed by an adult six or seven times a day it would result in a high steady concentration of caffeine in the blood. As little as four cups a day can result in appreciable omnipresent amounts of caffeine in the body.

Caffeine can accumulate in severe liver disease (Stratland, 1976) when its half-life can increase to 96 hours. If these patients drink coffee they should be closely monitored. Caffeine is known to interact with other drugs resulting in a modified effect. For example, caffeine administered with nardil (an MAO inhibitor) caused headaches and high blood pressure (Pakes, 1979). This potentially dangerous interaction was first noted by Berkowitz et al., (1971) and implicated serotonin in the mechanism.

Caffeine and barbitol are antagonistic, with caffeine (in coffee) reducing the sleeping time induced by barbitol. Decaffeinated coffee had no effect (Aeschbacher et al., 1975). In another study, caffeine resulted in reduced sleeping time which was counteracted by pentobarbitol in hospitalized patients (Forrest et al., 1972).

PSYCHOLOGICAL EFFECTS OF CAFFEINE Because of the wide spread use of caffeine and its known potent physiological effects, caffeine has been the subject of research in psychological related studies. This work has been stimulated by personal experiences and observations as well as by efforts to understand its action and mechanism.

Habituation and Tolerance: Caffeine ingestion and coffee drinking have been investigated with regard to the degree that this habit results in tolerance and withdrawal effects. These studies look beyond the obvious social implications and psychic dependence (Ritchie et al., 1975) of coffee consumption which may be related to the "first cup of coffee to wake me up" or "the coffee break" or to its association with smoking. In the latter case, it is of interest that coffee drinkers were shown to take more nicotine when deprived of coffee (Kozlowski, 1976).

Caffeine has not only been considered habit forming, but also addicting. Crothers considered morphinism and caffeinism to be similar, with caffeine causing loss of self-control, spells of agitation and depression as well as psychotic behavior (Stephenson, 1977). Ritchie mentions a report by Colton that tolerance can develop for the diuretic, salivary stimulation and sleep disturbance effects of caffeine.

Cola consumed in amounts of 48 to 111 ounces per day (144 to 333 mg of caffeine per day) was reported to have caused physical effects on withdrawal (Diamond and Pfifferling, 1974). The resultant effects were depression, nervousness, decreased alertness,, sleeping difficulty, frequent mood changes, and various other behavioral difficulties which we reattributed to caffeine withdrawal.

The dependence of coffee drinkers on caffeine was illustrated in a study by Kozlowski (1976) in which coffee drinkers drank more coffee if the caffeine content was lowered.

Abrams (1977) says "There is no doubt that a certain degree of psychic dependence, that is habituation, develops from the use of xanthine beverages".

A questionnaire completed by more than 200 young housewives showed that the perceived effects of caffeine depended on previous use (Goldstein et al., 1969). The heavy coffee drinkers had few sleep disturbances and less evidence of nervousness after their morning coffee as compared to nondrinkers. If the morning coffee was stopped, the habitual coffee

drinkers experienced nervousness, headache and irritation. The non-coffee drinkers reacted negatively to coffee, experiencing effects opposite to the coffee drinkers. An experiment was devised to verify the results of the questionnaire involving 18 housewives, non-coffee drinkers, and 38 who drank five or more cups per day. The results confirmed those obtained from the questionnaire previously administered (Goldstein et al., 1969). This experiment was double-blind and placebo controlled and caffeine was administered in coffee at 0, 150 and 300 mg. Coffee drinkers showed a dose-response effect whereas non-coffee drinkers showed signs such as nervousness, jitters and upset stomachs at all doses of caffeine but not on placebo.

Ritchie (1975) says that tolerance and psychological dependence to caffeine beverages does occur to some extent but he feels that this does-not present a problem. He says that coffee or tea drinking are socially acceptable and are apparently not harmful when practiced in moderation.

However, it does appear that at least in some persons excess consumption of caffeine can result in severe psychological dependence and withdrawal effects and is a problem to be reckoned with.

Behavioral Effects: Caffeine's stimulating activity on the central nervous system as well as other body organs results in certain physiological effects which may be considered to be behavior oriented. Caffeine produces more rapid, clearer flow of thought, allays drowsiness and fatigue, increases the capability of a greater sustained intellectual effort and more perfect association of ideas. It also causes a keener appreciation of sensory stimuli, and reaction time is diminished. Motor activity is increased; typists, for example, work faster with fewer errors. Tasks requiring delicate muscular coordination and accurate timing may, however, be adversely affected. All of this occurs at doses of 150 to 250 mg of caffeine (approximately two cups of coffee) according to Ritchie (1975).

In 1912, Hollingsworth who was a psychologist reported caffeine's effect on mental and motor efficiency in a study sponsored by Coca-Cola. In nine double-blind tests, he found beneficial effects for both mental and motor performance at doses of 65 to 130 mg of caffeine. At a dose of 300 mg, caffeine caused tremors, poor motor performance and insomnia. These results have withstood the test of time (Stephenson, 1977).

Goldstein (1965) showed no effect of caffeine on objective measures of performance although most subjects "felt" more alert and physically active. However, some subjects felt nervous.

Mitchell, Ross and Hurst showed caffeine to prevent attention lapses in a visual monitoring

test which simulated night driving. The effect persisted for the two to three hour experiment (Stephenson, 1977).

A 200 mg dose of caffeine resulted in decreased decision time scores and improved motor time scores in volunteers (Smith et al., 1977). Hand steadiness, however, was impaired.

After a caffeine intake of 200 mg, introverts performed less well on a verbal ability test as compared to extroverts when time pressure was applied (Ritchie et al., 1975).

Wayner et al. (1976) reported on the effects of caffeine on dependent and induced behavior in mice. Caffeine, (3.125, 6.25, 12.5, 25, 50 and 100 mg/kg) was tested on lever pressing, schedule induced licking and water consumption of mice. The effect on mice at 80 percent of body weight was different than when mice were allowed to recover the lost weight. At the lower weight, caffeine had little effect except at the highest dose (equivalent to 100 cups of coffee given at once). At their ordinary weight, the mice were more sensitive to caffeine, with all measures enhanced, even at the lowest dose (equivalent to approximately three cups of coffee). At high doses, all measures decreased; the mice became tolerant.

Castellano (1976) studied mice behavior under two sets of conditions. One involved a natural preference (swimming towards a light-"L") and the other involved an acquired behavior pattern (swimming toward the dark-"D"). A facilitation of learning and consolidation after caffeine dosing was noted in naive mice after the "D" procedure. Natural tendencies were also enhanced by caffeine as noted by improved performance in the "L" procedure. Animals pre-trained in the "D" procedure exhibited behavioral disruption after treatment. Animals pre-trained in the natural -U procedure needed very high doses to cause disruption. Caffeine decreases fiveHT turnover in rat brain. Amphetamines do not show the results as demonstrated in this paper, whereas other drugs such as hallucinogens show a similar effect. The implication is that the mechanism of caffeine's action may be similar to hallucinogenic drugs.

Effect on Sleep: Caffeine is known to cause insomnia because of its central nervous system stimulating activity. In fact, its major therapeutic use is to allay sleep and drowsiness, being the only OTC stimulant approved by the FDA. Several studies investigating this action in some detail have been published.

Karacan (1976) found that caffeine given half an hour before sleep adversely affected the sleeping process in normal subjects. The effect is dose related. Caffeine's effect simulates clinical insomnia and gave the same response as coffee containing an equivalent amount of caffeine. Decaffeinated coffee showed no effect on sleep.

Dorfman and Jarvick (1970) showed a dose-response effect of caffeine on the self

estimation of sleep latency (which was increased) and quality (which was decreased). This was a double-blind study in which 0, 60, 120, and 250 mg of caffeine was administered one hour before bedtime.

Mikkelsen (1978) notes that caffeine seems to inhibit deeper stages of sleep as opposed to disturbances of the REM stage. Other studies show contradictory evidence, REM being affected by caffeine, leaving the situation to be resolved.

The tolerance developed to caffeine's effect on sleep by coffee drinkers has been documented by Colton (Stephenson, 1977). Non-coffee drinkers were more sensitive to coffee's insomniac effect whereas coffee drinkers were relatively insensitive in this regard. Non-coffee drinkers experienced disturbed sleep patterns and delayed onset of sleep. Mueller-Limmroth (Stephenson, 1977) showed that the quality of the first three hours of sleep was impaired by the ingestion of coffee before retiring. This is approximately equal to the half-life of caffeine in the body.

Goldstein did extensive work on the effect of coffee and showed that coffee drinkers slept more soundly when they took placebo as opposed to caffeine in coffee. If 150 to 200mg of caffeine was taken before bedtime, there was an increased sleep latency which was less pronounced in persons who were heavy ingestors of caffeine (Goldstein et al., 1965). These studies show that caffeine has a profound effect on sleep. Heavy and continued use of caffeine results in tolerance so that heavy users have less sleep disturbance or need more to obtain its stimulating effect.

Treatment of Hyperkinetic Children: Hyperkinetic children have been shown to respond to central nervous system stimulants, resulting in improved attention, concentration, -and decreased activity. Side effects are usually disturbing with the more powerful drugs and include insomnia, anorexia, nervousness, weight loss and abdominal pain.

A study by Schnackenberg (1975) showed that 200 to 300 mg of caffeine was similar in effect to methylpheniclate in treating hyperkinetic impulse disorder secondary to minimal brain dysfunction syndrome. Some hyperkinetic children, he observed, drank coffee to calm down. Sixteen children who had shown improvement on methylphenidate but who had annoying side effects were given one cup of coffee at breakfast and lunch. Test scores showed a similar improvement with coffee as compared to methylpheniclate and the annoying side effects disappeared when the children were on caffeine. Schnackenberg recommends 200 to 300 mg of caffeine in a time-release form.

In 1977, Reichard and Elder published an article on caffeine's effect on reaction time in hyperkinetic children. They tested the effect on a choice reaction time task and simple

reaction time as compared to normal children. Caffeine increased the accuracy of stimulus identification and processing and decreased lapse of attention in the hyperkinetic group. This is what might be expected based on caffeine's known effects on such tasks in normals. Hyperkinetic children have a slower reaction time, are less able to maintain attention and have a lower rate of correct responses on a vigilance performance task as compared to normal children. In this study, six normal and six hyperkinetic children were compared in a double-blind design. Caffeine significantly raised the rate of correct responses on simple reaction time in the hyperkinetic group. The reaction time was reduced with caffeine but was not significantly less than the control period or placebo. Similar results were found with choice reaction time. The response is a function of the initial state of the children, i.e., the more severely afflicted had a larger response. The authors note that other studies have shown methylpheniclate was more effective than caffeine in controlling certain aspects of clinical behavior (impulsivity and hyperactivity). This result does not contradict those obtained in this study; they are compatible.

Garfinkel was unable to confirm the results of caffeine's effectiveness in controlling the behavior of children with minimal brain damage (Stephenson, 1977). Children responding to methylpheniclate did not necessarily respond to caffeine.

Firestone and associates in a study funded by the Ontario Mental Health Foundation(1978) showed a significant improvement with methylphenidate as rated by mothers and teachers on tests of impulsivity and motor control. No significant improvement was noted with caffeine although some children showed a slight improvement. Side effects with both drugs were minimal. Each of 21 hyperactive children received 500 mg of caffeine, 300 mg of caffeine, and 20 mg methylpheniclate. This was a carefully controlled study consisting of 17 boys and four girls. In 1978, Firestone did a study comparing 300 mg of caffeine with placebo in a double-blind crossover design. In this study, subjective ratings by teachers and parents as well as a reaction time task showed caffeine to be better than placebo although the difference was not statistically significant. Firestone concludes on the basis of the most recent study that caffeine is not a meaningful alternative as a treatment for hyperkinetic children.

The use of caffeine in the treatment of hyperkinetic children remains unresolved at this time. Further work seems warranted to ensure that if caffeine is useful in this prevalent condition that it be available as a viable alternate treatment in lieu of more powerful CNS stimulants.

"Restless Legs, Anxiety and Caffeinism" (Lutz, 1978) Restless legs is a syndrome which may

be associated with anxious – depressed as well as other clinical states. Dr. Lutz, in an article titled as above, suggest that this syndrome is primarily caused by caffeine. Anxiety is not a causative factor. Caffeine stimulates the nervous system and has a direct contractile effect on striated muscle. This is reflected in anxiety, depression, insomnia: and the heightened proprioceptive awareness may result in restless legs. This manifestation consists. of nervousness and movement of legs as a result of a distressing creeping sensation. Its symptoms are most obvious at night when the patient is trying to be still, and results in insomnia. Dr. Lutz describes cases of this disorder in detail and cites examples, all of which were alleviated when caffeine was removed from the diet. This condition has been attributed to many causes including psychiatric disturbances, e.g. restless legs is a frequent symptom of hysteria, anxiety, depression. In periods of stress, "normal" persons are also afflicted. All of these states are associated with high central nervous system arousal. Also, rest. less legs syndrome, was first described in England at the time when coffee and tea first were introduced in the country. Thus, diagnosis of the restless legs syndrome, as has also been observed in certain psychological disorders, may simply be the result of overdosage of ubiquitous caffeine.

Psychological Disorders: Dr. John Greden, a professor of psychiatry at the University of Michigan, says . caffeineism can be found among those who have psychiatric problems". Symptoms of excessive caffeine consumption are similar to anxiety neurosis(Avery, 1980) and include nervousness, irritability, recurrent headache. twitching, and gastrointestinal disturbance among other symptoms (Greden, 1974). This is a known effect of caffeine and Greden adds "...all medications including caffeine have a potential for abuse and many individuals clearly ingest symptom-producing doses daily".

Other studies support the relationship indicated above. For example, a prisoner with severe anxiety symptoms admitted to drinking 50 cups of coffee per day (Niolde, 1975). The symptoms remitted after the coffee drinking stopped. Excess drinking of coffee by prisoners is not uncommon and may initiate a vicious cycle: a bored person drinking more coffee resulting in caffeineism which may result in more consumption.

The intake of caffeine (coffee, etc.) has been correlated with the degree of mental illness in psychiatric patients. it is not clear if the caffeine intake intensifies the psychiatric disorder or whether those with more severe problems tend to drink more coffee. In any event, in another study by Dr. Greden and associates (Greden, 1978) 83 hospitalized psychiatric patients were interviewed and showed an association to symptoms with high caffeine intake. This may provide an explanation of some problems which have been experienced in

diagnosing outpatient disorders. Eighteen of the 83 patients (22percent) were high caffeine consumers (7~0 mg or more). They scored significantly higher on the State-Trait anxiety index and the Beck Depression Scale than lower caffeine consumers. The high consumers had more clinical symptoms: their physical health was worse; they used more sedatives, hypnotics, and minor tranquilizers. These patients showed a tolerance to sleep effects which could be due to a change in body kinetics or metabolism. Catecholamines contribute to the anxiety profile and patients may drink more coffee in response to stress, accentuating a neurotransmitter response cycle. Since caffeine affects catecholamine levels and inhibits phosphodiesterase breakdown of C-AMP, sensitizing receptor sites, the association of caffeine with anxiety and depressive symptoms is indeed a possibility. Dr. Greden considers caffeine to be a psychotropic drug and 25 percent of the population may take more than 500 mg per day, a large physiologically active dose. He describes three cases in which caffeinism may be misdiagnosed as an anxiety syndrome.

Dr. Greden concludes that caffeine is found among a fairly large percentage of hospitalized patients with psychiatric symptoms. Caffeine should not be used as part of psychiatric treatment routines, e.g., to reduce drowsiness from psychotropic medications as has been occasionally suggested.

Dr. John Neil and associates (1978) reported on the possible complication of caffeinism in diagnosing psychiatric patients. He suggests that self-medication may confound behaviors of patients. Caffeine has been considered the most popular "psychotropic" drug in North America and coffee and tea drinking are not usually in the records of psychiatric patients. In this experiment, hypersomniac patients with various diagnoses and caffeine consumption participated, The authors conclude that "self medication with large doses of caffeine is a likely response to the anergia and hypersomnia experienced during certain types of depression". This may lead to diagnostic confusion and a complicated course of therapy. Mixed depressive states may be caused by excess caffeine consumption and they suggest, also, that unipolar 11 depressives may use more caffeine as they become depressed.

Caffeine, in these patients, provides only transitory relief as it is not a true antidepressant. Caffeine also may render anxiolytic and anti-psychotic medications less effective.

Mikkelsen (1978) noted caffeine's involvement in schizophrenic-like states similar to that observed by Greden in anxiety/neurosis symptoms of patients who consumed large quantities of caffeine (coffee). One case cited was of a white male in a catatonic state who threatened his mother after having gone on a coffee jag over injustices caused to him by

his mother. He developed paranoid delusions which he felt were, at least in part, due to the coffee. A 30-year-old white single female exhibited paranoid and auditory hallucinations. An anxiety state had resulted in increased coffee consumption. In the hospital she noted the correlation of these strange feelings with coffee consumption. Other examples of psychotic behavior as noted in the literature are described in this paper. Forty years ago a case of psychosis was reported in which a 24-year-old female took 60 gr (about four g) of caffeine. Manic symptoms developed. He theorizes that adenylylase which is increased by caffeine may be a receptor for dopamine. If this system is abnormal in schizophrenics, caffeine may further sensitize the patient. Certainly, coffee should be considered as a factor in this disease.

Reimann (1967) noted that symptoms of a psychoneurotic woman disappeared when coffee was reduced. She presented with an irregular fever, insomnia, anorexia and irritability, having consumed large amounts of coffee.

Clearly, as recommended by Drs. Greden, Mikkelsen and Neil, caffeine intake should be considered as a factor in diagnosing and treating psychiatric patients.

SUMMARY A review of the literature reveals that caffeine is an important factor in modifying the psychological state of its consumers under the present condition of usage. Caffeine is probably the most widely used drug and those who drink coffee, tea, cola or take OTC caffeine containing drugs are all potential and susceptible candidates. Those of us who're "normal" can expect manifestations which may be subtle at low doses, overt at high doses, with the possibility of being the victims of a habit which results in intolerance and possible severe withdrawal symptoms. The pleasant stimulant feeling which often occurs at low doses may be replaced by psychological symptoms which resemble anxiety and depressive neuroses at high doses. Those with more severe psychological problems may have their symptoms exaggerated with excessive caffeine usage, or such symptoms can actually be caused by excess. Diagnosis of such conditions must take caffeine usage into account. As a result of its potent physiological activity, caffeine can alter our behavior. It affects our sleeping habits generally resulting in insomnia and hyperactivity. Task oriented performance, attention, and concentrations may be modified by caffeine. At lower doses, these effects appear to be beneficial. At higher doses, we can expect the reverse, including toxic and rebound effects.

The common "Restless Legs Syndrome" which has often been related to psychological disturbances may, in fact, be primarily a symptom of caffeinism according to Lutz.

Caffeine has been investigated as a possible treatment for hyperkinetic children since

central nervous system stimulants have been shown to be effective in this condition. Results of caffeine treatment are controversial, some studies showing a beneficial effect with little adverse reactions and other studies showing little or no benefit. Caffeine's effect on our body, our nervous system, our mind, our psychology is no illusion. It is a potent drug. That it may cause symptoms of mental illness as recently published is no small concern. With these findings we see that caffeine abuse is more prevalent than we may imagine. These facts should be brought to the attention of the medical community as well as the public in order that we may have the opportunity of being aware of the possible interactions between ourselves and our environment.

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