The Nutritional Significance of Regulation of Intestinal Nutrient Transport

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I. Introduction

A two-way relationship exists between the nervous system and the gut that affects the digestion of food and the organization of behavior. The integration and regulation of the various steps of the digestive process are achieved, in part, through the action of intrinsic nerve plexuses, which are modulated by the central nervous system, and a variety of regulatory substances, mainly peptides, which function as neurotransmitters or neuromodulators. In turn, the digestive process potentially affects the nervous system through its effect on the plasma concentration of nutrients which affect particular brain neurotransmitters. Whatever part intestinal absorption plays in this two-way interaction between gut and brain is largely unknown for at least two reasons. First, historically, research on intestinal absorption has concentrated on the mechanisms by which nutrients are absorbed and less so on the regulation of these mechanisms. Second, the entire digestive-absorptive process involves many steps and we are generally unable to identify which steps are most important under various conditions in affecting end results such as the postprandial time course of a plasma nutrient concentration.

Our knowledge and interpretation of regulation of intestinal nutrient absorption has increased markedly in recent years, and in this chapter I will discuss this regulation within the context of nutrition. I will first consider some examples of regulation of nutrient absorption in relation to factors such as food consumption and diet composition, focusing on the mechanistic bases for the regulation. This will be followed by discussion of whether regulation of nutrient absorption actually has nutritional or physiological
significance. The question of reciprocal interactions between intestinal absorption and the nervous system will be taken up in the final section, which will discuss the role that nerves and hormones might play in the regulatory process, and the ways in which nutrient absorption might mediate some of the interactions between diet and behavior. Primary emphasis is placed on the carrier-mediated transport of amino acid and monosaccharide constituents of protein and carbohydrate, because these nutrients, along with choline and lecithin, are known to affect particular brain neurotransmitters (Wurtman, 1982–1983). Choline uptake occurs by passive and carrier-mediated pathways, as do monosaccharide and amino acid uptake, but there is a dearth of information on regulation of its transport (though see Sheard and Zeisel, 1984; Karasov et al., 1987b).

II. NUTRITION AND THE REGULATION OF INTESTINAL NUTRIENT ABSORPTION

The digestive tract responds to changes in at least three kinds of nutritional variables: the quantity of food eaten per day, the qualitative aspects of the food, and the nutritional or physiological state of the animal. For the step of digestion involving enzymatic breakdown of food, examples of responses to possibly all three situations include the increase in pancreatic amylase concentration in rats fed high carbohydrate rations and the decrease in rats that are fasted (Solomon, 1981).

There is increasing evidence that the absorptive step of the digestive process is also involved in the gut’s adaptation to variation in these nutritional variables. The mechanisms involved in the adaptation of nutrient absorption include nonspecific mechanisms such as changes in absorptive surface area, which likely affect absorption of all nutrients, and also more specific responses as in the induction and repression of a specific transport mechanism. The following sections present examples of adaptation of intestinal nutrient absorption to changes in the nutritional variables, and the likely mechanisms involved.

A. VARIATION IN THE AMOUNT OF FOOD EATEN

Hyperphagia (increased food intake) occurs under a variety of conditions. One of the best-studied situations with respect to nutrient absorption is the increased food intake during lactation. For example, in laboratory mice food intake increases as much as four times during lactation (Studier, 1979). In spite of the much greater nutrient flux through the digestive tract, lactating mice are able to extract a similar proportion of energy from the food as nonreproductive mice (Studier, 1979). Lactating mice absorb glucose and proline faster than nonreproductive mice (Fig. 1). Studies with rats and hamsters have yielded similar results (Cripps and Williams, 1975; Pénzes and Simon, 1968), with some exceptions (Craft, 1970). Absorption of trace elements such as Zn, Cu, and Fe also increases (Davies and Williams, 1977; Linder and Munro, 1973).

The main basis for the nonspecific increase in absorption rate in lactating mice appears to be an increase in absorptive surface area reflected in an increase in mass per centimeter length intestine (Fig. 1). In rats, villus height and total villus area per centimeter length increases in lactating animals (Foll, 1972).

Another situation in which hyperphagia occurs and for which the same kind of nonspecific increase in absorption rate may occur is the increased food intake in animals exposed to cold temperatures. Rats and voles maintained at low temperatures exhibit...
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Fig. 1. Measures of amount of intestinal tissue and nutrient transport rate in mice in two situations: the nonspecific adaptation to lactation (figures on the left) and the specific adaptation to high dietary carbohydrate (figures on the right). Figures show intestinal mass per centimeter intestine (top figures), carrier-mediated D-glucose uptake per centimeter (middle), and total L-proline uptake per centimeter (bottom) at three or five intestinal positions. The transport measurements are unidirectional uptake rates close to Vmax across the brush border of intestinal alveoli measured in vivo as described in Karasov et al. (1983). Note that in the example of nonspecific adaptation the mass of intestinal tissue per centimeter is significantly increased as well as transport of both glucose and proline. In contrast, in the example of specific adaptation, glucose transport is significantly enhanced without any significant change in intestinal mass or proline transport. Tests for significant differences were by analysis of variance. N.S. means not significant at the p < 0.05 level. The results for mice fed chow and meat are from Karasov et al. (1983).
an intestinal hyperplasia (Jacobs et al., 1975; Gross et al., 1985), and enhanced glucose absorption was noted in the rats.

In these examples hyperplasia occurs in response to increases in the demand for energy and possibly all nutrients (in the case of lactation). Many animals can also be made hyperplagie through dilution of the calories in their rations with cellulose-type nonnutritive filler or kaolin. It is not clear what effect this hyperplagia has on intestinal structure and nutrient absorption. In rats, calorie dilution with kaolin has been variously reported to have no effect on (Dowling et al., 1967) or to cause enlargement of (Kennedy and McCance, 1958) the small intestine. In voles, calorie dilution with cellulose had no effect on small-intestine length or dry mass (Gross et al., 1985). Intestinal glucose absorption by rats has been reported to increase when the ration was diluted with kaolin (Dowling et al., 1967) and decrease when the ration was supplemented with cellulose (Schwartz and Levine, 1980).

An important distinction between hyperplasia during lactation or cold stress and hyperplagia in response to diet dilution is that the rate at which absorbable nutrients flow through the digestive tract is increased only in the former situations. Because retention time of digested products in the small intestine does not necessarily decrease when the ration is diluted (e.g., Dowling et al., 1967; Savory and Gentle, 1976), there may be no physiological advantage to increased glucose or amino acid absorption rate in this situation. In order to rationalize the apparently conflicting results on hyperplagia in response to diet dilution, we need additional studies on the relationship between intake rate and retention time of digested products in the small intestine, and possible direct interactions between the type of nonnutritive filler and the intestinal mucosa.

B. VARIATION IN DIET MAKEUP

Intestinal transport of sugar and amino acids varies with ration carbohydrate or protein content, respectively. This type of specific adaptation is illustrated for glucose in Fig. 1. Laboratory mice fed chow (50% carbohydrate) exceeded mice fed meat (0% carbohydrate) in carrier-mediated D-glucose uptake. This change in glucose transport was not paralleled by changes in the amount of intestine nor by changes in transport of the amino acid L-proline, in contrast to the nonspecific adaptation of glucose transport seen during lactation. In vitro (Ginsburg and Heggeness, 1968; Hahn and Koldowsky, 1966) and in vivo (Bode et al., 1981; Diamond and Karasov, 1984; Wolfbrandt and Scharrer, 1984) studies with rats and mice fed other rations have yielded similar results and indicate that monosaccharide absorption increases monotonically with dietary carbohydrate content.

For rations with dietary protein content above the maintenance level, there is a similar increasing relation between transport of amino acids and dietary protein level in rats and mice. When the casein content of synthetic rations is increased, the uptake rate of dispensable and indispensable amino acids is enhanced both in vitro (Lis et al., 1972; Sugiyma et al., 1983) and in vivo (Scharrer et al., 1982; Karasov et al., 1983, 1985, 1987).

The simplest mechanistic explanation for the specific adaptation of monosaccharide transport is induction or repression of the number of glucose transport sites per enterocyte. The kinetic basis for the change in transport is a change in V_{MAX} (considered to reflect carrier numbers), with little change in apparent K_{M} (considered to reflect carrier affinity for substrate) (Diamond and Karasov, 1984). Phlorizin binding measured of enteroc transport c hyperglyc transport is (1984) and 1986).

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measurements have indicated that the number of transport sites at the brush border of enterocytes is increased in mice fed a high carbohydrate ration (Ferraris and Diamond, 1986). Recent studies with membrane vesicles have shown that in rats glucose transport can also be induced at the basolateral membrane by a different stimulus—hyperglycemia (Maenz and Cheeseman, 1986). In both cases induction of glucose transport is fairly rapid—within 24 h at the brush border in mice (Diamond and Karasov, 1984) and within 5 h at the basolateral membrane in rats (Maenz and Cheeseman, 1986).

The functional significance of enhanced monosaccharide or amino acid uptake on rations with high levels of carbohydrate or protein, respectively, has been explained as follows (Karasov and Diamond, 1983, 1987). Intestinal tissue and specific transport systems represent an ongoing biosynthetic expense that is justified only by a payoff in calories or in availability of an indispensable nutrient. Carbohydrate and protein at high dietary levels are a readily available source of calories. Omnivorous species potentially faced with fluctuation in dietary carbohydrate or protein have evolved mechanisms to suppress the biosynthetic machinery when the payoff drops and to mobilize it when the payoff increases.

C. VARIATION IN NUTRITIONAL OR PHYSIOLOGICAL CONDITION

When animals are deficient in iron or calcium, the intestinal absorption rate for these nutrients is increased (Linder and Munro, 1977; Wasserman and Taylor, 1976). For iron, repression of iron absorption protects against iron toxicity when body stores are not depleted but when eating excess iron.

Monosaccharide and amino acid absorption have also been found to vary with animals' nutritional or physiological condition. Numerous studies have demonstrated increased in vitro glucose or amino acid absorption in diabetic animals, primarily in alloxan- or streptozotocin-diabetic rats (for review see Karasov and Diamond, 1983). The aforementioned changes in absorption in lactating animals might be considered another example.

One might wonder whether amino acids share with iron and calcium the characteristic of being indispensable nutrients whose intestinal absorption depends on the body's nutrient balance. The evidence for this is equivocal. Low protein rations have been variously reported to increase, decrease, or not to change the absorption of amino acids (see Karasov et al., 1987b), and references therein). Results may be so mixed because of different authors' choices of different bases of normalization, lack of discrimination between amino-nitrogen deficiency and deficiency for particular indispensable amino acids, and the complexities of working on transport of amino acids for which there exist at least six carrier mechanisms of somewhat overlapping specificities for the 20 or so amino acids (Munck, 1981; Karasov et al., 1987a).

A recent study compared uptake of several dispensable and indispensable amino acids (whose carrier specificities had been partly established) in mice fed either a maintenance ration or a ration deficient in total nitrogen but not in any particular indispensable amino acid (Karasov et al., 1987b). A noteworthy result was that uptake was depressed at low nitrogen for the dispensable amino acids aspartate and proline, each of which is transported on the "semiprivate" carriers for either acidic or imino acids. In contrast, the uptake rate was unchanged or increased for the indispensable amino acids methionine, leucine, lysine, and histidine, and the dispensable amino acid
alanine, all of which are transported on at least one of the more "public" carriers for neutral and basic amino acids. These data suggest that the pattern of regulation of amino acid carriers may relate to the physiological roles in metabolism of the primary substrates of the carriers (Karasov et al., 1987b).

III. PHYSIOLOGICAL SIGNIFICANCE OF REGULATION OF INTESTINAL NUTRIENT TRANSPORT

While it is clear that rates of nutrient absorption by the small intestine vary in response to many factors, are these changes really adaptive? Objections to the view that adaptations as discussed here are physiologically significant include the following: (1) there may be plenty of spare capacity to absorb nutrients under normal conditions; (2) a change in transport rate might be physiologically insignificant if transport is not the rate-limiting step in the digestive process; and (3) compensatory adjustments in some other steps of the digestive process might nullify the effect of the adaptation in transport (Snook, 1974). In this section I discuss evidence that addresses these objections and that suggests some ways in which regulation of nutrient absorption is important to digestive physiology and nutrition in general, and to interactions between the gut and the brain in particular.

A. THE INTESTINE'S ABSORPTIVE CAPACITY IS NOT INFINITE

Normally, dietary carbohydrate and protein that enter the small intestine from the stomach are >95% absorbed by the time that the intestinal contents reach the ileocecal valve. Evidence for this can be found in the natural gradients in nutrient concentrations down the gut and in experimental studies in which the fate of ingested protein or of glucose infused into the stomach is determined (Reynell and Spray, 1956; Chung et al., 1979). Studies with humans given oral fructose loads, and with resected rats, however, illustrate how the absorptive capacity of the small intestine can be temporarily exceeded when the ratio of solute load in the lumen to available transport capacity is increased.

In the first example, in a test of fructose absorptive capacity (Ruvich et al., 1983), 50 g fructose given in a 10% solution was incompletely absorbed in 38% of humans tested. Incomplete absorption was associated with symptoms of cramps or diarrhea, or both. If humans have the adaptive abilities of rats, however, their absorptive capacity for fructose can be increased. In rats, a high-fructose diet enhances fructose transport (Bode et al., 1981).

In rats, resections of 25% of the proximal intestine are not followed by increased nitrogen excretion (Nygaard, 1966), perhaps reflecting some extra absorptive capacity in the normal situation. Resections of more than 25% of the proximal intestine, however, are associated with postoperative elevations of fecal nitrogen. Subsequently, mucosal hyperplasia and enhanced uptake per centimeter compensate for the loss of intestinal length, and in the adapted resected rat nutrient absorption is again nearly complete by the time digested products reach the ileocecal valve (Reynell and Spray, 1956).

If the body's normal absorptive capacity does not constitute an infinite reserve, then adaptation of nutrient absorption likely plays an important role in the brain's regulation of food intake. Insofar as gut distension has an inhibitory effect on food intake [either directly or indirectly through a negative feedback on stomach emptying (Van Itallie and Kissileff, 1985, and references therein)], nutrient absorption must occur at the proper rate re further ingest lactating or col rate, which we At the very le increased foo

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proper rate relative to stomach emptying, and to completeness, in order not to inhibit further ingestion of food. If nutrients are absorbed more rapidly from the gut of a lactating or cold-stressed animal, one might expect the stomach to increase its emptying rate, which would lead to an increased food intake (Van Itallie and Kissileff, 1985). At the very least, higher nutrient absorption rates would seem to be a requisite for increased food intake to occur.

II. THE ABSORPTIVE STEP IN RELATION TO OTHER STEPS OF THE DIGESTIVE PROCESS

It is a reasonable supposition that regulation of nutrient absorption will be physiologically significant only when it is the rate-limiting step in the digestive process, and only if compensatory adjustments in some other steps do not nullify the effect of an adaptation in transport. While we can identify some situations where transport does appear to be the rate-limiting step, this supposition also appears to be overly simple when considering a complex, multistep process such as digestion. In humans, absorption of glucose from either sucrose or maltose occurs at the same rate as from the equivalent monosaccharide mixture (Gray and Ingelfinger, 1965; Gray and Santiago, 1966), and glucose molecules derived from maltose actually accumulate intraluminally. These findings have been taken as evidence that absorption, not hydrolysis, is the rate-limiting step in the digestion and absorption of sucrose and maltose by humans. Similarly, when readily soluble and digestible protein is fed to animals, the rate-limiting step in protein digestion and absorption may be amino acid transport (Snoek, 1974). Alternatively, enzymatic digestion is considered the rate-limiting step in the digestion and absorption of more complex proteins and starch.

The step of the digestive process that is rate-limiting varies with the makeup of the food and probably other factors. It is not surprising, then, that adaptation often occurs in several steps of the digestive process simultaneously, rather than occurring in only the single step that is immediately rate-limiting. Thus, even though starch and sucrose digestion and absorption may be rate-limited by different processes (hydrolysis in the first case, transport in the second), high-starch and high-sucrose meals induce both carbohydrate-splitting enzymes (Solomon, 1981; Koldovsky et al., 1982) and intestinal monosaccharide uptake.

These issues are pertinent to a consideration of the relationship between nutrients in foods and their effect on plasma nutrient concentration and, in some cases, neurotransmitter levels. Dietary carbohydrate, for example, influences plasma glucose level and insulin secretion, and the latter indirectly affects the movement of plasma tryptophan into the brain where it is synthesized into the neurotransmitter serotonin (Wurtman et al., 1981). Thus, factors that affect the time course of plasma glucose concentration potentially affect neurotransmitter levels. Glucose tolerance has been observed to improve after both acute and chronic ingestion of specific types of fiber, but different mechanisms may be involved. In the acute situation, fiber may alter glucose homeostasis by delaying gastric emptying. In the chronic situation, the effect of fiber may be to decrease the rate of glucose absorption in the small intestine (Schwartz and Levine, 1980, and references therein). If dietary fiber has an effect on brain neurotransmitter levels, determination of the role of changes in intestinal absorption may require an analysis of the entire digestive process under varying conditions.
IV. INTESTINAL NUTRIENT ABSORPTION AND INTERACTIONS BETWEEN THE GUT AND THE NERVOUS SYSTEM

The discussion so far has concentrated on evidence that regulation of intestinal nutrient absorption occurs, and has emphasized the likely nutritional or physiological significance of this regulation. This section now considers some of the reciprocal relationships between nutrient absorption and the nervous system. They are represented by the nervous system's role as a proximate mediator of regulation of nutrient absorption, and the role of absorption as a mediator in the effect of diet on behavior. In both cases the extent and depth of our understanding is limited.

A. NERVES AND HORMONES IN THE REGULATION OF INTESTINAL NUTRIENT ABSORPTION

A basic question in the regulation of intestinal nutrient absorption is whether the transported substrates themselves serve as the regulatory signals, or whether there are intermediate hormonal or nervous signals. Evidence that nervous or hormonal signals are involved comes from studies in which changes in mucosa structure or function are observed in the absence of direct contact with luminal nutrients (Dworkin et al., 1976). Both lactation and bowel resection are associated with intestinal hyperplasia and enhanced absorption of monosaccharides, amino acids, and minerals (see above and Garrido et al., 1979; Menge et al., 1982; Urban and Campbell, 1984; Urban and Pena, 1974), but this might be a direct trophic effect of increased luminal nutrients on mucosal mass. Suggestive of a humoral or neural agent, however, is the observation of hyperplasia and/or enhanced glucose transport in Thiry-Yella fistulas of lactating rats (Elias and Dowling, 1974) or of rats with intestinal resection (Hanson et al., 1977).

A humoral or neural signal is also suggested by Debnam's (1985) finding that glucose infused into the distal ileum of rats was followed within 3 h by an increase in in vivo monosaccharide absorption in jejenum proximal to the infusion site.

While there is evidence for a humoral or neural signal in regulation of intestinal transport, in most cases the exact physiological signal is unknown. The list of candidates for the humoral effects includes gastrin, glucagon, enterogluconag, cholecystokinin, secretin, insulin, prolactin, somatostatin, prostaglandin E₂, and androgenic and estrogenic steroids (for details see Karasov and Diamond, 1983, 1987). The effect of chronic glucagon administration for 2 or 3 days on nutrient transport has been studied in several laboratories and has been found, with some exceptions (Lorenz-Meyer et al., 1977), to result in increased glucose transport (Rudo and Rosenberg, 1973; Debnam, 1982; Csupo and Lieske, 1976). Glucagon administration decreases villus height and so, if it does have a direct effect on transport, perhaps it is on some other characteristic of the mucosa such as the membrane potential difference (Debnam and Thompson, 1985).

Somatostatin, which is released during meals, inhibits the postprandial appearance of several nutrients in the blood. It has been proposed that it could be a physiological regulator of the rate at which ingested nutrients enter the circulation by delaying intestinal absorption (Wahren and Felg, 1976). In vivo experiments in rats, dogs, and humans suggested that absorption rates for glucose and amino acids are inhibited following somatostatin administration (Krejs et al., 1980; Ross and Shaffer, 1983), but other in vivo and in vitro studies in rats showed no effect (Daumerie and Henquin, 1982; Wilson et al. and extend concentration).

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Hen biological nutrient transport between the blood and the brain has been studied extensively (see above, 1984; Urban and Maram 1984). However, the observation of lactating rats and the fact that glucose is not a significant nutrient for newborns (Lorenza and Rosenberg, 1984) raises questions about its role in nutrient transport. The effect of lactation on nutrient transport in the brain has been studied by Dehnam and colleagues (1984), who found that the appearance of nutrients in the brain following intestinal feeding is not significantly different from that in humans (Wein, 1982; Tappin, 1983).

TAPPIN et al. (1983) have shown that the transport of nutrients across the blood-brain barrier can be applied at the level of the intestinal nutrient absorption also. The transport mechanism that mediates the nutrient transport from the lumen into the brain must be unsaturated with its substrate so that it can become more nearly saturated when the lumen concentration rises.

What are the lumen concentrations of amino acids following a meal, and how do they compare with the apparent affinity constants (K_m) for intestinal amino acid transport? Surprisingly, few measurements have been made of lumen nutrient concentrations following meals. In humans, digestion of a mixed test meal containing 59 g purified bovine serum albumin for 2 h resulted in jejunal luminal free amino acid concentrations ranging from 0 mM (for proline) to 12 mM (isoleucine) and averaging 3.6 mM (Chung et al., 1979). (Parenthetically, 2.5 times as much amino acid was in the form of peptides, which can also be transported, as free amino acids.) The K_m values for alpha-amino acid uptake across the brush border by rabbit intestinal tissue in vitro range from 1.4 mM to 24.8 mM, averaging around 10 mM (Munck, 1981), and are higher in humans in vivo (Munck, 1981), probably, in part, because of unstirred layer effects (Winne, 1976). Thus, carriers would appear not to be saturated. A complicating factor, however, is that each carrier transports several amino acids, each of which can inhibit the others’ uptake, and most amino acids are transported by several carriers. The most realistic analysis of the situation would therefore require for each amino acid the determination of its apparent affinity constant, apparent inhibitor...
constant, and maximal transport rate for each of the six or more amino acid carriers. If carriers were saturated, amino acids would still be absorbed by concentration-dependent passive influx. This fact, plus the relatively low concentrations of amino acids in the gut compared with the apparent affinities of the carriers, makes it reasonable that when meal protein content varies, absorption rates of amino acids will also vary and affect amino acid concentrations in the hepatic portal vein.

The adaptation of amino acid transport to dietary protein is also likely to be permissive for the direct relationship between dietary protein content and plasma amino acid concentration. In laboratory mice fed high-protein rations, the transport of all four major classes of amino acids (neutral, basic, acidic, and imino) is enhanced (Karasov et al., 1987b). This adaptation to high dietary protein occurs within 24 h (Karasov et al., 1983). Thus, in laboratory rodents, at least, even if amino acid carriers were saturated such that absorption rate could not rise when plasma amino acid concentration rose following a high-protein meal, absorption rates would be more rapid for subsequent meals after adaptation had occurred.

A poorly understood aspect of the adaptation of amino acid uptake in mice to high diet protein was that the enhancement of uptake by carriers for neutral and basic amino acids appeared to be less than that for the acidic amino acid and the imino acid carriers (Karasov et al., 1987b). Tryptophan and tyrosine, precursors for neurotransmitters (amines), are transported by the former mechanisms (Karasov et al., 1986), whereas the acidic amino acid carrier transports glutamic and aspartic acids whose metabolites are thought to be neurotransmitters. Could the differential pattern of adaptation of amino acid transport in any way relate to the roles that these amino acids play in the production of neurotransmitters?

Other intriguing questions are raised by the changes in glucose and amino acid transport in diabetes. Recall that diabetes is associated with increased rates of intestinal glucose and amino acid absorption. The changes in absorption rates, and the changes in plasma sugar and amino acid concentrations possibly associated with them, could have important effects on rates of neurotransmitter synthesis in diabetics.

V. SUMMARY

Possible reciprocal relationships between the nervous system and intestinal nutrient absorption have been poorly defined because the regulation of nutrient absorption was for many years a neglected area, and absorption is just one of several possibly rate-limiting steps in the chain of events leading from ingestion of nutrients in a meal to the production of neurotransmitters. Nutrient absorption is regulated in response to changes in nutritional factors such as amount of food eaten, nutrient makeup of the food, and the nutritional or physiological condition of the animal. Mechanisms of regulation include nonspecific mechanisms such as changes in absorptive surface area that affect the absorption of many nutrients and specific mechanisms that affect the absorption of only one nutrient. These two types of mechanisms are exemplified in the adaptations of mice to lactation and to high-carbohydrate diets, respectively. While there is evidence for a hormonal or neural signal in the regulation of sugar and amino acid transport, there are no instances where the signal has been well identified. The changes or adaptations of intestinal sugar and amino acid transport are significant for their effects on both the rate and completeness of nutrient absorption.
no acid carriers, y concentration-rat- ions of amino acids takes it reasonable- ly long for the small intestine to be permissive. The amino acid transport system is multifaceted and complex. The concentration of amino acids in the small intestine is high, and the transport systems are highly efficient. The presence of specific transporters for amino acids allows for selective uptake and utilization of these nutrients. The regulation of amino acid transport is influenced by various factors, including hormonal signals and nutrient availability. The transport systems are sensitive to changes in the environment and can adapt to meet the body's needs.


REGULATION OF NUTRIENT TRANSPORT


