Coca Chewing: A New Perspective

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ABSTRACT
Arguments presented by researchers to explain "why" Andean peasants chew coca leaf have traditionally centered around the fact that coca leaf is the source of cocaine alkaloids. Following the presentation of several cocaine-models of coca chewing, this paper argues that we must begin moving toward a new perspective on this complex problem since recent research shows that during the process of chewing, hydrolysis and metabolism, cocaine alkaloids are degraded into ecgonine. Arguing for an ecgonine-model this paper suggests that the chewing of coca leaf may be an important cultural mechanism for the control of the problems of blood glucose homeostasis and carbohydrate utilization which Bolton (1973) has suggested may be widespread in highland Peru.

INTRODUCTION

The genus *Erythroxylon* [family *Erythroxylaceae*] is widely distributed throughout the tropical regions of South America, mainly in Peru and Bolivia, and less extensively in Colombia, Ecuador, Venezuela, and Brazil. The number of different species said to belong to the genus range from as low as 75 (de los Ríos 1868) to as high as 250 (Machado 1968). There is general agreement that only a limited number of these species have been cultivated through time. Because of the great plasticity of the plant under varying ecological conditions, Martín (1970:422) argues that several of these might best be considered as cultivated varieties rather than distinct species. In Peru, the two most widely cultivated species (or perhaps varieties) of the

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The leaves of these small woody bushes are popularly referred to as “coca.”

Coca leaf was probably first cultivated somewhere in the ecological zone referred to as the montaña or yunga on the eastern slopes of the Andes in Peru or Bolivia sometime in the distant past. Recently, Patterson (1971) has shown that coca was being cultivated in the middle- and upper-river valleys on the western or coastal slopes of the Andes as long ago as 1900–1750 B.C. The antiquity of coca cultivation in Peru therefore is at least 4,000 years and probably longer. Although ethnobotanical data indicate that coca was widely cultivated on the coastal side until the sixteenth century (cf. Rostworowski 1967), today its cultivation is almost totally limited to the eastern slopes of the Andes, or the montaña, between altitudes of 1,000 and 6,000 feet (Machado 1968).

The focus of this paper is on the problem of “why” Andean peasants chew coca leaf. Following a background on coca chewing and the general pharmacological and behavioral effects of cocaine hydrochloride, I shall outline several major cocaine-models of coca chewing, or what I refer to as the “food scarcity” hypothesis (Gutiérrez-Noriega 1949, Verzar 1955), and a recent argument by Bolton (1973) which he refers to as the “hypoglycemia hypothesis.” My major aim in this paper is to move us away from a traditional cocaine-model of coca chewing and toward an egonine-model, since egonine rather than cocaine is probably the central alkaloid involved in coca chewing.

THE CHEWING OF COCA LEAF

Shortly after the arrival of Europeans in the New World the use of coca leaf as a masticant was reported as far south in South America as the Rio de Plata in Argentina and as far north as the Caribbean. It was even reported in Nicaragua in Central America (Cooper 1946:549). The first description of coca use in northern South America was given in 1499 by Tomas Ortiz, a Dominican missionary (Gagliano 1961:27). Other early descriptions were presented by Alfonso Niño and Cristóbal Guerra who explored the Cumana region of Venezuela in 1500, and Amerigo Vespucci even commented on coca use in a report of 1504 (Bues 1935:2; Gagliano 1961:28).

In the Andean area, the Spanish chronicler, Pedro Cieza de Leon wrote in 1550 that: “All throughout Peru it was and is the custom to have ... coca in the mouth, and they keep it there from morning until they go to sleep without removing it” (Cieza de Leon 1959:259). Several years later, still
another Spanish observer, Juan de Matienzo, was writing in 1567 that:

In Peru, from Quito to the ends of the city of La Plata, in all parts, they use and carry [coca] in their mouth. And with it, they put certain powders which they call *hipita* made from certain ground bones and ash from certain grasses... which they call quinua, and in certain places with quicklime. And they don’t eat it or do anything else with it other than carry it in their mouth (Matienzo 1967 [1557]: 162).

Over four centuries have passed, yet there are probably as many as two million individuals in Peru who can be classified as coqueros, or coca chewers. In 1966, Peru produced a total of 9,091,517 kilograms of coca leaf (Banco de la Nación 1967:4). Of this total production, 18,184 kilograms of coca leaf were used within Peru for the government controlled manufacture of cocaine hydrochloride, 261,291 kilograms were exported outside Peru, with 500 kilograms going to France and 260,791 kilograms to the United States, mainly for the extraction of flavorides used in the manufacture of Coca-Cola. The remainder of this official production total, or some 8,812,062 kilograms is said to have been consumed within Peru, principally by the peasant population, for the purpose of “mastication” (Ibid.: Anexo II).

The mechanical procedures of chewing coca leaf, or of *chaqchando*, have been described by various researchers (cf. United Nations 1950:20–21; Stein 1961:99–100). I shall only note that it involves little more than a handful of coca leaf and some basic equipment. In the Department of Huanuco, for example, this equipment consists of a *shuti* [coca pouch], an *ishkupuru* [small gourd containing iskha (lime) or cal], and a *chapadero* [spatula] which serves to extract the iskha from the puru as well as a cap for the *ishkupuru* when not in use.

The chewing of coca leaf is both ritualized and stereotyped. The coca leaves are taken from the *shuti*, cupped in both hands and their advice on a certain past or future event or action is requested, generally with the phrase “María Santísima, Mamita kuquita, avisa me...” The hands are uncupped and the leaves are examined by the *coquero*. At times the requested information is made public, but more generally it is private knowledge. The leaves are then inserted into the mouth, one or several at a time, and masticated until a *bola* [quid] begins to take shape. The coca *bola* is tucked into the side of the mouth, between the teeth and cheek. The *ishkupuru* is tapped several times against the thumb, knee, chest, or elbow after which the *chapadero* is withdrawn and wet with saliva. It is then reinserted into the *puru*, withdrawn, and tipped with iskha, inserted into the *bola* in the mouth. Coca leaf is added throughout the process of mastication until the *bola* has the “correct” size, and iskha is added throughout
the process until the bola is considered to have the "correct" taste and consistency.

"WHY DO YOU CHEW COCA?"

The chewing of coca leaf has long contributed to the visibility of Andean peasants as well as to both curiosity and scientific interest on the part of the non-chewer to know why coca leaf is chewed in the first place. The simplest way to answer this question would appear to be a direct approach to the problem: merely ask. White (1951:242) writes: "Probably the first question asked by the visitor to the sierra and Altiplano is, why do the Indians chew coca?"

The question is not only a logical one, but an old one. Four centuries ago, Cieza de Leon wrote: "When I asked some of the Indians why they always had their mouth full of this plant, which they don't eat, but only keep in their mouth, they said they do not feel hunger, and it gives them great strength and vigor" (Cieza de Leon 1959:259). He added, "I think it probably does something of the sort, though it seems to me a disgusting habit, and what might be expected of people like these Indians" (ibid.).

In 1567, the Spanish licentiate, Juan de Matienzo wrote: "Asking them why they carry it [coca] in their mouth, they say they feel little hunger or thirst, and they find themselves with more vigor and strength" (Matienzo 1967[1557]:162). The same question was asked by other early observers and they likewise recorded similar responses to their question (cf. Cobo 1890 (1):475; Zarate 1944[1555]:37).

The contemporary researcher who approaches the Andean peasant with questionnaire in hand, and asks: "Why do you chew coca leaf?" will without a doubt receive much the same answers as have individuals who have posed this question over the past four hundred years or more. For example the United Nations Commission of Experts (United Nations 1950:53) noted that "the most prevalent and important belief held by peasants is that coca chewing dispels and relieves hunger, thirst, fatigue, weariness, and even the desire for sleep." Fine (1960:8) writes that his informants claimed that "with coca we are in high spirits, we feel stronger. We want to work." Goddard et al. (1969:579) write that according to their study of "attitudes" centering around coca-use, it "reduces sensations of hunger, although apparently not in every case." "In general," they write, "chewers associate coca use primarily with the work situation; it being very common to hear that 'without coca it is impossible to work,' or 'it helps to work,' and 'we always use it because of work.'"

Of the several hundred coca chewers with whom I had contact during
the course of fieldwork many dozens of them were asked the same question: “Why do you chew coca?” With few exceptions, I received the same answers as did Cieza de Leon and Juan de Matienzo during the Colonial period, as well as contemporary researchers. On the other hand, many informants simply responded that chewing coca was “customary,” and one informant merely stated: “I am an Indian, therefore, I chew coca.”

It is relatively easy to document the continuity in peasant responses to their inquisitors over a period of more than four centuries. Since the Spanish conquest to the present day, numerous hypotheses have been presented in an effort to explain these responses. Since the isolation of cocaine alkaloids from coca leaf by Niemann in 1859, the problem of “why” peasants chew coca leaf in the Andes, and elsewhere, has appeared to be rather straightforward. La Barre (1948:67) writes of coca leaf: “These plants contain a certain amount of cocaine, and it is for the purpose of obtaining the stimulation of this drug that coca leaf is chewed.”

COCA LEAF ALKALOIDS

At least 14 different alkaloids have been isolated from coca leaf varieties (Martin 1970:422). They belong in the tropine series, together with atropine and scopolamine from the solanaceous genera Datura, Hyoscyamus, Atropa, etc., and are a combination of ecegonines, tropines, and hygrines. Ecegonine derivatives consist of methyl benzoyl ecegonine [cocaine], methyl ecegonine, and cinnamyl cocaine; tropines include tropine and pseudotropine, dihydrozypeine, tropacocaine, and benzoyl tropine; and the hygrines include hygrine, hygroline, and cuscohygrine (Ibid.:422). The stereoisomers α and β — truxilline have also been isolated, and nicotine has been reported (Ibid.).

Since the isolation of cocaine alkaloids, both the pharmacological and behavioral effects of cocaine hydrochloride on humans and non-humans have served as the model for explaining coca-use in Peru and elsewhere.

COCAINE: PHARMACOLOGICAL AND BEHAVIORAL EFFECTS

*Classification:* Cocaine is generally classified as a CNS [central nervous system] sympathomimetic, or stimulant, since moderate oral doses produce signs of electro-physiological stimulation or arousal of the CNS and peripheral effects indicative of the activation of the sympathetic [adrenaline-like] part of the automatic nervous system (Commission of Inquiry 1973:312). Cocaine is often grouped together with amphetamines [benzodrine]
tive effects, if not the actual mechanics of these effects, are almost identical.

Subjective Effects: Whereas Lewin (1964[1924]: 82) may have been able to comment in 1924 that the "method of its introduction into the body is of no importance," such a statement today would and should be considered as fallacious. Jaffe (1970:293) states that the subjective effects of CNS stimulants are (a) dependent upon the user, (b) the environment [setting], (c) the dose, and (e) the route of administration, i.e. whether taken intravenously or orally since rate and extent of absorption differ with route of administration. Taken intravenously the subjective effects of cocaine are described by Jaffe as follows:

...elevation of mood that often reaches proportions of euphoric excitement. It produces a marked decrease in hunger, an indifference to pain, and is reputed to be the most potent antifatigue agent known. The user enjoys a feeling of great physical strength and increased mental capacity and greatly overestimates his capabilities [my emphasis]. The euphoria is accompanied by generalized sympathetic stimulation... [resulting in greater epinephrine output — my insertion]. Cocaine is rapidly metabolized and its duration of action is brief, lasting only minutes after intravenous injection. The contrast between the euphoria and the usual affective state, which for many compulsive users is one of chronic depression, motivates the user to repeat the experience. In this way, multiple injections may be taken over a very few hours. When large amounts of cocaine are used, the euphoria becomes mixed with anxiety and suspicion (Jaffe 1965:298–299).

In humans, moderate oral doses [5–30 mg] of cocaine and other CNS stimulants also result in an elevation of mood [euphoria], which may not, however, occur at all times: a sense of increased energy and alertness or decreased fatigue and boredom; and a reduction in appetite (Jaffe 1970:293; Kosman and Unna 1968:21).

Increase in Energy: According to Jaffe (1970:380) there is "no evidence that cocaine increases the strength of muscular contractions" and he therefore concludes that the subjective effect of increased energy or reduction of fatigue, "seems to result from central stimulation which masks the sensation of fatigue."

Anorexigenic Effect [Reduction of Appetite]: The reduction of appetite by cocaine and other CNS stimulants probably results from some depressing action on the appetite-regulating centers of the brain (Commission of Inquiry 1973:214). In non-human animals, experimental studies show that cocaine reduces food intake in rats, although chronic doses of cocaine had little effect on the weight-gain curve, except when given in very high doses [200–1200 mg per kg] (Kosman and Unna 1968:243). In humans the appetite returns to normal following the discontinuance of CNS stimulants (Commission of Inquiry 1973:23).

Anesthesia: When applied to an abraded area of the skin or mucous
membrane, cocaine blocks conduction in the terminal sensory nerve fibers in concentrations as low as 0.02 percent (Ritchie et al. 1970:376). Investigations show that the addition of an alkaline substance to local anesthetics will potentiate their activity, and the duration of anesthetic action is proportional to the time it is in direct contact with nerve tissue (ibid.).

**Body Temperature:** Cocaine, according to Jaffe, is "markedly pyrogenic" and a combination of three factors leads to a rise in body temperature: (1) increased muscular activity leads to greater heat production; (2) vasoconstriction leads to decrease in heat loss; and (3) cocaine may have direct effect on the heat-regulating centers. For Jaffe states, the onset of cocaine-fever [sudden rise in temperature following a toxic dose] suggests that the body is adjusting its temperature to a higher level (1970:381).

**Respiration:** The stimulation of the medulla oblongata by moderate oral doses of cocaine leads to increased respiratory rate, the duration of which is at first unaffected, but later is diminished so that breathing becomes rapid and shallow (Jaffe 1970:380).

**Cardiovascular Effects:** After moderate oral doses of cocaine, heart rate, pulse rate, and blood pressure increase, and there is widespread vasoconstriction due to stimulation of the vasomotor centers in the medulla oblongata (Grollman and Grollman 1970:405). The rise in blood pressure will later diminish and fall (Jaffe 1970:390). Cocaine intake will also result in a rise of blood glucose levels (Frombach 1967).

**Tolerance:** Little evidence exists to indicate that cocaine use results in tolerance (Jaffe 1970:294; Commission of Inquiry 1973:23).

**Toxicity:** Excessive doses [particularly intravenous] may cause seizures and death by respiratory failure (Jaffe 1970:294).

**Physiological and Psychological Dependency:** Most researchers agree that the chronic use of cocaine does not result in "physiological dependence" and that its abrupt discontinuance will not be characterized by grossly observable withdrawal symptoms or abstinence syndrome (Jaffe 1970; Kosman and Umma 1968). Researchers do argue, however, that chronic cocaine use may result in "psychological dependency" or "habituation" which is defined by Jaffe as follows:

... In the use of drug to alter mood and feeling... some individuals eventually consider the effects produced by the drug, i.e., the condition associated with its use, are necessary to maintain an optimal state of well-being. Such individuals are said to have a psychological dependence on the drugs [habituation] (1970:276).

He continues:

The intensity of this dependence may vary from a mild desire to a 'craving' or
'compulsion' to use the drug. This need or psychological dependence may give rise to behavior (compulsive drug use) characterized by the preoccupation with the use and procurement of the drug.

The Canadian Commission of Inquiry on the Non-Medical use of Drugs (1973), points out, however, that:

In one sense, psychological dependence may be said to exist with respect to anything which is part of one’s preferred way of life. In our society, this kind of dependency occurs regularly in respect to such things as television, music, books, religion, sex, many favorite foods, certain drugs, hobbies, sports or games, and often other persons. Some degree of psychological dependence is, in this sense, a general and normal psychological condition (1973:26).

The Commission of Inquiry continues [citing the Addiction Research Foundation of Ontario]:

It should be recognized, however, that dependency is not necessarily bad in itself, either for the individual or for society. The question to be evaluated, therefore, is not whether dependence can occur, but whether dependence in a given case results in physical, psychological, or social harm.

Addiction: The concept of “addiction” has little meaning and is often used interchangeably with physiological or psychological dependence, as well as with “drug abuse” (Ibid.). The classical model of the addiction-producing drugs were the opiate narcotics and required the presence of tolerance, as well as psychological and physiological dependency (Ibid.). The classical model does not hold up for the non-opiate drugs. In regard to cocaine, its use may produce psychological dependence without tolerance or physical dependence (Ibid.: 27). Researchers emphasize that the term “habituated” (Jaffe 1970) or “dependent” be used instead of describing an individual by the term “addict” (Commission of Inquiry 1973:27).

The preceding pharmacological and behavioral effects of cocaine hydrochloride have been incorporated into several cocaine-models of coca chewing in the past few decades.

COCAINENE-MODELS OF CHEWING

One of the most active and influential groups of proponents of the cocaine-model of coca chewing in Peru over the past several decades has been the late Carlos Gutierrez-Noriega and his associates, particularly Vicente Zapata Ortiz (cf. Gutierrez-Noriega and Zapata Ortiz 1947). Gutierrez-Noriega (1949: 143) states that “the main cause of the coca habit is the deficiency of foodstuffs in the affected areas.” Zapata Ortiz (1970:292)
likewise writes that “the lack of food is the principal cause of cocaism.”
I shall refer to the following argument, therefore, as the “food scarcity” hypothesis.

*The “Food Scarcity” Hypothesis*

“One of the most characteristic actions of coca and cocaine,” Gutierrez-Noriega (1949:146) writes, “is the suppression of the sensations of hunger and fatigue.” He argues that because of widespread scarcity of food in highland Peru and because coca “suppresses hunger,”

the coca chewer, as a consequence, takes coca to suppress the disagreeable sensations that result from chronic intution. But the use of the drug occasions, after some years, the loss of appetite. The habituated chewer prefers the drug to food.... From this, a vicious cycle is established; one begins to chew coca to suppress hunger but later the subject loses his appetite and eats little because he chews coca.

With such a deficient eating regime, the organism becomes debilitated and as always occurs in poorly fed subjects, they experience chronic tiredness, fatigue at the slightest effort, and apathy. But coca, as well as suppressing the hunger, is a powerful stimulant, perhaps superior to benzedrine and desoxycphedrine. In this way the second condition of the habit of coca chewing establishes itself, the need to counteract the fatigue of the organism with the drug. As a result, those habituated to coca refuse to undertake whatever physical work unless they have previously taken stimulating doses of coca leaf.

Thirdly, coca is chewed to produce a state of euphoria. The habituated chewers are, in general, depressed and apathetic, which in large part is a result of the bad diet and their chronic intoxication (1952:118).

According to Gutierrez-Noriega, many of the physiological effects of coca chewing also fit within the model of cocaine hydrochloride as well. He writes:

In general coca addicts do not show, after an ordinary period of coca chewing, strong excitation symptoms as cocaine addicts do. I have found, nevertheless, some physiological changes during the period of chewing, slight mydriasis, moderate increase in respiratory rate, rise in blood pressure, and a definite increase in heart rate.... The metabolic alterations are very constant, even with small doses of coca; there is an increase in body temperature and remarkable increase in basal metabolic rate. The blood sugar always increases in experimental animals, but only in a very few cases does it increase in man after a period of coca chewing [my emphasis] (Gutierrez-Noriega and Von Hagen 1950:85).

Moreover, he states that the “symptoms of habituation of the coca chewer are relatively weak” and that “tolerance phenomena are not observed” (Gutierrez-Noriega 1949:148).

A similar position is held by the United Nations Commission of En-
quiry on the Coca-Leaf (1950), and I shall also include it under the label of the “food scarcity” hypothesis. This position is best summarized by Verzar (1955), a former member of the above Commission. Verzar writes:

The alkaloid cocaine which is contained in these leaves in a concentration of 0.5 to 0.6 per cent is the oldest known local anesthetic....

Cocaine has an inhibitory action on peripheral nerves and on sensory nerve endings. It also has a specific action on the central nervous system, even in very low concentrations.... It abolishes the sensation of pain and also of taste and smell. An anesthesia of the stomach with cocaine takes away the sickish feeling in stomach disease. Mucous membranes after cocaine show a vasoconstriction. Stomach secretion is stopped....

This anesthetic action depends on the presence of the free alkaloid base. At a very acid reaction, as in the gastric juice, a dissociation of the alkaloid takes place and action is destroyed. Only in alkaline solution is the alkaloid stable and active and absorbed in the active state. Obviously, this must be one of the reasons why the coquio [sic] mixes a strong alkaline substance (lipta) [sic] with the leaves [my emphasis].

The action on the central nervous system is what is desired by the addicts, the cocaists.... Eichholtz describes the central cocaine action and also the result of coca leaf chewing by saying: “It satisfies the starving, gives new strength to the tired and exhausted, and makes the unfortunate forget his unhappiness.”

It is... certain that cocaine stops the painful sensation of hunger. The frequently quoted story of Indians who walk for days without food and rest over enormous distances may be true.... The capacity of coca to stop the feeling of hunger is not the result of some nutrient value of the coca leaves.... It is the result of central inhibition of hunger feelings....

We come to hold the conviction that the chewing of coca leaves is a method for inhibiting the feeling of hunger in a chronically underfed population.... The habit is, however, so much surrounded by mysticism that it is difficult to explain it on a biological basis. No doubt there are many who use this habit without being in the condition for which it was originally used [my emphasis] (Verzar 1955:366-367).

This latter point by Verzar of course raises the question of what exactly was the “condition” for which coca chewing was “originally used”? At this point, then, let me turn to still another hypothesis concerning “why” highland peasants chew coca leaf.

The “Hypoglycemia Hypothesis”

In a recent paper “Aggression and hypoglycemia among the Qolla: a study in psychobiological anthropology,” Bolton (1973) has presented what surely will become one of the more widely debated arguments for years to come, centering around the “roots of social conflict” in highland peasant communities in Peru. In his paper, Bolton argues that (a) there
is a high incidence of "aggressive" behavior in the highland community of Incawatana, (b) that glucose homeostasis problems are widespread among the inhabitants, and (c) that there is a causal relationship between these two phenomena, or what he refers to as the "hypoglycemia hypothesis" (Bolton 1973:241-242). Because it is not possible to outline Bolton's arguments in great detail in this paper, I shall limit my comments only to those aspects of his hypothesis that have to do with the chewing of coca leaf. Although I believe Bolton to be mistaken, I also believe that he has provided us with a starting point from which we can begin moving toward a new perspective on the chewing of coca leaf in Peru. Bolton writes:

If one postulates that the human organism attempts to maintain glucose levels at or above the nominal level, which seems to be the case, then when blood glucose falls below the nominal level, processes occur which will raise the glucose level. In the normal, healthy organism these processes are internal metabolic processes. If, however, these metabolic processes are not operating properly, e.g., because of adrenal exhaustion or liver disease, then behavioral and emotional means might be sought to produce the same effects. The individual may find that by becoming angry or by expressing aggression his glucose level is raised. Anger -- the fight-flight reaction -- may serve as a stimulus to sluggishly operating glands and organs. Consequently, a person's aggression is reinforced because of the physiological feeling of well-being which accompanies the emotions or aggressive actions. In this way hypoglycemia may lead to aggressiveness because this type of stimulation is extremely effective as a short-run booster of glucose levels.

Consequently, it can be seen that aggressive behavior may become part of the set of mechanisms which are involved in glucose homeostasis. To be sure, this solution to problems of glucose homeostasis, while markedly effective in the short run, is detrimental to the organism if continued for any length of time. While it is probably maladaptive for the individual it has potential eutential consequences, too, if, for example, it leads to spacing out or increased access to scarce resources important for an adequate diet (Bolton 1973:249).

Bolton then outlines a number of ecological, biological, and behavioral variables which he feels are interrelated in a systemic way and which account for the etiology of the hypoglycemia present in the community. He presents them in the form of a model of the "Bio-aggression system of the Qolla" (Ibid.:251). Included in this discussion are such variables as high population density; low per capita resource base -- particularly land; inadequate food production; dietary deficiencies, including hypocaloric intake, low protein intake; vitamin deficiencies, e.g., A,B complex, and C; mineral deficiencies, e.g., calcium; high carbohydrate diet coupled with heavy muscular work; disease, e.g., cirrhosis of the liver; unpredictable weather; anxiety; hypoxia; excessive alcohol intake;
and finally, the chewing of coca leaf (Ibid.: 251–253).

Let me briefly illustrate Bolton's argument concerning the systemic interrelationship between these variables and hypoglycemia by focusing on two of them, i.e., hypoxia and coca chewing. Bolton writes:

Hypoxia, too, may serve as a stressor and may be partly responsible for the widespread hypoglycemia in Incawatana residents. But this question is complex. The data (Baker 1969) seem to suggest that permanent residents at high altitude have attained an adaptation which permits equivalent or higher levels of oxygen consumption than is normal for sea-level subjects. However it may be that not all individuals in the Andes are equally adapted to the hypoxic conditions. An individual might in fact be overadapted or underadapted to the hypoxic environment. If a person is overadapted, he would necessarily burn more glucose at a faster rate than is considered normal elsewhere. Thus he might more readily experience glucose deficits, particularly if nutrition is poor and if he encounters other forms of stress. If a person is underadapted, the low oxygen pressure would serve as stressor and possibly lead to the eventual deterioration of the adrenal glands and from there to the development of hypoglycemia. One or both of these situations may exist (cf. Van Liere and Stickney 1963). Picon (1962, 1963, 1966) has been studying the effects of chronic hypoxia on carbohydrate metabolism, comparing groups of subjects at sea level and at high altitudes in Peru. His studies show that there are important differences in metabolic processes between the two groups. Among his findings is the fact that during the intravenous GTT the blood glucose concentration diminishes more rapidly in high-altitude subjects than in sea-level ones, and that the initial hyperglycemic response to glucose is less pronounced in the high-altitude subjects. It was pointed out above that the highest correlation between aggressiveness and glucose levels occurred when the glucose level in GTT had dropped by the time of the fourth glucose reading, two hours after the beginning of the test. Consequently, the rapidity of the drop seems to be related to both altitude and aggressiveness. Unfortunately, it is not known what causes this rapid drop among high altitude natives [my emphasis] (Ibid.: 252–253).

In regard to the "reciprocal interaction between coca chewing and hypoglycemia," Bolton writes:

The person with hypoglycemia [brought on by such stresses as inadequate food production, dietary deficiencies, hypoxia, etc. — my insertion] becomes hungry and chews coca to dull his hunger pains and to provide himself with energy. The coca has immediate effects in raising the glucose level, probably by stimulating the transformation of glycogen stores, but it probably has long-term detrimental effects which complicate glucose homeostasis problems for the individual who chews (Ibid.: 253).

"Hypoglycemia," Bolton writes, "leads to high involvement in aggression"
The circle, therefore, becomes complete with aggression becoming, as pointed out earlier, a behavioral means by which the individual attempts to maintain blood glucose homeostasis.

In summary, both the "food scarcity hypothesis" and the "hypoglycemia hypothesis" have a number of points in common. First, both are predicated on a cocaine-model, one explicitly and the other implicitly, of coca chewing. Second, both emphasize food deprivation as playing a major causal role in the chewing of coca leaf to "suppress the sensation of hunger" or to "dull hunger pains," as well as to increase the energy level. Third, both argue that while this may have short-term advantages it probably has long-term disadvantages; in one case, it is argued, the individual eventually "loses his appetite" and "prefers the drugs to food," thereby resulting in a "deficient eating regime" and "debilitation of the organism" (Gutierrez-Noriega 1952:118), and the other argues that it probably "complicates glucose homeostasis problems for the individual who chews" (Bolton 1973:253).

There is, however, one difference between the two models. On the one hand, Gutierrez-Noriega states that "the blood sugar always increases in experimental animals, but only in a very few cases does it increase in man after an ordinary period of coca chewing" (1950:85). On the other hand, Bolton is arguing that "coca has immediate effects in raising the blood glucose level, probably by stimulating the transformation of glycogen stores" (1973:253). The remainder of this paper will be directed at showing that chewing coca leaf does in fact raise blood glucose levels and may in reality be a cultural mechanism for the management of blood glucose homeostasis problems.

TOWARD AN ECgonine-MODEL OF COCA CHEWING

I believe that Bolton has provided us with an important clue to the problem of "why" Andean peasants chew coca leaf when he showed that there is a widespread problem of hypoglycemia in the community of Incawatana and probably throughout the Andean highlands. Frombach (1967) has clearly demonstrated that the chewing of coca leaf does in fact result in a rise in blood glucose concentration levels (see Figure 1).

What is interesting about the pattern in blood glucose rise during coca chewing that Frombach determines, is that it remains high over an extended period of time, surpassing four hours and still remaining significantly above the initial fasting glucose level of about 72 mg per 100 ml of blood. Using the same criteria as Bolton used for the administration of the oral GTT [glucose tolerance test], i.e., that "a rise in glucose [above fasting level] at the end of four hours or a drop [below fasting level] of 5 mg or less to be normal" (1973:246), then I would say that the chewing of coca
Figure 1. Blood glucose levels in coca chewers after chewing 1-11/2 ounces of coca leaf. Median values of 18 cases, after Frombach (1967: 392)

leaf may turn out to be of significance in the management of glucose homeostasis problems, since it may help to “normalize” glucose levels. But this rise in blood glucose is probably not taking place in exactly the way that Bolton would probably argue, i.e., by cocaine stimulation of the sympathetic part of the autonomic nervous system, and the transformation of liver glycogen by increased levels of catecholamines [epinephrine and norepinephrine].

As I pointed out earlier, at least 14 different alkaloids, including methyl benzyol ecgonine or cocaine, have been isolated from coca leaf varieties (Martin 1970). Several of the most cherished conceptions of the chewing of coca leaf that we may have to put aside are that cocaine is in fact the central alkaloid in coca chewing, and that the coquero adds alkaline substances [cat, lipta, tocr, etc.] to the coca bula during the process of chewing to both facilitate the extraction of cocaine alkaloids and to potentiate their action [refer back to the cocaine-model as outlined by Verzar 1955].

Recently, Montesinos (1965) and Nieschulz and Schmersahl (1969) have demonstrated that the addition of an alkaline substance to coca leaf does in fact facilitate the extraction of the alkaloids from coca leaf, but it does so only by bringing about the degradation of cocaine. Moreover, Montesinos (1965) argues that, beginning in the mouth itself, cocaine alkaloids are further degraded, and ecgonine, not cocaine, is the end product of hydrolysis and metabolism. Montesinos writes:
The amount of cocaine circulating in the addict's [sic] organism is insignificant and the amount of ecgonine very appreciable.

It is quite possible that the addict [sic] may absorb directly through the buccal and gastric mucosa very small amounts of cocaine which are disintegrated by the cocaine-esterase in the blood, without any appreciable quantities remaining in the circulation or in the tissues (1965: 16).

Montesinos outlines the pharmacological effects of ecgonine on mice as follows:

Ecgonine modifies the degree of blood pressure producing slight hypotension, has no influence on the salivary and sudoriferous glands, slightly reduces the rate of breathing, produces slight myosis without altering the pupillary reflex, has no effect on the contraction of the striated muscle, and produces moderate relaxation of the muscles of the small intestine in the rat, while maintaining the intensity of its peristaltic movements; the toxic dose of ecgonine is between 100 and 110 mg for a mouse weighing 25 to 30 g (Ibid.: 15).

More recently, Nieschulz has shown ecgonine to be about 80 times less toxic than cocaine; that it has little or no central stimulating effect on the sympathetic nervous system; no anesthetic or euphoric properties; and that oral doses do increase the exertion capacity of mice, although less so than similar oral doses of cocaine (1971: 285). Moreover, Nieschulz demonstrates that the addicting, euphoric, and anesthetic action of cocaine can only occur when the molecule is intact, and because cocaine is degraded into ecgonine, he states that the distinction between the chewing of coca leaf and "cocainism" is "pharmacologically supported" (1971: 285).

That cocaine is not the central alkaloid involved in coca leaf mastification should, if nothing else, lead researchers in the future to be very cautious about conducting experiments with pure cocaine hydrochloride on either mice or men and attempting to generalize from this to the mastification of coca leaf, a situation which has been all too common in the past (cf. Gutierrez-Noriega and Zapata Ortiz 1947). Moreover, this should provide us with insights into why, as even the principal opponents of coca cultivation and use agree, and, as one of them states, "the symptoms of addiction to coca leaves... are weak..." (Zapata Ortiz 1970: 290).

That cocaine is not the central alkaloid involved in coca-use makes it easier to understand some of the contradictions that appear in the arguments of those researchers who support a cocaine-model of coca-use, especially statements such as those by Gutierrez-Noriega that "the use of the drug occasions after some years the loss of appetite," or "the habituated chewer prefers the drug to food" (1949: 143), or the statement by Wolff (1950: 147) that "it is evident that the chewing of these leaves for pleasure is incompatible with the regular taking of normal meals."
Close observation by researchers, including myself, who have spent time in the field undertaking participant observation in Andean communities where coca is important indicates that there is no correlation between the chewing of coca and the “loss of appetite” or the preference of the “drug to food.” Webster writes: “the conjecture that coca use is actually a cause of inadequate food consumption because it deadens normal hunger drives... is discredited if one appreciates the Indian’s nearly obsessive preoccupation with food and food needs as reported by Stein (1961) and Margin (1954)” (Webster 1970:94). In a comparative study of two brothers from the highland community of Vicos, one of whom chewed coca regularly and the other of whom did not chew coca, Fine (1960) reports that their respective food consumption was equally adequate and their eating patterns almost identical. More recently, Murphy et al. (1969) conducted controlled research on a group of long-term coca chewers and a group of non-chewers. Although their research was not specifically directed at the problem of coca-use and food, nevertheless they write:

Regarding appetite, we had expected that since coca was chewed by the local population to suppress hunger when traveling or working in the fields, the continuing-users might eat less than other experimental subjects. However, they ate amply, and explained their behavior by saying that coca did not kill their hunger, but merely made it easier to hear and forge: [my emphasis] (Murphy et al. 1969:45).

During the course of my own fieldwork, there were literally dozens of times when, after sitting and chewing coca with peasants for periods of several hours, we were invited to a meal. In these many times, I never once saw a coca chewer refuse to eat because chewing coca resulted in a “loss of appetite,” or had suppressed his desire for food. On the contrary, everyone, including myself, ate with great gusto. I have seen both men and women who have spent from 8 to 10 hours in a session [chagchupada] where coca chewing is continuous, end the session with a large meal. When I confronted one of my informants with these contradictions, since he too had told me that he chewed coca to “suppress hunger” [“me quita el hambre”], he turned to me and replied: “Coca is good to chew but you have to eat food to live. All people have to eat food, no matter if they chew coca or not.”

I do not mean to imply by the above remarks that when coca chewers say, as they have since and probably even before the Spanish conquest, that they chew coca because it “suppresses hunger” or that it permits them to “work harder,” that it may in fact not do just that. What I mean to say is that although these responses, as well as some, but apparently not all, of the physiological responses (cf. Frombach 1967) involved in
Coca chewing happen to fit within a cocaine-model, perhaps the overemphasis on a cocaine-model of coca chewing and the powerful symbolism of the “cocainist” have been leading us astray in these many years of trying to explain “why” peasants chew coca leaf.

Is it possible, therefore, that in a population where over 70 percent of the diet is based on carbohydrate intake (Mazess and Baker 1964), where hypoxia may impair certain enzymes involved in the transformation of glycogen into glucose and where glucose is used at a much higher rate than at sea level (Picon 1966), and where it has been shown that there are problems of blood glucose homeostasis (Bolton), that since the chewing of coca leaf does result in not only a rise in blood glucose but maintains concentration over an extended period of time, that this may in fact be what the peasant means to imply when he says that it helps to “suppress hunger” and to “work harder”?

I believe that it is, but before I make additional comments, let me turn briefly to the complex problem of coca chewing and peasant health. Although Bolton has argued that chewing coca does result in a rise in blood glucose levels, he also argues that it may have long-term detrimental effects which complicate glucose homeostasis problems for those who chew (1973:253). In fact, the entire problem of coca chewing and peasant health is the center of major debate in Peru and elsewhere today. It is outside the scope of this paper to outline all the arguments that have been brought forth on the subject, but let me present some examples of the complexity of the problem.

Although it is commonly stated that all coca chewers suffer from chronic undernourishment (Gutiérrez-Noriega 1949; Zapata Ortiz 1952, 1970; Ricketts 1952, 1954; Buck et al. 1970), Baker (1969) has undertaken a nutritional survey of the highland district of Núñoa, located at an altitude of between 4,000 and 5,000 meters, with a population of some 7,750 inhabitants, most of whom are probably coca chewers. Baker writes:

The results to date indicate that the Núñoa population has a very delicate, but adequate balance, between nutritional resources and needs... if malnutrition exists, it is probably no more common than in U.S. society (1969:1154).

As an indication of just how delicate the balance is between nutritional needs and resources, Baker points out that the native foods of the area are generally low in calcium, as well as low in certain vitamins, including ascorbic acid [vitamin C] (Ibid.). Yet elsewhere, Baker and Mazess (1964) indicate that cal and ilipta, both of which are used in the chewing of coca, are sources of needed calcium. Cal contributes between 300 and 1200 mg and ilipta between 200 and 500 mg of calcium per day to the diet of coca
chewers (Ibid.:1466). In regard to the deficiency of vitamins in the diet, it should be noted that, the United Nations Commission of Experts states that coca leaf has a relatively high level of vitamin B1, B2, and C (United Nations 1950:26). They report that "a quantity of 100 grams of dried leaves could supply a considerable part of the daily human requirements" in the above mentioned vitamins. But they add, because cocaine is involved in coca chewing, that this overshadows any vitamin content that coca leaf might have. But, because cocaine is insignificant, perhaps the vitamin content, as well as the minerals, involved in coca chewing may be significant after all.

In a recent epidemiological study, Buck et al. (1968, 1970) point out that they found several indicators which they believe demonstrate the lower nutritional status of coca chewers as opposed to non-chewers. For example, they indicate that coca chewers in the montaña community of Cachicota, Huancuco, Peru, have lower serum protein levels than non-chewers. They write: "Differences in total protein average 0.53 gm% and are on the borderline of statistical significance [P = .07]" (Buck et al. 1970:26). Yet, in another study, Chahud et al. (1969:216) found the total serum proteins of highland coca chewers to be 6.50 gm%, and that of non-chewers to be 6.25 gm%, although both groups were lower than coastal "normals" at 6.89 gm%. This indicates that the problem of coca chewing and "poor" health is not as clear cut as we might think on the basis of any one single investigation.

Let me now turn to the topic of coca leaf and blood glucose. Again, I wish to point out that although I am referring to this argument as an "ecgonine-model," that there are other alkaloids in coca leaf, i.e., tropine and hygrine, and I use the term mainly to try and place emphasis elsewhere than on cocaine. Ecgonine is an amino alcohol base, closely related to tropine, the amino alcohol and active component of atropine (Ritchie 1965:375). Therefore, the combination of ecgonine and the tropine alkaloids also found in coca leaf may in fact be acting on the parasympathetic nervous system as opposed to the sympathetic, and a parasympathetic response has been reported (Risemberg 1950). Atropine also results in a rise in blood glucose (Berk et al. 1970). Moreover, atropine is among the drugs recommended by Miller and Keane (1972:461) "to help control the symptoms of hypoglycemia." Gray (1973:121) points out that malnutrition appears to play a role in the reduced capacity to digest and absorb carbohydrates. He writes: "A study of atropine effect in man...showing an increased absorption of xylose [a sugar] suggests a possible mode of drug therapy that might be of benefit in patients with rapid intestinal transit" of glucose. He notes that atropine increases monosaccharide [of which
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Glucose is the principal one] absorption by increasing the contact-time between carbohydrates and mucosa (Ibid.:123).

In short, I believe that the chewing of coca leaf, since tropine is a part of it, may be doing the same thing, and in fact may be important in the control of problems of the malabsorption and too rapid transit of glucose.

CONCLUSION

Bolton (1973:254) writes: "We will need detailed studies on the relationship between coca chewing and hypoglycemia...." This paper, although not as "detailed" as might be needed, is nevertheless, a direct response to the call.

It remains to be seen if the blood glucose rise resulting from coca chewing is due to the transformation of glycogen stores in the liver or merely a result of the concentration of the existing glucose pool and reduced peripheral utilization. Nevertheless, it may very well be that chewing coca leaf is an important cultural mechanism for managing problems of glucose utilization since coca leaf is chewed just as frequently after a high carbohydrate meal as before. After all, Andean peasants classify coca as a medicine and not as a "substitute for food" as many researchers who adhere to a cocaine-model of coca chewing have for too long led us to believe (cf. Gagliano 1961; Hughes 1946; Gutierrez-Noriega 1949; Zapata Ortiz 1952).

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