Gastrointestinal effects and energy value of polydextrose in healthy nonobese men

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ABSTRACT We studied seven healthy volunteers before and during acute (PD1) and chronic (PD2) ingestion of 30 g polydextrose (PD)/d. The energy value of PD was assessed after [U-14C]PD was added to the 10-g morning dose of PD during PD1 and at the end of PD2. Thirty-one ± five percent (± SD) (PD1) and 29 ± 4% (PD2) of the dose appeared in breath within 48 h. A small fraction of the ingested radioactivity was recovered in urine (4 ± 1%) and excreted in flatus (± 1%) and in feces as volatile fatty acids (VFAs) (< 1%) and bacteria (3–4%); the remaining radioactivity in stools, 33 ± 3% (PD1) and 32 ± 4% (PD2), was assumed to be intact PD. Breath excretion of the label was 49 ± 5% after intracolonic infusion of [U-14C] acetate. The energy value of PD, calculated by means of Miller and Wolin’s stoichiometric equation of colonic fermentation, was similar during PD1 and PD2: 4.0 and 6.1 kJ/g, respectively, when breath 14CO2 and VFA production from PD were used for calculation. Am J Clin Nutr 1994:59:1362–8

KEY WORDS Polydextrose, colonic fermentation, energy value

Introduction

The energy value of unabsorbed or incompletely absorbed carbohydrates is of interest because of the growing use of bulking agents, which share some properties and health benefits with dietary fibers as low-energy food ingredients that replace sugars and fats. There is, however, much controversy regarding the energy value of these compounds, which, in addition to nutritional aspects, is important to determine for academic and legislative reasons. Several methods have been used in attempts to measure the energy value of poorly absorbed carbohydrates (1, 2). They are either difficult to carry out in humans or inappropriate. The most accurate and noninvasive is the disposition method, provided the site of digestion and absorption of the substance studied is known (1, 2).

When a metabolizable carbohydrate is absorbed in the small intestine, its energy value is 16.7 kJ/g; when it is fermented in the large intestine, energy is provided by absorbed volatile fatty acids (VFAs) arising from colonic fermentation. Production of VFAs is described by Miller and Wolin’s equation (3):

\[34.5 \text{C}_6\text{H}_{12}\text{O}_6 + 37 \text{H}_2\text{O} \rightarrow 48 \text{acetate} + 11 \text{propionate}
\]

\[+ 5 \text{butyrate} + 34.25 \text{CO}_2 + 23.75 \text{CH}_4\]

The majority of VFAs are absorbed via a mechanism that may involve secretion of bicarbonate into the colonic lumen (4). After absorption, VFAs are metabolized in the colonic wall and liver, giving less adenosine triphosphate (ATP) than glucose (5–7), and the end product—carbon dioxide—is excreted in breath or trapped in the bicarbonate pool (8). Another source of breath and flatus carbon dioxide is that formed during fermentation itself. A fraction of energy from fermented carbohydrate is utilized by bacteria for growth and maintenance; bacteria are excreted in feces, which also contain small amounts of VFAs in extracellular water (9). Currently, no available study has taken into account energy losses by all these routes in the assessment of the energy value of poorly absorbed carbohydrates.

Among bulking agents, polydextrose (PD) is a water-soluble, randomly bonded condensation polymer of glucose containing minor amounts of sorbitol and citric acid. It is primarily used as a bulking agent in many low- and reduced-energy food products (baked goods, confections, dressings, and frozen dairy desserts), where it replaces significant amounts of both sugar and fat. For example, in baked goods, PD typically replaces from 25% to 50% of the sugar and in frozen dairy desserts from 10% to 25% of the fat (10). The random nature of the glycidosic bonds in PD makes it resistant to small bowel enzymes and only partially fermented in the colon (10, 11). Thus, its energy value should be very low. However, actual data from studies using the disposition method are conflicting in both acute and chronic administration of PD. Although Grossklaus et al (12) concluded that a chronic load of PD resulted in bacterial adaptation, Figdor and Rennhard (13) found a low energy value of 4.2 kJ/g during both acute and chronic administration of PD to rats. Conversely, Cooley and Livesey (14) in acute experiments in rats found a higher energy value of 7.9 kJ/g. In the single human study (15), the energy value of PD was found to be 4.2 kJ/g, but the test dose of [U-14C]PD was given only after a short period of administration of this carbohydrate, and energy losses in bacteria and flatus were not studied.

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Thus, in this study we reevaluated the energy value of PD in healthy nonobese men, using the disposition method, and determined whether changes occurred as a result of its prolonged administration. Special care was taken to examine how much energy was lost in bacteria, unabsorbed VFAs, and flatus. In addition, because a part of PD is fermented in the colon, we investigated the influence of PD on clinical tolerance, stool weight, and intestinal transit.

**Subjects and methods**

**Polydextrose**

Commercial and [U-14C]PD were a gift from Pfizer (Eastern Point Road, Groton, CT). To prepare the [U-14C]PD, a mixture of 89 parts [U-14C] glucose, 10 parts [U-14C] sorbitol, and 1 part nonlabeled citric acid were heated under vacuum at 161 °C for 3 h. The monomer and polymer contents of the resulting [U-14C]PD were characterized by HPLC. The compositions of labeled and nonlabeled PD were similar.

**Subjects**

Seven healthy male volunteers aged 27 ± 2 y (± SD) were studied. An eighth subject was excluded because of incomplete stool recovery. All subjects had normal body weight (within 10% of the ideal weight), had no history of gastrointestinal disease or recent treatment with antibiotics, had never taken laxatives, and had demonstrated a lactose breath-hydrogen test consistent with normal lactose absorption. To avoid undetected loss of carbon in the form of methane, subjects were nonmethane producers, i.e., their breath methane concentrations during the lactose breath test did not exceed 1 ppm above room atmosphere (16). All subjects gave informed written consent to the protocol, which was approved by the Lariboisière—Fernand-Widal—Saint-Lazare Hospital Ethics Committee.

**Experimental design**

The study was divided into three periods: control period (CP) (days 1–8), acute PD ingestion period (PD1) (days 9–16), and chronic PD ingestion period (PD2) (days 17–38).

From days 1 to 16 and 31 to 40 a controlled diet was given, and volunteers consumed all their meals at the study site. Four 1-d menus were designed and fed in rotation. The controlled diet was free of pits and skins, and fiber intake was moderate (=11 g/d) and the same each day. During PD1 and PD2, volunteers ingested in each of the three daily meals 10 g PD mixed into a fruit juice. From days 17 to 30 volunteers were allowed to eat their usual diet at home, where they continued to ingest 10 g PD mixed into a fruit juice in each of the three daily meals.

On days 13 and 35, 740 kBq (20 μCi) [U-14C]PD was added to the morning dose of PD, and consumed with breakfast at 0800. On days 5, 13, and 35, subjects received with the morning meal 10 g polyethylene glycol 4000 (PEG) as a fecal recovery marker. For 3 consecutive days starting on days 5, 13, and 35, volunteers also ingested with the morning meal 20 radiopaque pellets of different shapes in order to measure the mean gastrointestinal transit time (17).

Lastly, to determine the amount of 14CO2 exhaled in breath that derived from oxidation of VFAs in the body (see below), a colonic infusion of [U-14C] acetate was done in all subjects. On day 39, subjects were intubated via the nose with a double-lumen tube whose transit down the gut was aided by a terminal inflatable bag containing mercury. When the bag had reached the cecum, as confirmed fluoroscopically, it was deflated and subjects had to remain in a semirecumbent position. On day 40, 60 mL distilled water containing 4.3 mmol NaCl and 5 g PD, adjusted to pH 6.5, was infused at a rate of 0.5 mL/min into the cecum; after 2 h, the same solution containing 370 kBq (10 μCi) [U-14C] acetate was infused at the same rate for 2 h. The total body radiation exposure from [U-14C]PD and [U-14C] acetate was 2 mSv (18).

**Sample collection**

On days 5–8, 13–16, and 35–38, subjects were asked to record any symptoms, and urine and feces were collected and immediately frozen at –20 °C. A radiograph was taken of the first stool passed ≈ 24 h after the last pellet was ingested. In the last two subjects studied, stools were collected for 4 d after [U-14C] acetate infusion.

Starting on days 5, 13, and 35, breath collections were obtained hourly from 0 (0800 h) to 15 h and at 18, 21, 24, 30, 36, and 48 h. Because 14CO2 excreted in breath was very small 24 h after the cecal infusion of [U-14C] acetate, sampling was stopped at that time on day 41. At sampling times an end-expiratory breath sample for hydrogen analysis was collected into a 50-mL syringe and then subjects breathed over 5 min into a 50-L rubber bag for 14CO2 analysis. Total carbon dioxide production was determined by indirect calorimetry (MMC Beckman Horizon System, San Diego). To avoid contamination of the gas collection system by 14C, the determination was done on a day when subjects received the menu served on days 13 and 35 but without [U-14C]PD. Ten measurements were taken for 16 h, starting at time 0 (0800 h) in the fasting state. The value for carbon dioxide production obtained at a given time was used on the following day.

On days 13 and 35 in three subjects, flatus was collected for 12 h by means of a flexible gas impermeable rubber tube, whose tip was inserted into the rectum and the other end attached to a laminated gas bag that was impermeable to gas diffusion. The gas was aspirated from the bag with a greased glass syringe and its volume was noted. An attempt had been made to collect flatus from all subjects; however, collection was complete for only these three subjects.

**Analytical methods**

Hydrogen in breath was measured by gas chromatography (microlyser model DP; Quintron, Milwaukee). Gas composition of flatus was obtained by mass spectrometry (QMG 511; Balzers, Balzers, Lichtenstein) (19). For breath or rectal 14CO2, the contents of 50-L rubber bags or of flatus bags were slowly bubbled through 3 mL of a solution containing 1 mL hyamine hydroxide (1 mol/L), 2 mL ethanol, and 2 drops of phenolphthalein to indicate when the solution had become saturated by 1 mmol CO2. The carbon dioxide absorbing capacity of ethanolic hyamine hydroxide solution was confirmed in preliminary experiments by titration with 0.1 mol HCl solution/L. When the solution became colorless, 15 mL scintillation fluid (Omnifluor; Dupont de Nemours, Rungis, France) was added, and the radioactivity was determined.

Each day’s collection of urine was pooled and assayed for radioactivity. Triplicate aliquots (0.2 mL) were dissolved in counting vials containing the scintillation fluid for radioactivity determination.
After partial thawing, each daily collection of feces was pooled, homogenized in distilled water, and an aliquot was lyophilized. In nonlyophilized stool samples, PEG was measured by turbidimetry (20). For \(^{14}\)C determination, the soleuene method was used (21). Duplicate aliquots (20 mg) of the lyophilized samples were rehydrated with 0.1 mL distilled water in a counter vial for 30 min. Feces were then solubilized with 1 mL Soleuene-350 (Packard Instruments, Rungis, France). After 2 h incubation at 40 °C, 0.2 mL isopropanol (cell destroyer) and 0.5 mL H₂O₂ (decolorization agent) were added and the material was further incubated for 2 h at 40 °C. The contents of the vials were then dissolved in 10 mL Hionic-Fluor (Packard Instruments) as scintillating fluid and radioactivity was assayed. This method of fecal \(^{14}\)C assay was compared with the more usual combustion technique in two subjects. The results of the soleuene procedure were in good agreement with those from combustion (16 determinations; \(r = 0.97, P < 0.001\)). Efficiency of counting was 95 ± 1% (\(\bar{x} \pm SD\)) and reproducibility was ±1.5%.

In five subjects the 4-d fecal collections from each of the CP, PD1, and PD2 periods were separately pooled and homogenized with a blender, and an aliquot was lyophilized. The bacterial fraction was isolated by the fractionation procedure of Stephen and Cummings (22). For the PD1 and PD2 periods, the bacterial fraction was assayed for radioactivity. Another fecal aliquot was steam distilled and radioactivity in distilled VFAs was assayed (23). In two subjects the feces for each period were not pooled; the weight and radioactivity of the bacterial fractions were measured in daily stools. In the two subjects in whom stools were collected after \([1\text{-}^{14}\text{C}]\text{acetate infusion, radioactivity in bacteria and VFAs was assayed.}]

Calculations and statistics

The mean transit time was calculated according to the single stool method (17). Total volumes of hydrogen excreted in breath during the 24-h sampling periods were determined by integrating the areas under the hydrogen concentration curves. Tidal volumes were determined from the Radford nomogram and data were expressed in milliliters per total test period (24 h) (24).

The rate of \(^{14}\)CO₂ excretion in breath was calculated by multiplying the specific activity by the rate of carbon dioxide production. The latter was calculated as the mean of the two measured values closest in time to the corresponding radioactivity measurement. The rate of \(^{14}\)CO₂ excreted in breath on the second day after \([1\text{-}^{14}\text{C}]\text{acetate infusion was estimated from the difference between the two extreme assumptions (excretion continues at the rate noted at the 24-h time point, or drops immediately to zero after the 24-h time point). Radioactivity in flatus was calculated by multiplying the specific activity by carbon dioxide output.}]

We used commercial and labeled PD, which are mixtures of monomers (5% as anhydro-\(\alpha\)-glucose, \(\alpha\)-glucose, and \(\alpha\)-sorbitol), polymers (93%), and water. The 5% monomeric component of PD is likely to be completely absorbed in the small intestine and to provide 16.7 kJ/g. The percentage of ingested \([1\text{-}^{14}\text{C}]\text{PD (and thus PD) fermented in the colon was 95%} (100 - 5\%) \text{ of ingested dose minus the percentage excreted in stools; we assumed that excreted PD in stools was equal to the total fecal radioactivity minus radioactivity in bacteria and VFAs, and we did not correct for the unknown amount of radioactivity excreted in stools as labeled bicarbonate. From the amount of PD that was fermented, we used the equation of Miller and Wolin applied to nonmethane-producing subjects (25):}

\[
34.5 \text{ C}_6\text{H}_{12}\text{O}_6 + 37 \text{ H}_2\text{O} \rightarrow 48 \text{ acetate} + 11 \text{ propionate} + 5 \text{ butyrate} + 58 \text{ CO}_2 + 95 \text{ H}_2
\]

From this we calculated the energy value of PD in two different ways: 1) from the amount of VFA produced and absorbed in the colon, and 2) from the percentage of radioactivity expired in breath as \(^{14}\)CO₂, corrected by \(^{14}\)CO₂ from bacterial fermentation.

One-way analysis of variance was used to detect period differences. When \(F\) values were significant (\(P < 0.05\)), comparisons among periods were made by using the Newman-Keuls test (26). Data are expressed as mean ± SD.

**Results**

During PD1 and PD2, no volunteer complained of abdominal symptoms. Compared with the control period, total fecal weight tended to be higher during PD1 and PD2 than during CP (\(P = 0.06, \text{Table 1}\)). For both dry matter and wet weights of stools a similar nonsignificant trend was observed. During the three periods, bacterial mass and transit time did not change, and PEG recovered in stools averaged 90% of the ingested amount (Table 1). The total volumes of hydrogen excreted in breath per 24 h were not significantly different during CP, PD1, or PD2 (143 ± 50, 162 ± 109, and 222 ± 115 mL/24 h, respectively).

**Table 2** summarizes the values for \(^{14}\)C excretion in breath, feces, and urine during PD1 and PD2. Thirty-one (PD1) and 29% (PD2) of the label were recovered in breath during the first 48 h after oral ingestion of PD. The time between ingestion of \([1\text{-}^{14}\text{C}]\text{PD and peak} \text{^{14}CO₂ excretion averaged} 6\text{ h;} (21\% \text{ (PD1)} \text{ and 19}\% \text{ (PD2) were excreted during the first 16 h after PD ingestion, then} \text{^{14}CO₂ excretion plateaued at a low level and was still detected at 48 h (Fig 1). The final pulmonary label probably originated from endogenous pools. Volume, composition, and radioactivity of 12-h rectal gas in PD1 and PD2 periods are given in Table 3. Excretion of the radioactivity in rectal gas was very small.}

**Table 1**

**Mean transit time and mean 24-h fecal wet weight, weight and percentage of dry matter, bacterial mass, fecal water, and percentage of polyethylene glycol 4000 (PEG) excreted in stools during the control (CP), acute (PD1), and chronic (PD2) ingestion periods**

<table>
<thead>
<tr>
<th></th>
<th>CP</th>
<th>PD1</th>
<th>PD2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Transit time (h)</td>
<td>44 ± 6</td>
<td>47 ± 7</td>
<td>46 ± 10</td>
</tr>
<tr>
<td>Fecal weight (g/d)</td>
<td>122 ± 46</td>
<td>164 ± 53</td>
<td>158 ± 61</td>
</tr>
<tr>
<td>Dry weight (%)</td>
<td>24 ± 2</td>
<td>24 ± 2</td>
<td>25 ± 3</td>
</tr>
<tr>
<td>(g/d)</td>
<td>29 ± 11</td>
<td>38 ± 10</td>
<td>40 ± 12</td>
</tr>
<tr>
<td>Bacterial mass (%)</td>
<td>36 ± 0.1</td>
<td>32 ± 0.1</td>
<td>33 ± 0.0</td>
</tr>
<tr>
<td>(g/d)</td>
<td>10 ± 1</td>
<td>13 ± 6</td>
<td>13 ± 5</td>
</tr>
<tr>
<td>Fecal water (%)</td>
<td>76 ± 2</td>
<td>77 ± 2</td>
<td>75 ± 3</td>
</tr>
<tr>
<td>(g/d)</td>
<td>93 ± 36</td>
<td>129 ± 43</td>
<td>119 ± 49</td>
</tr>
<tr>
<td>PEG (%)</td>
<td>92 ± 10</td>
<td>90 ± 11</td>
<td>88 ± 3</td>
</tr>
</tbody>
</table>

\(\bar{x} \pm SD; n = 7. PD, polydextrose. There were no significant differences (one-way ANOVA).\)
In the PD1 and PD2 periods, urine recovery of radioactivity was 4.3 ± 1.2% and 3.8 ± 0.7% of the ingested dose, respectively; most of the radioactivity was recovered in the 0–24 h collection interval. In the PD1 and PD2 periods, fecal radioactivity accounted for 36% of the ingested dose; most of the radioactivity was recovered on the second and third days after [U-14C]PD ingestion.

Radioactivity in bacterial mass was 3.0 ± 0.6% and 3.6 ± 1.2% during PD1 and PD2, respectively. In the two subjects studied, the time course of the daily excretion of labeled bacterial mass followed the same pattern as that of the total fecal radioactivity. Radioactivity in fecal VFAs was lower than 1% of the ingested dose: it averaged 0.4% (range 0.1–1.0%) and 0.2% (range 0.1–0.3%) during PD1 and PD2, respectively.

Radioactivity recovered in stool after [U-14C]acetate infusion in the two subjects studied was 11.6% and 10.5%, one-third to one-half being passed after the third day after infusion; radioactivity was 6.5% and 3.8% in bacterial mass and 0.6% and 0.3% in VFAs. After colonic infusion, 46 ± 4% of the absorbed amount of [U-14C] acetate was recovered in breath during the first 24 h after colonic infusion, 41% being excreted within 12 h, with a sharp peak at 2–4 h. After 16 h, 14C excretion from [U-14C] acetate plateaued at a low level (Fig 2) and was parallel to 14C excretion from PD. Data at the 24-h time point extrapolated to 48 h indicated that an additional 3% of the absorbed amount of [U-14C] acetate was excreted in breath.

From the 10-g dose of PD containing the 740-kBq (20-μCi) [U-14C]PD tracer, 5% was absorbed in the small intestine as monomers and an average of 33% (total fecal radioactivity minus radioactivity in bacteria and VFAs) was excreted in the feces and was assumed to be unchanged PD. The remaining 62% was fermented with 3% being incorporated into bacterial mass and 59%

![FIG. 1. Breath 14CO2 excretion curves after [U-14C]polydextrose (PD) administration in the acute (PD1) and chronic (PD2) ingestion periods. 0 refers to time at which [U-14C]PD was given. \( \bar{x} \pm SD; n = 7 \).](image1)

![FIG. 2. Breath 14CO2 excretion curve during and after cecal infusion of [U-14C]acetate. 0 refers to time at which the 2-h cecal infusion of [U-14C]acetate was started. Data at the 24-h time point were extrapolated to 48 h. \( \bar{x} \pm SD; n = 7 \).](image2)
converted to VFAs and gas. On the basis of the stoichiometric equation, 59% (ie, 5.9 g) would be incorporated into 2.7 g acetate, 0.8 g propionate, and 0.4 g butyrate. Multiplication of the respective gross energy values of these VFAs (14.57, 20.63, and 24.81 kJ/g for acetate, propionate, and butyrate, respectively) (27) indicates a total of 65.8 kJ. This is converted to ATP with an efficiency of 80% (5–7), ie, 52.6 kJ would be available to the host. To this must be added the 8.4 kJ available from directly absorbed monomers. Thus for the total 10-g dose the effective energy value of PD is 61.0 kJ, or 6.1 kJ/g.

As stated above, 59% of the ingested dose was fermented by bacteria to carbon dioxide and to VFAs. Thirty-one percent of the label was recovered in breath within 48 h. $^{14}CO_2$ in breath was derived from 1) bacterial $^{14}CO_2$ produced during the fermentative process, 2) oxidation of VFAs produced during the fermentative process and metabolized in the body after colonic absorption, and 3) oxidation of absorbed monomers in the small intestine. When labeled glucose is ingested approximately 60% of the label is recovered in breath as labeled carbon dioxide within 24–48 h (13): the amount of label expected to be exhaled in breath within 48 h during oxidation of the 5% monomeric fraction is hence 3%. According to the equation of Miller and Wolin, 28% of the fermented PD carbon, ie, 16.5% of the dose, was converted to carbon dioxide during the fermentative process. To derive the amount of $^{14}CO_2$ that must have come from VFA oxidation, 3% of the dose coming from oxidation of the absorbed monomers and 16.5% from bacterial carbon dioxide are subtracted from the total amount of exhaled label in breath for 48 h. Hence, $31\% - (16.5 + 3\%) = 11.5\%$ of the label was derived from VFA oxidation.

To calculate from this value the amount of VFAs formed, it is necessary to use the data derived from colonic infusion of labeled acetate. In consideration of the findings regarding $^{14}C$ fecal excretion in the two subjects studied, [U-$^{14}C$]acetate available for absorption was estimated to be 90% of the infused amount (see Discussion) and 49% of this amount appeared in breath within 48 h. This fraction can be used to calculate the amount of VFAs that must have been formed to place 11.5% of the dose in breath, ie, 23.5% of the ingested dose. This amount of VFAs is converted to ATP with an efficiency of 80% relative to glucose (5–7), and thus the energy yield from VFAs formed is 18.8% of the energy content of PD. To this must be added 5% from absorbed monomers. The total 23.8% applied to the gross energy of PD indicates an effective energy value of 4.0 kJ/g.

**Discussion**

In view of the controversy and uncertainty regarding the energy value of PD (12–15) we designed a further study with [U-$^{14}C$]PD, whose main goals were to assess under acute conditions and after prolonged administration the energy value of PD and some physiological effects. From the fermented amount of [U-$^{14}C$]PD, fecal radioactivity being corrected for that contained in bacteria, the energy value was evaluated by means of Miller and Wolin’s equation in two ways: 1) from VFAs produced, and 2) from $^{14}CO_2$ in breath corrected by $^{14}CO_2$ from the fermentative process.

Alterations in concentrations of VFAs in the rat cecum have suggested that the bacterial flora may adapt to PD and improve its capacity to ferment PD (12). Conversely, it has been shown by recycling experiments that, after a first passage through the digestive tract of rats, 90% of [U-$^{14}C$]PD was still recovered in feces after a second passage (13), indicating that fermentation of the PD molecule is incomplete and cannot be increased by repeated exposure to bacterial enzymes unless new bacterial enzymatic activities can be induced. In our study there was no evidence of modified fermentation of PD after daily administration of 30 g PD for 19 d. Fecal and breath radioactivity remained unchanged compared with the initial acute load, and bacterial mass did not vary significantly. Moreover, areas under the curves of breath hydrogen did not change significantly. In both periods, radioactivity incorporated into bacterial mass and VFAs was low, indicating that most of the fecal label represented the resistant part of PD.

In the two subjects studied, the time course of radioactivity in the daily fecal bacterial mass followed the same pattern as did total fecal label and was consistent with transit measured with radiopaque pellets. Because radioactivity is probably incorporated in bacteria during the first 16 h after ingestion, it is likely that the colonic transit times of bacteria and resistant PD are similar. The very low incorporation of radioactivity into fecal VFAs is similar to that found by Figdor and Rennhard (13) in rats; this could be anticipated from the large capacity of colonic absorption, even if there was increased fermentation (9). Colonic absorption of VFAs may involve secretion of bicarbonate into the colonic lumen, and hydration of bacterial carbon dioxide in the lumen may also provide bicarbonate (4). Thus, a small part of the radioactivity excreted in stools may come from labeled bicarbonate. Because we did not measure radioactivity in fecal bicarbonates, the calculated amount of fermented PD was underestimated.

None of the volunteers experienced adverse symptoms during PD administration. Wet and dry weights of the feces were higher in five to six subjects during the two PD periods than during CP, which could be accounted for by the weight of excreted PD. Because the number of subjects studied was limited, the increase in these indexes was not statistically significant. The mean transit time did not vary among the three periods. In rats, Oku et al (28) concluded that PD significantly increased wet and dry weights of the feces, whereas its effects on transit time were dependent on the nature of associated fibers in the diet.

The energy value of PD depends on the extent of absorption in the small bowel, the amount fermented in the large intestine, and the proportion of resulting VFAs available to the body. We calculated these indexes from data obtained in feces and breath after the ingestion of [U-$^{14}C$]PD together with 10 g PD, using the stoichiometric equation of Miller and Wolin (3). Because label in fecal bicarbonate was not specifically measured, the amount of fermented PD in the large intestine was slightly underestimated. We assumed also that absorption and metabolism of low-molecular-weight carbohydrates present in commercial PD was 100%, ie, 0.5 g, or 5% of the oral $^{14}C$ load. Whether PD itself may be partly digested and absorbed in the small intestine is disputed (12–15, 29). The bulk of evidence suggests minimal or mild digestion and absorption of PD. Urinary excretion of radioactivity was 4%, but this does not interfere with calculation of PD energy value, because it represents incorporation into normal catabolic products, such as urea (13).

In our study the mean total recovery of the label, taking into account the conversion factor of [U-$^{14}C$] acetate infused in the colon, was $86 \pm 8\%$ and $81 \pm 7\%$ during PD1 and PD2 periods
The deficit can be explained by either incomplete collection of the label and/or by overestimation of the conversion factor based on acetate infusion or the amount of bacterial carbon dioxide calculated from the stoichiometric equation. The deficit cannot be explained by incomplete collection of radioactivity in stools, because fecal recovery of PEG was high and taken into account when 14C fecal excretion was calculated. Likewise, excretion of 14CO2 in flatus measured in three subjects was very low during the two periods of PD administration (≈ 1%). This contradicts the hypothesis of Fidor and Bianchine (15), who found a large deficit in 14C recovery. Whereas others used a theoretical endogenous production rate of 9 mmol kg−1 h−1 for carbon dioxide, we measured this index several times during the period after [U-14C]PD ingestion. In this way, we found a mean breath excretion of 14CO2 of 31% (PD1) and 29% (PD2), instead of 38% (PD1) and 37% (PD2) when the theoretical endogenous carbon dioxide production rate was used. We also measured the conversion factor of acetate to carbon dioxide individually, by perfusing this labeled VFA into the colon during steady-state PD fermentation. Measurements in two subjects showed that ≈10% of 14C from infused acetate was found in stools, one-third to one-half being present after 2 d; this fraction was assumed to be unavailable for absorption. Some of the label was indeed incorporated in bacteria and unabsorbed VFAs, but the remaining label was presumably labeled bicarbonate excreted in stools, possibly resulting from hydration of bacterial carbon dioxide in the lumen (as stated previously) or from bicarbonate secreted from blood into the colonic lumen, leading to an underestimation of the absorbed amount of 14C and to an overestimation of the conversion factor (acetate to carbon dioxide). This overestimation is a further cause of error when breath 14CO2 coming from VFA oxidation in the body is calculated. Finally, the validity of Miller and Wolin’s equation is crucial. This formula was established for the rumen and adapted to the human large intestine, assuming that VFAs, methane, and carbon dioxide were the sole products of fermentation (7). It can be easily adapted to subjects producing hydrogen and no methane (25). Other equations have been established from the ruminal or large bowel fermentations of mammals (30–32); although they give similar values, ie, 72% of carbon converted to VFAs and 28% to bacterial carbon dioxide, it is obvious that these equations provide only a rough quantitative estimate of the colonic fermentative process. Any alteration in these values could lead to large changes. For example, Hosoya et al (21) found in vitro anaerobic incubations of rat cecal contents that only 9.6% of [U-14C] fructooligosaccharides was converted to bacterial 14CO2. If instead of 28% only 9.6% of the PD carbon had been released as bacterial carbon dioxide during colonic fermentation, the total recovery of the label (after correction by the conversion factor of carbon dioxide from VFAs) would have been 93% of the ingested dose, and the calculated energy value of PD would be close to 5.9 kJ/g. A great improvement would result from a method to experimentally distinguish between bacterially produced carbon dioxide in breath and that resulting from VFA oxidation.

Our calculation of the energy value of PD from theoretical production of VFAs and from VFA- derived 14CO2 resulted in values of 6.1 and 4.0 kJ/g, respectively. These values are not completely independent of each other because they both rely on Miller and Wolin’s equation and on the estimate of radioactivity absorbed in the small bowel. However, the first method takes into account only the amount of PD metabolized in the colon, whereas the second also includes measurement of a metabolite derived from this process. Our results are similar to those of Fidor and Renhard (13) and Fidor and Bianchine (15) obtained in rats and humans. These authors calculated the energy value of PD from breath 14CO2 excretion, which was lower than in our study. In contrast, fecal excretion of radioactivity was higher, at least in three of four subjects, in whom most of the fecal 14C was excreted within 2 d after ingestion of [U-14C]PD. This is, however, unusual compared with the expected transit time of undigestible compounds (33). That the PD energy value was similar to ours is explained by their failure to subtract bacterial carbon dioxide. Cooley and Livesey (14) did not take into account energy losses from fermentation and, although 48% of ingested label was excreted in rat feces, they found an energy value of 7.9 kJ/g.

In conclusion, the present work confirms that the energy value of PD in normal men is low, whether it is calculated from fecal data only or with VFA-derived carbon dioxide in breath. It must be stressed, however, that both calculations rely on the validity of assumptions, which need further confirmation. Second, colonic flora do not adapt to prolonged administration of PD, and tolerance to a high daily intake of PD is excellent.

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References