Differential Effect of Gluten and Casein Diets on Rat Liver HMP Shunt Dehydrogenases

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ABSTRACT The effect of diets containing as their protein component wheat gluten or casein, fed ad libitum to adult male rats for 1 to 6 days, after 6 days of protein deprivation, on the liver levels of glucose-6-phosphate dehydrogenase (G6PD) and 6-phosphogluconate dehydrogenase (6PGD) were studied. In rats fed the gluten diet the values of body weight gain, liver size, liver protein, liver xanthine oxidase, G6PD and 6PGD activities were lower in comparison with rats fed the casein diet. Amino acid supplements to the gluten diet improved the nutritive value of this diet, as shown by the enhancement of body weight gain, liver size, liver protein and xanthine oxidase activity to levels found in rats fed the casein diet, but failed to increase G6PD and 6PGD activities. Essential fatty acids added to the casein diet, in order to bring their level up to that of the gluten diet, proved incapable of decreasing the levels of G6PD and 6PGD activities found in rats fed the casein diet. It was concluded that the differential effect of gluten and casein on liver G6PD and 6PGD is independent of differences in the nutritive value and in the essential fatty acid content of these proteins, and is dependent upon one or more factors different from those known to affect the liver HMP shunt dehydrogenases.

INDEXING KEY WORDS gluten - casein - liver - HMP shunt dehydrogenases

It has been previously found that the increase in the hepatic levels of glucose-6-phosphate dehydrogenase (G6PD, EC 1.1.1.49) in rats fed a normal protein diet after protein deprivation was greater when casein replaced wheat gluten or soy protein as the dietary protein component (1, 2). To explain these findings the investigation was limited to gluten and casein, since these proteins demonstrated the greatest influence over the hepatic levels of G6PD. This, however, does not imply that the results concerning casein and gluten can be implicitly applied to soy protein or to other proteins, which may have the same action upon G6PD as gluten or casein.

It is known that wheat gluten, compared to casein, has a lower nutritive value (3) and contains higher amounts of essential fatty acids (4). Since dietary proteins increase (5-7) and, conversely, essential fatty acids decrease (8-15) liver G6PD, it was relevant to enquire if the different enzyme response to gluten and casein may be ascribed to the different nutritive value of these proteins or to the essential fatty acid content of gluten. It has been demonstrated not only that the nutritive value of dietary protein is highly correlated with the activity of liver xanthine oxidase (EC 1.2.3.2) (16-20) and of kidney transamidinase (21), but also that the essential fatty acids are largely involved in the lowering effect manifested by wheat gluten on dietary hypercholesterolemia (22). Therefore, in this study we have examined the effects on G6PD, firstly, of amino acid supplements to the gluten diet and, secondly, of essential fatty acid increases in the casein diet. The research has also been extended to the other oxidative enzyme of the hexose monophosphate (HMP) shunt, 6-phosphogluconate dehydrogenase (6PGD, EC 1.1.1.44).

The results demonstrate that both HMP...

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shunt dehydrogenases are coordinately influenced by dietary proteins and that neither low nutritive value nor high fatty acid content are involved in the lower activity of the liver enzymes in response to gluten when compared to that in response to casein.

MATERIALS AND METHODS

Male albino rats of the Wistar-Glaxo strain, fed a commercial standard diet from weaning, were used. The animals were housed in individual cages in an environment at constant temperature (23°) and had free access to water and food throughout all the experiments.

First, a carbohydrate-rich, protein-free diet containing 33% water and the following ingredients,1 (g by dry weight): olive oil, 3; corn oil, 1; Hawk-Oser salt mixture with additional Zn and Mn,2 4; vitamin mixture,3 1; dextrinized starch up to 100 was fed for 6 days. Successively, a diet containing as its protein component either 20.4% casein or 21.3% wheat gluten20 was fed. The nitrogen content of the diet was 2.6%, supplied by either casein or wheat gluten. The nitrogen content of the casein and of the wheat gluten used was 12.9% and 12.4%, respectively. These and other additions, reported in the tables, were made by replacing equal amounts of starch.

At preselected times, between 9 and 10 AM, the animals were decapitated and their livers removed. The enzyme activities were assayed at 25°, using Bottomley’s II method for the G6PD and 6PGD, and Della Corte’s method for the total xanthine oxidase. Total liver protein by biuret method and glycogen were also determined. In all cases body weight changes and food intake were recorded. The results were related to body weight at the end of protein deprivation and in the majority of cases the enzyme activities were related, as well, to the protein values.

RESULTS

Rats fed a gluten or a casein diet ate the same amount of food; therefore, differential effects brought about by these diets are not to be ascribed to a different energy intake but rather to specific components of the dietary proteins.

The effects of feeding a gluten or a casein diet for 1, 2, 4 and 6 days after protein starvation are shown in table 1. Both diets caused a gain in body weight and, in comparison with rats at the end of protein deprivation, an evident increase in liver size, in liver protein content and in liver 6PGD and G6PD activities. However, the casein diet, compared with the gluten diet, tended to increase more markedly body weight, liver size, liver protein—but the differences were statistically significant only for liver protein on day 6—and caused significantly higher values of G6PD and 6PGD activities from day 2 and day 1, respectively. Liver glycogen was high with both diets and tended to be higher during the first 2 days in rats fed the gluten diet. Since there were significant differences in the levels of the HMP shunt dehydrogenases between rats fed the diets with gluten and those fed the diets with casein on each day from day 2 to day 6 of feeding, the enzyme assays in subsequent experiments were carried out only on rats fed gluten or casein diets for 6 days.

The results in table 2 show that with larger numbers of rats the differences between the effects of casein and gluten diets on weight gain, liver size and liver proteins were all significant. Moreover, the supplementation of the gluten diet with lysine; lysine and threonine; or with an amino acid mixture comprising lysine, threonine, histidine, isoleucine, leucine,
### Table 1

**Effect of feeding gluten or casein diets on several body and liver parameters**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>0 day</th>
<th>1 day</th>
<th>2 day</th>
<th>4 day</th>
<th>6 day</th>
</tr>
</thead>
<tbody>
<tr>
<td>Days of feeding and nature of the dietary protein</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Original body wt, g</td>
<td>305 ± 3.6¹</td>
<td>300 ± 5.2</td>
<td>302 ± 5.2</td>
<td>310 ± 6.5</td>
<td>315 ± 6.0</td>
</tr>
<tr>
<td>Body wt at end of protein deprivation, g</td>
<td>275 ± 5.1</td>
<td>273 ± 6.6</td>
<td>275 ± 5.4</td>
<td>286 ± 7.3</td>
<td>291 ± 3.9</td>
</tr>
<tr>
<td>Cumulative post-deprivation food intake, dry wt, g/100 g body wt</td>
<td>—</td>
<td>5.6 ± 0.23</td>
<td>6.1 ± 0.55</td>
<td>13.0 ± 1.3</td>
<td>12.5 ± 0.93</td>
</tr>
<tr>
<td>Body wt at time of killing, g</td>
<td>—</td>
<td>279 ± 7.5</td>
<td>281 ± 4.9</td>
<td>293 ± 6.8</td>
<td>303 ± 2.7</td>
</tr>
<tr>
<td>Cumulative post-deprivation body wt gain, g/100 g body wt</td>
<td>—</td>
<td>1.95 ± 0.44</td>
<td>2.24 ± 0.85</td>
<td>2.26 ± 0.66</td>
<td>3.90 ± 0.78</td>
</tr>
<tr>
<td>Liver size g/100 g body wt</td>
<td>3.40 ± 0.06</td>
<td>4.12 ± 0.04</td>
<td>4.33 ± 0.14</td>
<td>4.46 ± 0.07</td>
<td>4.32 ± 0.11</td>
</tr>
<tr>
<td>Liver protein mg/100 g body wt</td>
<td>573 ± 14.5</td>
<td>693 ± 13.0</td>
<td>705 ± 22.0</td>
<td>686 ± 11.0</td>
<td>719 ± 20.8</td>
</tr>
<tr>
<td>Liver glycogen mg/100 g body wt</td>
<td>348 ± 19.9</td>
<td>559 ± 39.0</td>
<td>474 ± 49.0</td>
<td>571 ± 52.0</td>
<td>286 ± 19.0 **</td>
</tr>
<tr>
<td>G6PD units/100 g body wt</td>
<td>7.1 ± 0.66</td>
<td>10.1 ± 1.2</td>
<td>25.4 ± 7.3</td>
<td>16.6 ± 7.0</td>
<td>49.6 ± 6.4 **</td>
</tr>
<tr>
<td>G6PD units/g protein</td>
<td>12.5 ± 1.3</td>
<td>14.7 ± 1.8</td>
<td>35.4 ± 9.3</td>
<td>34.4 ± 10.3</td>
<td>69.1 ± 8.4 *</td>
</tr>
<tr>
<td>6PGD units/100 g body wt</td>
<td>14.4 ± 0.88</td>
<td>13.5 ± 0.34</td>
<td>18.5 ± 1.6 *</td>
<td>21.3 ± 2.9</td>
<td>32.8 ± 1.8 *</td>
</tr>
<tr>
<td>6PGD units/g protein</td>
<td>25.2 ± 1.5</td>
<td>19.5 ± 0.79</td>
<td>26.2 ± 2.1 *</td>
<td>31.1 ± 4.3</td>
<td>45.4 ± 2.5 *</td>
</tr>
</tbody>
</table>

¹ Value at end of protein deprivation. 
² In parentheses is given the number of rats. 
³ Mean ± SE. 
⁴ One asterisk indicates a statistical significance between values on the same day at *P < 0.05. 
⁵ Two asterisks indicate a statistical significance between values on the same day at **P < 0.01. 
⁶ One unit represents the reduction of 1 μ mole of NADP per minute under the assay conditions.
TABLE 2
The effect of l-amino acid supplements to gluten diet on several body and liver parameters

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Glutens 1% (14)</th>
<th>Glutens Lys-HCl 1% (15)</th>
<th>Glutens Lys-HCl 0.4% (16)</th>
<th>Glutens amino acid mixture (16)</th>
<th>Casein (33)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Original body wt, g</td>
<td>282 ± 5.2 *</td>
<td>290 ± 4.6</td>
<td>292 ± 3.7</td>
<td>286 ± 5.0</td>
<td>294 ± 3.3</td>
</tr>
<tr>
<td>Body wt at end of protein deprivation, g</td>
<td>270 ± 4.8</td>
<td>280 ± 4.5</td>
<td>289 ± 3.8</td>
<td>281 ± 6.0</td>
<td>271 ± 3.3</td>
</tr>
<tr>
<td>Cumulative post-deprivation food intake, g</td>
<td>30.1 ± 1.3</td>
<td>35.3 ± 1.0</td>
<td>38.6 ± 1.1</td>
<td>39.1 ± 2.0</td>
<td>38.7 ± 1.0</td>
</tr>
<tr>
<td>Body wt at time of killing, g</td>
<td>291 ± 5.6</td>
<td>311 ± 5.0</td>
<td>300 ± 3.7</td>
<td>285 ± 5.7</td>
<td>301 ± 3.2</td>
</tr>
<tr>
<td>Cumulative post-deprivation body wt gain, g/100 g body wt</td>
<td>8.00 ± 0.77 a</td>
<td>11.14 ± 0.88 b</td>
<td>11.39 ± 0.60 B</td>
<td>12.79 ± 1.32 B</td>
<td>11.14 ± 0.66 B</td>
</tr>
<tr>
<td>Liver size g/100 g body wt</td>
<td>3.89 ± 0.11 A</td>
<td>4.23 ± 0.11 BC</td>
<td>4.22 ± 0.08 B</td>
<td>4.49 ± 0.14 c</td>
<td>4.29 ± 0.07 b</td>
</tr>
<tr>
<td>Liver protein mg/100 g body wt</td>
<td>722 ± 22.7 A</td>
<td>848 ± 22.5 B</td>
<td>831 ± 20.0 B</td>
<td>888 ± 25.8 B</td>
<td>875 ± 15.2 B</td>
</tr>
<tr>
<td>Liver glycogen mg/100 g body wt</td>
<td>251 ± 37.5</td>
<td>273 ± 31.5</td>
<td>259 ± 22.4</td>
<td>193 ± 21.6</td>
<td>245 ± 16.0</td>
</tr>
<tr>
<td>GPD units/100 g body wt</td>
<td>15.7 ± 2.9 A</td>
<td>21.0 ± 2.7 A</td>
<td>17.6 ± 1.7 A</td>
<td>19.0 ± 2.8 A</td>
<td>41.2 ± 3.1 B</td>
</tr>
<tr>
<td>GPD units/g protein</td>
<td>18.9 ± 2.7 A</td>
<td>24.2 ± 2.8 A</td>
<td>20.9 ± 1.7 A</td>
<td>21.1 ± 3.1 A</td>
<td>40.2 ± 2.8 B</td>
</tr>
<tr>
<td>6PGD units/100 g body wt</td>
<td>17.6 ± 1.0 A</td>
<td>24.4 ± 1.4 b</td>
<td>21.4 ± 1.4 AB</td>
<td>21.4 ± 1.2 AB</td>
<td>29.6 ± 1.3 d</td>
</tr>
<tr>
<td>6PGD units/g protein</td>
<td>24.6 ± 1.5 A</td>
<td>28.6 ± 1.2 a</td>
<td>25.4 ± 1.4 A</td>
<td>24.3 ± 1.7 A</td>
<td>33.8 ± 1.3 b</td>
</tr>
</tbody>
</table>

*In parentheses is given the number of rats. 1Lys-HCl 1%; Thr 0.4%; His 0.15%; Ile 0.3%; Leu 0.4%; Met 0.15%; Tyr 0.3%; Val 0.4%. *Mean ± se. *Different small letters indicate a statistical significance at P < 0.05. If at least one of the different letters is in capitals, statistical significance is at P < 0.01. *One unit represents the reduction of 1 mole of NADP per minute under the assay conditions.
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TABLE 3
The effect of amino acid supplements to gluten diet on total liver xanthine oxidase

<table>
<thead>
<tr>
<th>Dietary nitrogenous component</th>
<th>Glutamin</th>
<th>Lys-HC1 1%</th>
<th>Gluta</th>
<th>aminoacid</th>
<th>Casein</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(5)</td>
<td>(5)</td>
<td></td>
<td>mixture²</td>
<td>(5)</td>
</tr>
<tr>
<td>Xanthine oxidase units/100 g body wt</td>
<td>780 ± 50.1 A</td>
<td>1178 ± 52.6 B</td>
<td>1323 ± 133.2 B</td>
<td>1103 ± 81.6 B</td>
<td></td>
</tr>
<tr>
<td>Xanthine oxidase units/g protein</td>
<td>1042 ± 101.7 A</td>
<td>1437 ± 59.6 B</td>
<td>1478 ± 82.4 B</td>
<td>1339 ± 68.6 B</td>
<td></td>
</tr>
</tbody>
</table>

1 In parentheses is given the number of rats. ² See table 2. ³ Mean ± se. ⁴ Different capitals letters indicate statistical significance at P < 0.01. If different letters are not both capitals, the statistical significance is at P < 0.05. ⁵ One unit represents the production of 1 µmole per minute of uric acid under the assay conditions.

In table 5 are shown the levels of HMP shunt dehydrogenases after a suitable supplementation of the casein diet by essential fatty acids in order to make its essential fatty acid content comparable to that of the gluten diet. It may be seen that an increase in essential fatty acid supply, within our limits, did not produce any relevant effect upon HMP shunt dehydrogenases, and further that differences between the rats fed the gluten diet and those fed the casein diet were still statistically significant.

DISCUSSION
According to our previous work (1, 2), the levels of both HMP shunt dehydrogenes...

...methionine, tyrosine, tryptophan and valine (all in L-form) improved the nutritive value of the gluten diet. However, the increase in the nutritive value of the gluten diet brought about by amino acid supplements had little effect upon the HMP shunt dehydrogenases.

Since some liver parameters associated with protein biosynthesis are not correlated with the nutritive value of several dietary proteins on the basis of body weight gain (30), and since liver xanthine oxidase is a more reliable index than liver protein of the fulfillment of amino acid requirements (31), it was relevant to assay this enzyme in order to enquire if the amino acid supplements were effectively capable of removing the effects upon the liver caused by the deficiency of amino acids. Table 3 shows that the gluten diet induced a lower level of xanthine oxidase than did the casein diet, and that amino acid supplements, under our conditions, were capable of increasing the levels of xanthine oxidase activity observed in rats fed the gluten diet compared with those found in rats fed the casein diet.

It has been suggested that liver HMP shunt dehydrogenases are increased by carbohydrate intake (32-34). Therefore, since amino acid supplements to the gluten diet slightly diminished starch content, we enquired whether a stimulation of HMP shunt dehydrogenases elicited by amino acids might be masked by a reduction in starch supply, although it seemed highly unlikely that minor differences in dietary carbohydrate content could influence the HMP shunt dehydrogenases. Table 4 shows that, in accordance with our expectations, increasing proteins by 5% at the expense of starch did not modify the levels of HMP shunt dehydrogenases. Therefore, the general failure of amino acid supplements to increase enzyme levels did not depend upon the slight reduction in carbohydrate supply.

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TABLE 5

Effect of essential fatty acid supplements to casein diet upon liver HMP shunt dehydrogenases

<table>
<thead>
<tr>
<th>Dietary protein component</th>
<th>EFA source g% of dry wt diet</th>
<th>EFA g/100 g of diet, dry wt</th>
<th>Food intake*</th>
<th>G6PD*</th>
<th>6PGD*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>C18:2</td>
<td>C18:3</td>
<td>C18:2</td>
<td>C18:3</td>
<td>C18:2</td>
</tr>
</tbody>
</table>
| Casein (8)               | 1.2   | 0.040 | 46.3±1.3
|                          |       |       |       |       |       |       |       |       |       |       |       |       |
| Gluten' (8)              | 1.2   | 0.058 | 45.2±1.6
|                          |       |       |       |       |       |       |       |       |       |       |       |       |
| Casein (6)               | 0.06  | 0.031 | 43.0±2.0
|                          |       |       |       |       |       |       |       |       |       |       |       |       |
|                          |       |       |       |       |       |       |       |       |       |       |       |       |
| Casein (6)               | 0.06  | 0.031 | 43.0±2.0
|                          |       |       |       |       |       |       |       |       |       |       |       |       |
|                          |       |       |       |       |       |       |       |       |       |       |       |       |
|                          |       |       |       |       |       |       |       |       |       |       |       |       |
|                          |       |       |       |       |       |       |       |       |       |       |       |       |

* Dietary proteins were dissolved in 30% KOH and subsequently fat saponificated in ethanolic (50%) 15% KOH. Fatty acids were extracted by conventional methods and analyzed by gas-liquid chromatography. A similar procedure was adopted for oils. Absolute values were obtained by the method of internal standard (acid C17:0). * Cumulative post deprivation food intake, dry wt g/100 g body wt. * Units/100 g body wt. One unit represents the reduction of 1 n mole of NADP per minute under the assay conditions. * In parentheses is given the number of rats. * Mean ±SE. * Different letters indicate statistical significance at P < 0.01. * Gluten and supplements of Lys-HCl, 1% and Thr, 0.4%. * Carlo Erba, Milano, Italy.

ases were higher in the liver of the rats fed the casein diet, as compared to those fed the gluten diet.

Amino acid supplements increased the nutritive value of the gluten diet to that of the casein diet, as shown by body weight gain, liver size, total liver proteins and liver xanthine oxidase activity, but they failed to modify the hepatic levels of the HMP shunt dehydrogenases. Such a finding allows us to assert that the difference in nutritive value between gluten and casein is not involved in their differential effect on liver HMP shunt dehydrogenases. Moreover, their different essential fatty acid levels do not account for this effect since the increase in essential fatty acid levels of the casein diet up to that of the gluten diet was not able to modify the enzyme activities.

The differential liver response to the dietary proteins is quite selective because, when the amino acid requirements were fully supplied, the levels of protein, of xanthine oxidase activity and of some other enzymes, as glutamic oxalacetic transaminase, glutamic pyruvic transaminase, serine dehydratase, tryptophan pyrrolase and histidase, regulated like HMP shunt dehydrogenases by protein intake, were the same in the livers of rats fed either the gluten or the casein diet. Mauron et al. (35) have recently shown that in the livers of rats fed a nonsupplemented gluten diet the serine dehydratase activity curve, as a function of the dietary protein level, is still similar to that of rats fed the casein diet.

It is well known that food intake stimulates hepatic levels of the HMP shunt dehydrogenases (32, 36). Recently, it has been postulated that some factors influencing these levels may act through their effect on the appetite (33, 34, 37). However, in our experiments the intake of the gluten or casein diet was the same and, therefore, the differences elicited by these diets cannot be ascribed to such an effect.

A number of dietary manipulations regarding the amount of protein (5–7, 38) or cholesterol (39), the qualitative and quantitative supply of carbohydrate (33, 34, 40–42) or fatty acids (8–15) as well as mineral (43) or vitamin (44) deficiencies are known to affect liver HMP shunt dehydrogenases. Yet none of these dietary actions can explain the differences observed in our research. Indeed a) there were only slight differences in the amount of protein or in carbohydrate consumed between rats fed the gluten diet and those fed the casein diet; b) the supply of essential fatty acids when perfectly equalized did not modify
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the difference in enzyme levels; c) the low enzymatic levels encountered in the liver of rats fed the gluten diet cannot be ascribed to cholesterol because of its low content in wheat lipids (45); d) the supply of vitamins and minerals was sufficient to meet the growth needs of the rat.

Therefore, it may be suggested that the differential impact of gluten and casein on the liver, at least as concerns HMP shunt dehydrogenases, is ascribable to one or more gluten or casein factors, different from any factors previously listed, and able to cause a decrease or an increase, respectively, in enzyme levels. Research on their features is now in progress.

Camus et al. (46) have reported that rats previously fed a 7% gluten diet and successively, after 24 hours starvation, a 18% casein diet showed an increase in the liver HMP shunt dehydrogenases which was not observed when the gluten diet replaced the casein diet. The low stimulative capacity of the gluten diet was attributed by the authors to amino acid deficiency. However, after our findings, such a result cannot be so easily explained because factors other than the low nutritive value of the gluten diet may be involved in the differential stimulation upon liver HMP shunt dehydrogenases by casein and gluten.

Nevertheless, it is possible that at a low gluten content the amino acid deficiency may wholly account for the effect observed by the above-mentioned authors.

Incidentally, an increase of the liver HMP shunt dehydrogenases has been reported in rats fed semisynthetic casein-containing diets, in comparison with those fed standard laboratory diets (33, 42, 47, 48). Our results do not allow us to exclude quantitative differences between these two diets in factors so far not recognized as acting upon the HMP shunt dehydrogenases.

The possibility of influencing the HMP shunt dehydrogenases is noteworthy since it is generally felt that modifications of such levels are associated with important metabolic repercussion. In fasting (49–51), in refeeding after starvation (47, 52), in meal-feeding (53–56), in feeding high fat (15, 52) or orotic acid diets (57) and in diabetes (36, 58, 59) it was found that the changes in the levels of the HMP shunt dehydrogenases are closely correlated to variations in the rate of fatty acid synthesis. The modification in the enzyme levels has been related to NADPH requirements for the reductive synthesis of the fatty acids. Although it is held that an increase in the rate of fatty acid synthesis is the stimulus triggering the enzymatic adaptation (54, 60), it seems highly likely that the levels of HMP shunt dehydrogenases may influence the maximal capacity for fatty acid synthesis. However, it is known that 1) the rate of fatty acid synthesis without corresponding modifications in the HMP shunt dehydrogenases is increased by refeeding a protein-free diet after starvation (61) or by B-6 deficiency (44); 2) the regulation of the levels of the HMP shunt dehydrogenases has been ascribed to NADPH-dependent metabolic processes different from fatty acid synthesis like elongation or desaturation of the fatty acids (59, cholestrol synthesis (39) and steroid reduction (62). Therefore, more work is necessary in order to clarify the metabolic functions of the dietary actions on liver HMP shunt dehydrogenases shown in this paper.

LITERATURE CITED

38. Szepesi, B. & Freedland, R. A. (1968) Time-course of changes in rat liver enzyme...