Digestion and Absorption of Carbohydrates in Fowl and Events through Perinatal Development

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ABSTRACT Starch is the main carbohydrate in the food of poultry. Starch granules are digested by pancreatic α-amylase in the small intestine. Intestinal villi have enterocytes that project microvilli with a fibrous glycocalyx from the surface. These fine structures are envisaged to entrap water that is mixed with mucin from nearby goblet cells to form the "unstirred water layer." Maltose, maltotriose and α-limit dextrins must diffuse across this first barrier to absorption to be hydrolyzed by maltase and sucrase-isomaltase immobilized at the membrane; however, the resultant glucose, once formed, accrues at the surface to provide a concentration advantage. Fowl adjust to changes in dietary starch by altering the amount of amylase released, intestinal surface area and enterocyte carbohydrase concentration. Enterocytes arising during embryonic development have no carbohydrases and are not involved with glucose absorption, but they appear to be specialized for maternal immunoglobin transfer in ovo. Embryonic villi are stimulated by transfer activity, and their growth depends on enterocytes arising from the crypt. Mature crypt cells are capable of digestion-absorptive activities and dominate the villus shortly after the chick hatches when yolk sac reserves are depleted. J. Nutr. *115*: 665–674, 1985.

INDEXING KEY WORDS amylase • carbohydrate • chick • digestion • immunoglobulins • intestine • malatase • starch • sucrase-isomaltase

Plants provide an overwhelming amount of dietary carbohydrate for fowl, and starch is by far the major digestible form. Fowl have evolved to be particularly adept at coping with extremes in amount and sources of starch. Mammals are unable to utilize starch after birth, and they depend on lactose as the sole dietary carbohydrate until their digestive system matures. Fowl develop digestive capacity for starch while in ovo and are fully competent in this respect shortly after emergence from the shell. Little is known of the sequence of events in this development; however, considerable research has been done on the transition from immature to mature systems with the piglet. The present review will describe the mature system that is postulated to function in fowl, then speculate on in ovo development by using the piglet as a model.

THE MATURE SYSTEM

Intestinal anatomy. Villi protrude from the small intestinal wall greatly expanding the surface exposed to the luminal contents. Villi in fowl may vary in shape from finger- to leaflike and closely resemble those found in mammals (1). For the most part, enterocytes comprise the villus epithelium; however, only those cells located on the upper half are capable of digestive and absorptive activities. These "mature" enterocytes further increase luminal exposure by having microvilli at their apical surface. Contractile filaments within each microvillus may provide strength and some convective movement (2).

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The glycocalyx is a network of glycoprotein fibers that are superimposed on the microvilli (3). Fowl have been reported to have longer microvilli but a less dense glycocalyx than mammals (4, 5). Extensive peristaltic activity is a characteristic of intestinal motility with fowl; presumably, abrasion is responsible for the thinness of their glycocalyx.

Goblet cells also populate the villus epithelium. Motility aids the release of mucin, and its function has largely been associated with lubrication of bolus movement. More recently, mucin's contact with the intestinal wall is believed to play a central role in nutrient absorption (6, 7). The microvilli-glycocalyx composite is viewed as providing a "suprastructure" in which water is immobilized because of the viscosity from accompanying mucin. Nimmerfall and Rosenthaler (8) speculated that the rate at which molecules may move through this mucin-water complex will depend on their charge, hydration radius, ability to form hydrogen bonds and molecular weight. All but the simplest of saccharides are expected to have difficulty in passing through this unstirred water layer.

Enzymes finalizing the reduction of starch to glucose appear to be attached to the enterocyte membrane below the unstirred water layer. Both maltase and the sucrase-isomaltase complex have been established as being on the chick's jejunal mucosa (9). Kushak et al. (10) removed the glycocalyx from the small intestinal surface by placing liquid agar on the mucosa and then peeling the gel. Digestive enzyme activity remained almost exclusively with the "denuded" surface (table 1). Thus, saccharides restricted by the unstirred water layer would not have access to the carbohydrases.

Starch reduction. I (11) reviewed the structure of starch granules and the factors affecting their utilization by fowl. Essentially, amylose is a helix of glucose units having α-1,4 connections, whereas amylopectin has many amylose helices bound together in a bush-like fashion by α-1,6 linkages. Both polymers are packed together in an orderly manner to form the granule. The plant source determines the proportions of each polymer, nature of their crystallization, and granule size. These factors taken together determine the granule's resistance to digestion. Heat combined with moisture causes the granule to gelatinize and improves enzyme access to polymers for digestion.

Pancreatic α-amylase is the only enzyme produced by fowl that has amylolysis as its primary function. Avian and mammalian amylases are very similar in composition, mode of action and susceptibility to inhibitors (11). Basically, the enzyme attaches to the amylose helix, then sequentially cleaves maltose units until the nonreducing end is reached, then maltotriose arises. The α-limit dextrins occur when the helices in amylopectin are disassembled around the α-1,6 linkages that hold them together.

Amylase action on the starch granule is limited to its interface with water. Rate of digestion is related to granule surface area and occurs faster through amorphous areas than where crystallized. The greatest part of starch digestion usually takes place through

TABLE 1

Enzyme activities from the jejunum of the chick as found in the glycocalyx and mucosa after glycocalyx removal[1]

Enzyme	Substrate	Glycocalyx		Mucosa less glycocalyx	
		mmol/(g · min)	*% of total*	*mmol/(g · min)*	*% of total*
Alkaline phosphatase	*p*-Nitrophenyl-phosphate	0.67	7.9	7.79	92.1
Maltase	Maltose	6.58	16.5	33.45	83.5
Sucrase	Sucrose	0	0	8.60	100.0
Dipeptidase I	Glycyl-L-leucine	23.70	2.8	533.90	97.2
Dipeptidase II	L-Valyl-L-valine	26.70	3.7	688.00	96.3
Tripeptidase	Glycyl-L-leucyl-L-valine	2.41	4.1	53.19	95.7

[1]Reprinted with permission from Kushak et al. (10). Relationship of intrinsic enzymes of the apical glycocalyx and mucosa of the small intestine of chicks. Comp. Biochem. Physiol. *70A*, © 1981 Pergamon Press.

the upper jejunum because grains provide the bulk in feed, and their granules are labile. Tuber and legume starches are usually difficult to digest, and product release is distributed over the entire length of the small intestine. Extremely stable granules, such as those from potatoes, may pass through into the large intestine for microbial degradation if not gelatinized.

Maltose, maltotriose and α-limit dextrins are all soluble and may rapidly diffuse through the unstirred water layer (fig. 1). Once at the enterocyte membrane, each molecule is reduced to glucose by the appropriate carbohydrase. In his review of carbohydrate utilization by fowl, Levin (12) cited evidence that glucose derived from sucrose has a greater absorptive rate than if given in free form. This advantage is postulated to occur because glucose formed at the membrane is not readily "swept" into the luminal contents but is "held" there by the unstirred water layer to accrue near active transport sites.

The unstirred water layer is also envisaged to protect enzymes immobilized on the enterocyte surface from degradation by pancreatic proteases in the lumen. Alpers and Tedesco (13) observed that elastase is particularly effective in releasing disaccharidases from the rat's small intestinal surface. The mucin network is estimated to impede protease diffusion through to the surface in a manner that is inversely proportional to the square root of its molecular weight.

Crane (14) originally proposed the sodium gradient hypothesis to explain active absorption. Freel and Goldner (15) reviewed the research relating to active absorption

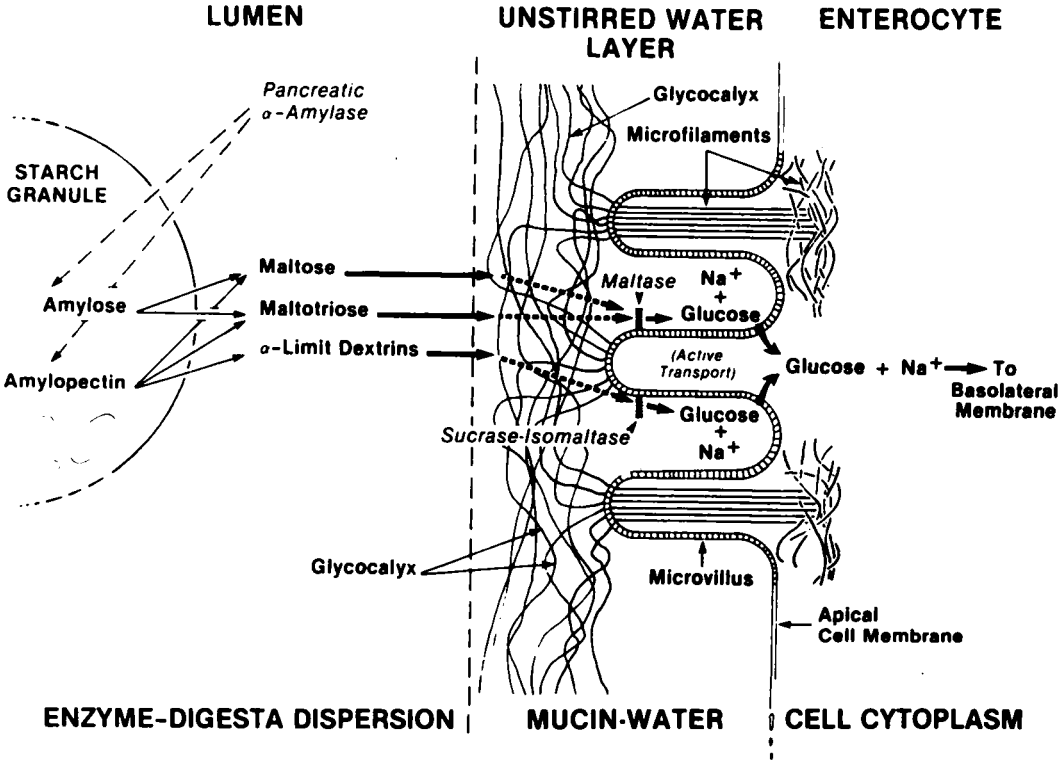

Fig. 1 Postulated sequence of events in the digestion of starch by fowl. Starch is normally in granular form and pancreatic α-amylase progressively hydrolyzes constituent amylose and amylopectin to maltose, maltotriose and α-limit dextrins. Enterocytes lining the small intestine project microvilli and a fibrous glycocalyx into the lumen. An aqueous dispersion of mucin from nearby goblet cells is immobilized in these structures to form the "unstirred water layer." Dissolved products from starch digestion must diffuse through this barrier to reach carbohydrases anchored on the surface in order to finalize digestion; however, glucose accrues near active transport sites, and its rate of absorption is improved.

over the 20 yr since the presentation of this hypothesis and concluded that little has been discovered that would meaningfully modify the concept. Essentially, ATP-driven pumps within the enterocyte's basolateral membrane generate an Na^+ potential at the luminal surface. Glucose, galactose and xylose move into the cell by using Na^+ and a carrier protein. Na^+-dependent and independent transporters move accumulated monosaccharides out of the enterocyte through the basolateral membrane. Vascular turnover within the villus continually maintains a concentration gradient that favors absorption.

Mechanisms for adaptation. The capacity of fowl to digest and to absorb carbohydrates is not a fixed circumstance but is highly adaptable and multifaceted.

As the amount of dietary starch changes, the concentration of α-amylase in the pancreatic juice is altered accordingly. Hulan and Bird (16) fed chicks isonitrogenous feeds in which fat content increased from 4.5 to 14.5% while starch was conversely decreased. Each feed was alternated over 4-d periods. Amylase, lipase and protease activities in the pancreatic juice were found to adjust with intake of the respective nutrients.

Adaptation of pancreatic zymogen levels to optimize digestive need within the lumen has been shown to occur with mammals as well as fowl (17). The mechanism for adjustment appears to involve the nature and extent of stimulation that the pancreatic acinar cells receive from the intestine for zymogen release. Neural impulses, cholecystokinin and pancreozymin are distinctly different stimuli that effect expulsion of stored granules that contain the full array of zymogens. However, an additional mechanism is in evidence that superimposes the release of individual zymogens. Presumably, amylase is preferentially increased with neural stimulation, whereas lipase and chymotrypsin respond to cholecystokinin and pancreozymin, respectively. Because these stimuli arise from immediate conditions at the small intestine, adjustments in the pancreatic juice are continual. Zymogen concentrations of the granule are also adjusted over the long term with these changes involving the same stimuli only at the nuclear level.

The absorptive surface also adapts to dietary conditions. Implicit in this adjustment is the necessity for a rapid turnover of the villus epithelium, which, in turn, permits change in its length. Imondi and Bird (18) reported that the chick's intestinal epithelium has a turnover time of approximately 2 d. The mechanism of turnover involves the generation of cells in the crypt of Lieberkühn at the base of each villus, then they attain digestive and absorptive capability while ascending the shaft. Cells are finally extruded from the tip with senescence. Controlling the rates of division and extrusion permits lengthening or shortening of the villus accordingly.

General observation indicates that villi have a "critical" length, which is determined by the benefit to nutrient retrieval and its cost of maintenance. Physical restriction of feed intake such that the intestine is extensively idle has been shown to reduce villi length but not to impair nutrient utilization (19, 20). On the other hand, villi lengthening occurs when the animal has ad libitum access to feed and additional throughput is necessary to meet extended requirements. This circumstance may appear by lowering the level of nutrition from one feed to another, or if the productive and maintenance needs of the animal were to increase while receiving the same ration (21–24).

Villi may also lengthen in response to "competition" for nutrients with normal microflora (25). Transient lengthening has also been documented with loss of effective surface area from coccidial parasitization (26, 27). Regardless of reason, adjustment in villi length does not involve the whole tract to the same degree but is usually localized. For the most part, the upper jejunum undergoes the greatest change because maximal digestion and presentation of nutrients for absorption occur through this area.

Enzymes that finalize digestion of carbohydrates to yield absorbable products are localized at the mucosal surface. The disaccharidases have been shown to vary in their concentration with substrate "load." Each enzyme is viewed as being anchored on the luminal side of the enterocyte membrane in a manner that permits a high degree of

molecular freedom and independence from one another (28).

With rats, altering dietary sucrose has been shown to cause a parallel change in sucrase activity. Raul et al. (29) suggested that sucrose seeps through imperfections in the crypt, then basolateral membrane uptake provides enterocytes with information during their development for sucrase synthesis. More recently, Cezard (30) reported that this response to sucrose maximizes within 12 h and involves all mature cells covering the villus rather than being restricted to those in transition. Regardless of mechanism for controlling the disaccharidases, fowl have been shown to respond as the rat. Blum et al. (31) varied dietary carbohydrate for chicks and observed that mucosal maltase and sucrase concentrations changed accordingly.

THE IMMATURE SYSTEM

Embryonic intestine. The fowl's intestinal system is anatomically complete early in embryonic development. Villi having enterocytes showing rudimentary microvilli are in place by 16 d of incubation for the chicken (32–34).

Early completion of the small intestine appears to be necessary if complete transfer of maternal passive immunity is to occur. Immunoglobulin G (IgG) is passed into ova by the hen during follicular development, then taken up by the embryo during yolk sac resorption (35–38). IgA is intended for protection of mucosal surfaces, and this immunoglobulin appears to cross the oviduct as one of the albumen proteins. Rose et al. (39) noted that IgA could only be found in the white of the fresh egg and after incubation was detected in the digestive tract of the 19-d embryo.

Uptake of IgA from the albumen is indicated to occur after 14 d of incubation when the embryo actively consumes the contents of the amniotic sac (40). Immunologically identifiable albumen proteins appear in embryonic circulation concurrent with this consumption, and they reach maximal concentration by 19 d. Rose et al. (39) likened the conveying of passive immunity in fowl by yolk and albumen to that in mammals by placenta and colostrum, respectively.

Piglet development. The small intestine of the piglet at birth resembles that of the 16-d chick embryo. Villi are physically underdeveloped, and the enterocytes were placed there during embryonic development. The enterocytes absorb colostral immunoglobulins only at the embryonic stage, and their capacity to do so ceases in about 2 d after birth (41–44).

Consumption of colostrum and milk stimulates villi growth that is particularly rapid. Moon (45) observed a 9- to 10-d turnover time for enterocytes in place following parturition as opposed to a 2- to 4-d turnover 1 wk later. Smith and Jarvis (46) microscopically monitored villi development during this early period by thymidine pulse labeling of crypt cells. Based on movement of label from the crypt, they concluded that growth through the first 6 d of life was due to rapid crypt cell emergence combined with an absence of extrusion from the tip.

Enterocytes of embryological origin appear to be limited in their ability to digest carbohydrate to lactose. Aumaitre and Corring (47) measured mucosal carbohydrase activities throughout early development in the piglet. Total lactase was observed to be prominent at birth, to maximize 1 wk later then to decline (table 2). Conversely, maltase and sucrase were negligible at birth, detectable 1 wk later, then rapidly increased. These enzyme changes can be explained in terms of villus growth and if enterocytes arising from the crypt have maltase and sucrase-isomaltase activities but no lactase.

Presumably, embryonic enterocytes located low on the villus at birth would not express their lactase potential until maturation several days after birth. Absence of villus extrusion would permit lactase to peak at a time when relative dependence on milk maximizes. Prolonged retention of lactase on the villus appears to result from the failure of emerging crypt enterocytes to uniformly displace the embryonic cells. Smith and Jarvis (48) followed the movement of labeled crypt cells as they ascended the villus of the pig. Frequent lateral and oblique directions led to mixing such that an estimated 19 d would be required for complete removal of embryonic cells. This mixing would explain

TABLE 2

Total small intestinal mucosa, protein content and disaccharidase activities through perinatal development of the piglet[1]

Parameter	Fetus @ 105 d	Piglet,[2] *wk of age*			
		0	1	2	3
Mucosa wt, *g*	23	26	61	124	187
Mucosal protein, *g*	1.9	7.0	4.1	5.7	22.9
Substrate hydrolyzed in whole intestine, *μmol/min*					
Lactase	111	1222	2963	2102	2778
Maltase	74	106	588	1361	4166
Sucrase	0	0	393	2242	2578

[1]From Aumaitre and Corring (47) with permission of S. Karger AG, Basel. Fetus, n = 40; piglets, n = 20 ♂ + 20 ♀/wk. [2]Animals were suckled until killed.

the absence of any marked transition in the location of lactase and maltase along the rat's villus-crypt axis after birth (49).

Embryonic enterocytes of the piglet can actively transport glucose; however, their capacity in this respect is seriously impaired when immunoglobulins are being absorbed. DeJesus and Smith (50, 51) reported that mucosal uptake of sodium diminishes by one-half, while the K_m increases about 10-fold with access to colostrum. Immunoglobulin uptake has been shown to involve passage between microvilli into a tubovesicular system for internal processing and release at the basolateral membrane (44, 52, 53).

Temporary reduction in nutrient absorptive capacity by the small intestine appears to be compensated for in the colon. Embryonic cells lining the colon of the postnatal piglet have been shown capable of active transport (54–56). "Closure" and return of absorptive competence in the small intestine precedes cell turnover in the large intestine and loss of active transport.

Fowl development. Intestinal transfer of immunoglobulins with fowl is indicated to occur in ovo rather than postnatally as with the piglet. Absence of carbohydrases on the fowl's embryonic mucosa is understandable because yolk sac resorption supplies nutrients by a nonalimentary system. Cessation of immunoglobulin uptake is suggested to occur from the plateauing of its concentration in circulation 2 d after amniotic sac consumption and corresponds to when the chick embryo initiates entry into the air cell for emergence from the shell. The piglet operates in the same relative time frame as the chick from parturition to closure, i.e., ca. 1–2 d.

The capacity of fowl to utilize plant carbohydrates is detectable at 18 d of incubation, moderate at hatching and established a few days later (57). Dautlick and Strittmater (58) found the wet weight and protein content of the chick's upper jejunum to increase dramatically after 18 d of incubation and to parallel total maltase and sucrase activities through to 4 d after hatching (fig. 2). Pancreatic α-amylase also appears around 18 d of incubation and reaches its maximum specific activity 4 d after hatching (59). Lactose utilization in fowl is low at all times and generally assumed to arise because of overlap in specificity by enzymes other than lactose.

Stimulation of villus growth in ovo is probably initiated with intestinal presentation of amniotic contents. This growth would depend on cells emerging from the crypt, and their maturation along with the embryo would explain development of disaccharidase activity (fig. 3). Solid food intake after hatching would further encourage villus elongation. Baranyiova and Holman (60) measured morphological changes in the intestines of fed and fasted chicks through the first week of life. Access to food was observed to markedly increase villus height, whereas fasting prevented villi growth and reduced epithelial cell turnover time.

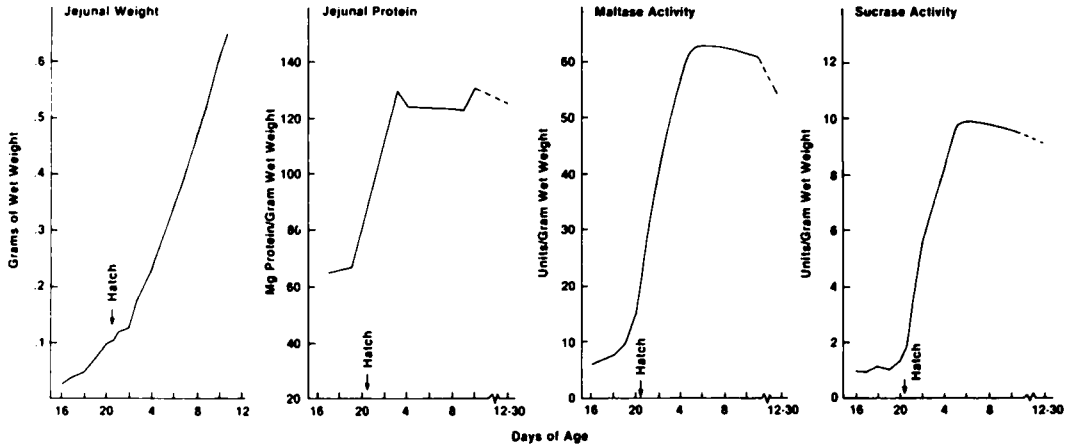

Fig. 2 Development of the small intestine and total disaccharidase activity in the pre- and posthatch chick. The intestine is anatomically complete by 16 d of incubation. Subsequent growth is largely attributable to villi elongation, which parallels the increase in maltase and sucrase. Ability to digest the major plant carbohydrates is detectable when the embryo initiates hatching at 18 d, then it rapidly develops upon emergence from the shell. Full digestive capacity occurs concurrent with depletion of yolk sac reserves. [Redrawn from Dautlick and Strittmatter, (58).]

Active transport of glucose by embryonic enterocytes would seem to be different in fowl than in piglets and also different when produced by replacement crypt cells. Levin (12) and Shehata et al. (61) provided evidence that the nature of active absorption changes in fowl during the hatching process. Two days prior to hatching, anaerobic metabolism provides most of the energy, while sodium is not required for glucose transfer;

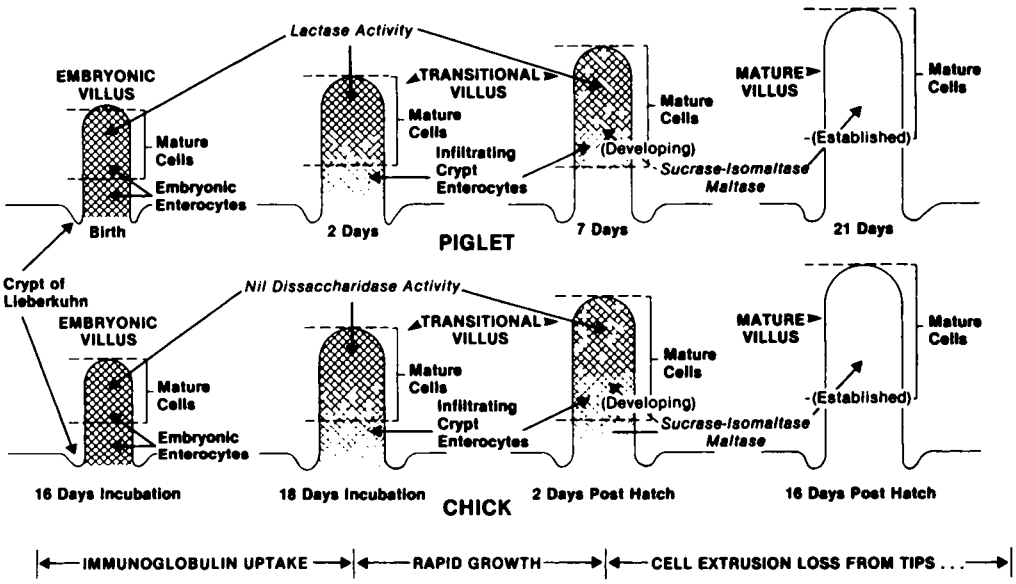

Fig. 3 Cellular changes postulated to occur on the villus during perinatal development of the piglet and chick. Enterocytes placed on the villus during embryonic development are oriented to immunoglobulin transfer. Immunoglobulin uptake stimulates villus growth which, in turn, depends on enterocytes formed in the crypt. Maturation of crypt enterocytes leads to the appearance of carbohydrases capable of digesting plant saccharides. Competence to utilize starch progressively develops with dominance of crypt enterocytes on the villus and eventual displacement of those from embryonic origin. These events with the piglet occur after birth and embryonic enterocytes must have lactase. The chick is viewed as developing in parallel with the piglet, only its development takes place largely in ovo and no carbohydrases are needed.

2 d after hatching, 80% of total uptake becomes aerobically driven and sodium dependent. The V_{max} increases dramatically throughout this period while the K_m remains essentially the same.

The expansion of surface area that occurs with villus growth has been used to explain the increased absorptive capacity. Transition from embryonic to crypt source enterocytes where transfer proteins closely resemble one another would rationalize the consistent K_m values. Minimal access to oxygen and absence of glucose in the small intestine while in ovo is the converse of that after hatching. Presumably, the basolateral membrane pumps that would use large amounts of ATP to generate a sodium potential are absent from the chick's embryonic enterocytes.

Mixing of embryonic and crypt enterocytes on the villus is expected to occur for fowl as was described earlier for the piglet. This dispersion may account for the microscopic appearance of surface discontinuities on the villus at day of hatch (1) and the observation of a transient reduction in nutrient utilization through the chick's early life (62).

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