The Influence of Pancreatic Secretions on the Fat Tolerance of Humans

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A high fat meal was given to 78 volunteers. Total esterified fatty acid, cholesterol, and lipase levels were determined in a fasting blood specimen and in specimens obtained 3 and 6 hours after the meal. Attempts were made to correlate the fat tolerance of the individual with the level of fat splitting enzymes in the various serum specimens. The influence of Pancreatic Hormone, whole pancreatic preparations, secretin, and heparin on the fat tolerance as well as on the lipase level was determined. Attempts were made to correlate the findings of fat tolerance and glucose tolerance tests done on the same individuals.

At the present time there is no agreement concerning the pathogenesis of atherosclerosis. The majority of authors believe, however, that a high fat diet and a disturbed fat metabolism are among the factors contributing to this disease. Several tests are available which indicate such abnormalities in fat metabolism and/or demonstrate atherogenic susceptibility. The fat tolerance test has proven to be especially valuable.

Some authors feel that the disturbance in fat metabolism is due to abnormalities in enzyme systems, and that it is most probably related to a deficiency in fat splitting enzymes. Experiments in this regard have been carried out but they have not resulted in agreement. Deficiencies in lipase or clearing activity in old people or in persons with atherosclerosis or related disorders have been reported by several authors. Other authors reported that there is no decrease in lipolytic or clearing activity in older people. Variations in results may have been related to differences in methods employed. In addition, all determinations referred to were done on fasting patients. Such studies, however, should include the investigation of enzyme behavior in relation to different nutritional states. This paper, therefore, refers to changes in the total esterified fatty acids (TEFA), lipase and cholesterol levels before and after test meals associated with the use of certain drugs.

Methods

Total Esterified Fatty Acids (TEFA)

The determination of the total esterified fatty acids has been carried out according to Nailor, Bauer, and Hirsch. We used 100 per cent ethanol and ether (1:1) for lipid extraction, however.

Total Cholesterol

The total cholesterol was determined with a method employing the Liebermann-Burchard reaction.

Serum Lipase

The determination of serum lipase was done according to Tietz, Borden, and Stepleton.

Glucose

Determinations of glucose in blood were carried out with a revised Folin-Wu method.

Test Meals

Meal no. 1: toast, 2 slices; bacon, 5 strips (25 Gm. fat); butter, 2 pats (10 Gm. fat); and cream, q.s. to 2 Gm. fat/Kg. body wt., maximum dose 400 ml. Meal no. 2: test meal no. 1 plus 8 tablets whole pancreatic preparation (Enseals Pancreatin, 0.325 Gm. triple strength, Eli Lilly & Co.). Meal no. 3: skim milk, 250 ml; toast, 2 slices; and 8 tablets whole pancreatic preparations (Enseals Pancreatin, 0.325 Gm. triple strength,

†See footnote on next page.
Results

The Change of Serum TEFA, Lipase, and Cholesterol Levels after a High Fat Meal (Fat Tolerance Test)

We mentioned above that we consider the levels of fat splitting enzymes in fasting specimens less significant than the levels found after test meals. In order to measure the response of human beings in regard to lipase activity we administered test meal number 1 to 78 human volunteers and determined the TEFA, lipase, and cholesterol levels in a fasting serum specimen as well as in specimens drawn 3 and 6 hours after the meal. Each specimen consisted of 20 ml. of blood which was obtained by venous puncture and allowed to clot. No preservative or anti-coagulant was used.

TEFA Levels

The levels of total esterified fatty acids (TEFA) found in the fasting, 3 hour and 6 hour specimens were plotted in the form of a curve which represented the fat tolerance of the individual. Presumably normal individuals had a fasting TEFA level within the normal range (7 to 16 mEq/L) which was followed by an increase in the 3 hour specimen. The 6 hour specimen showed a decline in TEFA levels. Individuals who demonstrated a further TEFA increase in the 6 hour specimen (from here on referred to as high type curve) were considered as having a low fat tolerance. In this interpretation of the findings we agree with reports given by Hirsch and co-workers,\(^8\) Winston, and Dutrey,\(^2\) and many others.

Five volunteers showed an extremely small increase in the TEFA level. (Fasting: 12.2 mEq/L ± 1.92 S.D.; 3 hr.: 12.9 mEq/L ± 1.51 S.D.; 6 hr.: 11.3 mEq/L ± 1.17 S.D.) It is of interest that all 5 of these persons were females below 45 years of age. All 10 volunteers who had had a previous myocardial infarction exhibited a high type TEFA curve. This was observed without exception.

We found only a limited correlation between the age of a volunteer and the type of the TEFA curve. Curves of all types were found in all age groups. This is in contrast to the findings of Becker, Meyer, and Nacheless\(^4\) who reported that the optical density of serum after a high fat meal increased in all older people much more prominently than in the younger control group.

Lipase Levels

Lipase levels determined simultaneously with the TEFA levels showed an increase in lipase activity in most representatives of the normal type curve (6 hour specimen lower than 3 hour specimen). This increase was usually found in both the 3 hour and 6 hour specimens. Most volunteers with a high type TEFA curve, however, showed lipase levels in the 3 and 6 hour specimens which were lower than the fasting levels. When our TEFA and lipase data were examined it was clear that the way in which TEFA and lipase levels changed was more significant than the magnitude of the levels alone. Thus we found that the values for a particular individual underwent daily fluctuations but the shapes of the curves remained unchanged. Winston and Dutrey\(^2\) came to the same conclusion and reported their values in per cent difference from the second to the third specimen.

\(^1\)We would like to thank Dr. James B. Hammond, Eli Lilly & Co., Indianapolis, Indiana, for the generous supply of Heparin, Enseals Pancreatin and Secretin, and Dr. Louis Berlinrood, Amfre-Grant Co., Brooklyn, New York, for Pancreatic Hormone, injectable.

\(^2\)Pancreatic Hormone is the trade name for a commercial product supplied by the Amfre-Grant Co., Brooklyn, New York. It is a deproteinized extract from the pancreas and is said to be free of enzymes and insulin and essentially free of choline and histamine.\(^2\) The composition and nature of the compound is not exactly known. Recent reports indicate that the active factor of Pancreatic Hormone is a hormone\(^2\) which increases the oxidation of fatty acids in the liver and the phospholipid exchange.\(^2\)
Figure 1

Relation between TEFA and Lipase Values. The results on all volunteers with a normal type TEFA curve and an increase in serum lipase can be found in the right upper quadrant. Results on volunteers with a high type TEFA curve and a decrease in lipase are in the left lower quadrant. The numbers demonstrate the frequency of points in each quadrant. Calculations which led to R and N values are explained in the text.

without giving any absolute values. We decided, therefore, to present our findings on 78 cases shown in figure 1 by means of the derived quantities R and N.

Values for R and N were calculated as follows:

\[ R = \frac{\text{Change of TEFA values in mEq/L during 1st 3 hour period}}{\text{Change of TEFA values in mEq/L during 2nd period}} \]

\[ N = \frac{\text{Difference in lipase values in units from fasting to 3 hr. specimen}}{\text{Difference in lipase values in units from fasting to 6 hr. specimen}} \]

R is greater than 1.00 if the TEFA value in the 6 hour specimen is lower than the value in the 3 hour specimen which is characteristic for all normal type curves. All R values less than 1 as well as all negative values indicate that the TEFA levels continued to increase from the fasting to the 6 hour specimen (high type curve).

N represents the average change in lipase units in the 3 and 6 hour specimen as compared to fasting values.

Chi-Square was calculated for the numbers given in each quadrant of figure 1 and found to be 22.301. With one degree of freedom this figure is highly significant.

There was no strict correlation between age and the lipolytic activity of serum. While a number of volunteers of high age showed lower lipase levels, others showed values above the normal average.

Administration of Enseals Pancreatin (meal no. 3; no fat) to 16 volunteers who showed in previous experiments a normal type TEFA curve, resulted in a lipase increase in 12 cases. Average lipase values for all 16 individuals before the meal (base) were 0.42 units ± 0.15 S.D. and the average lipolytic activity in the specimens drawn 2 hours after the meal was 0.55 units ± 0.16 S.D. Corresponding lipase values for 13 persons with a high type TEFA curve were 0.45 ± 0.18 and 0.44 ± 0.16 S.D.

Cholesterol Levels

Fasting cholesterol determinations done simultaneously with the TEFA levels revealed that high TEFA levels were usually accompanied by high cholesterol levels. The average values for the 48 persons with a normal type curve were found to be 224 mg./100 ml. serum ± 48.4 S.D. and values of 300 mg./100 ml. ± 67.4 S.D. were found for the 35 persons with low fat tolerance. However, there was no close correlation between the type of TEFA curve and the curve of cholesterol values if plotted in the same way. Some cholesterol values increased over the 6 hour test period, some stayed almost constant, and some decreased.

The Influence of Various Drugs on the Fat Tolerance (TEFA Curve) of Human Volunteers

Whole Pancreatic Preparations

Seventeen of our volunteers with a high type TEFA curve received an identical high fat meal and in addition whole pancreatic preparations (test meal no. 2). The fat tolerance curve of 9 of these volunteers was changed into a curve of the normal type. At the same time lipase levels increased after the administration of the drug (See table 1).
PANCREATIC SECRETIONS AND FAT TOLERANCE

Table 1
COMPARISON of TEFA and Lipase Values after High Fat Meals Associated with Various Drugs

<table>
<thead>
<tr>
<th>Type of test meal</th>
<th>Total no. of patients</th>
<th>Lipase units (Normal 0.6-1.0)</th>
<th>TEFA in mEq./L (Normal 7-16)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Fast±S.D.  3 hr±S.D. 6 hr±S.D.</td>
<td>Fast±S.D.  3 hr±S.D. 6 hr±S.D.</td>
</tr>
<tr>
<td>No. 1 (Fat)</td>
<td>14</td>
<td>0.37±0.10 0.28±0.13 0.32±0.09</td>
<td>14.0±2.9 18.4±2.6 22.8±4.7</td>
</tr>
<tr>
<td>No. 2 (Fat &amp; Pancreatin)</td>
<td>9</td>
<td>0.32±0.09 0.45±0.15 0.44±0.23</td>
<td>12.7±2.9 18.7±2.8 15.9±3.0</td>
</tr>
<tr>
<td>No. 1 (Fat &amp; Pancreatin Horm.)</td>
<td>14</td>
<td>0.52±0.04 0.46±0.16 0.51±0.04</td>
<td>13.3±0.9 21.9±5.3 23.6±4.6</td>
</tr>
<tr>
<td>No. 4 (Fat &amp; Pancreatin Horm.)</td>
<td>4</td>
<td>0.55±0.02 0.54±0.13 0.58±0.12</td>
<td>13.4±1.9 19.8±1.3 18.3±1.8</td>
</tr>
<tr>
<td>No. 5 (Fat &amp; Secretin)</td>
<td>9</td>
<td>0.44±0.09 0.42±0.16 0.39±0.11</td>
<td>14.4±0.9 18.0±0.5 25.0±6.5</td>
</tr>
<tr>
<td>No. 6 (Fat &amp; Secretin)</td>
<td>9</td>
<td>0.65±0.09 0.83±0.20 0.83±0.20</td>
<td>13.4±2.1 18.9±3.1 18.7±2.9</td>
</tr>
</tbody>
</table>

We mentioned above that the shape of the curve is of more significance than the absolute values. Therefore, calculation of R, as outlined above, can be considered as a better means of expressing the response to pancreatic preparations. R for the groups of 9 volunteers with a definite change in the type of TEFA curve was calculated to be —0.19 ± 0.90 S.D. without drug and +1.47 ± 0.30 S.D. with drug. The remaining 8 volunteers had the corresponding R values of —1.50 ± 4.58 and —0.321 ± 0.89 respectively. The last two values indicate that there was also a slight improvement in this group, although these TEFA curves remained among the high type.

Administration of whole pancreatic preparations was more successful in cases with slight abnormalities in the fat tolerance than in persons demonstrating an extremely high type curve. This is expressed by the better R value (—0.19 compared with —1.50) found in the group with a significant response to the drug. The same observation was made when using Pancreatic Hormone.

Pancreatic Hormone

Pancreatic Hormone given shortly before a high fat meal (meal no. 4) resulted in a definite increase in the fat tolerance in 4 of 14 patients (See table 1). R-values for the same group were calculated and found to be —0.13 ± 1.31 S.D. when no drug was given and +1.18 ± 0.30 S.D. when meal no. 4 was given. The corresponding R-values for the remaining 10 patients were —0.13 ± 0.86 and +0.24 ± 0.77 respectively which represents also a slight but not very significant improvement.

Three milliliters of Pancreatic Hormone given to volunteers without a test meal resulted in 12 of 13 cases in a significant increase in serum lipase activity. Average values found in the fasting specimen were 0.43 units ± 0.25 S.D. and values found 2 hours after the administration of Pancreatic Hormone were 0.63 units ± 0.17 S.D.

Secretin

Secretin, a hormone secreted by the intestinal mucosa, is known to stimulate the flow of pancreatic juice. Although this hormone does not have its primary function in stimulating the secretion of pancreatic enzymes (like lipase), it has been successfully used in this respect. Secretin, administered to 9 volunteers directly before the high fat meal (meal no. 5) caused considerable differences in response among the various individuals. Two patients exhibited a definite increase in serum lipase activity.
crease in fat tolerance (See table 1) but the other 7 individuals did not respond to the drug although lipase levels increased in all but one individual. Average N values (lipase) were found to be 0.07 ± 0.13 S.D. in the control experiments and 0.56 ± 0.75 S.D. after Secretin. R-values (TEFA) for the 2 individuals with response were −0.53 ± 1.52 S.D. without and +1.04 ± 0.28 S.D. with drug. The corresponding values for the remaining individuals were −0.37 ± 0.87 and -0.33 ± 0.86, showing practically no alterations.

Heparin

Literature of the recent past has emphasized the possible importance of heparin in controlling the lipid levels in blood. A comparison was made of the TEFA curve in 8 individuals after receiving the high fat meal no. 1 and the TEFA curve after receiving an identical meal shortly after the administration of heparin I.V. (meal no. 6). The comparison revealed that the 3 hour specimens after heparin were somewhat lower than observed in the control experiments. After 6 hours, however, patients showed a prominent increase in the TEFA level which in some individuals greatly exceeded the control results (See table 1). The fasting specimen, of course, was not affected since it was drawn before the administration of heparin.

Heparin given to 5 volunteers without any test meal resulted also in an initial drop in TEFA values which was followed by an increase exceeding the original value in all but one specimen. TEFA values for the base and the specimen drawn 3 and 6 hours after heparin were 15.4 mEq/L ± 9.9 S.D.; 10.5 mEq/L ± 3.4 S.D.; and 17.4 mEq/L ± 4.8 S.D.

Table 2

<table>
<thead>
<tr>
<th>Type of TEFA curve</th>
<th>Fasting</th>
<th>1% hr. ± S.D.</th>
<th>3 hr. ± S.D.</th>
<th>4% hr. ± S.D.</th>
<th>6 hr. ± S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>High</td>
<td>0</td>
<td>5.95 ± 2.39</td>
<td>12.07 ± 3.59</td>
<td>15.25 ± 1.34</td>
<td>12.44 ± 2.90</td>
</tr>
<tr>
<td>Normal</td>
<td>0</td>
<td>3.88 ± 2.29</td>
<td>9.73 ± 2.56</td>
<td>13.93 ± 0.52</td>
<td>13.00 ± 0.40</td>
</tr>
<tr>
<td>Low normal</td>
<td>0</td>
<td>4.00 ± 0.23</td>
<td>9.77 ± 2.99</td>
<td>12.29 ± 3.02</td>
<td>11.77 ± 2.97</td>
</tr>
</tbody>
</table>

The Rate of Absorption of I^{131} Labeled Triolein in Volunteers with Different Types of Fat Tolerance Curves

In a previous paragraph we pointed out that the fat tolerance curve can show impressive differences in various groups of volunteers. There can be an increase in the TEFA level of 100 per cent or more (high type curve) after fat meals as compared to the extremely small increase in some volunteers with a normal type curve (in table 2 referred to as "low normal" curve). These findings motivated us to determine the rate of absorption of I^{131} labeled triglycerides in 3 volunteers with a normal type curve, in 5 volunteers with an extremely high type curve and in 3 with an extremely low type curve.*

Table 2 demonstrates that the rates of absorption in the group of volunteers with either a low or a medium type curve, were essentially the same. Representatives of the high type curve, however, showed a higher rate of absorption in the 1/2 hour specimen. This higher rate persisted but to a lesser degree in the 3 and 4 1/2 hour specimen.

The tests were carried out in the following manner: 1 ml/Kg. of body weight of an emulsion of I^{131} labeled Triolein in peanut oil was administered. The emulsion was prepared by mixing 50 ml. peanut oil, 3 Gm. Gum Arabic (acacia), 50 ml. H_2O and I^{131} labeled triolein (50 μc.) in a blender. The amount of I^{131} found in 1 ml. of a blood sample taken 1/2, 3, 4 1/2, and 6 hours after administration of the emulsion was multiplied by the blood volume and the per cent of I^{131}
absorbed calculated. The blood volume was assumed to be 7.5 per cent of the body weight. In obese volunteers the blood volume was calculated on the basis of the ideal weight plus 1/3 of the overweight. Uptake of I$^{131}$ by the thyroid gland was prevented by administration of 10 drops Lugol's solution 30 min. prior to the test.

Absorption studies with I$^{131}$ labeled triolein have been of great help in fat absorption studies. It should be kept in mind, however, that this method has several sources of error. Uptake of free or bound Iodine by tissues and rate of excretion of Iodine through the urine must be considered. It seems obvious that a significant difference in the rate of absorption would have a great influence on serum lipid levels and that further efforts to study the rate of absorption are indicated.

The Glucose Tolerance in Patients with Low Fat Tolerance

An oral glucose tolerance test was given to 43 of our volunteers who showed a high type TEFA curve (low fat tolerance). These volunteers received orally 0.75 Gm. of glucose/Kg. of body weight (but not less than 75 Gm. and not more than 125 Gm.). Blood specimens were drawn and checked for glucose in the fasting state and 1/2, 1, 2, 3, 4, and 5 hours after administration of glucose.

Among these 43 volunteers we found only 4 with a normal type curve. Three volunteers had glucose tolerance curves said to indicate diabetic tendencies and 23 volunteers showed a normal or slightly decreased fasting blood sugar level which was first followed by an increase of 100 per cent or more and then by a pronounced hypoglycemic phase (decrease of at least 30 per cent below fasting level). Values as low as 39, 45 and 49 mg./100 ml. were recorded. This type of curve has often been considered as characteristic for liver impairment. The remaining 13 patients showed a tendency toward this liver type curve, but their increase in blood sugar was only between 60 and 100 per cent and their hypoglycemic levels were between 10 and 30 per cent below fasting level. Extensive long term experiments are under way in our laboratory to evaluate these findings as to their possible importance in relation to fat metabolism.

Discussion

The majority of investigators believe that a high fat diet can produce atheroma. Results which support this theory have been obtained in animals as well as in human beings. However, individual animals within any given species, including human subjects, show variable susceptibility to atherosclerosis. Some experimental animals (e.g. rabbits) when placed on a high fat diet will develop definite atherosclerotic lesions while others of the same group will fail to develop recognizable lesions during the test period. These factors strongly suggest that the etiological factors in atherosclerosis are related to a block in the metabolism of fat, or possibly, to an enzyme deficiency.

The reason for the development of atherosclerosis is not known. Since it is believed that a number of factors, such as heredity, sex, diabetes, stress, high blood pressure, diet, exercise, hormone imbalance, and others influence the formation of atheroma, we cannot expect a single answer to this complex problem. It is our intention, therefore, to investigate one of the factors which might contribute to this disorder. Our investigation has concentrated on measuring the response of individuals to high fat meals and on the effect of compounds normally found in the pancreatic secretions of humans on the fat tolerance of these same individuals.

There was a suggestive relationship between the type of TEFA (total esterified fatty acids) curve and the release or secretion of lipase as measured in the 3 and 6 hour specimens (fig. 1). The increase in fat tolerance of patients receiving whole pancreatic extract with their fat meal (table 1) might also point to the importance of the lipolytic enzyme to fat metabolism.

Our findings, that some individuals show high type TEFA curves despite the fact that their lipase content increased, do not argue against the possible importance of pancreatic secretions. Many other factors could be responsible for this behavior. One of the reasons...
might be a decreased uptake of fat by the liver. This possibility we will discuss later.

More than 80 per cent of our patients with a normal TEFA curve showed lipase levels in the 3 and 6 hour specimens which were above the fasting level (fig. 1). It is our conclusion, however, that lipase excretion alone is unlikely to influence the TEFA curve to any significant degree. In fact, we observed normal TEFA curves accompanied by an increase in serum lipase as small as 0.05 or 0.1 units. This conclusion was strengthened by the observation that when secretin was administered with fat tolerance tests, high serum lipase levels were obtained. At the same time, no significant changes in the TEFA levels accompanied this elevation of serum lipase.

It therefore remains to be considered whether or not the secretion of lipase is accompanied by the secretion of another compound with influence on fat metabolism. Our attention was directed to a preparation described in the literature as Pancreatic Hormone, anti-fatty liver factor, lipo-caic, or Lipotrat. Indeed, we found that Pancreatic Hormone (and Pancreatin, which contains the active factor of Pancreatic Hormone) does increase the fat tolerance in persons with a high type curve (table 1). The active factor of Pancreatic Hormone is said to prevent (or remove) fatty infiltration of the liver and to aid fat metabolism by activating the oxidation of fatty acids and the phospholipid exchange. If this is true, pancreatic deficiency would impair fat metabolism. This would result in an increase in serum lipids, and, in addition, it would cause fatty infiltration of the liver. It is of great interest that the majority of our patients with a high type TEFA curve showed a glucose tolerance curve said to be characteristic for liver impairment. Further experiments are in progress to evaluate the importance of these findings.

Pancreatic Hormone was found to increase lipase activity when given with or without a meal (see table 1 and text). With these results in mind one might consider the possibility that the increase in serum lipase values found after administration of Enseals Pancreatin (which contain lipase as well as Pancreatic Hormone) is not caused by absorption of lipase but by the stimulating effect of the Pancreatic Hormone. Such an effect of Pancreatic Hormone could have significant importance for fat metabolism, and, therefore, further experiments are planned to evaluate the role of this pancreatic secretion.

The present findings still leave many questions unanswered. It seems apparent, however, that the early observations of Dragstedt et al. should be considered and that more emphasis should be placed on the role of the pancreas in the development of disorders in fat metabolism.

Holman reported that patients with fibrocystic disease of the pancreas show less atherosclerotic plaques than "healthy" individuals. This observation does not speak against the above considerations. In fibrocystic disease of the pancreas, absorption of fats is so greatly diminished that such cases cannot be properly compared with cases in which fat absorption is unimpaired or is nearly normal.

Summary

A high fat meal was given to 78 human volunteers, and the TEFA, cholesterol and lipase levels were determined in the fasting, 3 hour, and 6 hour specimens.

Eighty per cent of the persons with normal type TEFA curve showed an increase in their lipase levels in the 3 and 6 hour specimens. Seventy per cent of the representatives of the high type curve showed lipase levels in the 3 and 6 hour specimens which were below the fasting level.

It was pointed out that this suggestive correlation between TEFA and lipase levels cannot be the only factor which determines the TEFA curve. Pancreatic Hormone was suggested as a possible compound which stimulates lipase secretion.

No correlation was found between TEFA and cholesterol curves. There was also no strict correlation between age and TEFA curve, nor was there a significant correlation between age and lipase levels.
PANCREATIC SECRETIONS AND FAT TOLERANCE

The effect of whole pancreatic preparations, pancreatic hormone, secretin, and heparin on the fat tolerance of humans was determined. The fat tolerance curves of 33 per cent of the volunteers with a high type curve were changed into curves of the medium type when whole pancreatic preparations were given. Four of 14 patients improved after Pancreatic Hormone injections, but only a slight or no improvement was noticed after Secretin.

Administration of 5000 units heparin with or without a high fat meal resulted in a decline of TEFA levels in the 3 hour specimen. This was followed, however, by a rise in lipids in the 6 hour specimen. This increase exceeded by far the one found in control experiments without heparin.

The rate of absorption of 1\textsuperscript{131}I labeled triolein was determined in volunteers with extremely low, normal, and extremely high type TEFA curves. The first two groups showed no significant difference in the absorption rate. Persons with a high type curve, however, absorbed the labeled triolein faster during the first 4½ hours of the test period.

Oral-Glucose tolerance tests were done on 43 of the volunteers with a high type TEFA curve. In 4 of these volunteers we found a normal glucose tolerance curve, in 3, a curve which is said to indicate a diabetic tendency and in 36 a type of curve which had been considered as characteristic for liver impairment.

The possible role of pancreatic secretions, especially Pancreatic Hormone, in the regulation of blood lipids is discussed.

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Summario in Interlingua

Un repasto ric in grassia esseva administrate a 78 voluntarios human, e le nivellos de total esterificate acido grassa (TEAG), de cholesterol, e de lipase esseva determinate in specimens prendite in stato jejun ante le repasto e 3 e 6 horas post illo.

Octanta pro cento del subjectos con un typo normal de curva pro TEAG exhibiva un augmento de lor nivellos de lipase post 3 e 6 horas. Septanta pro cento del subjectos con un typo supranormal de curva pro TEAG monstrava post 3 e 6 horas nivellos de lipase que esseva infra le nivellos constatate in stato jejun.

Es signalate che iste suggestive correlation inter le nivellos pro TEAG e lipase non pote esser le sol factor que determina le curva pro TEAG. Hormones pancreatic are mentioned as a composite that stimulates possibly the secretion of lipase.

Nulle correlation esseva constatate inter le curvas pro TEAG e cholesterol. Similmente, nulle stricte correlation esseva trovate inter le etates del subjectos o le nivellos de TEAG. Il etiam existeva nulle significative correlation inter le etates del subjectos o le nivellos de lipase.

Esseva determinate le effete exercite super le tolerantia pro grassia in humanos per le administratio de hormon pancreatic, preparatos de pancreas total, secretina, e heparina. Le curvas de tolerantia pro grassia in 53 pro cento del voluntarios con un alto typo de curva esseva rimplicate per curvas del typo intermedii quando preparatos de pancreas total esseva administrate. Quatro de 14 patientes se meliorava post injectiones de hormon pancreatic, solmente leve grades de melioration o nulle melioration del tutto esseva notate post administratio de secretina.

Le administratio de 5000 unitates de heparina con o sin repasto a alte contento de grassia resultava in un reduction del nivellos de TEAG in le specimen de 3 horas. Tamen, isto esseva seguito per un augmento del lipidos in le specimen de 6 horas. Iste augmento excedeva per multo le augmento constatate in experimentos de controlo sin heparina.

Le intensitate del absorption de triolein marcate con 1\textsuperscript{131}I esseva determinate in voluntarios con curvas pro TEAG del typo extrémemente basse, del typo normal, e del typo extrémemente alte. Le duo prime gruppos mostrava nulle significative differentia in le intensitate del absorption. Tamen, subjectos con curvas del typo alte absorbeva le marcaute trioleina plus rapido durante le 4½ horas del periodo experimental.

Tests de tolerantia pro glucosa oral esseva effectuate in 43 voluntarios con un curva pro TEAG del typo alte. In 4 de iste voluntarios non trovava un curva normal de tolerantia pro glucosa; in 3 le curva esseva del typo considerate comme indicative de un tendentia diabetic; e in 36 le curva esseva del typo considerate comme charateristic de dysfunction hepatic.

Es discutite le rolo possibile de secretiones pancreatic, specialmente hormon pancreatic, in le regulation del lipidos del sanguine.
References


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