samples came from, and it is still open to question whether much of the volume and of the protein was not of adventitious origin.

All that one can say in conclusion is that the form in which the products of protein digestion enter the body is still not established. No one has been able to to show that more than a minority of it enters the blood stream as amino acids. It seems unlikely that more than a very little passes in through the lymphatics as amino acids. It is certainly not established that the amount of protein that can be secreted into the lymphatics by the intestinal mucosa is sufficient to make up the difference. But enough work has not yet been done to resolve the question. The crucial experiments in this field are very difficult to design and execute. But they are still worth doing.

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Recent studies on carbohydrate absorption

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In recent years, studies on the intestinal absorption of carbohydrates have tended to fall into one of two categories, (a) the mechanism of active sugar transport and (b) the absorption of disaccharides. In the first it is necessary to consider sugar transport in the intestine as a phenomenon closely linked with electrolyte and water absorption on the one hand and on the other with active transport processes in general. As these aspects are discussed in other contributions to this Symposium, this review is concerned with disaccharide absorption.

In the last decade, by a remarkable 'cross-fertilization' of interests, biochemists, nutritionists, physiologists, histologists and clinicians have combined to elucidate the localization and mechanism of disaccharide absorption and to explain in bio-chemical terms the clinical findings concerning human disaccharide intolerance.

As early as 1901, Waymouth Reid suggested that maltose may be absorbed intact at appreciable rates from the lumen of the small intestine (Reid, 1901). Starling (1906) drew attention to the fact that the hydrolytic activity of the intestinal contents

during digestion was too low to account for the digestion of disaccharides. Cajori (1933) similarly observed that the rate of absorption of sucrose and of lactose by the jejunum and ileum of dogs seemed faster than expected if prior hydrolysis to monosaccharides in the lumen was required. None of these observations received attention and textbooks of biochemistry and physiology continued to present the view that hydrolyses of disaccharides are catalyzed by enzymes present in the 'succus entericus' and that the monosaccharides produced are then absorbed from the luminal contents by the intestinal villi.

Localization of disaccharidase function

In 1957 Borgström and co-workers again drew attention to the low activity of the disaccharidases in the intestinal content and suggested that these enzymes might be located in the mucosal cells (Borgström, Dahlqvist, Lundh & Sjövell, 1957). Using ^{14}C-labelled starch hydrolysate and sucrose, Chain, Mansford & Pocchiari (1960) found a more rapid increase in the fructose content of the serosal fluid of a preparation of rat small intestine during sucrose absorption than could be explained by the lumen concentration of fructose. Similarly, serosal glucose increased more rapidly during luminal perfusion of starch hydrolysate than could be explained by the actual free glucose content of the starch hydrolysate. These results suggested that the sucrose and maltose were taken up intact and hydrolysed intracellularly. Dahlqvist & Borgström (1961) then used human subjects, giving them test meals containing a non-absorbable marker and sucrose, maltose, lactose or starch. The limited hydrolysis of disaccharides in the intestinal contents and the extremely low glycosidase activities relative to the calculated amount of disaccharide absorbed suggested a direct uptake followed by intracellular hydrolysis.

A series of ingenious experiments was then carried out by Crane and his co-workers. Miller & Crane (1961a) observed that when sucrose was incubated with everted sacs of hamster intestine, the concentration of glucose, resulting from sucrose hydrolysis, was from ten to thirty times greater in the tissue than in the medium, suggesting hydrolysis of the sucrose primarily in the tissue. Inhibition of glucose transport by dinitrocresol failed to affect the accumulation of glucose in the cells when sucrose was in the medium. Further support for the intracellular hydrolysis of disaccharides came from a study in vitro in which glucose oxidase was present in the medium. In this instance any glucose liberated from the disaccharide, maltose, in the lumen would promptly have been oxidized by the glucose oxidase. However, here again tissue accumulation of glucose took place.

Following up these observations, Miller & Crane (1961b) were able to isolate the epithelial brush border membrane as a morphologically distinct entity from homogenates of intestinal mucosa and to show that virtually all the invertase and maltase activities of the homogenate was contained in the brush border fraction. Dahlqvist & Brun (1962), on the basis of histochemical studies, suggested that some invertase may also be present in certain cytoplasmic granules within the epithelial cell but, with an improved technique, this finding has been shown to be an artifact and the disaccharidase activity has been demonstrated histochemically in the brush border

region. The distribution of the disaccharidase activity in the different parts of the villi also has been studied and it has been found that the activity is highest in the apical half of the villus while there is essentially no activity in the crypts.

Following this demonstration by Miller & Crane, that the maltase activity in the epithelial cell is located in the brush border and is on the luminal side of the glucose transfer mechanism, Newey, Sanford & Smyth (1963) carried out further experiments using everted sacs of rat intestine. They found that glucose and maltose had similar effects on fluid transfer, and with both the fluid transfer was greater when the sugar was initially present in the mucosal fluid than when initially present in the serosal fluid. Phlorrhizin inhibited the stimulation of fluid transfer when either glucose or maltose was initially present in the mucosal fluid and when maltose was initially present in the serosal fluid but not when glucose was initially present in the serosal fluid. These observations could be explained by postulating locations for each cellular function or 'functional topography' as the authors termed it. In this hypothesis the epithelial cell contains three zones arranged in order from the luminal side, a zone of maltase activity lying external to a phlorrhizin-sensitive glucose transfer mechanism which in turn leads to a fluid transfer mechanism which is dependent on the metabolism of glucose. This model has recently been further elaborated (Barry, Smyth & Wright, 1965) to take account of the relation between hexose transfer and the sodium pump (Crane, Miller & Bihler, 1961; Schultz & Zalusky, 1964).

Disaccharidase activity along the intestinal tract

In their original studies in man Dahlqvist & Borgström (1961) suggested that sucrose was absorbed mainly in the distal jejunum and ileum whilst lactose and maltose were absorbed in the upper and mid jejunum. These suggestions were consistent with Dahlqvist's (1961) findings in the pig. More recently in the rat, however, Dahlqvist & Thomson (1963) have shown that most of the sucrose is absorbed in the upper third of the small intestine and Blair & Tuba (1963) have demonstrated sucrose activity mainly in the upper half of the small intestine. Mansford (1965) found higher sucrose absorption in vitro in human jejunum than in terminal ileum. Similarly, Gryboski, Thayer, Gryboski, Gabrielson & Spiro (1963) have observed that patients with gastrojejunostomies may show sucrose malabsorption. These findings suggest that the major site of sucrose absorption may be the upper jejunum.

Development of disaccharidase activity

Doell & Kretchmer (1962) showed that in both rat and rabbit the lactase activity increases late in gestation, reaches a maximum shortly after birth and then gradually decreases. Similar findings in the pig have been reported by Walker (1959). Blair, Yakimets & Tuba (1963) did not find sucrase activity in the rat intestine to increase significantly until the age of 3 months. Rubino, Zimbalatti & Auricchio (1964) found a complete absence of sucrase, isomaltase, and trehalase in suckling rats until the 15th day after birth. Various workers (Bailey, Kitts & Wood, 1956; Dollar,

Mitchell & Porter, 1957) have shown in the pig that maltase and sucrase activities are low at birth and take at least 10 days to reach high levels. All of the intestinal disaccharidases are already active in the 3-month-old human embryo (Auricchio, Semenza & Rubino, 1965). The α-disaccharidase activities reach the normal adult level between the 6th and 7th months of foetal life. The β-disaccharidases reach their maximum in man toward the end of foetal life (Auricchio, Rubino, Tosi, Semenza, Landolt, Kistler & Prader, 1963).

Specificity of disaccharidases

Parallel to the work on the localization of disaccharidases, a number of authors in Sweden (Dahlqvist, 1962a,b) and in Zurich (Auricchio, Dahlqvist & Semenza, 1963; Semenza & Auricchio, 1962) have carried out detailed investigations on the specificity of these enzymes. In order to obtain satisfactory results two obstacles had to be overcome: (1) the fact that in homogenates of intestinal mucosa the disaccharidases are not in a soluble form, and (2) the absence of a rapid and specific analytical procedure for glucose (i.e. overcoming the carbohydrase activity of commercial preparations of glucose oxidase (Crowne & Mansford, 1962)).

The first of these problems was partly overcome by using trypsin which was found to be effective in making disaccharidases soluble in the pig and rat. Human disaccharidases have been more recently found to be made soluble by 'autolysis' and papain and to be stabilized by potassium ion. The second difficulty was overcome by Dahlqvist (1964a), who developed a very satisfactory quantitative assay for the individual disaccharidases based on a glucose oxidase reagent made in tris buffer, thus overcoming previous difficulties in lack of specificity (Mansford & Opie, 1963).

Studies in both pig and man on the chromatographic separation of intestinal disaccharidases and of their specificity have revealed a complex pattern which may still be found to be incomplete (see Table 1). In man, five different intestinal maltases can be identified by inhibition and heat inactivation studies and by gel filtration chromatography. Maltase 1 and maltase 2 split only maltose, maltase 3 and maltase 4 split also sucrose, whilst maltase 5 splits also isomaltose and palatinose. There is some question whether maltase 4 and 5 are two individual molecules or

Table 1. *Specificity of human intestinal disaccharidases and their relative importance* (*Auricchio, Semenza & Rubino*, 1965)

Enzyme	Percentage of total activity against each substrate					
	Maltase	Sucrase	Trehalase	Isomaltase	Palatinase	Lactase
Maltases 1+2	15	0	—	trace	—	—
Maltase 3 (=sucrase 1)	5	10	—	—	—	—
Maltase 4 (=sucrase 2)	}80	90	—	—	—	—
Maltase 5 (=isomaltase)		—	—	100	100	—
Trehalase	—	—	100	—	—	—
Lactase 1*	—	—	—	—	—	85
Lactase 2†	—	—	—	—	—	15

*Lactase/cellobiase activity 5.
†Lactase/cellobiase activity 2.

Table 2.   *Disaccharidase activities in human jejunum mucosa (from Auricchio, Rubino, Tosi, Semenza, Landolt, Kistler & Prader,* 1963)

(Enzyme activities in units/g protein)

Enzyme	Activity
Maltase	593 (310–1120)
Sucrase	173 (70–325)
Isomaltase	159 (65–268)
Lactase	107 (39–258)
Cellobiase	14 (9–21)

one molecule containing two active sites. Of the five maltases, the greatest part of the total maltase activity is exhibited by the maltases 4 and 5. Gel filtration on Sephadex G 200 reveals two human intestinal lactases both of which exhibit cellobiase activity (Dahlqvist, 1964*b*; Semenza, Auricchio & Rubino, 1965; Semenza, Auricchio, Rubino, Prader & Welsh, 1965).

The normal values of the disaccharidase activities in adult jejunum obtained by biopsy are given in Table 2. The absolute activities show considerable variation but, apart from the lactase activity, the ratios between the various disaccharidases are reasonably constant, i.e. total maltase activity is three to four times the sucrase activity.

The pattern of specificity is of great importance because it has become increasingly evident that a number of specific congenital and hereditary diseases exist in which disaccharide intolerance occurs as a consequence of a deficiency or complete absence of one or more of these enzymes. The specificity studies described and shown in Table 1 enable certain predictions to be made concerning the probable nature and extent of these disaccharide intolerances. It is obvious that maltose intolerance should not occur, at least as a result of a single enzyme defect, since five maltases have been described. Sucrose intolerance may be expected to be accompanied by iso-maltose intolerance if the combined maltase 4 and 5 is absent. Lactose intolerance should be accompanied by cellobiose intolerance. These predictions are supported by the available clinical data (Dahlqvist & Brun, 1962).

To date, only two types of hereditary disaccharide intolerance have been reported, (a) hereditary sucrose intolerance and (b) hereditary lactose intolerance. It is necessary here to stress the distinction between these two congenital malabsorption syndromes and the acquired type of disaccharide malabsorption arising from (1) specific acquired lactose malabsorption and (2) the secondary non-specific disaccharide absorption seen in patients with various intestinal disorders.

The most prominent symptom of disaccharide malabsorption is diarrhoea caused partly by bacterial degradation of the unabsorbed disaccharide and partly by osmotic effects. The stools contain large amounts of lactic acid and of the disaccharide; confirmation of the diagnosis can be achieved by oral loading tests with the suspected disaccharide and by the direct assay of the disaccharidase activities in a peroral suction biopsy of the intestinal mucosa (Weijers, van de Kamer, Dicke & Ijsseling, 1961).

Congenital malabsorption of sucrose and isomaltose

Over sixty human cases of deficiency of intestinal sucrose are documented (Auricchio, Dahlqvist, Mürset & Prader, 1963). In all of these a concomitant iso-maltose intolerance has been shown although the intestinal isomaltase activity is seldom reduced as much as the sucrase activity. Consequently only some of these patients exhibit starch malabsorption due to the isomaltose content (Burgess, Levin, Mahalanabis & Tonge, 1964).

Assay of the disaccharidase activities in biopsy specimens has revealed a complete absence of sucrase activity and a severe deficiency of isomaltase activity. Maltase activity is considerably reduced and is mainly of the maltase 1 and maltase 2 types. Lactase and trehalase activity is normal (Semenza, Auricchio, Rubino *et al.* 1965). These results indicate an apparent deficiency of maltase 4 and 5 with a possible deficiency of maltase 3, the activity of which is low even in the normal subject.

Congenital malabsorption of lactose

Two types of lactose intolerance are known—congenital and acquired. Malabsorp-tion of lactose in adults is not rare but to date has been of the acquired type. In the nine reported patients with congenital lactose intolerance, assay of intestinal disac-charidase activity has shown a severe deficiency of lactase activity and normal levels of the α-disaccharidases (Semenza, Auricchio, Rubino *et al.* 1965; Davidson, Sobel, Kugler & Prader, 1964).

Acquired lactose intolerance

In a study of disaccharidase activity in samples of small intestine obtained at surgical operation for duodenal ulcer, Auricchio, Rubino, Landolt *et al.* (1963), found a specific deficiency of lactase and cellobiase in three out of eighteen patients. All three patients had tolerated milk as infants and had developed milk intolerance later, thus ruling out the congenital type of lactose intolerance. Haemmerli, Kistler, Ammann, Marthaler, Semenza, Auricchio & Prader (1965) used oral loading tests and suction biopsy analysis to study intestinal lactase activity in adults with so-called 'milk allergy'. Nine patients with lactase deficiency were revealed in this group, all of whom had tolerated milk when young children. Twelve similar cases have been reported by Dunphy, Littman, Hammond, Forstner, Dahlqvist & Crane (1965).

These findings suggest that in human development there may be a regression of lactase activity such as Bailey *et al.* (1956) reported in pigs and Doell & Kretch-mer (1962) found in rats and rabbits.

'Secondary' disaccharidase deficiencies

It is not surprising that in many pathological conditions of the intestinal mucosa there is a reduction of the intestinal disaccharidase activity (Littman & Hammond, 1965). This has been shown in cases of infectious diarrhoea, kwashiorkor, coeliac disease, sprue, gastrectomy and intestinal resections. McMichael, Webb & Dawson

(1965) measured jejunal lactase activity in forty-four patients with various gastro-intestinal symptoms and found fourteen with lactase deficiency. In discussing these results they point out that the enzyme deficiency may frequently be asymptomatic.

In contrast to the selective deficiencies already discussed, the symptomatic disaccharidase deficiencies generally affect all the disaccharidases. Clinically lactose malabsorption frequently dominates, which is not surprising when the normal values of lactase, maltase and sucrase are compared. It is, however, of interest that, in coeliac disease and in sprue treated with gluten-free diet, the lactase activity may remain extremely low even after the other disaccharidase activities have returned to within the normal range.

The increasing awareness of the concomitant lowering of disaccharidase activity with these diseases of the intestine has recently provoked speculation (Paulley, 1963) of the possible effects of 'primary' disaccharidase deficit on predisposition to another disease. For example, could not the presence of unsplit lactose favour bacterial colonization of the proximal intestine and lead to disease? Is it not possible that continuous bathing of the intestinal mucosa with the fermentation products of unsplit disaccharides could favour subsequent inflammatory or ulcerative disease? To date there is little direct evidence bearing on these questions but continuation of the progress of the past few years will doubtless provide satisfactory answers to these and other questions.

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Absorption of fats

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The general principles of the digestion and absorption of triglyceride fat have been outlined during recent years and are summarized in Fig. 1. Intestinal digestion of fat involves a physico-chemical transformation of the emulsified non-polar triglyceride fat catalysed by pancreatic lipase in the presence of bile, to a mixed bile salt micellar solution including mainly the polar end-products of lipolysis, monoglycerides and fatty acids. The mixed micelles of bile salts, monoglycerides and fatty acids have a diameter of 40–80 Å, assuming that they are spherical, and they appear to be highly hydrated. Recent work seems to indicate that they are spherical with the bile salt attached to the periphery, its hydroxy groups bound to the hydroxy groups of the monoglycerides, the latter forming the central core of the micelle. The micellar form seems to be the physical form preferred for absorption of lipids even if the mucosa cell can accept lipids in other forms. For detailed discussions of the various phases of triglyceride digestion the reader is referred to original articles (Borgström, 1964, 1965; Laurent & Persson, 1966; Hofmann, 1963) and I will change over to another related subject, which has interested us during the last year, the mechanism of specificity in absorption of non-glyceride fat. It has long been known that other fats, usually present dissolved in the dietary fat, are absorbed to a varying degree. It is thus known that cholesterol is absorbed only to a limited extent and that plant sterols, which chemically are very similar to cholesterol, are almost completely non-absorbable.