suggest inhibition of protein synthesis. In order to prove this protein synthesis was investigated in slices of mouse pancreas[11–13]. We regard the inhibition of protein synthesis by allylisothiocyanate, phenylisothiocyanate, cheirolin $CH_3SO_2CH_2CH_2NCS$ and iberin $CH_3SOCH_2CH_2NCS$ as proved, because it has been simultaneously observed that phenylisothioalanine and phenylisothioleucine do not inhibit protein synthesis and that also other N-substituted derivatives of amino acids are without inhibiting effect. It is improbable that the slowing down of protein synthesis by isothiocyanates is caused by the elimination of some amino acid through bonding with isothiocyanates. When the concentration of amino acids is increased so that even the amino acid, present in the lowest concentration, is present in a concentration at least double with respect to the amount of inhibitor, the inhibiting effect of the isothiocyanates tested remains unchanged. Isothiocyanates belong to the most powerful inhibitors of protein synthesis known.

Zusammenfassung. Es wurde ein inhibitorischer Effekt des Allylisothiocyanates auf die Keimung von Erbsen, Weizen und Raps beobachtet, und dabei die Veränderungen des Stickstoff- und Zuckermetabolismus verfolgt: Isothiocyanate setzen die Atmung und anaerobe Glykolyse um etwa 30% herab und legen die Proteosynthese vollständig still.

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Lipogenesis in Rats Adapted to Intermittent Starvation or Continuous Underfeeding

In previous papers (FÁBRY et al.[1]) we have demonstrated that in rats accustomed to intermittent starvation, i.e. alternating periods of fasting and days of free access to food, a series of adaptive changes develop, most of which differ markedly from commonly known sequelae of simple continuous caloric restriction. In the present paper we are submitting the results of experiments in which we investigated changes in the carcass composition (a) of male rats submitted to intermittent starvation for 11 weeks, where the total caloric intake amounted to 46% of that in controls; (b) in rats subjected to continuous underfeeding, pair-fed the same amount of food divided into daily rations; (c) in *ad libitum* fed controls. In addition, the *in vitro* hepatic lipogenesis in female rats, intermittently starved for 3 weeks, was investigated by assessing the incorporation of 1-C^{14}-acetate into fatty acids by liver slices (for method see BARUCH and CHAIKOFF[2]).

Adult albino rats, Wistar strain, fed a standard laboratory diet (Larsen mixture[3]) were used. The intermittently starving animals were fed on alternate days during the first two weeks of the experiment, subsequently three times a week. In the pair-feeding experiment groups of animals killed by decapitation after a standard test meal were compared; hepatic lipogenesis was investigated after an unrestricted test meal over night (absorptive phase) and after a subsequent 48 h fast. For carcass analysis, a technique described by MICKELSEN[4], was used; the extracted fat was estimated gravimetrically, protein was calculated from total nitrogen values obtained by microkjeldahlization.

All assessed parameters revealed that periodic hyperphagia on days of free access to food, by which the intermittently starving rats partly compensate for the period of starvation[5], leads, in addition to other metabolic sequelae, to a markedly enhanced lipogenesis, which persists even after fasting. In the absorptive phase, the *in vitro* lipogenesis by liver slices (Table I) is about four times greater in intermittently starving rats than in the controls. After subsequent 48 h acute starvation, when in agreement with literary data lipogenesis is considerably suppressed in the controls, the incorporation of radioactive acetate in intermittently fasting rats is even of a higher order than in the comparable group of controls fasting for an equal period, and is still three times greater as compared with values of controls in the absorptive phase.

Tab. I. *In vitro* incorporation of 1-C^{14}-acetate into fatty acids by rat liver slices, expressed as percentage of activity added (mean ± S.E., five animals per group)

Group	Absorptive phase	Fasting 48 h
Controls	4.22 ± 1.01	0.72 ± 0.19
Intermittent starvation (3 weeks)	15.50 ± 3.10[a]	13.51 ± 1.64[b]

[a] Difference, as compared with the control group, is significant for $P < 0.02$.
[b] for $P < 0.001$.

The accentuated lipogenesis manifests itself also in the final carcass composition (Table II). The weight decrement of both underfed groups being equal, the carcass of intermittently starved animals contains a greater percentage of fat not only as compared with the continuously underfed group but also as compared with controls fed an unrestricted diet. The ratio of body protein, on the other hand, is lower in these animals than in the other two groups. The enhanced lipogenesis in intermittently starving animals manifests itself also by a different percentage of fat and protein increase or decrease during the period of experimental feeding (Table II).

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Tab. II. Body-weight and carcass composition of rats after 11 weeks of experimental feeding (mean ± S.E., body-weight as the nearest whole number)

Group	No. of rats	Average food intake (g/rat/day)	Body-weight (g) initial	Body-weight (g) final	Body-fat %	Body-fat change[a] %	Body-protein %	Body-protein change[a] %
Controls	17	27.6	163 ± 4.2	291 ± 9.3	10.53 ± 0.21	+ 99.0	20.00 ± 0.35	+ 78.0
Intermittent starvation	18	11.6	164 ± 3.3	156 ± 3.5	11.74 ± 0.25[b]	+ 10.0	17.00 ± 0.16[b]	− 21.3
Continuous underfeeding (pair-fed)	17	11.6	164 ± 3.6	155 ± 3.1	9.93 ± 0.22	− 6.8	19.14 ± 0.24	− 12.0

[a] Based on the amount of fat and protein in control animals, comparable as to body-weight, killed at beginning of the feeding period.
[b] Difference, as compared with both remaining groups, is significant for $P < 0.01$.

The results are in keeping with our previous work where we assumed enhanced lipogenesis in intermittently starving rats in view of their raised respiratory quotient[6]. Periodic concentration of food ingestion into brief periods thus leads to markedly accentuated lipogenesis not only when the caloric intake is nearly adequate[7] but also when the supply of calories is substantially reduced. This fact could possibly interfere with adipose tissue reduction in some obese patients whose self-imposed dietary habits sometimes resemble the dietary pattern of intermittent starvation.

Zusammenfassung. Albinoratten, bei denen zwischen die Fütterungstage 1–2tägige Hungerperioden eingesetzt wurden, zeigten nach 3 Wochen eine wesentlich erhöhte Inkorporation von 1-C^{14}-Acetat in die Fettsäuren der Leberschnitte. Selbst bei Herabsetzung der Kalorienzufuhr um ca. 50% stieg hier, im Gegensatz zu kontinuierlich unterernährten Tieren, der prozentuale Körperfettgehalt. Die Resultate sprechen für eine Steigerung der Lipogenese.

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Antigen-Distribution in Rat Liver Mitochondria

It has been shown that particulate components, sedimented from homogenates of rat liver by means of differential centrifugation and cell sap itself, have immunological properties[1].

These findings suggest the possibility that a study of the antigenic properties of cellular components might yield information as to the pattern of distribution of intracellular proteins.

Results on the immunological properties of rat liver mitochondria and their deoxycholate sub-fractions will be reported here.

The perfused livers were homogenized in 0.44 *M* sucrose in water and the mitochondria isolated by centrifugation at 10000 *g* for 10 min, after removal of cell debris and nuclei at 1000 *g* for 15 min. The microsomal fraction was obtained by centrifugation for 1 h at 105000 *g* of mitochondria supernatant, previously, however, centrifuged at 30000 *g* for 30 min. Both the mitochondrial and microsomal fractions were washed twice with sucrose. By treatment of the particulate fractions with sodium deoxycholate 3% at pH 7.8 (0.3% final concentration), deoxycholate soluble (dc-sol) and deoxycholate-insoluble (dc-ins) fractions were obtained. The dc-ins fraction from mitochondria will be referred to as mitochondria membranes[2]. Cell sap was obtained by centrifugation at 105000 *g* for 1 h of the supernatant of microsome preparations.

To obtain the antisera, the samples were injected into rabbits using adjuvant technique[3]. 6 rabbits were injected with each antigen preparation. For the immunological experiments the double diffusion agar technique was used[4] and the reactions were allowed to proceed at 20°C. After completion, the agar plates were washed and recorded photographically.

Mitochondria were tested with sera produced against mitochondria themselves, dc-sol mitochondria fraction, mitochondria membranes, microsomes, and cell sap.

As shown in Figure 1, mitochondria preparations tested with a homologous antiserum gave rise to several precipitation lines. When the same mitochondria suspension was allowed to react with an antimembrane serum, only two groups of antigens were demonstrated by precipitation lines joining with identical lines present in the reaction with mitochondria antiserum. One of these has further proved to be identical with one of the several lines present in the reaction between mitochondria and anti-dc-sol mitochondria fraction, while the other one crossed all of them.

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