Toxicity of Pure Foods
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The studies described in this volume demonstrate that there is a dose or amount of all pure foods which can produce a lethal toxicity syndrome in laboratory animals. Pure foods are usually regarded as nontoxic apart from an occasional stomach upset from eating too much of certain foods, especially in children, and the continued overeating of pure foods, which may produce obesity.

The concept that pure foods can produce death, if administered in a sufficiently large dose, arose a number of years ago in studies by the author and his associates. In 1963, following the thalidomide disaster, the government of Canada introduced new legislation that before a new drug could be sold in Canada, the manufacturer had to provide, among other things, proof of the safety of the new drug under the conditions of use. Evidence of safety, therefore, became a necessary part of any application for acceptance of a new drug by the government of Canada. It then became necessary to define what was meant by safety. Safety obviously meant the absence of toxic effects, especially death. Toxic effects, in turn, are related to dosage so that the required evidence should include a study of toxic effects of doses ranging up to those which produced death. Such studies obviously had to be performed largely on laboratory animals and the science of Predictive Toxicometrics came to maturity as a result.

Predictive Toxicometrics is concerned with predicting toxicity in man from studies on laboratory animals. The subject is divided into three main phases. The first of these is Factorial or Etiological Toxicometrics, which is concerned with the various factors that affect the response of laboratory animals to toxic doses of drugs and other agents. Acute or single dose toxicity is discussed under the heading of Uniposal Toxicometrics. Subacute and chronic toxicity is described under the heading of Multiposal Toxicometrics. These topics have been discussed by the author in a volume entitled Predictive Toxicometrics, Scientechnica Publishers Ltd., Bristol, England, 1972.

In the course of these studies it was noted that skin reactions to drugs rarely appeared in laboratory animals but are frequent in man. Since skin reactions are common manifestations of certain vitamin deficiencies, it was postulated that they might occur in persons with a marginal vitamin deficiency. To test this hypothesis, laboratory animals were placed on various vitamin-deficient diets and then given toxic doses of drugs such as benzylpenicillin. It was found that the vitamin deficiency augmented susceptibility to drug toxicity but that the augmentation was not due to the absence of vitamins in the diet.

To make a long story short, it was finally discovered that augmentation of drug toxicity in vitamin deficient animals was due to the presence of large amounts of certain pure foods, such as sucrose, in the vitamin deficient diet fed to the animals. This observation prompted a study of the uniposal and multiposal toxicity of pure foods.

The results demonstrated that there are toxic and lethal doses of all pure foods. The amount or dose required to produce a lethal syndrome is within the limits of possible human consumption for some pure foods and beyond these limits for others. Since toxic doses vary with body weight, a child is more likely to consume a lethal dose of pure foods than is an adult. For example, based on animal data, a child of 22 lb eating and retaining half a pound of candy would receive the equivalent of a dose of sucrose of 25 g/kg, which is a fatal dose in albino rats.

Data on lethal doses of other pure foods in laboratory animals suggest that death in man from single doses would be extremely unlikely due to the large lethal dose. For example, in a single meal a man would have to eat half his body weight of starch before getting doses that killed albino rats in terms of g/kg body weight. On the other hand, it is possible that sublethal doses could produce signs of sublethal toxicity, such as gastrolith from large amounts of starch.

The chronic toxicity of pure foods is such that large fractions of the LD₅₀ (1 dose) have to be given daily to produce severe toxicity and death.
The LD₅₀ (100 days), or dose which kills 50% of animals over a period of 100 days of daily administration, is about 90% of the LD₅₀ (1 dose) in the instance of sucrose. The LD₅₀ (100 days) expressed as a percentage of the LD₅₀ (1 dose) is termed the 100-day LD₅₀ index. The higher the 100-day LD₅₀ index, the relatively more safe is a substance for chronic use. Pure foods, therefore, are relatively safe for repeated daily use, which is a well-known fact.

The survey of the toxicity of pure foods described in this book has been confined of necessity largely to clinicopathological syndromes at lethal doses. Time did not permit extensive studies on the toxicity of sublethal doses. Such studies obviously should be conducted and could yield valuable information. For example, the author and his associates found that phenacetin given daily for two thirds of a year to albino rats in amounts that produced no other obvious toxic effect caused a complete inhibition of spermatogenesis in the animals. There remains a great deal to be done in the science of predictive toxicometrics.

Finally, the author wishes to acknowledge the receipt of many grants in aid of research on the toxicity of pure foods in his laboratory. In particular should be mentioned generous financial assistance from the Department of National Health and Welfare of Canada and the Medical Research Council of Canada.

Eldon M. Boyd
Kingston, Ontario, Canada
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Part I. INDICATIONS THAT PURE FOODS MAY HAVE TOXIC EFFECTS

Chapter 1

FOOD INTAKE AND LIPID METABOLISM

Scope of the Book

This volume will not deal with aspects of food toxicity due to food additives and contaminants. It will not be concerned with toxicity from substances in food such as coloring agents, antioxidants, pH modifiers, and synthetic flavoring agents which have been discussed by authors such as Furia. Nor will it consider topics such as anticaking and bleaching agents, emulsifiers and stabilizers, preservatives, glazers, polishers, and sequestering agents, which have been reviewed by Chapman and Pugsley. The toxicity of "food chemicals," as noted by the National Academy of Sciences, will likewise not be described. "Food chemicals" are defined as substances added directly or indirectly to food for a functional purpose without intending that they remain in the final product. Finally, this book will not deal with food contaminants which have been extensively reviewed, for example, at a Symposium of the Food and Drug Directorate at Ottawa.

Rather, this book will present recent evidence on the toxicity of pure foods, carbohydrates, fats, proteins, salts, water, vitamins, and food adjuvants such as caffeine. Most of the evidence to be described was obtained by the author and his associates in studies which defined the range of lethal doses of pure foods and the clinico-pathological syndrome of toxicity. Pure foods are popularly believed to be largely free of poisonous effects to most persons. It will be shown that while the lethal dose of some pure foods is so high as to be unlikely of consumption by man, that of others is within the range of possible human intake. It will also be shown that animals fed large amounts of certain pure foods, in diets upon which they grow and appear normal, become highly susceptible to stresses such as to toxic doses of drugs and other agents, including pesticides.

The basic concept that arises from these studies is that there is probably a toxic dose or amount of everything. This dose is an amount which overwhelms the body mechanisms for dealing with the substance in question. The toxic amount is small for substances popularly known as poisons and large for those called pure foods. Conversely, in amounts that can be satisfactorily dealt with by body mechanisms, no substance is toxic or poisonous. This is not a new concept. It was proposed over 400 years ago by Paracelsus, who stated: "All things are poisons, for there is nothing without poisonous qualities. It is only the dose which makes a thing a poison."

Fasting and Blood Lipids

A piece of knowledge becomes personally significant only when it may be used to solve a personal problem. The earliest impression of the author was that while diet was undoubtedly important, it had little influence on body functions as long as it was adequate. This impression arose during studies on lipid metabolism. Following graduate studies under Professor W. R. Bloor at Rochester, N.Y., the author transferred to a department of obstetrics and gynecology for a postdoctoral research program. A method for the differential lipid analysis of blood plasma was developed and applied to the study of a series of problems in obstetrics and gynecology. The author was then appointed to the staff of the Department of Pharmacology at Queen’s University in 1934 and had a research laboratory in the Kingston General Hospital. This arrangement was ideal for studies of blood lipids in patients with various diseases.

At that time it was believed that samples of blood for lipid analysis should be collected from patients in a fasting state, i.e., in the morning before breakfast. To obtain such prebreakfast samples of blood meant that the wards of the hospital had to be visited very early in the morning. The author reviewed the evidence indicating that blood should be collected with the patient in a fasting condition and reached the conclusion that such evidence was not too conclusive. He therefore collected samples of blood from eight unfasted persons at intervals over a
The constancy of lipid levels in the blood plasma of nonfasted human subjects*

<table>
<thead>
<tr>
<th>Composition of total lipid</th>
<th>Fatty acids</th>
<th>Cholesterol</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hour of sampling</td>
<td>Total lipid</td>
<td>Neutral fat</td>
</tr>
<tr>
<td>8 a.m.</td>
<td>595</td>
<td>150</td>
</tr>
<tr>
<td>11 a.m.</td>
<td>591</td>
<td>146</td>
</tr>
<tr>
<td>2 p.m.</td>
<td>591</td>
<td>156</td>
</tr>
<tr>
<td>5 p.m.</td>
<td>570</td>
<td>114</td>
</tr>
<tr>
<td>8 p.m.</td>
<td>581</td>
<td>128</td>
</tr>
<tr>
<td>12 p.m.</td>
<td>563</td>
<td>131</td>
</tr>
<tr>
<td>4 a.m.</td>
<td>586</td>
<td>140</td>
</tr>
</tbody>
</table>

*The results are expressed as mean mg/100 ml of plasma. (From Boyd7 with the permission of The Journal of Biological Chemistry.)

This period of 24 hr from 8 a.m. one morning until 4 a.m. the next morning.

Blood plasma of these samples was analyzed for its lipid composition and the results are summarized in Table 1. It was found that the mean values of the various lipids noted in Table 1 did not change significantly over a period of 24 hr in persons who ate regular meals. Subsequent studies disclosed that conditions such as toxic goiter,18 nephritis,19 and parenchymatous hepatic diseases20 produced greater alterations in the concentration of plasma lipids than did fasting.

Regeneration of Blood Lipids

Other evidence of the stability of concentrations of lipids in blood plasma was obtained by Boyd and Stevenson.21 One quarter of the blood volume of rabbits was removed via the marginal ear veins and the lipids of blood plasma and of the red blood cells determined at subsequent periods of 0, 3, 6, 12, 24, and 48 hr. The removal of blood produced no significant changes in the lipid content of the erythrocytes. Changes in the levels of lipids in blood plasma are summarized in Table 2. It will be noted that the concentration of blood hemoglobin was reduced by about one quarter of the value before hemorrhage and that hemoglobin was not regenerated in significant amounts over the period of 48 hr of measurement.

The level of plasma phospholipid was reduced by a mean of 39% following the hemorrhage. Plasma phospholipid levels were restored to normal over a period of 24 hr. The pattern followed by the plasma cholesterol fractions was similar to that of phospholipid. The concentration of plasma triglyceride (neutral) fat was not affected by hemorrhage until 48 hr, when it was significantly increased to values above those initially present in the animals. Boyd and Stevenson21 reviewed evidence suggesting that the slight lipemia at 48 hr may have been a response to oxygen deficit.

Prolonged Fasting

It is well known that prolonged fasting at normal body temperature produces a loss of body fat due to the need for calories to maintain body temperature. Baker et al.22 investigated changes produced by prolonged starvation in a form of life which could exist at a low temperature and under conditions in which there was little or no need to maintain body temperature. They measured the water, lipids, glycogen, and iodine content of Nova Scotia oysters stored at 4°C. Representative values are noted in Table 3.

Without the need for calories to maintain body temperature, prolonged starvation had little effect on the constituents of oyster flesh. There was some loss of water after two months of storage. The concentration of neutral fat and free sterol was unaffected by storage. The level of sterol esters slowly declined and of phospholipid slowly increased in the second and third months. The concentration of glycogen and of iodine also...
TABLE 2

The Regeneration of Lipids in the Blood Plasma of Rabbits Following a Single Massive Hemorrhage*

Composition of total lipid

<table>
<thead>
<tr>
<th>Time after bleeding (hr)</th>
<th>Hemoglobin</th>
<th>Total lipid</th>
<th>Neutral fat</th>
<th>Total fatty acids</th>
<th>Cholesterol</th>
<th>Phospholipid</th>
</tr>
</thead>
<tbody>
<tr>
<td>3</td>
<td>-25</td>
<td>-14</td>
<td>+18</td>
<td>-4</td>
<td>-37</td>
<td>-39</td>
</tr>
<tr>
<td>6</td>
<td>-26</td>
<td>-4</td>
<td>+21</td>
<td>+1</td>
<td>-30</td>
<td>-40</td>
</tr>
<tr>
<td>12</td>
<td>-26</td>
<td>-7</td>
<td>-15</td>
<td>-10</td>
<td>+15</td>
<td>+7</td>
</tr>
<tr>
<td>24</td>
<td>-29</td>
<td>+1</td>
<td>+8</td>
<td>+4</td>
<td>-6</td>
<td>-7</td>
</tr>
<tr>
<td>48</td>
<td>-26</td>
<td>+34</td>
<td>+88</td>
<td>+45</td>
<td>+9</td>
<td>+5</td>
</tr>
</tbody>
</table>

*(From Boyd and Stevenson with permission of The Journal of Biological Chemistry.)

TABLE 3

Mean Changes in the Composition of the Flesh of Nova Scotia Oysters Stored at 4°C

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Weeks of storage</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>3–5</td>
</tr>
<tr>
<td>Body water (g/100 g dry wt)</td>
<td>486</td>
</tr>
<tr>
<td>Total lipid (mg/100 g dry wt)</td>
<td>6,292</td>
</tr>
<tr>
<td>Neutral fat (mg/100 g dry wt)</td>
<td>4,225</td>
</tr>
<tr>
<td>Total fatty acids (mg/100 g dry wt)</td>
<td>4,985</td>
</tr>
<tr>
<td>Free sterol (mg/100 g dry wt)</td>
<td>430</td>
</tr>
<tr>
<td>Sterol ester (mg/100 g dry wt)</td>
<td>274</td>
</tr>
<tr>
<td>Phospholipid (mg/100 g dry wt)</td>
<td>1,193</td>
</tr>
<tr>
<td>Glycogen (mg/100 g dry wt)</td>
<td>-</td>
</tr>
<tr>
<td>Iodine (µg/100 g dry wt)</td>
<td>440</td>
</tr>
<tr>
<td></td>
<td>7–9</td>
</tr>
<tr>
<td>Body water (g/100 g dry wt)</td>
<td>428</td>
</tr>
<tr>
<td>Total lipid (mg/100 g dry wt)</td>
<td>6,940</td>
</tr>
<tr>
<td>Neutral fat (mg/100 g dry wt)</td>
<td>4,420</td>
</tr>
<tr>
<td>Total fatty acids (mg/100 g dry wt)</td>
<td>5,539</td>
</tr>
<tr>
<td>Free sterol (mg/100 g dry wt)</td>
<td>352</td>
</tr>
<tr>
<td>Sterol ester (mg/100 g dry wt)</td>
<td>254</td>
</tr>
<tr>
<td>Phospholipid (mg/100 g dry wt)</td>
<td>1,744</td>
</tr>
<tr>
<td>Glycogen (mg/100 g dry wt)</td>
<td>10,900</td>
</tr>
<tr>
<td>Iodine (µg/100 g dry wt)</td>
<td>393</td>
</tr>
</tbody>
</table>

slowly fell. These various results indicated that prolonged fasting had little effect when there was no need for calories to maintain body temperature.

Obesity

Obesity is popularly believed to be due to overeating, although there are many examples of overeating producing obesity in one person but not in another. Obesity occurs in albino rats as they grow older and providing they have food available ad libitum. The amount of food that albino rats eat actually declines with age if food intake is calculated as g/kg body weight/day. Is obesity in the albino rat due to overeating, in the sense of total g intake of food, or to some other factor?

To investigate the cause of obesity in albino rats, a series of studies was made upon the activity of the genital, perirenal, mesenteric, skinfold, and omental fat depots. It was first confirmed that as the total neutral fat content of the body increases, the amount of neutral fat in the fat depots also increases. This is exemplified in Figure 1, in which the amount of neutral fat in the genital fat depots of albino rats, expressed as g/kg body weight, is plotted against total body neutral fat in animals weighing from 200 to over 500 g. It may be seen that as the total amount of body neutral fat increased, per kg body weight, the amount of neutral fat stored in the genital fat depots also increased.

While concentrations of neutral fat are usually expressed as shown in Figure 1, this method has the disadvantage that the substance being compared — neutral fat — is part of the denominator —
body weight — as well as of the numerator. The disadvantage may be overcome by expressing concentrations of neutral fat as g/100 g of nonlipid dry weight, as exemplified in Figure 2. Nonlipid dry weight of the tissues illustrated in Figure 2 is practically synonymous with tissue protein. There was positive correlation, significant at P = 0.01, between concentration of neutral fat in the omentum of albino rats and concentration of body neutral fat, as shown in Figure 2.

If storage of neutral fat is an active rather than passive function of the fat depots, one might anticipate that with storage of increasing amounts of neutral fat there would be evidence of increased physiological activity in the depots. Many years ago Professor W. R. Bloor established that increase in physiological activity of tissues is associated with an increase in the concentration of phospholipid and of free cholesterol in such tissues. The Bloor hypothesis was applied to a study of the postoperative activity of the blood leukocytes by Boyd.12

The concentration of neutral fat in the fat depots was, therefore, compared with the concentration of phospholipid in the same depots. This was done by plotting concentration of neutral fat, as g/100 g nonlipid dry weight, against concentration of phospholipid, similarly expressed, in five body fat depots and in the total body. The correlations were mostly positive and correlation coefficients are assembled in Table 4. Correlation coefficients in Table 4 of 0.200 or higher were statistically significant at P = 0.05 or less, with the available value of N. By this criterion, it may be concluded that a primary active function of the mesentery, omentum, and skin is to store neutral fat. Similar active storage would appear to be a possible secondary function of the genital and perirenal depots.

When concentrations of neutral fat were plotted against concentrations of free cholesterol in a similar manner, correlation coefficients in all five fat depots were significantly positive, as shown by data in Table 5. The author had previously obtained evidence that with increase in the physiological activity of a tissue there is an increase in the concentration of water per unit of
TABLE 4

Correlation Coefficients Obtained from the Regression of Concentration of Neutral Fat on Concentrations of Phospholipid in the Fat Depots of Albino Rats*

<table>
<thead>
<tr>
<th>Fat depot</th>
<th>Males (N=22 to 63)</th>
<th>Females (N=24 to 47)</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Genital fat depot</td>
<td>+0.100</td>
<td>+0.211</td>
<td>23</td>
</tr>
<tr>
<td>Mesenteric fat depot</td>
<td>+0.365</td>
<td>+0.374</td>
<td>25</td>
</tr>
<tr>
<td>Omental fat depot</td>
<td>+0.465</td>
<td>+0.423</td>
<td>27</td>
</tr>
<tr>
<td>Perirenal fat depot</td>
<td>+0.003</td>
<td>+0.175</td>
<td>24</td>
</tr>
<tr>
<td>Skinfold fat depot</td>
<td>+0.688</td>
<td>+0.806</td>
<td>26</td>
</tr>
<tr>
<td>Total body</td>
<td>+0.275</td>
<td>−0.009</td>
<td>23–27</td>
</tr>
</tbody>
</table>

*Concentrations were calculated as g/100 g nonlipid dry weight. Correlation coefficients of 0.200 or greater were significant at P = 0.05 or less.

TABLE 5

Correlation Coefficients Obtained by Plotting the Regression of Concentration of Neutral Fat on Concentration of Free Cholesterol in the Fat Depots of Albino Rats*

<table>
<thead>
<tr>
<th>Fat depot</th>
<th>Males (N=22 to 63)</th>
<th>Females (N=24 to 47)</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Genital fat depot</td>
<td>+0.696</td>
<td>+0.259</td>
<td>23</td>
</tr>
<tr>
<td>Mesenteric fat depot</td>
<td>+0.847</td>
<td>+0.658</td>
<td>25</td>
</tr>
<tr>
<td>Omental fat depot</td>
<td>+0.914</td>
<td>+0.816</td>
<td>27</td>
</tr>
<tr>
<td>Perirenal fat depot</td>
<td>+0.882</td>
<td>+0.575</td>
<td>24</td>
</tr>
<tr>
<td>Skinfold fat depot</td>
<td>+0.554</td>
<td>+0.648</td>
<td>26</td>
</tr>
<tr>
<td>Total body</td>
<td>−0.107</td>
<td>+0.204</td>
<td>23–27</td>
</tr>
</tbody>
</table>

*Concentrations were calculated as g/100 g nonlipid dry weight. Correlation coefficients of 0.200 or greater were significant at P = 0.05 or less.

protein. Concentrations of neutral fat were, therefore, plotted against concentrations of water in the same five fat depots and the correlation coefficients are listed in Table 6. It may be seen that by the criteria tested in Tables 5 and 6, storage of neutral fat appears to be an active, rather than a passive physiological function of the body fat depots.

When the composition of adipose tissue is expressed as g/100 g wet or dry weight, results indicate that it contains neutral fat and very little of other ingredients. A different picture is obtained when concentration is calculated as g/100 g nonlipid (or protein) dry weight. The mean concentrations of phospholipid, free cholesterol, and water, as g/100 g nonlipid dry weight, have been listed in Table 7 and compared with corresponding values in a variety of other tissues. It may be seen that appreciable amounts of these three indicators of physiological activity are present in the fat depots when concentration is expressed as g/100 g nonlipid dry weight. From the values listed in Table 7, it would be estimated that the level of physiological activity in the fat depots is not as high as that in brain, liver, and testicles, although activity of the omentum was similar to activity in tissues such as lung, heart, and kidneys.

These various results indicate that obesity in the albino rat is not a passive response to overeating. As the animal grows toward adult weight, it eats less food/kg body weight, presumably because less nutrient is required for the formation of body proteins and related essential growth elements. As the needs for growth decline, adipose tissue becomes increasingly active and a greater proportion of the food intake is actively stored as neutral fat. The earliest function of the body appears to be growth and when that is satisfied, food is eaten and much of it stored as
**TABLE 6**

Correlation Coefficients Obtained by Plotting the Concentration of Neutral Fat, in g/100 g Nonlipid Dry Weight, Against Concentrations of Water, in g/100 g Nonlipid Dry Weight, in the Fat Depots of Albino Rats*

<table>
<thead>
<tr>
<th>Fat depot</th>
<th>Males (N=22 to 63)</th>
<th>Females (N=24 to 47)</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Genital fat depot</td>
<td>+0.853</td>
<td>+0.901</td>
<td>23</td>
</tr>
<tr>
<td>Mesenteric fat depot</td>
<td>+0.929</td>
<td>+0.392</td>
<td>25</td>
</tr>
<tr>
<td>Omental fat depot</td>
<td>+0.559</td>
<td>+0.923</td>
<td>27</td>
</tr>
<tr>
<td>Perirenal fat depot</td>
<td>+0.944</td>
<td>+0.892</td>
<td>24</td>
</tr>
<tr>
<td>Skinfold fat depot</td>
<td>+0.894</td>
<td>+0.904</td>
<td>26</td>
</tr>
<tr>
<td>Total body</td>
<td>+0.498</td>
<td>+0.111</td>
<td>23-27</td>
</tr>
</tbody>
</table>

*Concentrations were calculated as g/100 g nonlipid dry weight. Correlation coefficients of 0.200 or greater were significant at P = 0.05 or less.

**TABLE 7**

Mean Concentrations of Phospholipid, Free Cholesterol, and Water, Expressed as g/100 g Nonlipid Dry Weight, in the Tissues and Organs of Albino Rats

<table>
<thead>
<tr>
<th>Organ</th>
<th>Reference</th>
<th>Phospholipid</th>
<th>Free cholesterol</th>
<th>Water</th>
<th>Organ</th>
<th>Reference</th>
<th>Phospholipid</th>
<th>Free cholesterol</th>
<th>Water</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brain</td>
<td>28</td>
<td>40.4</td>
<td>9.81</td>
<td>702</td>
<td>Spleen</td>
<td>32</td>
<td>5.9</td>
<td>1.28</td>
<td>370</td>
</tr>
<tr>
<td>Liver</td>
<td>28</td>
<td>13.6</td>
<td>0.82</td>
<td>326</td>
<td>Thymus</td>
<td>33</td>
<td>5.7</td>
<td>1.00</td>
<td>472</td>
</tr>
<tr>
<td>Testicle</td>
<td>29</td>
<td>12.5</td>
<td>1.35</td>
<td>770</td>
<td>Leg muscle</td>
<td>29</td>
<td>5.5</td>
<td>0.35</td>
<td>353</td>
</tr>
<tr>
<td>Testicle</td>
<td>29</td>
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fat, presumably for possible use in periods of famine.

**Conclusions**

Evidence obtained in early studies on lipid metabolism in the author's laboratory, between 1930 and 1960, suggested that diet, providing it was adequate, had minor influence on body function. This evidence was as follows:

1. Levels of lipids in human blood plasma are not affected by ordinary meals.

2. Amounts of blood lipids removed by a single massive hemorrhage are replaced within 24 hr.

3. Prolonged fasting does not affect levels of tissue lipids, providing there is no need to produce calories in order to maintain body temperature.

4. Obesity is not due to overeating but to the active storage of neutral fat, in adipose tissue, from food that is no longer needed for body growth.


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