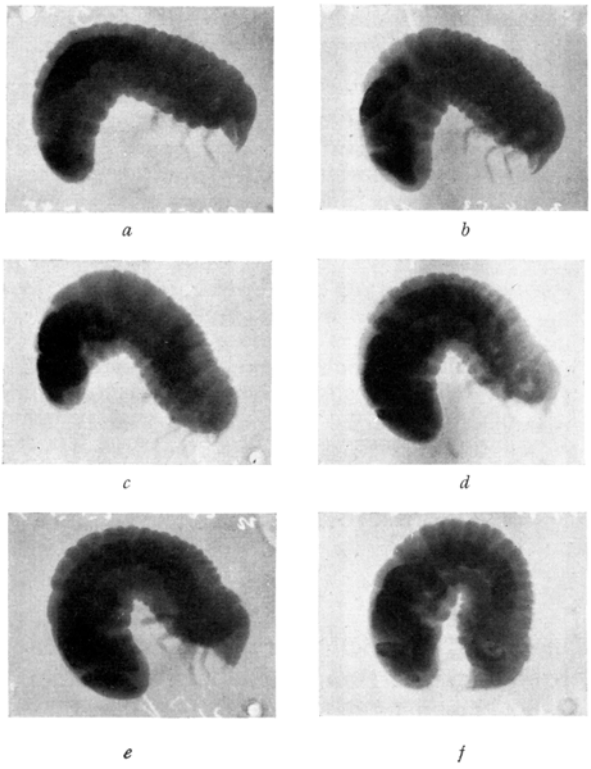

6 Stadien aus einem Röntgenfilm über die Nahrungspassage im Darmkanal der Nashornkäferlarve. *a* 29. April, 17.45 Uhr; *b* 30. April 11.15 Uhr; *c* 2. Mai, 9.50 Uhr; *d* 4. Mai, 21.10 Uhr; *e* 6. Mai, 23.00 Uhr; *f* 8. Mai, 17.40 Uhr. Bei *c* ist der gesamte kontrastmittelhaltige Nahrungsbrei im Saccus; bei *d* ist der Rücktransport im Mitteldarm deutlich zu sehen.

primären Stickstoffgewinn erfolgt somit ein erneuter Verbrauch. Das auffallende Faktum ist, dass die Larve des Nashornkäfers im Kot die dreifach grössere N-Menge ausscheidet, als sie mit dem Futter aufgenommen hat. Parallel hierzu ergaben die kalorimetrischen Verbrennungen für das Futter einen Energiegehalt von 4,09 Cal/1 g Futter (Streuung 4,2–4,03 Cal/1 g) und für den Kot 4,30 Cal/1 g Kot (Streuung 4,42–4,23 Cal/1 g). Keimzählungen im Mitteldarm, im Saccus und im Kot ergaben eine auffallende Verschiedenheit in den Populationszahlen. Für den Mitteldarm fanden wir 188 Millionen Keime pro 1 cm³, für den Saccus 470 Millionen Keime und für den Kot 76 Millionen Keime pro 1 cm³. Wie bei den N-Gehaltsbestimmungen zeigen auch diese Zahlen, dass die N-Quelle in der Bakterienvermehrung im Saccus zu sehen ist. Ob die Bakterien des Saccus hauptsächlich die im Holz vorliegende Zellulose oder andere Zucker (Pentosane) zum Aufbau ihres Körpereiweisses verwenden, konnten wir noch nicht klären. Die Untersuchungen darüber werden fortgesetzt.

*Wirkungsort des Fermentes.* Die vorstehenden Befunde liessen uns an den Verdauungsmodus bei Wiederkäuern denken und an die Möglichkeit eines Rücktransportes von Nahrungsbrei und Bakterienflora im Verdauungstraktus der Orycteslarve zum Mitteldarm.

Röntgenologisch haben wir den Weg, den die Nahrung im Darm zurücklegt[1], verfolgt (Abb.). Die Aufnahmen zeigen, dass der Darmbrei (Sägemehl mit Bariumsulfat als Kontrastmittel) zunächst in etwa 17 h durch den Mitteldarm hindurch in den Saccus gelangt und dort ungefähr 3–4 Tage verweilt. Hierauf erfolgt ein fraktionierter Rücktransport in den Mitteldarm. Noch nach 7 Tagen konnte bariumsulfathaltiger Nahrungsbrei im Mitteldarm festgestellt werden[1].

Damit ist nachgewiesen, dass in der Tat die Orycteslarve das Wiederkäuen von Zellulose im Insektenreich verwirklicht. Die bakterielle Vorverdauung ist im Saccus lokalisiert, während die proteolytische Hauptverdauung offenbar mit dem Wiederkauakt verknüpft ist und nur im Mitteldarm stattfindet.

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*Summary*

The larva of *Oryctes nas.* L. shows a bacterial predigestion in the rectal-saccus. The main breakdown of proteins takes place in the middle gut, closely connected with the act of regurgitation.

[1] TH. WILDBOLZ, Mitt. Schweiz. Entomolog. Ges. *27*, 193 (1954). TH. WILDBOLZ hat bei der Larve von *Melolontha melolontha* L. die Verweildauer der Nahrung im Darmkanal untersucht. Er findet eine sehr variable Passagegeschwindigkeit und im Mitteldarm Gasblasen, die er als beim Schlingakt aufgenommene Luft interpretiert. Auf Grund der eigenen Befunde ist anzunehmen, dass es sich dabei um Gärgase aus dem Saccus handelt.

## Fat Metabolism in Various Forms of Experimental Obesity. VI. Instantaneous Lipogenesis in Hypothalamic Obese Mice[1]

In a series of reports[2] it was shown that various forms of experimental obesity differed as far as lipogenesis was concerned. Mice with the hereditary obese-hyperglycemic syndrome incorporated more acetate into fatty acids than did their non-obese siblings not only, as predictable, when both groups were fed *ad libitum*, but also when the obese were pair-fed with the controls, when both groups were underfed in similar proportion and even when both groups were fasted. By contrast, goldthioglucose obese mice and hypothalamic hyperphagic rats only incorporated more acetate into fat than their controls in the measure that they were allowed to overeat. The first type of obesity (increased lipogenesis under fasting, etc., conditions) has been termed "metabolic" obesity, the second type (increased lipogenesis dependent upon increased caloric intake) has been termed "regulatory obesity"[3]. Since lipogenesis in genetic obesity and goldthioglucose obesity had been studied in mice, and hypothalamic obesity in rats, species differences could vitiate the comparison between the three forms. Hypo-

[1] Supported in part by grants-in-aid from the National Institute of Arthritis and Metabolism (Grant No. A49C2R), National Institutes of Health, Public Health Service, U. S. Department of Health, Education, and Welfare; Sugar Research Foundation, New York City; Kellogg Co., Battle Creek, Michigan; and the J. M. Kaplan Fund, Inc., New York.

[2] M. W. BATES, J. MAYER, and S. F. NAUSS, Amer. J. Physiol. *180*, 304 (1955). – M. W. BATES, C. ZOMZELY, and J. MAYER, Amer. J. Physiol. *181* 187 (1955). – J. MAYER, N. C. HAGMAN, N. B. MARSHALL, and A. J. STOOPS, Amer. J. Physiol.(in press, June 1955).

[3] J. MAYER, Proc. IIIrd Internat. Nutrition Congr., Voeding, *16*, 62 (1955).

[1] E. T. NIELSEN, Ent. Medd. *23*, 255 (1943).

$C^{14}$-acetate Incorporation into Carcass and Liver Fatty Acids in Hypothalamic Obese Mice and Controls

| Nutritional Status | Type * | Body Weight g | Liver Weight g | $C^{14}$-Incorporation | | |
|---|---|---|---|---|---|---|
| | | | | Total | Carcass | Liver |
| Nonfasted | Obese | 54·7 ± 5·7 | 2·228 ± 0·78 | 1266 ± 236 | 930 ± 32 | 384 ± 43 |
| Nonfasted | Nonobese | 34·0 ± 1·3 | 1·875 ± 0·06 | 3705 ± 561 | 318 ± 17 | 52 ± 17 |
| Fasted | Obese | 59·8 ± 6·6 | 2·965 ± 0·40 | 231 ± 69 | 205 ± 28 | 42 ± 13 |
| Fasted | Nonobese | 28·1 ± 2·0 | 1·810 ± 0·11 | 249 ± 23 | 187 ± 42 | 27 ± 6 |

* 8 animals of each type. Incorporation is expressed as percentage counts retained × $10^3$.

thalamic obesity has since been achieved in mice[1]. It was therefore logical to repeat the most informative lipogenesis experiment using hypothalamic hyperphagic mice rather than rats, to check that the characteristics previously described were typical of the syndrome itself and not limited to one species.

Sixteen 4 month old female mice were used. The experimental animals were 8 hypothalamic obese swiss mice, weighing from 50 to 65 g and their unoperated controls, weighing 27 to 36 g. The obese animals had been operated according to the procedure described previously[1]. The obese mice used in the "non-fasted" experiment had been operated 4 months previously and were putting weight on slowly. The obese mice used in the "fasted" (16 h fast) experiment had been operated 1 month previously and were still putting on weight rapidly. (Thus, the demonstrated failure to demonstrate an increased acetate incorporation in the fasted state, contrasting with the picture in the fed state could not be attributed to the fasted animals being in a less "dynamic" form of obesity[2].) All mice were maintained in individual cages, in an air-conditioned room and were fed Purina Laboratory Chow *ad libitum*. The animals were injected with 0·5 $cm^3$ of saline solution containing 0·075 mg of $C^{14}$-carboxyl-labeled acetate containing 1 500 000 cpm in the case of the obese animals, 150 000 cpm in the case of the nonobese. They were killed half an hour later and the procedure previously described[3] was followed to isolate the liver and carcass fatty acids and count the radioactivity. Results were expressed in percent counts retained multiplied by $10^3$.

From the data in the table, it is readily apparent that the fed hypothalamic obese mice incorporated considerably more acetate into fatty acid than the nonobese (approximately 4 times as much). Lipogenesis was greater both in the carcass (3 times) and in the liver (over 7 times). On the other hand, in the fasted state, total incorporations were not significantly different between obese and nonobese mice. Carcass lipogenesis was similar in obese and nonobese mice. There was some difference between liver incorporation in the two types of animals, of borderline significance ($p \leqslant 0{\cdot}05$) as had been observed in the hypothalamic obese rats. The significance of this slight residual difference, not demonstrated by isolated liver slices[4] is obscure, but is most probably related to the differences in liver weights between obese and nonobese animals. Actually, if liver incorporation is expressed on the basis of counts per gram, the counts in the obese mice are identical to those in the nonobese.

Incidentally, it is of interest to note that the livers of the obese mice used in the "fasting" experiment were larger than those of the group used in the "nonfasted" experiment. The former had been in a state of "dynamic" obesity (rapid weight gains), while the latter had gained weight more slowly. The animals of both groups were of similar over-all size. Furthermore, liver weight is very sensitive to the nutritional status and was most probably decreased somewhat by the 16 h fast. This finding would tend to indicate that the increase in liver weight encountered in various forms of experimental obesity[1] is probably related, at least in part, to the hyperphagia rather than to the excess weight as such.

The results presented here on lipogenesis confirm that the results previously obtained on hypothalamic hyperphagic rats are generally valid for hypothalamic hyperphagia in other species. They thus confirm the suggestion that this syndrome is of "regulatory" rather than "metabolic" nature.

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*Zusammenfassung*

Bei Mäusen und Ratten mit hypothalamischer Fettsucht beobachtet man bei Fütterung *ad libitum* vermehrte Fettbildung, die zurückgeht, wenn man die Tiere hungern lässt. Diese Befunde lassen auf einen «regulatorischen» und nicht «metabolischen» Charakter dieser Fettsucht schliessen.

[1] R. G. French, C. Y. Zighera, and J. Mayer, Fed. Proc. *14*, 433 (1955). – J. Mayer, R. G. French, C. Y. Zighera, and R. J. Barrnett, Amer. J. Physiol. (in press, July 1955).

[2] J. Mayer, Physiol. Revs. *33*, 472 (1953).

[3] M. W. Bates, C. Zomzely, and J. Mayer, Amer. J. Physiol. *181*, 187 (1955).

[4] J. Mayer, N. C. Hagman, N. B. Marshall, and A. J. Stoops, Amer. J. Physiol. (in press, June 1955).

## Paléontologie et expérimentation

Contre les hypothèses voulant expliquer par des actions mécaniques la morphogénie dentaire, il était facile de trouver des arguments; en particulier, l'existence de dents de forme normale, bien qu'hétéropiques ou qu'implantées de travers, pouvait être invoquée. Des faits de même ordre ont pu être provoqués grâce à des cultures de tissus. Déjà, Glasstone[1], en explantant des germes de dents de Rat, avait prouvé que la formation, la forme et le nombre des cuspides n'étaient déterminés que par des facteurs endogènes[2]. Verne[3] a montré que des germes dentaires, prélevés sur des embryons, évoluaient avec

[1] A. H. Glasstone, Dent. Rec. *1938*.

[2] En 1940, A. G. Lapchinsky et A. A. Malinovsky[4] réussissaient également à obtenir des molaires ou des incisives, d'apparence normale, en prélevant des germes de rat ou de chien et en les insérant, soit dans une autre région des mâchoires, soit à la surface du fémur.

[3] J. Verne, Rev. odontol. *64*, 55 (1942).

[4] A. G. Lapchinsky et A. A. Malinovsky, C. r. Acad. Sci. U.R.S.S. *26*, 722 (1940); *29*, 268 (1940).