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Current Anthropology, Vol. 37, No. 5 (Dec., 1996), 831-842.

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Reports

The Non-Thrifty Genotype[1]

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In 1962, the geneticist James Neel suggested that diabetes mellitus might be due to a "thrifty genotype" rendered detrimental by "progress." By this he meant that the relatively high prevalence of diabetes, a disease whose ultimate effects are quite debilitating, could be explained if the diabetes genotype were viewed as an adaptation that arose in hunter-gatherer populations in response to periodic famine conditions or to frequent periods of food stress. This too efficient or thrifty genotype manifested itself as diabetes only in a continually "nutrition-rich" (i.e., "Westernized" or "civilized") environment; in a marginal environment, individuals in possession of this genotype were selected for because they made more efficient use of available food resources than individuals lacking it. Over the past 30 years, the thrifty genotype has become one of the orienting concepts of nutritional and biomedical anthropology. For example, in a recent edited volume on diabetes in native North Americans (Joe and Young 1994), half of the 18 contributors cite the thrifty-genotype concept, almost all positively (but see Jackson 1994:394).

Although the thrifty-genotype concept presupposes the existence of a "non-thrifty genotype," there has been very little discussion of this in the literature. One reason for this neglect may be that the thrifty genotype was originally defined in contrast to a Euro-American norm, and Euro-American anthropologists, more concerned with the anthropological "other" than with themselves, have been willing to accept the standard and rather facile explanations for why the thrifty genotype does not exist in all human populations. In fairness, it should also be noted that the interest in the thrifty genotype has also been driven by an attempt to understand and treat health problems associated with diabetes and obesity, which are very common in recently Westernized populations. However, the perspective that led to the invention and acceptance of the "thrifty-genotype" label also identified in the 1960s a "disease" known as lactose intolerance. Here the European norm (lactose tolerance) turned out to be quite abnormal in a worldwide context and required a specific evolutionary explanation. We suggest that the non-thrifty genotype is analogous to lactose tolerance in that both are putative dietary adaptations found in high prevalence in European and European-derived populations but rare in most human populations. Furthermore, we suggest that the coexpression of these two features is not due to chance but may reflect a single evolutionary trajectory.

The thrifty-genotype concept itself has been evolving. Neel (1982) revised the concept after it became clear that there were at least two major forms of diabetes mellitus: insulin-dependent (IDDM, "juvenile-onset" or Type 1) and non-insulin-dependent (NIDDM, "adult-onset" or Type 2) (Bennett 1985). Whereas in 1962 Neel had relied on the severe and acute effects of IDDM as the agent of selection against the thrifty genotype in nutrition-rich populations, in 1982, with knowledge that IDDM and NIDDM are not etiologically related and that most recently Westernized populations suffer in considerable numbers only from NIDDM, Neel had to modify his hypothesis. Still, he argued that negative selection due to the effects of NIDDM (had) occurred in nutrition-rich populations despite the late age of onset of the disease; he also noted the relationship between NIDDM and gestational diabetes, which could exacerbate selection against the thrifty genotype.

Metabolic evidence indicates that the thrifty-genotype concept is physiologically plausible. First, diabetic and non-diabetic Pima Indians show increased insulin resistance (compared with Europeans) coupled with maintenance of the antilipolytic action of insulin; this leads to more efficient fat storage at any given nutritional intake level (Knowler et al. 1982, Nagulesparan et al. 1982). Second, in the early stages of starvation, increased resistance to insulin-mediated glucose utilization would conserve glucose for use in important insulin-independent tissues, such as the brain and red blood cells, and prevent it from being utilized by peripheral tissues, such as muscle; in addition, insulin-resistance leads to diminished nitrogen excretion, and nitrogen conservation is probably more important than caloric conservation for growth and health (Cahill 1979:389–90). Third, different types of diabetes developed in laboratory mouse strains often exhibit thrifty

1. © 1996 by The Wenner-Gren Foundation for Anthropological Research. All rights reserved 0011-3204/96/3705-0006$1.00. This research has been supported by funds from the New Zealand Dairy Advice Bureau and the Health Research Council. An earlier version of this paper was presented at the 1994 Australasian Clinical Nutritionist Society meeting in Auckland, New Zealand; we thank the organizer, B. Swinburn, for inviting our participation. We also thank A. Brewis, P. Baker, T. Baker, S. McGarvey, K. O'Dea, V. Sarich, and two anonymous referees for comments on earlier versions of this paper. JSA thanks Fatima Jackson for inspiring his work in this area, Roger Green for archaeological consultation, and the organizers of the Summer Institutes for Comparative Anthropological Research (NSF grant #SBR-8911173).

metabolic processes (Coleman 1982). Fourth, studies by Neel and others on the Yanomama indicate that they have normal glucose tolerance levels when living under traditional conditions; this is a necessary demonstration of the hypothesized thrifty genotype's ability to do more with less (Neel 1982:286–87). All in all, these studies amply demonstrate that the thrifty genotype is theoretically of adaptive value.

Further evidence in its support comes from studies of the epidemiology of NIDDM in Westernizing or recently Westernized populations. The Pima Indians (Bennett et al. 1982) and the people of Nauru in Micronesia (Zimmet et al. 1977) are well-known for their very high rates of NIDDM, which almost undoubtedly result (in a proximate sense) from the relatively recent adoption of a Western diet. Under similar circumstances of Westernization, elevated NIDDM rates have also been observed in Australian, Polynesian, Asian, and African-derived populations (see table 1).

Reconsiderations of the thrifty-genotype concept have recently been provided by McGarvey (1994) and Crews and Gerber (1994). McGarvey states that the wide acceptance of the concept illustrates the generative role of metaphorical thinking in bioanthropology. Whether the concept is correct or not (in the narrow sense), "it allows for the generation of concrete studies of metabolic processes and their fertility, mortality and morbidity concomitants" (1994:30). Crews and Gerber emphasize the pleiotropic effects of thrifty genotypes (there could be several working in different metabolic domains), which in a well-nourished, long-lived populations may result in the development of chronic degenerative diseases associated with aging. Using Williams's (1957) concept of antagonistic pleiotropy, they propose a model exploring the development of these "thrifty-pleiotropic phenotypes." Neither of these reconsiderations rejects the thrifty-genotype concept, however, and McGarvey (1994:30) reiterates that "metabolic efficiency and energy balance would have been a selective advantage in most human populations during the last 1,000,000 years of subsistence systems."

Although some workers have previously explained the presence of the thrifty genotype in particular populations as the result of unique historical processes, it has become increasingly clear that its geographic distribution is widespread. On the basis of current data, one would predict that as more populations of the world adopt a Westernized diet and lifestyle, diabetes and obesity rates will increase. While Western diets do vary, the common response to them in recently Westernized populations indicates some shared features. Eaton, Shostak, and Konner (1988:67) point out that "our industrial present includes increased levels of saturated fat and salt, decreased levels of fiber and exercise, the widespread use of alcohol and tobacco, a state of continual abundance, and universal caloric concentration." In contrast to the thrifty genotype, the non-thrifty genotype characterizes a small minority of the world's populations; this indicates that it may be a derived characteristic of these populations requiring an evolutionary explanation.

There is abundant evidence, then, to support the idea that a thrifty genotype would be beneficial to individuals living in any population under conditions of food stress and that the adoption of a Western diet, high in fat, calories, and simple sugars, negates the benefit of a thrifty metabolism, instead making an individual possessing the genotype more prone to diabetes, obesity, and the health consequences of those conditions (Eaton, Shostak, and Konner 1988, Crews and Gerber 1994). These have been characterized as the "price of civilization" (Prior 1971). Implicit in the thrifty-genotype concept is that there are basically two kinds of people, those who are currently paying the price of civilization and those who already have (being descended from long-civilized populations characterized by nutritional abundance). Possessors of a non-thrifty genotype have an advantage over others in a Westernized environment because their "slow insulin trigger" allows them to avoid diabetes and obesity (at least until an older age) despite exposure to a high-calorie, high-fat, high-sugar, low-fiber, low-complex-carbohydrate diet.

The current explanation for the existence of a non-thrifty genotype—that it has been selected for in the food-rich environment of long-"civilized" populations—is problematical. Although Neel (1982) certainly considered this issue carefully, most workers in the field have concentrated on the thrifty-genotype end of the spectrum and have basically accepted the explanation for the non-thrifty genotype as a given. However, while the thrifty genotype may extract a price for civilization, this does not suffice as an evolutionary explanation for what happened in populations in which it no longer exists (or is very rare).

Neel's basic assumption was that hunter-gatherers, in contrast to agricultural populations, live(d) with a highly uncertain food supply—he referred to the "feast or famine days" (1982:289). This is a far from generally accepted supposition. In writing about social adaptations to food stress, Minnis (1985:4) felt it necessary to point out that although "famines have been conspicuous events in recent history . . . food shortages are not just a modern phenomenon." Reflecting a commonly held viewpoint, Segraves (1977:181) writes: "It is clear that with the evolution of highly complex societies, the dangers inherent in agricultural specialization become increasingly more significant. The agriculturalist highly dependent on one or more staple crops is clearly at greater risk from food-supply failure and famine than is the forager." Although the characterization of hunter-gatherers as members of the "original affluent society" (Sahlins 1968) can be oversold, it is apparent that an individual in a hunter-gatherer society was likely to do as well as an individual in a large-scale agricultural population from a food-supply perspective. Over the past 2,000 years, periodic famine has plagued China, Russia, India, and Western Europe (Segraves 1977, Minnis 1985).

Since Europe is the implicit and explicit model for a rich nutritional environment (after all, we refer to the effects of Westernization), it is worthwhile to consider aspects of that region's recent nutritional history in

TABLE 1
Lactose Absorption and Diabetes (NIDDM) Rates

Population	Lactose	Diabetes	References
Africa			
Bantu	4.30	0.20	Durham (1991), Cook and Dahlqvist (1968), Jackson and Latham (1978), WHO (1992)
Nigeria	18.30	1.70	Olatun-Bosun and Adadevoh (1971), Kretchmer et al. (1971), Ohwovoriole, Kuti, and Kabiawu (1988)
South Africa	17.10	6.10	Levitt et al. (1993), Currie, Nurse, and Jenkins (1978), Segal, Gagjee, and Noormohamed (1983), Joffe and Saftel (1994)
Tanzania	38.00	0.80	Jackson and Latham (1978), Ahren and Corrigan (1984), McLarty et al. (1989)
Zambia	4.00	1.10	Cook, Asp, and Dahlqvist (1973), Davidson et al. (1969)
Asia			
Chinese Composite	14.20	10.50	Wang et al. (1984), Dowse et al. (1990), King, Rewers, and WHO (1993)
India, South	33.00	11.50	Tandon et al. (1981), King, Rewers, and WHO (1993)
Japan	0.00	11.00	Sasaki et al. (1970), Sekikawa et al. (1993), Ohmura et al. (1993)
Singapore	0.00	16.55	Bolin et al. (1970*a*), King, Rewers, and WHO (1993)
Thailand	1.90	5.75	Keusch et al. (1969), Flatz, Saengudom, and Sanguanbhokhai (1969), King, Rewers, and WHO (1993)
Thailand, North	0.00	5.95	Flatz, Saengudom, and Sanguanbhokhai (1969), King, Rewers, and WHO (1993)
Taiwan, Chinese	0.00	12.40	Sung and Shih (1972), Chou, Chen, and Hsiao (1992)
Europe			
Britain	94.00	0.60	Pena, Truelove, and Whitehead (1973), Ho, Povey, and Swallow (1982), Andrews (1957), Redhead (1960), Walker (1959), Harkness (1962), West (1978)
Czechoslovakia	100.00	1.80	Durham (1991), West (1978)
Denmark	97.50	0.40	Durham (1991), Lindhart (1954)
Finland	82.90	1.20	Jussila (1969), Jussila, Isokoski, and Launiala (1970), Sahi (1974), Eriksson et al. (1992)
Germany	85.00	1.30	Gudmand-Hoyer, Dahlqvist, and Jarnum (1969); Flatz et al. (1982)
Italy			
Composite	25.00	6.45	Bianchi, Parente, and Sangaletti (1983), King, Rewers, and WHO (1993)
North	49.00	2.10	Burgio et al. (1984), Cavalli-Sforza et al. (1987)
Poland	63.00	3.50	Socha et al. (1984), King, Rewers, and WHO (1993)
Russians	85.00	2.70	Flatz (1987), King, Rewers, and WHO (1993)
Sweden	100.00	1.90	Dahlqvist and Lindqvist (1971), Durham (1991), West (1978), Munke (1964)
Near East			
Arabs, (Jordan, Syria, Oman)	0.00	14.15	Durham (1991), King, Rewers, and WHO (1993)
Egypt	27.00	6.28	Hussein et al. (1982), Alwan and King (1992), Arab (1992)
Iraq	14.00	4.80	Kassir and Kellow (1978), Alwan and King (1992)
Saudi Arabia	12.00	4.80	Cook and Al-Torki (1975), Alwan and King (1992)
North America			
Alaska, Indian; Eskimo	17.00	1.60	Duncan and Scott (1972), Gohdes, Kaufman, and Valway (1993), Zimmet, Taylor, and King (1982)
Canada			
Eskimo	17.00	0.40	Ellestad-Sayed, Haworth, and Hildes (1978), Young et al. (1990)
Indians	8.70	14.00	Ellestad-Sayed and Haworth (1977), Delisle and Ekoe (1993), Brassard, Robinson, and Lavallee (1993)
White	94.00	0.80	Leichter (1972), Kenny and Chute (1953)
United States, African	25.60	10.30	Bayless and Rosensweig (1966), Welsh et al. (1968), Sasaki et al. (1970), Cuatrecasas, Lockwood, and Caldwell (1965), King, Rewers, and WHO (1993)
Apache	0.00	10.10	Gohdes, Kaufman, and Valway (1993), Simoons (1978)
Caucasians	80.70	0.70	Welsh et al. (1967), Cuatrecasas, Lockwood, and Caldwell (1965), Bayless and Rosensweig (1966), Huang and Bayless (1968), Woteki, Weser, and Young (1977), Silliman et al. (1984), Sheehy and Anderson (1965), Zimmet, Taylor, and King (1982)
Chippewa	29.80	14.75	Newcomer et al. (1977), Rith-Najarian, Valway, and Gohdes (1993), Simoons (1978), Gohdes, Kaufman, and Valway (1993)
Mexican	48.90	14.10	Dill et al. (1972), Sowers and Winterfeldt (1975), Woteki, Weser, and Young (1977), King, Rewers, and WHO (1993)
Papago	7.00	49.40	King, Rewers, and WHO (1993), Johnson et al. (1978)
Pima	5.00	50.25	King, Rewers, and WHO (1993), Simoons (1978)
Shoshone	0.00	12.50	Acton et al. (1993), Simoons (1978)
Pacific			
Australia			
Aboriginal	16.00	22.08	Brand et al. (1983), King, Rewers, and WHO (1993), O'Dea et al. (1993)
Caucasian	87.60	2.55	Bolin et al. (1970*b*), Davis and Bolin (1967), Bolin and Davis (1969), Bryant, Chu, and Lovitt (1970), Brand et al. (1983), Zimmet, Taylor, and King (1982)
Fiji	0.00	15.25	Masarei, Sharma, and Jansen (1972), King, Rewers, and WHO (1993)
New Zealand			
Samoans	46.00	6.95	Abbott and Tasman-Jones (1985), Zimmet, Taylor, and King (1982), King, Rewers, and WHO (1993)
Maori	36.00	9.60	Abbott and Tasman-Jones (1985), Prior et al. (1978)
Caucasians	91.00	2.80	Abbott and Tasman-Jones (1985), Zimmet, Taylor, and King (1982)
Tokelauans	45.30	6.15	Cheer unpublished, Prior et al. (1978)
PNG	11.40	15.0	Arnold, Perman, and Nurse (1981), Gibney et al. (1981), Cook 1979, Zimmet, Taylor, and King (1982), Martin et al. (1980)
South America			
Brazil, non-Caucasian	6.00	7.95	Troncon et al. (1981), King, Rewers, and WHO (1993)
Mexico	26.00	10.20	Lisker et al. (1974), Haffner et al. (1990)

more detail. The Greco-Roman era constitutes the beginnings of civilization in Europe, yet there is no indication that this also signalled the beginnings of "feast" days for the average person. While famines were relatively rare, Greco-Roman communities "were endemically vulnerable to food crisis through a combination of human and natural causes. . . . Subsistence crises were common in antiquity" (Garnsey 1988:16–17). There was "permanent anxiety about the food supply" in these communities, and the "alleviation of food crises by private benefactors" was institutionalized (Garnsey 1988:15). Post-Roman Europe apparently fared little better. In her survey of Anglo-Saxon foodways, Hagen (1992:109) finds that "the number of references to hunger in the literature, chronicles and legal documents implies that it was probably a normal experience of life. . . . A proportion of the population, in particular women, probably had to make do with a less than adequate diet throughout the period [A.D. 400–1100]." More than 50 separate famines are referred to in Anglo-Saxon records of this period (Hagen 1992:151–54). Paradoxically, the best nutritional times for the average European before the modern era were to be had in the days following the Black Death. Braudel (1981) emphasizes the unprecedented availability of meat and the high living standard of the average worker in this period of scarce manpower. This period was relatively short-lived, however; "in about 1600, the workers in the copper mines in Mansfeld, Upper Saxony, could only afford, on their wages, to eat bread, gruel and vegetables" (Braudel 1981:196). Braudel (p. 197) provides an apt summary for this brief review: "Europe remained hungry for a long time."

Civilization in Europe did not bring an abundant diet to its inhabitants, and the civilizations of Asia, Africa, and the New World apparently did not lead to the evolution of the non-thrifty genotype. Indeed, we suggest that the typical consumer in virtually any community in the world, living in the year 1650 (i.e., before the impact of New World foods on Old World cuisines), would have been better off with the thrifty genotype than without it. King (1992) suggests that the relative scarcity of diabetes in Highland Papua New Guinea may be due to the long history (perhaps greater than 9,000 years) of agriculture in this area, which has resulted in a steady food supply. Diabetes rates in Westernized Highland populations have yet to be reported; for coastal Papua New Guineans (who are genetically and linguistically distinct from Highland populations), urban dwellers have a diabetes rate 15 times higher than rural dwellers (Martin et al. 1980). King (1992) argues that if Westernized Highland populations do not exhibit the thrifty genotype, this would be further support for the concept in general. It should be pointed out that while Highland populations may have had steady food supplies (and if they are like other agricultural populations, they may not have), they were certainly not characterized by energetic abundance relative to a Western diet (Heywood and Jenkins 1992). The Highland Papua New Guinea situation remains unresolved but intriguing; however, on the basis of the pattern observed in most other populations, we would predict that they too will develop higher rates of diabetes upon the general introduction of a Western diet.

The thrifty genotype is certainly rare in European and European-derived populations today, and it seems highly unlikely that selection against it would have been strong enough to reach the current level in only a few hundred years. If we rule out civilization as a marker for nutritional abundance, how then do we explain the evolution of the non-thrifty genotype in these populations?

One possible explanation is chance. European populations could have gone through some kind of population bottleneck and lost the thrifty genotype through genetic drift. Working against this idea is the fact that the thrifty genotype is present in some individuals in contemporary European populations, and given the advantages conferred by it during periods of nutritional stress, it should have been selected for in Europe as it was in other populations. There is also no evidence of a very recent Europewide population bottleneck.

Besides a direct influence on exploiting particular foodstuffs, sociocultural factors could theoretically play a role in changing the selection environment in which the thrifty genotype is expressed. Colson (1979) lists five cultural adaptations to food stress observed in a wide range of societies: (1) diversification of food-gathering activities; (2) storage of foodstuffs; (3) storage and transmission of information on famine foods; (4) conversion of surplus food into durable valuables that could be stored and traded for food in an emergency; and (5) cultivation of social relationships to allow tapping of food resources in other regions. These cultural adaptations have probably characterized human populations for a long time, and for each of them possession of a thrifty genotype during periods of food stress would still be an advantage. However, the advantage could be reduced in a large-scale society covering a large geographic area with efficient mechanisms for the trade and exchange of food. Certainly this is the case in the modern worldwide food economy, where shortages in one region can be compensated for by increased trade with another. Palaeoeconomic studies indicate that this is unlikely to have occurred to a significant extent in any preindustrial large- or small-scale society (Jarman, Bailey, and Jarman 1982:26–48).

Another sociocultural factor that could influence the evolution of the thrifty genotype involves social stratification. By definition, social stratification results when different parts of a society have differential access to basic resources. Minnis (1985:9) points out that "the presence of social stratification represents significant change in a social system's response to food shortages. . . . a stratified society exhibits class variation in access to food." Segraves (1977:209) states: "The quietly systematic pruning of the population represented by the higher morbidity and mortality rates of the poor obviates a wider and considerably more serious system strain. Those of the poor who have not adapted to a chronically low dietary intake generally die in sufficient numbers to ensure some moderation of an overburdened resource base." In a stratified society, the ability to survive during

periods of food stress may depend less on genotype and more on position in the social hierarchy. This is less true in non-stratified societies, where the effects of food shortage are more evenly distributed throughout the population (Sahlins 1972). In caste societies, it is possible for lower-caste people to do better than upper-caste ones during periods of food stress because of dietary and economic proscriptions affecting only the latter (Minnis 1985:9–10). Thus social stratification may also serve to lessen the advantages of a thrifty genotype. Most people, however, are not elites, and in the competition amongst the poor for scarce food resources a thrifty genotype should still do better than a non-thrifty one.

The environment in which the thrifty genotype evolves is usually characterized in broad terms: Westernized versus traditional or famine versus feast. More specific components of the nutritional environment could, however, play a role in selecting against the thrifty genotype. The European population, for example, is different from most others not only in having low NIDDM rates but also in having high rates of lactose absorption. We suggest that there may be a relationship between these two dietary adaptations and that there may have been increased selection for the non-thrifty genotype in a nutritional environment characterized by a relatively high level of lactose consumption.

Lactose is the principal carbohydrate in most mammalian milk. It is hydrolyzed by the enzyme lactase, which is present in the small intestine of most mammalian infants (Scrimshaw and Murray 1988). In mammals, intestinal lactase levels are at their highest immediately after birth, decline after weaning, and fall to very low levels in adulthood (Saavedra and Perman 1989). Until the mid-1960s, researchers believed that humans were exceptional among mammals in retaining high adult lactase activity (Flatz 1987). In a pioneer study, Cuatrecasas, Lockwood, and Caldwell (1965) demonstrated that African-Americans had a much higher rate of lactase deficiency (lactose malabsorption) than Americans of European descent. There is much ethnic variation in levels of adult lactase production, and research has confirmed that in most human populations the mammalian pattern of lactase decline is the norm (Kretchmer 1972).

In most people, lactase levels decline in the postweaning period. The age at which this decline begins varies with ethnicity. Lebenthal, Antonowicz, and Shwachman (1975) found that lactase levels in lactose malabsorbers in European populations declined gradually to low levels by the age of 5 years. Johnson et al. (1977) determined that the prevalence of low lactose digestion capacity (lactose malabsorption) in Pima was 40% at 3–4 years, 71% at 4–5 years, 92% at 5–7 years, and 100% at 8 years and older. Lactase levels decline at 2–4 years in Thais (Keusch et al. 1969) and as late as 15 years in Finns. The precise mechanism associated with the decline is unknown; hormonal and cytokinetic factors have been suggested, and so has the notion that synthesis continues but in a less active form (Saavedra and Perman 1989).

Lactose is a disaccharide that is split into its component monosaccharides, glucose and galactose, by the enzyme lactase 1 (Scrimshaw and Murray 1988). Lactose "malabsorption" occurs when the intake of lactose exceeds the rate at which it can be digested. Because of the osmotic activity of lactose in the small intestine and the fermentation of lactose by colonic flora in the large intestine, lactose malabsorbers suffer from a variety of symptoms of gastric distress, including abdominal distention, flatulence, cramps, acidic stools, and diarrhoea (Ladas et al. 1991, Seakins 1983, Lee and Harvey 1989).

Most of the world's populations, excepting those of northwestern Europe and their descendants, some ethnic groups in Africa (Tussi, Fulani, and Hima), and those of northern India, have low lactase levels (Harris 1985, Durham 1991). A number of hypotheses have been advanced to explain this distribution of lactase phenotypes. Most researchers now agree that the variability in adult lactase activity represents a genetic polymorphism (Durham 1991). Evidence supporting this demonstrates that lactose digestion capacity varies across ethnic groups (Bayless and Rosensweig 1966, Cuatrecasas, Lockwood, and Caldwell 1965, Elliot, Maxwell, and Vawser 1967, Dalhqvist and Lindquist 1971, Sahi and Launiala 1978), the absorption rates in populations of mixed ancestry are intermediate to those of the parent populations (McMichael, Webb, and Dawson 1965, Cook and Kajubi 1966, Gudmand-Hoyer and Jarnum 1969, Gudmand-Hoyer et al. 1973, Bayless, Christopher, and Boyer 1969, Flatz and Rotthauwe 1971, Johnson et al. 1977), and low lactose digestion capacity (lactose malabsorption) in families can be calculated on the basis of autosomal recessive inheritance (Sahi 1974, Sahi et al. 1973).

Although there is general agreement that a genetic polymorphism is responsible for the present distribution of lactase phenotypes, the mechanism by which this polymorphism evolved is controversial. Most researchers favor natural selection and suggest that the use of fresh milk in dairying populations conveyed some kind of nutritional advantage (Flatz 1987). Two hypotheses currently account for the worldwide distribution of the lactase polymorphism: the *cultural-historical* hypothesis (Simoons 1970, McCracken 1971) and the *cultural-mediation* hypothesis (Durham 1991).

The cultural-historical hypothesis was presented independently by Simoons (1970) and McCracken (1971) after each identified a correlation between the frequency of lactose absorption and the history of dairying in some populations. They hypothesized that human hunter/gatherers, like other mammals, are lactose "malabsorbers" because of a drop in lactase activity levels after weaning. With the introduction of dairying, lactose-absorbing individuals gained a general nutritional advantage in populations where milk was a critical part of the diet, where dietary stress existed, and where milk was not processed to form low-lactose products (Simoons 1970, 1978; McCracken 1971).

In contrast, the cultural-mediation hypothesis (Durham 1991) is based on the fact that lactose acts physiologically like vitamin D, facilitating the absorption of

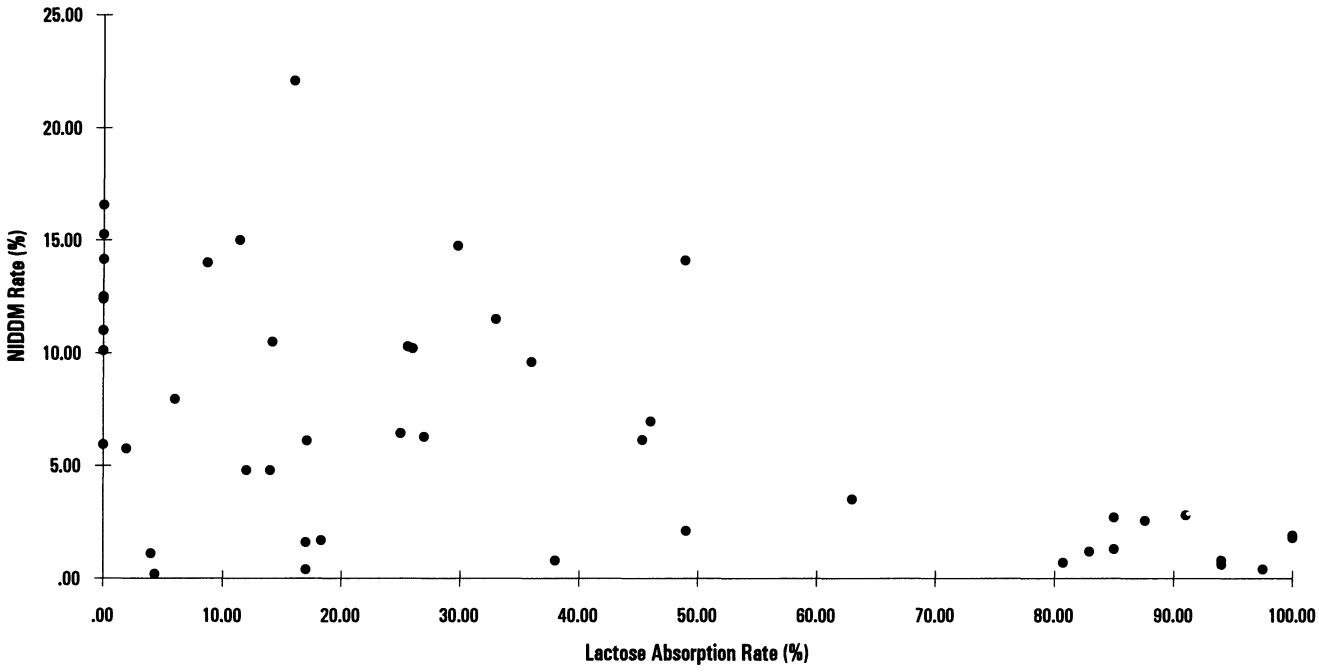

FIG. 1. *Lactose absorption rate vs. NIDDM rate for the populations listed in table 1 (Pima and Papago outlier values excluded).*

calcium and thus preventing diseases such as rickets (a disease of early childhood). Durham suggests that high frequencies of the lactose absorption gene should occur in populations living at high latitudes (where vitamin D production in the skin is insufficient because of low ultraviolet radiation) if in the past they evolved a cultural means of encouraging fresh milk consumption after weaning, had substantial consumption of milk, and lacked sufficient intake of vitamin D. While this hypothesis seems extremely plausible for the northern European situation, Durham's own cross-cultural analysis indicates that neither hypothesis can completely account for the global distribution of lactase phenotypes.

Table 1 presents lactose absorption and NIDDM rate data for 48 societies for which both values were available; in the cases where multiple values were available, the average of the values is provided. These data are plotted in figure 1 (with the exception of the outlier Pima and Papago figures). Table 2 presents correlations derived from these values. The correlation for all populations ($r = -0.44$, $r^2 = 0.20$) is statistically significant, although not particularly compelling from a qualitative perspective. Removal of the results for the outlier Pima and Papago (which have diabetes rates double that of any other population in the sample) has a strong effect on the overall correlation; r^2 for this sample of 46 populations is 0.32 ($r = -0.56$). Another potentially mediating factor in these figures is the inclusion of populations that are not sufficiently Westernized in diet to manifest the thrifty-genotype effect. Almost all of the low-lactose-absorption/low-diabetes-rate populations in the sample come from Africa and the Arctic, where the extent of Westernization of the diet at the time of testing may have been limited (although this is a complex issue [see Young et al. 1990]). If these are taken out of the correlation computation but Pima and Papago are left in, the r^2 is 0.30. If the African, Arctic, and Pima/Papago figures are taken out, then for the remaining 39 populations in the sample, 56% of the variance in observed diabetes rates is attributable to lactose absorption capac-

TABLE 2
Correlations between Lactose Absorption and NIDDM Rates

Sample	*N*	r^a	r^2	*p*
All	48	−0.44	0.20	0.002
Without Pima-Papago	46	−0.56	0.32	<0.001
Without Africa/Arctic	41	−0.54	0.30	<0.001
Without Africa/Arctic/Pima-Papago	39	−0.75	0.56	<0.001

[a] Pearson's (two-tailed) correlation coefficient.

ity. The Pima/Papago figures, which are in the "right" direction (i.e., low lactose absorption and high diabetes rates), clearly have a strong distorting effect on the derived correlations. If they are left out, then we broadly estimate that 30–50% of the variance in NIDDM rate is associated with lactose absorption. We predict that the actual figure might be higher if we could control adequately for degree of Westernization, age of subjects tested (all of the above are based on "adult" values, but clearly this is a source of variation), and extent of genetic admixture; it is unlikely to be lower unless there turns out to be an unexpectedly high correlation in the opposite direction in some populations.

These data strongly indicate a relationship between lactose absorption and NIDDM, even if it is perhaps best not to attach too much significance to any particular r value. Indeed, as indicated by the uniformly low rates of NIDDM in high-frequency lactose-absorbing populations (note the absence of populations in the upper right quadrant of fig. 1), it appears that the ability to absorb lactose confers some "protection" against developing NIDDM. From a physiological perspective, however, such a supposition would be incorrect. Factors important in the etiology of NIDDM have little or no influence on whether an individual continues to produce lactase in adulthood; the lactase enzyme itself does not have a role in the development of diabetes. Lerch et al. (1991) found no differences in the frequency of lactose malabsorption between a group of diabetic patients (including both IDDM and NIDDM) and the general population. Without knowledge of life-history data (both genetic and environmental), knowing the glucose tolerance status of an individual does not allow prediction of lactose absorption ability. Yet, as we have seen, at a population level the correlation between the two is reasonably strong.

This indicates that there may be a historical or evolutionary relationship between lactose and diabetes. The history of dairying in Europe begins with the influx of Indo-Europeans some 6,000–7,000 years ago (Renfrew 1987, Mallory 1989). Reindeer exploitation certainly occurred before this time (Jarman, Bailey, and Jarman 1982), and this may have involved milking in some way; however, no one has suggested that this was a critical factor in the evolution of lactose tolerance. The intensive use of milk in herding populations was one aspect of the "secondary-products revolution" associated with the development of agriculture (Sherratt 1981:275–82). Milk use is indicated archaeologically by iconographic evidence, changes in the shapes of pottery, and the presence of special hollow tubes that may have been inserted into the rectum or vagina of a cow to stimulate lactation and the milk ejection reflex.

A high rate of lactose absorption in a population is, of course, also evidence of a long history of dairying. Population genetics models (Aoki 1986, Feldman and Cavalli-Sforza 1989) of the evolution of lactose absorption (via a single dominant allele) indicate that the lactose absorption levels observed in contemporary northern European populations could have been achieved in 6,000 years. Aoki and Feldman/Cavalli-Sforza differ somewhat on the selection coefficient necessary to achieve these levels: Aoki says it is 5% (with an effective population size of 100), while Feldman and Cavalli-Sforza, using a somewhat more complex model, put the figure at closer to 10%. The two models agree that in order for lactose absorption to have reached contemporary levels, selection coefficients must have been higher than a few percent.

Ammerman and Cavalli-Sforza (1984) suggest that the spread of farming and genes associated with Indo-European expansion can be explained by gradual demic expansion—the "wave-of-advance" model. It is possible that biocultural selection for lactose use could have been very intensive at the "front" of the wave. As Sherratt (1981:287) points out, "As agricultural communities expanded . . . because of the uncertainty involved, in areas without alternative resources, hunting was not a viable economy. With a product like milk which could be continuously obtained and especially with the added mobility given by draught- and riding-animals, such areas could be exploited by pastoralism. . . . The evolution of lactose-tolerant populations removed an important brake." Given the added mobility that dairying confers, coupled with the likelihood that immigrating groups will be kin-structured, it is not surprising that high-frequency lactose-absorbing populations were established in northern Europe, far from the Fertile Crescent where milking probably first occurred on a large scale about 7,000 years ago (Sherratt 1981). In Feldman and Cavalli-Sforza's model, "there is an important effect of the initial frequency of the absorbing allele" (1989:169) in terms of determining how quickly high frequencies of lactose absorption can be achieved. Founding northern European populations probably had relatively high levels of lactose tolerance, and selection for it was intensified because of basic nutritional needs (including that for vitamin D, although see Robins 1991) in a relatively harsh and highly seasonal environment. It is possible that contemporary frequencies of the lactose-absorbing allele could have characterized northern European populations for several thousand years.

The thrifty genotype was thus placed in an environment characterized by regular access to lactose, but the European diet may have been otherwise unremarkable (relative to that of populations with similar kinds of agricultural and foraging practices) in terms of quality and quantity. The question is, then, could the regular (or semiregular) presence of lactose in the diet have led to or facilitated selection against the thrifty genotype or selection for the non-thrifty genotype? Studies of lactose metabolism in patients with NIDDM can shed light on this issue.

Although lactose is a disaccharide, in its metabolism it acts more like a monosaccharide than a complex carbohydrate. In a study of NIDDM patients, Ercan et al. (1993:1565) found that a 50-g lactose load after overnight fasting provokes a plasma glucose area response (an index incorporating the total response over the five-hour post-challenge period) 60% of that following ingestion

of 50 g of glucose and an insulin area response 85% of that seen in response to the glucose. In another study of NIDDM patients, Gannon et al. (1986) looked at serum insulin and plasma glucose responses to foods with lactose. As with the Ercan et al. results, lactose alone provoked a plasma glucose response lower than that seen with glucose. However, 50 g of lactose taken in the form of skim milk provoked an insulin response fivefold greater than would be expected on the basis of the increase in plasma glucose levels observed; indeed, the insulin response to milk was greater, although not significantly so, than that for ice cream (which contains glucose, sucrose, and lactose) or glucose alone. The insulin response to milk was significantly greater than that for lactose alone. Gannon et al. (1986:790; see also Nuttall et al. 1984) state that "animal protein is a potent insulin secretogogue in untreated Type 2 diabetic patients," which accounts for the increased insulin response to milk.

The studies of lactose metabolism of NIDDM patients are particularly relevant in that these are people who, in effect, have thrifty genotypes. Lactose consumption constitutes glucose loading, and milk in particular provokes a high insulin response. Although the etiology of NIDDM is not clear, the development of insulin resistance and diabetes in susceptible individuals could be exacerbated by either of these factors.

A scenario for the evolution of the non-thrifty genotype in European populations might be the following: In these high-latitude populations, access to simple sugars in the typical hunter-gatherer diet was very limited. With the influx of Indo-European agriculturalists and pastoralists, populations bioculturally adapted to the use of milk were established. At these higher latitudes, there was perhaps especially strong selection for lactose absorption ability to compensate for the loss of vitamin D in calcium uptake. This in turn introduced regular access to a simple sugar (in a form that promoted high insulin secretion) to a diet previously characterized by low simple-sugar intake. Diabetes became the "price" not of civilization but of the use of lactose. Given the great benefits afforded by lactose in childhood (i.e., preventing rickets) and as a nutritional supplement during the reproductive years (i.e., for pregnant women), it is likely that lactose absorption ability was the drive in this particular evolutionary sequence. Once the use of lactose became established, both culturally and genetically, individuals with the non-thrifty genotype (i.e., individuals not susceptible to NIDDM until postreproductive age) were selected for.

There are several ways in which this scenario may be falsified or tested:

1. *Chronology.* One of the weaknesses of the orthodox thrifty-genotype scenario is the amount of time of negative or relaxed selection necessary to eliminate the genotype in "feast" populations; without the highly debilitating effects of IDDM, the negative features are strongly associated with advanced age and may not have a strong reproductive impact. As we have seen, there is general agreement that there has been plenty of time for lactose absorption to evolve in the populations where it is found, and for European (or Indo-European) populations the amount of time available is somewhere on the order of 6,000 years. This time frame is the implicit one in the orthodox thrifty-genotype scenario; the faster the lactose absorption genotype spread throughout these populations, the more time available for negative selection against the thrifty genotype.

2. *Physiology.* Although it seems unlikely, if a direct physiological link (besides giving someone the ability to consume more simple sugars) were found between lactose absorption and the etiology of NIDDM, then this selection hypothesis would be unnecessary. In fact, the absence of the thrifty genotype in Europe would not have to be explained by "civilization" or any other factor but would be a direct result of the evolution of lactose absorption ability. A direct link between milk consumption and the development of IDDM has been controversially proposed but is not directly relevant here (Sheard 1993).

3. *Other dietary factors.* There may be other dietary factors in Europe responsible for the evolution of the non-thrifty genotype. We reiterate, however, that neither agricultural nor preagricultural European populations were *uniquely* exposed to rich nutritional environments relative to populations elsewhere that exhibit the thrifty genotype in contemporary settings.

4. *Other lactose-absorbing populations.* Supporting evidence for the hypothesis would be obtained if non-European lactose-absorbing populations also exhibited a non-thrifty genotype after being exposed to a Westernized diet. However, other cultural-historical and biological factors may have mediated for the maintenance of the thrifty genotype in these populations.

Another scenario is suggested by Crews and Gerber's (1994) discussion of the pleiotropic effects of the thrifty genotype. In this case, is it possible that the non-thrifty genotype is a pleiotropic effect of the lactose-absorbing allele? The continued production of lactase in adulthood is presumably governed by a regulatory gene inherited as an autosomal dominant allele. This constitutes a prime example of heterochrony, which is evolution occurring through changes "in the rate or timing of ancestral development patterns" (Shea 1989:70). More specifically, it is an example of neoteny, the retention of a juvenile feature in adulthood. With exposure to a Western diet, people with a thrifty genotype develop diabetes and obesity during early and mid-adulthood; people with the non-thrifty genotype are also at risk for developing these conditions but especially so with advanced age. The non-thrifty genotype, like lactose-absorbing ability, also arises from a developmental shift in which a metabolic pattern characterizing younger people is retained through all or much of adulthood. Given how little is known about the developmental genetic mechanisms underlying either of the two conditions, a direct relationship between the two may be hypothesized, but it is a hypothesis that will be very difficult to test at this time.

We take very seriously the notion of and the logic

behind the thrifty genotype. The human organism is "out of date," to use Richard Dawkins's (1982) evocative phrase (see also Eaton, Shostak, and Konner 1988)—selected for a low-density hunter-gatherer environment but living increasingly in a high-density urban one. The negative sequelae of the thrifty genotype with a Westernized diet are a manifestation of this more general phenomenon. At the same time, the currently accepted explanation for the evolution of the non-thrifty genotype seems inadequate. Superabundance has not been a feature of the European diet for very long, and we suggest that a particular dietary factor, lactose, may have been important. If it turns out that other populations, upon the adoption of a Western diet, seem to possess the characteristics of the non-thrifty genotype, then we should look for local and particularistic explanations for this derived feature of human metabolism.

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Birth Non-Seasonality on the Pacific Equator[1]

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Seasonality is a very significant source of human biological variation, including variation in fertility. Birth seasonality requires adequate explanation because, compared with other temporal variations in fertility, the degree of consistent change can be very large (Lam and Miron 1991), providing substantial opportunities for microevolutionary, biological, ecological, and sociocultural impacts. An abundance of studies demonstrating birth seasonality in humans has led to the general conclusion that the phenomenon is an "almost universal" (Madrigal 1993), "virtually universal" (Becker, Chowdhury, and Leridon 1986), or universal (Huntington 1938; James 1971; Lam and Miron 1991:77) feature of human demography. The assumption of universality makes evolutionary sense inasmuch as climatic (and food-source) seasonality was a basic component of the ecosystems in which hominids evolved (Foley 1993) and a vital facet of human ecologies before, during, and after the transition to agriculture (Bailey et al. 1992). Certainly, accounts of nonseasonal birth patterns are very rare in the literature. Our review, summarized in table 1, located between four and six cases (depending on how seasonality is defined)

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