THE EFFECTS OF ABOMASAL CASEIN INFUSIONS IN GROWING BEEF STEERS ON PORTAL AND HEPATIC FLUX OF PANCREATIC HORMONES AND ARTERIAL CONCENTRATIONS OF SOMATOMEDIN-C

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ABSTRACT

The net release of insulin, glucagon and somatostatin by the portal-drained viscera (PDV) and their net uptake by the liver in response to 3-d abomasal infusions of casein were measured in seven multicatheterized beef steers. The steers were fed 4.3 kg DM/d of a high-concentrate diet in 12 equal meals (13.1 Mcal ME/d and 95 g N/d). In two separate experiments, the abomasal infusion of 300 g casein/d (300C) or 150 g casein/d (150C) was compared to a water infusion. Plasma flow was measured by indicator dilution and net flux by venoarterial concentration difference x plasma flow. Arterial plasma concentrations of insulin were increased (P < .02) by either 300C or 150C. The 300C increased (P < .03) PDV insulin release but did not affect hepatic uptake, resulting in an increased (P < .03) total splanchnic (TSP) insulin flux. The 300C increased (P < .05) plasma concentrations of glucagon as the result of decreased (P < .06) hepatic extraction ratio and not as the result of increased portal release. The portal and hepatic flux of somatostatin measured as somatostatin-like immunoreactivity (SLI) were highly variable and not affected by casein infusions. Arterial plasma concentrations of somatomedin-C were not responsive to abomasal casein infusions. The abomasal infusion of 300C resulted in increased plasma concentrations of insulin via increased PDV release and increased plasma glucagon via decreased hepatic extraction ratio.

Key Words: Bovidae, Protein, Insulin, Glucagon, Somatostatin, Somatomedin


Introduction

Endocrine changes may be a mechanism by which nutrient supply regulates protein metabolism (Reeds and Fuller, 1983). The importance of insulin and glucagon as regulators of hepatic metabolism and the role of insulin as a key regulator of amino acid and glucose uptake by peripheral tissues has been discussed in numerous review articles (Trenkle, 1981; Prior and Smith, 1982; Buttery, 1983; Brockman and Laarveld, 1986). Potential changes in portal somatostatin-like immunoreactivity (SLI) release are important because of the interrelationships between pancreatic somatostatin, insulin and glucagon release.
Peripheral concentrations of pancreatic hormones are the net result of pancreatic release, hepatic removal and uptake by other tissues. Therefore, it is of interest to relate changes in peripheral concentrations in response to post-ruminally infused protein to changes in pancreatic release and hepatic removal.

The objectives of these experiments were 1) to quantify changes in portal-drained visceral, hepatic and total splanchnic (TSP) flux of insulin, glucagon and SLI by growing beef steers in response to abomasal casein infusions; 2) to relate flux changes to circulating hormone concentrations; and 3) to determine whether arterial concentrations of somatomedin-C (SmC) were affected by casein infusions.

**Materials and Methods**

The steers and their surgical preparation, diet composition, experimental design and flux calculations are described elsewhere (Guerino et al., 1991), so only a brief description will be given here. Seven multicatheterized Hereford × Angus beef steers were fed 4.3 kg DM/d of a high-concentrate diet in 12 equal meals. The diet provided 13.1 Mcal ME and 95 g N daily and met NRC requirements for growing beef steers (NRC, 1976). Two separate experiments were conducted. In Exp. 1, steers (284 ± 32 kg) were given an abomasal infusion of 300 g casein/d (300C) for 3 d. The casein, which had a DM content of 91.7% and was 96.2% CP (g Kjeldahl N × 6.38), was dissolved in 9 liters of water. The control was an abomasal infusion of 9 liters water/d and the order in which the seven steers received the casein or water infusions was randomized. The second experiment was identical to the first except that steers (299 ± 30 kg) were abomasally infused with 150 g casein/d (150C).

Blood samples were drawn simultaneously through the various blood sampling catheters (portal vein, hepatic vein and artery) at 30-min intervals for 4 h on the 3rd d of 300C, 150C or water infusions (resulting in eight sets for each steer and treatment). Arterial blood was obtained either through a temporary catheter in the right common carotid artery or a permanent catheter in the abdominal aorta. Blood (15 ml) was drawn into heparinized syringes and was immediately dispensed equally into three different test tubes. Tubes for glucagon analysis were treated with a benzamidine-EDTA solution that provided 4.74 mg benzamidine and 1.2 mg Na$_3$EDTA per milliliter of blood. Tubes for SLI analysis contained 500 KIU aprotinin and 1.2 mg Na$_2$EDTA per milliliter of whole blood. The remaining blood was placed in a tube containing 50 μl of heparin solution (3,507 U/ml) that provided 35.7 U of heparin per milliliter of whole blood and was used for insulin, SmC and plasma para-aminohippurate (PAH) analyses. Samples were kept on ice until they were centrifuged at 1,850 × g at 5°C for 20 min. Plasma was harvested, aliquotted into storage tubes and frozen immediately. Glucagon and SLI plasma samples were stored at −70°C. Plasma aliquotted for insulin, Sm-C and PAH was kept frozen at −20°C. Plasma for each hormone that was measured was stored in a separate tube; individual tubes were thawed on the day they were to be assayed. Plasma PAH concentrations were measured using automated procedures (Huntington, 1984). All hormones were assayed using double-antibody radioimmunoassays, and all samples from any one steer were contained within one assay to eliminate the influence of interassay variation on treatment comparisons.

The tracer that was purchased for the SLI assay was $^{125}$I-try-somatostatin, and $^{125}$I-glucagon was purchased for the glucagon assay. The tracer for insulin and Sm-C assays was iodinated using Iodogen to covalently attach $^{125}$I to tyrosine residues of these hormones. The iodination reaction for insulin was performed in a stopped gas chromatography vial (1.5 ml) for 12 min with 1.5 μg Iodogen, 10 μg bovine insulin and .5 mCi Na$^{125}$I in a .5-M phosphate solution at pH 7.5 in a total volume of 100 μl. The resulting iodinated hormone (assay tracer) was greater than 96% immunoreactive with a specific activity between 50 and 70 μCi/μg. The iodination procedure for Sm-C was the same, with the exception that 2 μg of recombinant threonine-59-substituted human Sm-C$^{13}$ was used to yield tracer; it had a...
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specific activity of approximately 150 µCi/g. Insulin was assayed using a nonequilibrium technique. The primary antibody was guinea pig anti-bovine insulin14. The precipitating second antibody was goat anti-guinea pig immunoglobulin G15. The minimal detectable amount of insulin (sensitivity), defined as 90% relative tracer binding, was 36 pg/tube. For each assay used in these studies, the slope of the displacement of tracer from antibody with increasing volumes of bovine plasma was parallel to the slope of the standard curve. Nonspecific binding was .83%. The intra-assay CV was 5.3% and the interassay CV was 15.3%.

Glucagon was assayed using an equilibrium assay. The primary antibody was Unger 04A rabbit anti-porcine glucagon16. The precipitating second antibody was goat anti-rabbit IgG. The assay sensitivity was 4 pg/tube and the nonspecific binding was 2.75%. The intra-assay CV was 5.9% and the interassay CV was 13.2%.

A delayed addition assay was used to assay SLI in plasma samples. This assay was described in detail by Reynolds et al. (1989). The antiserum to the central portion of somatostatin-14 was raised in rabbits. The assay sensitivity was 7 pg/tube and its nonspecific binding was 1.85%. The intra-assay CV was 12.0%. There is no estimate of interassay CV because the same plasma internal standard was not used for all assays.

Somatomedin-C in arterial plasma was measured via a nonequilibrium assay (Elsasser et al., 1988). The primary antibody was rabbit anti-bovine Sm-C, and the second antibody was goat anti-rabbit IgG. Assay sensitivity was 3 pg/tube and the nonspecific binding was 1.55%. The intra-assay CV was measured at 9.0%. All plasma samples were determined within one assay.

The calculation of plasma flow and flux measurements are as described by Bergman (1975). For each steer and treatment (eight sample sets), the mean venoarterial concentration difference was multiplied by the mean plasma flow to calculate the net flux value. These values were used in the statistical analyses. The hepatic extraction ratio of a hormone is hepatic uptake divided by the supply of the hormone to the liver (Brockman and Bergman, 1975).

Because of problems with catheter patency, we did not obtain blood from all sampling sites for each steer on all treatments. One steer became ill and did not complete the 150C.

Least squares means are presented in this paper. The GLM procedure of SAS (1985) was used for data analysis. Data were analyzed as two separate experiments; each casein infusion was compared to its own water control. Each experiment was a crossover design and the statistical model included steer and treatment as main effects, with steer x treatment as the error term. Period was not included in the model. Treatment differences were considered statistically significant if $P < .10$.

Results

The 300C infusion increased arterial ($P < .02$), hepatic ($P < .01$) and portal ($P < .05$) insulin concentration compared with the water control (Table 1). The 150C infusion also increased arterial ($P < .02$) and portal ($P < .05$) insulin concentrations. The arterial ($P < .02$), hepatic ($P < .02$) and portal ($P < .05$) glucagon concentrations all were increased by 300C (Table 1). The 150C had no effect ($P > .10$) on glucagon concentrations at any of the sampling sites. The concentration of SLI in arterial, hepatic and portal plasma was unaffected ($P > .10$) by either 300C or 150C (Table 1). There were no effects of either 150C or 300C casein infusion on arterial Sm-C concentrations (Table 1).

The 300C increased ($P < .03$) both portal and total splanchic insulin release compared with its control, whereas hepatic insulin uptake was unaffected (Table 2). Greater variation was associated with the insulin flux data from the 150C experiment, as evidenced by the larger SEM (Table 2). This probably was due, in part, to the loss of catheter function that resulted in fewer observations per treatment. There were no treatment differences in insulin flux in the 150C experiment, nor were there effects of 300C or 150C on portal, hepatic or TSP glucagon flux (Table 2).

The SLI data for the comparison of 150C to its water control are complicated by the fact that one steer had much higher SLI concentrations, concentration differences and flux

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TABLE 1. PLASMA HORMONE CONCENTRATIONS (ng/ml) IN GROWING BEEF STEERS ABOMASALLY INFUSED WITH A CASEIN SOLUTION OR WATER

<table>
<thead>
<tr>
<th>Item</th>
<th>Experiment 1&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Experiment 2&lt;sup&gt;b&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>300C Water SEM</td>
<td>150C Water SEM</td>
</tr>
<tr>
<td>Arterial</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Insulin</td>
<td>2.121&lt;sup&gt;d&lt;/sup&gt; (7)</td>
<td>1.676 (7)</td>
</tr>
<tr>
<td>Glucagon</td>
<td>2.73&lt;sup&gt;d&lt;/sup&gt; (7)</td>
<td>2.09 (7)</td>
</tr>
<tr>
<td>SLI&lt;sup&gt;e&lt;/sup&gt;</td>
<td>385 (7)</td>
<td>391 (7)</td>
</tr>
<tr>
<td></td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Hepatic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Insulin</td>
<td>2.498&lt;sup&gt;c&lt;/sup&gt; (6)</td>
<td>1.966 (7)</td>
</tr>
<tr>
<td>Glucagon</td>
<td>310&lt;sup&gt;d&lt;/sup&gt; (6)</td>
<td>236 (7)</td>
</tr>
<tr>
<td>SLI</td>
<td>416 (6)</td>
<td>404 (7)</td>
</tr>
<tr>
<td>Portal</td>
<td>2.688&lt;sup&gt;d&lt;/sup&gt; (6)</td>
<td>2.280 (7)</td>
</tr>
<tr>
<td></td>
<td>0.90</td>
<td>0.90</td>
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</tbody>
</table>

<sup>a</sup>Comparison of the abomasal infusion of 300 g casein/d (300C) to a control water infusion. Parenthetical number equals the number of observations.

<sup>b</sup>Comparison of the abomasal infusion of 150 g casein/d (150C) to a control water infusion.

<sup>c</sup>Casein infusion differs from its water control (P < .01).

<sup>d</sup>Casein infusion differs from its water control (P < .05).

<sup>e</sup>SLI = somatostatin-like immunoreactivity; SmC = somatomedin-C.

measurements than all other steers for the 150C infusion. These samples were reassayed and still yielded unusually high concentrations. We believe the values obtained from this steer on this treatment are outliers and that their use may lead to erroneous conclusions. Therefore, the SLI values for this steer were deleted from the values for both the 150C infusion and its

TABLE 2. PLASMA FLOW (liters/h) AND NET PORTAL, HEPATIC AND TOTAL SPLANCHNIC (TSP) FLUX<sup>a</sup> (ng/ml) OF INSULIN, GLUCAGON AND SOMATOSTATIN-LIKE IMMUNOREACTIVITY (SLI) IN GROWING BEEF STEERS ABOMASALLY INFUSED WITH A CASEIN SOLUTION OR WATER

<table>
<thead>
<tr>
<th>Item</th>
<th>Experiment 1&lt;sup&gt;b&lt;/sup&gt;</th>
<th>Experiment 2&lt;sup&gt;c&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>300C Water SEM</td>
<td>150C Water SEM</td>
</tr>
<tr>
<td>Plasma flow</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Portal</td>
<td>398 (6) 362 (7) 27</td>
<td>367 (6) 373 (6) 7</td>
</tr>
<tr>
<td>Hepatic</td>
<td>477 (6) 439 (7) 28</td>
<td>434 (5) 456 (5) 21</td>
</tr>
<tr>
<td>Insulin flux</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Portal</td>
<td>253&lt;sup&gt;d&lt;/sup&gt; (6) 218 (7) 9</td>
<td>273 (6) 240 (6) 31</td>
</tr>
<tr>
<td>Hepatic</td>
<td>-86 (6) -92 (7) 9</td>
<td>-165 (5) -111 (5) 39</td>
</tr>
<tr>
<td>TSP</td>
<td>172&lt;sup&gt;d&lt;/sup&gt; (6) 126 (7) 10</td>
<td>113 (5) 132 (5) 35</td>
</tr>
<tr>
<td>Glucagon flux</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Portal</td>
<td>28 (5) 27 (7) 2</td>
<td>24 (6) 26 (6) 1</td>
</tr>
<tr>
<td>Hepatic</td>
<td>-13 (6) -15 (7) 2</td>
<td>-15 (5) -14 (5) 1</td>
</tr>
<tr>
<td>TSP</td>
<td>15 (6) 12 (7) 2</td>
<td>9 (5) 10 (5) 1</td>
</tr>
<tr>
<td>SLI flux</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Portal</td>
<td>10 (6) 2 (7) 3</td>
<td>4 (5) 18 (5) 5</td>
</tr>
<tr>
<td>Hepatic</td>
<td>12 (6) 3 (7) 9</td>
<td>5 (4) -6 (4) 12</td>
</tr>
<tr>
<td>TSP</td>
<td>18 (6) 5 (7) 8</td>
<td>9 (4) 15 (4) 4</td>
</tr>
</tbody>
</table>

<sup>a</sup>Positive flux value indicates net release and a negative flux indicates net uptake.

<sup>b</sup>Comparison of the abomasal infusion of 300 g casein/d (300C) to a control water infusion. Parenthetical number equals the number of observations.

<sup>c</sup>Comparison of the abomasal infusion of 150 g casein/d (150C) to a control water infusion.

<sup>d</sup>Casein infusion differs from its water control (P < .03).
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TABLE 3. MOLAR INSULIN/GLUCAGON (I/G) RATIOS AND HEPATIC EXTRACTION RATIOS OF INSULIN AND GLUCAGON IN GROWING BEEF STEERS ABOMASALLY INFUSED WITH A CASEIN SOLUTION OR WATER

<table>
<thead>
<tr>
<th>Item</th>
<th>Experiment 1&lt;sup&gt;a&lt;/sup&gt;</th>
<th></th>
<th></th>
<th></th>
<th>Experiment 2&lt;sup&gt;b&lt;/sup&gt;</th>
<th></th>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>300C</td>
<td>Water</td>
<td>SEM</td>
<td></td>
<td>150C</td>
<td>Water</td>
<td>SEM</td>
<td></td>
</tr>
<tr>
<td>Arterial I/G</td>
<td>5.16 (7)</td>
<td>4.98 (7)</td>
<td>.43</td>
<td></td>
<td>6.17&lt;sup&gt;d&lt;/sup&gt; (6)</td>
<td>5.24 (7)</td>
<td>.34</td>
<td></td>
</tr>
<tr>
<td>Portal I/G</td>
<td>5.14 (6)</td>
<td>5.05 (7)</td>
<td>.41</td>
<td></td>
<td>6.43 (6)</td>
<td>5.56 (6)</td>
<td>.46</td>
<td></td>
</tr>
<tr>
<td>Extraction ratio</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Insulin</td>
<td>.078 (6)</td>
<td>.101 (7)</td>
<td>.010</td>
<td></td>
<td>.141 (5)</td>
<td>.106 (5)</td>
<td>.043</td>
<td></td>
</tr>
<tr>
<td>Glucagon</td>
<td>.083&lt;sup&gt;c&lt;/sup&gt; (6)</td>
<td>.132 (7)</td>
<td>.015</td>
<td></td>
<td>.126 (5)</td>
<td>.115 (5)</td>
<td>.011</td>
<td></td>
</tr>
</tbody>
</table>

<sup>a</sup>Comparison of the abomasal infusion of 300 g casein/d (300C) to a control water infusion. Parenthetical number equals the number of observations.

<sup>b</sup>Comparison of the abomasal infusion of 150 g casein/d (150C) to a control water infusion.

<sup>c</sup>Casein infusion differs from its water control (P < .06).

<sup>d</sup>Casein infusion differs from its water control (P < .09).

Discussion

In this study, the percentage of portal insulin release removed by the liver was 34.0% and 42.2% for 300C and its water control. For 150C and its control, hepatic removal of portal insulin release was 60.4 and 46.2%, respectively. Huntington and Eisemann (1988) reported a range for ruminants of between 50 and 83% of hepatic removal of portal insulin. However, only a single study with beef steers included a range of hepatic removal values. The hepatic extraction ratios in the present study (Table 3) ranged from .078 (300C) to .141 (150C), and they agree with published values (Huntington and Eisemann, 1988).

The 300C increased (P < .03) portal insulin release but did not affect (P > .10) hepatic uptake (Table 2); thus, TSP insulin release was increased (P < .02). The increased TSP release of insulin is reflected in increased arterial insulin concentration (Table 1). Therefore, in the case of the 300C infusion, a portion of the rise in circulating insulin concentrations (arterial, hepatic and portal) is explained by increased pancreatic release. Brockman and Bergman (1975) demonstrated a positive relationship between portal insulin release and arterial insulin concentrations in sheep. In goats, the same pattern for changes in insulin concentrations was seen in portal and jugular vein samples (De Jong, 1981).

The relationship between arterial concentrations and TSP insulin release is less clear for 150C. An explanation for increased arterial insulin concentrations without increased TSP flux for 150C (Table 2) could be that the clearance rate of insulin by peripheral tissues was decreased by 150C casein infusion. The number of insulin receptors and the binding of insulin to various tissues can be inversely related to the concentrations of insulin to which cells are exposed due to up- or down-regulation (Tepperman and Tepperman, 1985).
The concentration of insulin in peripheral blood of nonruminants increased with increasing dietary protein in previous studies (Edozien et al., 1978; Usami et al., 1982; Demigne et al., 1985; Jeffson et al., 1988). Increased jugular insulin concentrations have been measured in sheep abomasally infused with casein (Barry et al., 1982) and sheep fed formaldehyde-treated casein (Faichney and Weston, 1971). The insulin data presented here (Table 2) and the α-amino N data for these steers (Guerino et al., 1991) also demonstrate a positive relationship between amino acid absorption and pancreatic insulin secretion. A positive relationship between a postruminal supply of protein and plasma insulin concentrations in sheep has been demonstrated (Bassett et al., 1971). Further, Waghorn et al. (1987) found that insulin concentrations are higher in growing sheep fed high- vs low-protein diets.

Hepatic uptake of glucagon as a percentage of portal glucagon release was 46.4, 55.6, 62.5 and 53.8% for 300C and its control and 150C and its control, respectively. These values are within the range summarized previously for ruminants (Huntington and Eisemann, 1988). Decreases in hepatic glucagon extraction have been reported previously as a means by which circulating concentrations may increase (Dobbs and Unger, 1982). The tendency for decreased hepatic glucagon extraction measured in these steers in response to 300C is consistent with the concept of down-regulation of the hepatic glucagon receptor seen during exogenous glucagon administration (Srikant et al., 1977). Jugular glucagon concentrations have been shown to increase in growing sheep (Barry et al., 1982), lactating goats (Rodriquez et al., 1985) and lactating dairy cows (Cohick et al., 1986) given abomasal casein infusions. The increase in arterial glucagon by beef steers in response to 300C (Table 1) is consistent with the majority of previously published results and appears to be the result of decreased hepatic extraction, not greater portal release. These data raise the question of whether the increases in glucagon concentrations seen in previously published casein infusion studies are the result of increased pancreatic release or of decreased hepatic extraction.

Because insulin promotes protein anabolism and glucagon promotes protein catabolism, the molar ratio of insulin to glucagon (I/G) is a useful indicator of the metabolic state of an animal (Unger, 1971). Portal vein I/G ratio may be more meaningful in describing the relative actions of these hormones on the liver (Bassett, 1975). In these experiments with postruminal casein, portal vein I/G did not yield new information beyond the arterial concentration ratios.

In response to postruminal casein infusions, jugular vein plasma somatostatin concentrations were decreased in nonlactating sheep (Barry et al., 1982) and increased in lactating ewes (Barry, 1980). The portal and hepatic SLI flux measurements (Table 2) were highly variable, as noted previously in beef steers (Reynolds et al., 1986) and in dairy cows (Reynolds et al., 1989). In addition to the pancreatic D cell, somatostatin is produced throughout the gastrointestinal tract of cattle (Kitamura et al., 1985). This fact, together with the evidence that somatostatin may act via endocrine, paracrine and neurocrine mechanisms (Grossman, 1979; Schusdziarra, 1985), complicates the interpretation of portal SLI flux data.

Somatomedin-C concentrations were measured only in arterial blood because previous studies failed to detect portal or hepatic flux (McGuire et al., 1988; Lapierre et al., 1989; Reynolds et al., 1989). Our interest in measuring Sm-C concentrations was generated from the suggestion that plasma Sm-C concentrations may be positively related to energy and protein intake and possibly growth rate (Prewitt et al., 1982). Crude protein intake has been shown to increase plasma Sm-C concentrations in beef steers. This relationship also may be affected by ME intake (Elsasser et al., 1989). Those changes in Sm-C concentrations in response to dietary protein were measured over the course of 84 d and at lower protein intakes than in these casein infusion experiments. The failure of plasma Sm-C concentrations to increase in these steers abomasally infused with casein suggests that 3 d of increased protein intake is not sufficient time to cause a significant response. Much of the data that show the positive relationship between Sm-C concentrations and protein and(or) energy intake have been derived from studies in which animals were fasted or in negative energy and protein balance so that concentrations rose as nutritional status improved. The steers in our study were in both positive energy and protein balance prior to being given abomasal casein.
In response to abomasal casein infusions, portal insulin release and arterial concentrations of insulin were increased. The increased arterial glucagon concentrations seen in response to abomasal infusion of 300C were the result of a decreased hepatic extraction ratio (Table 3) and not an increased portal release (Table 2). Changes in circulating concentrations, portal or hepatic flux of SLI were not significant. Arterial Sm-C concentrations were not affected by casein infusions. Of the endocrine changes measured in this study, the increased insulin release and peripheral concentrations suggest that changed endocrine status may explain at least part of the improved N balances reported for growing ruminants in response to abomasal casein. However, the ability of chronic elevations of plasma insulin concentrations to have a direct and lasting effect on protein synthesis has not been demonstrated (Bell et al., 1987; Reeds, 1987).

Implications

Many hormones, including those produced by the pancreas, regulate how animals use nutrients. A better understanding of how hormone secretion responds to dietary changes may aid us in our ability to manipulate plasma concentrations. The data presented in this paper address how the secretion and uptake of insulin, glucagon and somatostatin by the splanchnic tissues of growing beef steers change in response to increases in dietary protein intake. The increases in insulin secretion by the pancreas caused by increased protein intake are consistent with insulin’s ability to promote nutrient storage by animal tissue.

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