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Glucose, Galactose, and Glutamine Metabolism in Pig Isolated Enterocytes during Development[1]

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ABSTRACT

In the pig, the gastrointestinal tract grows rapidly after birth and undergoes a short postnatal maturation. The objective of the present work was to assess the metabolic characteristics of the small intestinal mucosa during this period by investigating glucose, galactose, and glutamine metabolism in pig isolated enterocytes. Piglets were used immediately after birth or at various stages during suckling or postweaning. Fed animals were taken in a postabsorptive state. The jejunoileum was excised and perfused with an EDTA (5 mM)-containing buffer. The epithelial cell layer was further dissociated in the presence of hyaluronidase (0.01%). The resulting cell suspension (95% absorbing enterocytes; viability greater than 90%) was incubated with ^{14}C-labeled substrates to measure $^{14}CO_2$ production in parallel with substrate disappearance. The capacity to utilize glutamine was high and remained steady during the suckling period. Glucose utilization capacity was limited at birth and increased more than 3-fold during the first week of suckling. Such an increase was not observed in piglets kept unsuckled since birth. Galactose utilization capacity remained steady during the first week but afterward gradually disappeared. Lactate and pyruvate production through glycolysis was the major pathway accounting for glucose or galactose disappearance. A capacity for a net glucose production from galactose was evidenced during the first week of suckling. Thus, isolated newborn pig enterocytes exhibit specific and transient metabolic characteristics during the first postnatal week. (***Pediatr Res* 36: 175–181, 1994**)

Abbreviation

LDH, lactate dehydrogenase

In most mammals, birth is accompanied by profound changes in nutrition (1, 2). Several physiologic functions also change around birth. The gastrointestinal tract becomes operative and undergoes marked functional changes (3–5).

During the postnatal period, the ingestion of colostrum is followed by an immediate increase in intestinal tissue growth (6–9). However, the pattern of mucosal growth and cell replacement in the small intestine does differ from species to species. In the pig, the weight of the mucosal mass almost doubles in the jejunum during the first 24 h (6). A fraction of this mucosal gain probably corresponds to the sequestration of milk proteins, as previously noted (6, 10). This can be explained by the presence of vacuolated cells that transfer immunoglobulins from colostrum to the circulation (11, 12). Unlike rodents, the pig loses rapidly, *i.e.* within 24 to 48 h after birth, the capacity to transfer passive immunity (13). Marked modifications of disaccharidase and peptidase activities and of nutrient transport capacities (14–16) also represent developmental changes taking place in enterocytes after birth. Thus, both the structure and function of the small intestine, and particularly of enterocytes, dramatically change during the postnatal period. Most of these changes occur early after birth in the pig, whereas they occur later on, *i.e.* around weaning, in rodents (1, 3, 4). In this respect, the pig provides a useful model to study intestinal development because its intestine is morphologically similar to that of the human and exhibits a relatively short postnatal period of maturation (3, 17–19).

Although absorbing nutrients arising from intestinal digestion is its main function, the small intestinal mucosa also uses some specific substrates. Indeed, as reported in the rat (20, 21), glutamine, glucose, and ketone bodies represent the major oxidative substrates of the adult small intestine. During the suckling period, glutamine oxidation is high in intestinal tissue slices from rat (22), whereas glucose and 3-hydroxybutyrate oxidation are

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very low (22, 23). Moreover, as recently pointed out by Hahn (24), the small intestinal mucosa of suckling rats displays ketogenic and gluconeogenic capacities that disappear at weaning.

Thus, inasmuch as most of the information derives from rat intestinal tissue preparations, the metabolic characteristics of mucosal cells during early postnatal development have been poorly documented. The aim of the present work was to study glucose, galactose, and glutamine metabolism in enterocytes isolated from pigs at birth and at various stages of development.

METHODS

Animals. Pregnant Large White sows were delivered at the end of gestation on d 114 by injecting a prostaglandin analog (cloprostenol, 10 μg/kg body weight, Bellon, Neuilly, France). A total of 51 pigs was used. A group of 11 newborn pigs (0-d-old animals) was withdrawn from the mother at birth and thus never suckled. A group of newborn pigs (n = 6) was withdrawn from the mother immediately after birth and maintained unfed for 48 h at 34°C. Suckling newborns were left with their mother under a heating lamp that maintained a local temperature of 32–33°C. Newborn pigs were allowed to suckle *ad libitum* for 2 d (n = 16), 5 d (n = 6), or 12 d (n = 6) after delivery. A group of six pigs were weaned at 4 wk and used when 3 to 4 mo old. In 0-d-old animals, enterocytes were prepared within 30 min after birth. Suckling pigs were withdrawn from their mother 2 h before enterocyte isolation. This time was found to be sufficient for glucose concentrations to be identical in the portal vein and in the carotid artery and for galactose not to be detected in the portal vein (data not shown). Thus, it is suggested that suckling piglets were in the postabsorptive state. Enterocytes of postweaned animals were isolated after an overnight fast, inasmuch as a longer delay was required to reach a postabsorptive state. In the latter case, only a portion (40 cm) of the proximal jejunum, which represents the major site of nutrient metabolism (25), was taken out. All aspects of the protocol complied with the International Guiding Principles for biomedical research involving animals.

Enterocyte isolation. Newborn pigs were anesthetized with thiopental (Nesdonal, 25 mg/kg, Rhône Mérieux, Lyon, France), and the enterocytes were isolated according to a method previously described (26). Briefly, the jejunoileum was quickly excised, washed with a solution of NaCl (155 mM), and then perfused for 20 min at 37°C with a Ca^{2+}- and Mg^{2+}-free Krebs-Henseleit bicarbonate buffer (pH 7.4) containing 10 mM N-[2-hydroxyethyl]piperazine-N'-[2-ethanesulfonic acid] (HEPES), 5 mM EDTA, 1 mM DTT, and 0.25% BSA under continuous gassing (O_2/CO_2: 19/1 vol/vol). Then, the intestine was gently squeezed along its length, and the luminal fluid was collected and centrifuged (150 × g, 3 min). The cell fractions were submitted to an additional 15-min incubation at 37°C in the same buffer without EDTA but in the presence of 1.3 mM $CaCl_2$, 2 mM $MgCl_2$, and hyaluronidase (0.1 mg/mL). The isolated cells were washed twice and resuspended in the same bicarbonate buffer medium (pH 7.4) without DTT and containing 1.3 mM $CaCl_2$, 2 mM $MgCl_2$, and 1% fatty acid-free albumin (incubation buffer). Postweaned pigs were anesthetized with halothane and enterocytes from the jejunum were isolated as previously reported (27). To assess the origin of the cells removed, histologic examinations of the jejunum and ileum from 0- and 2-d-old animals were performed before and after EDTA treatment. Segments 2 cm long were taken, closed, and injected intraluminally with Carnoy fixative. They were postfixed for 12 h in the same fixative and processed for routine paraffin histology. Sections were stained with Alcian blue, periodic acid-Schiff, hematoxylin, and picroindigo carmine to visualize absorbing cells and goblet cells. The cell density of the final suspension was assessed by counting an aliquot in a Malassez hemocytometer. Starting from the cell suspension, cell smears were also prepared and further stained to identify cellular types in the final cell suspension from 0- and 2-d-old animals.

Enterocyte incubation and viability. Incubations were carried out in 25-mL polycarbonate Erlenmeyer flasks (Nalge Company, Rochester, NY) containing 1 mL of the cell suspension (20–30 × 10^6 cells) in a final volume of 4 mL, in the absence or in the presence of 2 mM D-[U-^{14}C]glucose, 2 mM D-[U-^{14}C]galactose, 2 mM L-[U-^{14}C]glutamine, or 10 mM dihydroxyacetone. The flasks were gassed with O_2/CO_2 (19/1 vol/vol), sealed, and incubated in a shaking-water bath at 37°C and 100 oscillations/min for 30 min, unless otherwise stated. Incubations were stopped by adding 0.5 mL of ice-cold perchloric acid (final concentration 4%).

The cell viability was checked by following the percentage of total LDH (EC 1.1.1.27) activity released in the extracellular medium during incubation in the presence of 2 mM glucose at 37°C. Simultaneously, the metabolic activity of the cell preparation was assessed by following the time course of lactate and pyruvate productions when enterocytes were incubated with 2 mM glucose.

Analytical methods. Dry matter (oven drying, 65°C, 24 h) and protein (28) contents of the cell suspension were determined. DNA content of the cell suspension was determined (29) after trichloracetic acid extraction as previously described (30). Glycogen content of the cell suspension was assayed using α-amylo 1–4,1–6 glucosidase according to Roehring and Allred (31). ATP content of cells isolated from 2-d-old suckling pigs and incubated for 30 min with 2 mM glucose was measured by the luciferin-luciferase system after extraction with dimethyl sulfoxide (32). Galactokinase (EC 2.7.1.6) activity was determined in homogenates of enterocytes (n = 3) isolated from 0-d-old, 2-d-old suckling, 12-d-old, and postweaning animals, according to the method of Ballard (33).

Glucose, galactose, glutamine, lactate, and pyruvate were assayed in the neutralized, deproteinized supernatant by using specific enzymatic methods (34). CO_2 pro-

duction was determined by measuring $^{14}CO_2$ released during incubation with ^{14}C-labeled substrates (1.7–3.4 MBq/mmol). After the incubations had been stopped with perchloric acid, $^{14}CO_2$ was trapped with methylbenzethonium hydroxide (90-min incubation, 25°C), and counted in a liquid scintillation counter (LKB-Pharmacia, St. Quentin-en-Yvelines, France). Blank values were obtained from incubation flasks containing ^{14}C-labeled substrates without any cell suspension added. The efficiency of the oxidation detection system was tested using [^{14}C]sodium bicarbonate as a standard. The average recovery amounted to 102 ± 2% (n = 12).

Chemicals. BSA (fraction V, fatty acid free), hyaluronidase (E.C. 3.2.1.35), DTT, HEPES, methyl benzethonium hydroxide, D-glucose, D-galactose, and L-glutamine were obtained from Sigma Chemical Co. (St. Louis, MO). Perchloric acid and Tris buffer were obtained from Merck (Darmsdadt, Germany). EDTA and all inorganic products were from Prolabo (Paris, France). All enzymes and coenzymes used for enzymatic assays were from Boehringer (Meylan, France). D-[U^{14}-C]glucose, D-[U^{14}-C]galactose, and L-[U^{14}-C]glutamine were purchased from Centre d'Etudes Nucléaires de Saclay (Gif-sur-Yvette, France). The radiochemical purity of all isotopes used was higher than 98%. The scintillation cocktail for radioactivity counting was obtained from LKB-Pharmacia (St. Quentin-en-Yvelines, France).

Presentation and analysis of data. The rates of D-glucose, D-galactose, or L-glutamine utilization corresponded to the net amounts of substrates that disappeared from the incubation medium. For all substrates, the rates of metabolite production corresponded to the net amounts of metabolites generated. All results are expressed as nmol per 10^6 viable cells, with the viability being taken at the onset of incubation. The values shown are the means ± SEM for the number of cell preparations given. For a single cell preparation, the data are based on duplicate or triplicate measurements.

Statistical analysis was performed using the unpaired t test.

RESULTS

Origin and viability of cell suspension. The histologic examination showed that the cells were efficiently removed from the villi without contamination by crypt cells (data not shown). Whatever the intestinal site (jejunum or ileum) or the age of the animals (0 or 2 d old), enterocytes represented 90 ± 1% (n = 15) and 95 ± 1% (n = 8) of intestinal cells in the intact tissue and in the final suspension, respectively. DNA content of isolated cells was stable (9.0 ± 0.3 μg · 10^6 cells^{-1}, n = 20). Protein content of isolated enterocytes represented a steady percentage of the cell dry weight (65 ± 1%, n = 37). However, protein content expressed per 10^6 cells was significantly higher in cells isolated from 2-d-old or 5-d-old suckling animals (0.36 ± 0.02 mg · 10^6 cells^{-1}, n = 10, and 0.30 ± 0.02 mg · 10^6 cells^{-1}, n = 6, respectively) compared with the other groups (0.20 ± 0.02 mg · 10^6 cells^{-1}, n = 21).

The mean viability was never less than 90% at all stages of development studied and never less than 85% after 30 min of incubation. Furthermore, incubating the cells isolated from 0-d-old pigs (data not shown) or from 2-d-old suckling pigs (Fig. 1) with 2 mM glucose resulted in lactate and pyruvate production that increased linearly for 60 min. Simultaneously, a minimal leakage of LDH activity in the extracellular medium was still observed at 60 min (Fig. 1). In contrast, when the incubation went on until 120 min, the lactate plus pyruvate production did not further increase and the leakage of LDH activity markedly increased, suggesting that the viability of the cell suspension was dramatically altered. Nevertheless, such a decrease of cell viability could be caused by glucose shortage. According to the criteria chosen, cell viability was preserved over a 30-min incubation. Furthermore, ATP concentration measured in cells isolated from 2-d-old suckling pigs and incubated with 2 mM glucose remained constant during a 30-min incubation (t_0, 4.6 ± 0.2 nmol · 10^6 cells^{-1}; t_{30}, 4.1 ± 1.1 nmol · 10^6 cells^{-1}, n = 4).

Glycogen content in enterocytes. At birth, isolated enterocytes exhibited a sizeable glycogen content (Fig. 2) that decreased 4-fold within 2 d after birth. One to 2 wk later, intestinal cells contained only 10% of the amount of glycogen found at birth.

Glutamine metabolism in enterocytes. The capacity for glutamine utilization (Fig. 3) was already high in intestinal cells isolated from newborn pigs and remained high during the suckling period. Thereafter, glutamine utilization significantly decreased compared with the birth value. The rates of glutamine oxidation (Fig. 3) were high in isolated cells at birth or during the first week of life because the conversion of the amino acid to $^{14}CO_2$ accounted for about 20% of its disappearance. Thereafter,

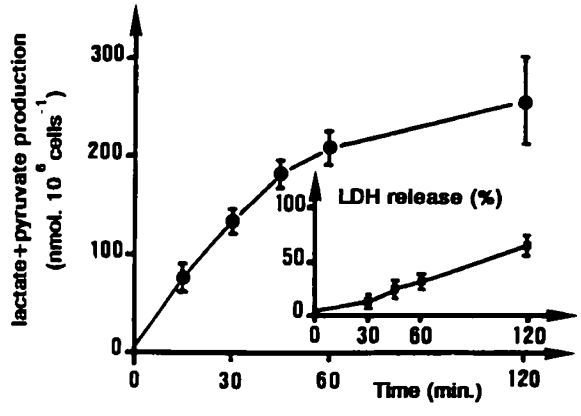

Figure 1. Time-course of net production of lactate and pyruvate from glucose by enterocytes isolated from 2-d-old-suckling pigs. Comparison with LDH release. Enterocytes isolated from 2-d-old-suckling pigs were incubated at 37°C in the presence of 2 mM glucose. Each data point represents the mean (± SEM) of four separate incubations performed in duplicate. Total LDH activity (cells + medium) remained constant during 120 min of incubation (0.73 ± 0.05 u · 10^6 cells^{-1}; n = 4).

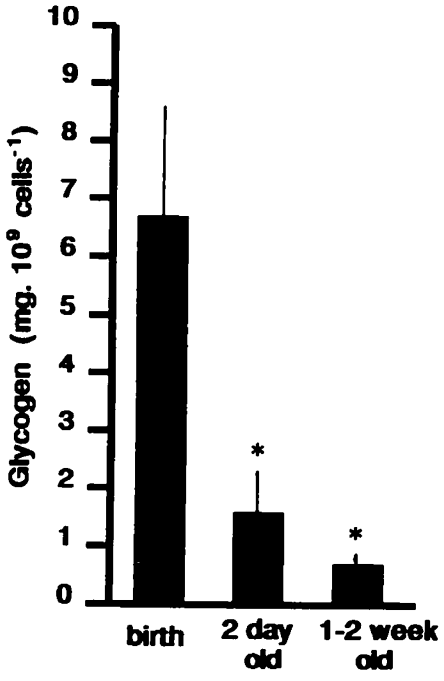

Figure 2. Glycogen concentration in enterocytes isolated from newborn pigs during the first 2 wk of life. Enterocytes were isolated as described in the Methods section. Cells were immediately frozen in liquid N_2 for subsequent analysis. Each value is the mean ± SEM of six to eight determinations. *, Values statistically different from those obtained at birth ($p < 0.01$; t test).

glutamine oxidation was significantly lower and dropped to only 10% of the amino acid utilization.

Hexose metabolism in enterocytes. The capacity for glucose utilization dramatically increased in cells isolated from 2- or 5-d-old suckling animals compared with the birth value (Fig. 3). Yet, despite a 3-fold rise in glucose utilization during the first postnatal week, the rates of glucose oxidation remained constant (Fig. 3). In older pigs (12-d-old and postweaned), the capacity for glucose utilization was similar to that of cells isolated immediately at birth, but the rate of glucose oxidation was significantly reduced and amounted to 10% of glucose utilization (Fig. 3). When cells were incubated in the presence of 2 mM D-galactose, a significant galactose disappearance could be evidenced in enterocytes isolated from pigs immediately at birth or during the first postnatal week. Thereafter, the capacity of these cells to utilize galactose dramatically decreased (Fig. 3). This decrease was accompanied by a sharp fall in galactokinase activity determined in homogenates of freshly isolated enterocytes (0 d: 1.40 ± 0.01 nmol/min · 10^6 cells; 2 d: 1.89 ± 0.01 nmol/min · 10^6 cells; 12 d: 0.44 ± 0.11 nmol/min · 10^6 cells; postweaned: 0.54 ± 0.09 nmol/min · 10^6 cells; n = 3 at all stages). The capacity to oxidize galactose was high in cells isolated from newborn pigs and progressively decreased thereafter (Fig. 3).

Whatever the age of the animals, lactate and pyruvate production was the main pathway responsible for glucose or galactose metabolism, inasmuch as it accounted for 70 to 80% of hexose disappearance (Table 1). Thus, intesti-

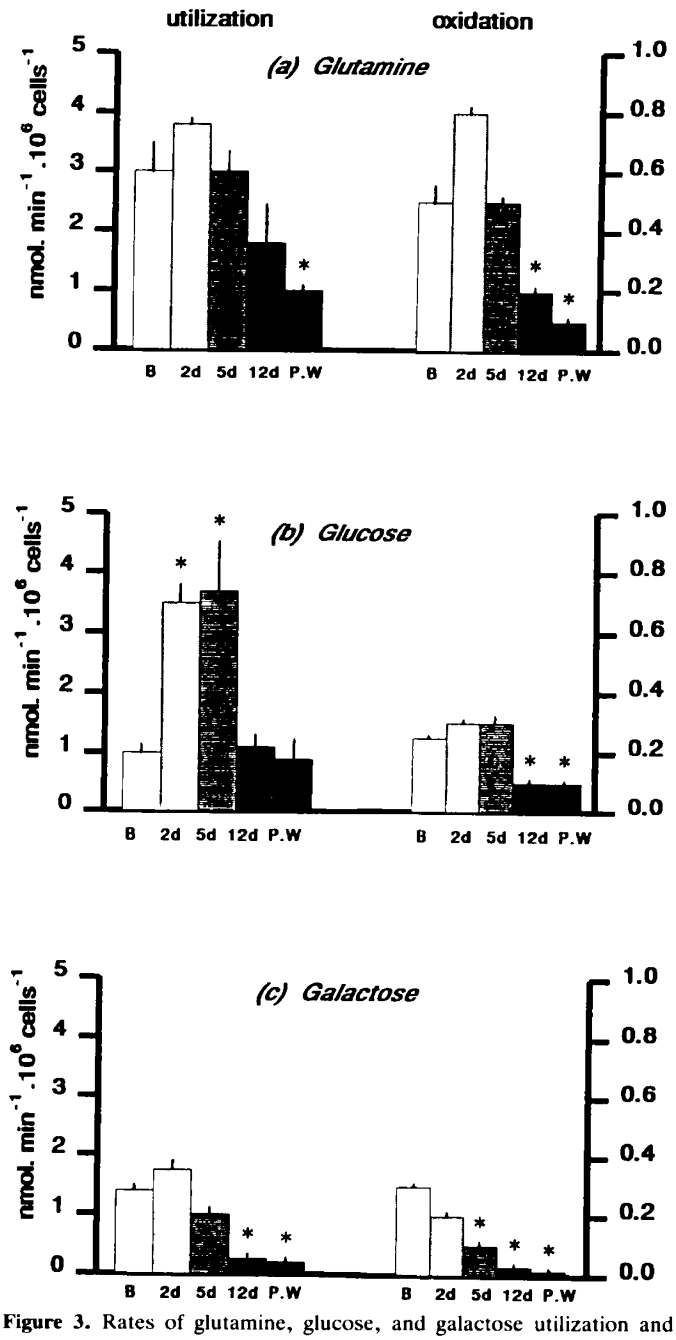

Figure 3. Rates of glutamine, glucose, and galactose utilization and oxidation in pig isolated enterocytes during development. Isolated enterocytes were incubated for 30 min at 37°C in the presence of [U-^{14}C]glucose, [U-^{14}C]galactose, or [U-^{14}C]glutamine at a final concentration of 2 mmol/L. Rates of substrate utilization correspond to the net amounts of substrates that disappeared from the incubation medium. Each value is the mean ± SEM of six to 16 determinations. *, Values statistically different from those obtained at birth ($p < 0.01$; t test).

nal cells isolated from 2- or 5-d-old suckling pigs exhibited a high rate of glycolysis when incubated with 2 mM glucose. Moreover, during the first postnatal week, isolated enterocytes incubated with 2 mM galactose also generated high amounts of lactate and pyruvate (Table 1).

Glucose metabolism in enterocytes from unsuckled 2-d-old pigs. To know whether the enhanced capacity for glycolysis in enterocytes from newborn pigs was triggered by chronologic or diet-related factors, glucose metabolism

Table 1. *Rates of lactate + pyruvate production from glucose or galactose in pig isolated enterocytes during development (nmol · min^{-1} · 10^6 viable cells^{-1})**

Age of animals	Substrate added		
	None	Glucose	Galactose
0 d old	0.3 ± 0.1 (11)	1.4 ± 0.2 (11)	2.0 ± 0.2 (11)
2 d old	0.8 ± 0.1† (16)	5.0 ± 0.4† (16)	2.5 ± 0.2 (16)
5 d old	0.4 ± 0.1 (6)	5.2 ± 0.8† (6)	1.4 ± 0.3 (6)
12 d old	0.3 ± 0.1 (6)	1.7 ± 0.4 (6)	0.4 ± 0.2† (6)
Postweaning	0.1 ± 0.1 (6)	1.0 ± 0.2 (6)	0.1 ± 0.01† (6)

* Enterocytes were incubated for 30 min at 37°C in the absence or in the presence of glucose (2 mM) or galactose (2 mM). Values are means ± SEM with the number of experiments performed in duplicate in parentheses.

† $p < 0.01$ as compared by t test with values obtained at birth.

was studied in enterocytes isolated from 2-d-old pigs kept unsuckled since birth. The capacities for glucose metabolism (glycolysis and oxidation) in these animals were identical with those evidenced in 0-d-old animals (compare Table 1 and Figs. 3 and 4). In contrast, their rates of glucose utilization and lactate plus pyruvate production were 3-fold lower than those in enterocytes from suckling pigs (Fig. 4). However, the rates of glucose oxidation were similar in enterocytes isolated from both groups of animals.

Capacity for net glucose production in enterocytes. Because dihydroxyacetone enters glycolysis or gluconeogenesis at the level of triose phosphates, the rates of lactate plus pyruvate and glucose production from dihydroxyacetone reflect the partitioning of the precursor into glycolysis and gluconeogenesis. The apparent amount of dihydroxyacetone converted into C_3 units was similar in enterocytes isolated from newborn pigs *versus* 2-d-old pigs (Table 2). In all the groups, glycolysis was predominant. However, a capacity to generate glucose from dihydroxyacetone could be detected only in enterocytes isolated from 2-d-old suckling pigs. Such a pathway accounted for 25% of dihydroxyacetone metabolism.

The capacity for a net glucose production in the presence of 2 mM galactose was also observed in enterocytes isolated from 2-d-old suckling pigs (0.31 ± 0.08 nmol · min^{-1} · 10^6 cells^{-1}, n = 12); it was 10-fold higher than that measured immediately after birth (0.03 ± 0.01 nmol · min^{-1} · 10^6 cells^{-1}, n = 11).

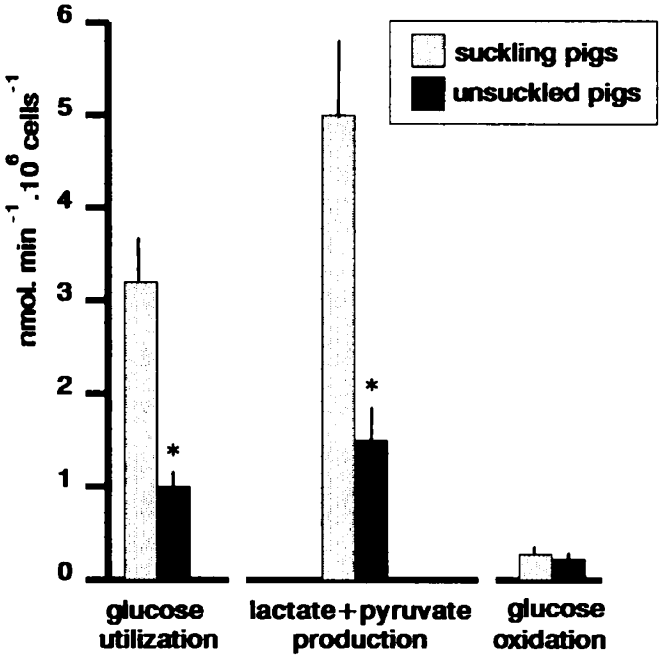

Figure 4. Rates of glucose utilization, lactate + pyruvate production, and glucose oxidation in enterocytes isolated from unsuckled *vs* suckling 2-d-old pigs. Isolated enterocytes were incubated for 30 min at 37°C in the presence of [U-^{14}C]glucose 2 mM. Rates of glucose utilization correspond to the net amount of substrate that disappeared from the incubation medium. Each value is the mean ± SEM of six determinations. *, Values statistically different from those of 48-h-old suckling pigs ($p < 0.01$; t test).

DISCUSSION

The present work informs on the capacities of pig enterocytes to utilize and oxidize substrates at birth, during the suckling period, or after weaning.

The cell suspension obtained in this study represents a well-defined population of villus absorptive cells without any contamination by crypt cells. According to the criteria used, cell viability remained high during incubation, making this preparation suitable for studying enterocyte metabolic capacities. Although exposing all faces of isolated enterocytes to nutrients at the same concentration could be considered a nonphysiologic situation, using this preparation essentially devoid of other intestinal cell types does permit a direct investigation of enterocyte metabolism.

Glutamine utilization and oxidation. At birth and during suckling, pig enterocytes exhibited a high capacity for glutamine utilization, as high as that previously found in enterocytes isolated from adult fed rats (35, 36). Data obtained from homogenates of rat intestine have already shown that phosphate-dependent glutaminase activity is high at birth and further increases during the suckling period (37–39).

After weaning, glutamine utilization declined in pig enterocytes. Whether this decrease corresponds to a

Table 2. *Rates of glucose and lactate + pyruvate production from dihydroxyacetone in isolated enterocytes from newborn or 2-d-old pigs (nmol · min^{-1} · 10^6 viable cells^{-1})**

		48-h-old pig	
	Newborn pig	Suckling	Unsuckled
Amount of dihydroxyacetone metabolized†	3.2 ± 0.3 (8)	4.2 ± 0.5 (11)	3.5 ± 0.4 (3)
Lactate + pyruvate	3.1 ± 0.3 (8)	3.3 ± 0.3 (11)	3.5 ± 0.4 (3)
Glucose	0.05 ± 0.02 (8)	0.5 ± 0.2‡ (11)	<0.05 (3)

* Enterocytes were incubated for 30 min at 37°C in the presence of dihydroxyacetone 10 mM. Values are means ± SEM with the number of experiments performed in duplicate in parentheses.

† Calculated as [2 × (glucose produced · min^{-1} · 10^6 cells^{-1}) + (lactate + pyruvate produced · min^{-1} · 10^6 cells^{-1})].

‡ $p < 0.01$ as compared with newborn pig by t test.

lower quantity of glutaminase, a lower activity of glutaminase, or both needs additional examination. At birth and during the first week of suckling, the rate of glutamine oxidation was 2-fold higher than that determined in 12-d-old suckling or weaned pigs or in adult rats (40). Even in enterocytes isolated from piglets kept unsuckled since birth, the capacity for glutamine oxidation was maintained (data not shown). This result agrees well with previous data showing that glutamine was highly oxidized by intestinal tissue slices from late suckling rats (22, 38, 41). Our data also emphasize that using glutamine as a fuel takes place early in pig intestinal cells compared with the rat mucosa.

Galactose utilization and oxidation. Our data show for the first time that intestinal cells from newborn pigs can metabolize galactose actively, and this metabolic capacity remains high during the first week of suckling. This high capacity to utilize galactose correlated with a high galactokinase activity. In turn, the rapid fall of galactokinase activity could explain the very low rate of galactose metabolism after the first week of life. Recently, Kaempf *et al.* (42) have identified the gastrointestinal tract as an extrahepatic site of galactose consumption in the newborn lamb. In the rat, the specific activity of intestinal galactokinase is high at birth (43). Then, it progressively declines during the suckling period, whereas the activities of galactose-1-phosphate uridyltransferase (44, 45) and uridine diphosphate galactose-4-epimerase (46) remain at a high level. Taken together, these data suggest that galactokinase is the rate-limiting enzyme of galactose metabolism in pig and in rat enterocytes.

Glucose utilization and oxidation. The present data show that newborn pig enterocytes are able to metabolize glucose at a high rate, similar to the findings in enterocytes isolated from adults of other species (35, 36). Moreover, this capacity for glucose consumption dramatically increased in enterocytes isolated from 2- and 5-d-old suckling pigs. These cells exhibited a high glycolytic activity only during the first week of suckling. In contrast, the rate of glucose completely oxidized remained constant. In intestinal slices from suckling rats, the rate of glucose oxidation to CO_2 represents 2 to 5% of the rate of glycolysis (23, 47). However, in contrast to the pig situation, the rates of glycolysis and glucose oxidation significantly increase at weaning in rat intestinal slices. After weaning, the small intestine always represents a potential site of glucose consumption and lactate production, but the extent of glucose metabolism depends on the nutritional status of the animals and also on the methodology used (21, 48–50).

The low rate of lactate and pyruvate production from glucose found in enterocytes isolated from 2 d-old unsuckled pigs suggests that the emergence of a high glycolytic activity in suckling pig enterocytes depends on colostrum ingestion. In addition, results obtained with dihydroxyacetone further indicate that the hexose transport, the initial steps of the glycolytic pathway, or both are potential regulatory steps in intestinal glucose metabolism after birth.

Glucose production. Besides a high glycolytic capacity, enterocytes isolated from 2-d-old suckling pigs also have the capacity to generate glucose from galactose or dihydroxyacetone. This result strengthens previous observations on the suckling rat mucosa (51) and suggests that fructose 1,6-bisphosphatase and glucose 6-phosphatase are active in newborn pig enterocytes during the early suckling period. Again, the emergence of gluconeogenesis in suckling pig enterocytes depends on colostrum ingestion, inasmuch as glucose formation from galactose was very low in 2-d-old unsuckled pigs (data not shown). Nevertheless, determining whether both pathways either metabolizing or producing glucose coexist in the same intestinal cells requires further investigation.

In conclusion, viable absorptive cells have been successfully isolated from the small intestine of newborn pigs. Our data demonstrate that these cells express transient metabolic capacities during the first week of suckling, probably induced by colostrum feeding. These metabolic changes parallel changes in both structure and function of enterocytes. In turn, they could be responsible for generating both ATP and specific metabolites required for intestinal adaptation at birth.

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