however, until the end of exercise, when it rises in a fashion similar to the free fatty acids (Harris *et al.* 1965). Presumably the reasons for this are similar to those which underlie the changes in the free fatty acids.

Such simple observations serve to illustrate how very complex is the metabolic response of the body to physical activity. At a more practical level they also show how important is the timing of sampling in any study designed to relate the concentration of a metabolite in the blood with some other physiological measurement during exercise.

REFERENCES

Asmussen, E. (1950). *Acta physiol. scand.* **20**, 125.
Bang, O. (1936). *Skand. Arch. Physiol.* **74**, Suppl. **10**, p. 49.
Havel, R. J., Naimark, A. & Borchgrevink, C. F. (1963). *J. clin. Invest.* **42**, 1054.
Harris, P., Bateman, M. & Gloster, J. (1962*a*). *Clin. Sci.* **23**, 531.
Harris, P., Bateman, M. & Gloster, J. (1962*b*). *Clin. Sci.* **23**, 545.
Harris, P., Bateman, M. & Gloster, J. (1962*c*). *Clin. Sci.* **23**, 561.
Harris, P., Fletcher, R. F., Gloster, J. & Gotsman, M. (1965). *Clin. Sci.* **28**, 343.
Huckabee, W. E. (1958*a*). *J. clin. Invest.* **37**, 244.
Huckabee, W. E. (1958*b*). *J. clin. Invest.* **37**, 255.
Lundin, G. & Ström, G. (1947). *Acta physiol. scand.* **13**, 253.
Margaria, R., Edwards, H. T. & Dill, D. B. (1933). *Am. J. Physiol.* **106**, 689.

Nutrition and its relation to body composition in exercise

By JANA PAŘÍZKOVÁ, *Physical Culture Research Institute, Prague, Czechoslovakia*

Increased energy output due to enhanced physical activity in sportsmen requires correspondingly increased calorie input which reaches 7000–8000 kcal per day in special kinds of sports disciplines during most intensive periods of training. Such an increased input does not result, as it surely would in subjects of an average population, in enhanced body fatness; on the contrary, typical body composition, i.e. a high ratio of lean body mass at the expense of fat, is one of the most remarkable features of the physique of top athletes. Matiegka (1921) was the first who evaluated these characteristic differences by means of anthropometric measurements. By a special method, densitometry, the body composition of sportsmen was studied by Behnke, Feen & Welham (1942) and Welham & Behnke (1942). In their work it was shown that even in instances of increased relative body-weight which in subjects not systematically interested in sports characterizes enhanced development of fat, the body of an athlete contains only a very low ratio of adipose tissue and the increased weight is due to highly developed muscle mass.

Not only sport but also different professional activity could cause differences in the ratio of body fat to lean body mass, which was demonstrated in subjects from various countries by Brožek (1956) and Keys & Brožek (1957). Tanner (1952) elucidated the differences of physique in trained and untrained subjects by anthropometric methods. In men during military service trained to different work loads the lowest skinfold thicknesses were always found in men exposed to greatest physical strain (Koldovský, Pařízková, Špaček & Hahn, 1959). In the Soviet Union the differences between athletes and subjects who did not take part in sports were followed by means of densitometry by Khanina & Chagovetz (1954) and later by Zhdanova (1962). In Poland the problems of body composition in sportsmen were studied by Drobny (1958).

The influence of systematic sport activity on body composition not only in adults but also in children, adolescents (Pařízková-Čapková, 1957; Pařízková, 1959, 1962, 1963) and in subjects of advanced age (Eiselt, Parízková & Zbuzek, 1961; Pařízková, 1963; Kuta, Pařízková & Eiselt, 1964) was studied in the Czechoslovak population. The magnitude of the differences in body composition was obviously caused by the intensity of physical strain, as the maximal differences were found among members of Olympic teams and normal population. Longitudinal investigations of growing as well as adult sportsmen revealed a dynamic relationship between body composition and changes in the intensity of training, and consequently also in the degree of physical fitness (Pařízková, 1963, 1965; Pařízková & Poupa, 1963): with increasing intensity of training the lean body mass in adult leading sportsmen increased at the expense of fat, together with unchanged or slightly decreased body-weight, and in growing girls with increased body-weight. In the periods of diminished physical activity the enhanced deposition of fat occurred in all subjects, with more significant increase of total body-weight both in adult and in growing subjects. In the average population without more marked changes in the intensity of physical activity no similar changes could be proved (Pařízková, 1959, 1962).

The enhanced deposition of fat after the interruption of systematic and intensive training is not due to impaired regulation of food intake, i.e. equal or higher calorie intake during both the period of very high and reduced intensity of training (Pařízková & Poupa, 1963). Quantitative and qualitative study of calorie intake showed that during the period of reduced physical activity the calorie input fell by 25% compared to that in the period of intensive training. The relation of calorie input from proteins, carbohydrates and fats during the period of intensive training was 13:50:37; during the period of relative rest the calorie intake from protein decreased significantly by 2·1% and that from carbohydrates rose significantly by 2·7%. These changes, however, were so slight that they can hardly be considered as having caused the increased formation of fat; it is therefore probable that increased laying down of fat during the period of relative rest that follows prolonged intensive physical exercise is not due to disturbed regulation of the quantity and quality of ingested calories, but that some further changes, probably adaptive transformations of inner fat metabolism involving the deposition and release of fatty acid in adipose tissue are probably of special importance. It is known that during prolonged intensive muscular work utilization of fatty acids in muscles is increased; this situation applies to man (Carlson & Pernow, 1959, 1961; Havel, Naimark & Borchgrevink, 1963) and some data from comparative physiology also support this concept (George & Naik, 1957; George & Vallyathan, 1963).

We had occasion to study some of these problems in a group of obese boys before and after summer camp with regulated diet (1700 kcal/day) and intensive physical training, which resulted in a remarkable reduction of superfluous fat together with significant improvement of physical fitness and aerobic capacity; this was evaluated according to changes of oxygen consumption during maximal work load on a treadmill (Šprynarová & Pařízková, 1965). The control of various blood indicator levels (glucose, esterified and non-esterified free fatty acids) before and after maximal work load measured before and after the stay in the training camp showed that though the free fatty acid (FFA) blood level before the camp was significantly higher (proportionately according to the amount of body fat) it decreased during maximal work and then remained unchanged. After the training camp when greater performance ability was proved the FFA blood level was significantly lower, but remained unchanged during the work and increased after the rest. A significant negative correlation was found between the increase in pulse frequency and the decrease in FFA in blood during maximal work load both before and after training camp; the higher the pulse frequency increase (i.e. the poorer the adaptation for intensive muscular effort) the greater decrease of blood FFA level during maximal work load (Pařízková, Staňková, Šprynarová & Vamberová, 1965). All the results mentioned, together with the conclusions from the literature, seemed to show that the adaptation for higher muscular activity characterized also by typical body composition, changes the ability to mobilize FFA from the fat depots according to the enhanced utilization in muscles during work load.

As we had no opportunity to sample the adipose tissue of athletes we performed our studies in an experimental model. In the second part of my communication I should like to present some results for male rats adapted gradually at various ages for long-term exercise on a treadmill. Several groups of seven to eleven male rats at the ages of 85, 125, 195 and 360 days were further subdivided according to physical activity. In the first three groups running on a treadmill was begun immediately after weaning, i.e. 35 days after birth and was gradually increased so that the animal under given conditions ran always to its maximal capacity, i.e. 180 min in youngest to 50 min in oldest animals), but in a manner not to evoke a stress. This was proved by identical growth curves and the fact that only in two instances significant differences were found in absolute weights of the adrenals between running animals and animals with limited physical activity, but not between running and controls.

All animals were given the Larsen diet *ad lib.*; repeated short-term controls of several groups showed an increase of calorie input in younger running animals by 10–13% (Pařízková, Zbuzek & Bartosová, 1962; Pařízková, Staňková, Fábry & Koutecký, 1966). In the groups of trained and control animals killed at the age of 360 days, and in which the training was begun at 160 days, the amount of diets of different composition eaten between the 250th and 270th days of life was also studied. The amount of calories eaten per day per 100 g body-weight was lower than for younger age groups and did not differ according to physical activity; but running rats selected spontaneously equal amounts of diets with high starch and high fat content. The control rats selected on the average twice as much of the diet with the high fat content as of that with the high starch content. Both groups ate the same amount of the diet with the high casein content (Pařízková & Staňková, 1964).

As the physical activity could influence spontaneous calorie input and its frequency during the day, which according to Teppermann, Brobeck & Long (1942), Cohn (1963), Fábry, Petrásek, Kujalová & Holečkova (1962) and others influences the intermediary metabolism and composition of adipose tissue, we also controlled in one group (195 days) the frequency of calorie input during certain periods. It was demonstrated that the animals accepted the diet continuously and that there was no difference between the groups according to physical activity. This led us to the conclusion that the observed changes in adipose tissue were due mainly to the influence of enhanced physical activity (Pařízková, Staňková, Fábry & Koutecký, 1966).

All running animals were killed together with controls 24 h after the last run on a treadmill. Like athletes, the running animals had a significantly lower ratio of body fat to muscle with unchanged total body-weight, and also the weight of selected muscles (m. tibialis, m. soleus) was mostly markedly increased. When comparing individual groups of various ages it was shown that in younger rats (85 days) there was a difference not only between control and running animals in which a significantly lower ratio of total body fat was found, but also between control animals and animals with limited physical activity in which a significantly higher ratio of fat was demonstrated. This latter difference was not proved in the 125-day-old group in which only running animals had a significantly lower ratio of body fat in comparison with the other two groups (Pařízková & Staňková, 1966) (Table 1).

Table 1. *Percentage of fat in the bodies of running and control rats and rats with limited physical activity at different ages* (*Pařízková* & *Staňková*, 1966)

Age	Running		Control		Limited activity	
(days)	Mean	SD	Mean	SD	Mean	SD
85	4·6	1·6	10·0	1·6	13·3	2·0
125	9·0	2·2	14·7	3·2	14·7	4·8
360	17·1	4·0	26·0	6·0	—	—

The amount of FFA released in vitro from epididymal adipose tissue during incubation in Krebs–Ringer phosphate buffer with 3% of albumin and adrenaline, titrated according to Dole (1956), was always higher in running animals; the most marked differences were found after 100 min incubation. In the group of growing animals (85 days) only the difference between running animals and animals with limited physical activity was significant, but not between controls and the other two groups though the intensity of physical activity in running animals of this age group was highest in comparison with other age groups. In older groups the running animals always differed most markedly and significantly from the control group and the group with limited physical activity, but there was no significant difference between the last two groups (Table 2). It seems, therefore, that in the period of growth a more significant stimulus was limited physical activity, leading to lower release of FFA and higher ratio of body fat to muscle; in full-grown and older animals, on the contrary, the enhanced physical activity led to relative increase of FFA

Table 2. *Mean values and standard deviations for release, in vitro by 2 μg adrenaline, of free fatty acids from the epididymal adipose tissue of male rats of different ages running on a treadmill or with limited physical activity*

Group	Age (days)	FFA released (μequiv./ml medium per g tissue) 60 min incubation	100 min incubation	210 min incubation
Running	85	8·5±2·9	11·1±3·0	17·5±6·0
Control		8·2±3·5	10·5±3·8	13·3±3·9
Limited activity		6·3±1·1	8·5±2·0	12·1±1·2
Running	125	7·9±1·6	10·7±1·5	16·1±2·1
Control		4·7±1·7	7·1±1·3	12·1±1·6
Limited activity		4·4±1·9	6·8±2·2	13·0±3·7
Running	360	3·9±0·8	7·4±1·3	12·4±3·4
Control		2·4±1·8	4·4±1·7	10·5±4·9

release and decrease of body fat. Between these indicators there existed a close relationship. A significant negative correlation between FFA release in vitro by adrenaline from epididymal adipose tissue and total body fat ratio was proved, the greater the ratio of body fat the lower the release of FFA. This relationship was proved only in adult animals; in growing animals it was not demonstrated (Table 3). With both high and low body fat ratios the release of FFA in vitro was always increased (Pařízková & Staňková, 1966) which was probably an effect of growth hormone (Engel, 1962). Further, this relationship was not proved in another group of animals of different age and weight which were not fed *ad lib.* and which did not differ in the degree of physical activity or in total body fat ratio (Pařízková & Staňková, 1966).

Table 3. *Correlation coefficients for the relationship between the release in vitro by adrenaline of free fatty acids from adipose tissue and the ratio of total body fat to lean body mass in male rats at different ages* (*Pařikozvá & Staňková*, 1966)

	No adrenaline				2 μg adrenaline					
Age (days)	60 min incubation		210 min incubation		60 min incubation		100 min incubation		210 min incubation	
	r	Significance	*r*	Significance	*r*	Significance	*r*	Significance	*r*	Significance
85	−0·452	0·05>*P*>0·02	−0·220	0	−0·158	0	−0·347	0	−3·051	0
125	−0·792	*P*<0·001	−0·305	0	−0·702	*P*<0·001	−0·719	*P*<0·001	−0·783	*P*<0·001
360	—	—	—	—	−0·395	0	−0·541	0·05>*P*>0·02	−0·494	*P*<0·001

Another comparison showed that running animals of older age groups did not differ significantly from control groups of younger animals either in total body fat or in FFA release in vitro (Tables 1 and 2).

Higher release of FFA in younger animals was demonstrated by many authors (Altschuler, Lieberson & Spitzer, 1962; Marshall & Engel, 1959–60; Tenorová & Hrůza, 1962). It was also proved that the adipose tissue of younger animals is relatively more cellular and contains therefore more deoxyribonucleic acids (Jelinková, Myslivečková & Hrůza, 1965). The measurement of DNA content in adipose tissue (Slabochová & Placer, 1962) in one of our groups (195 days) showed that the adipose tissue of running animals included also a relatively greater amount of DNA. A significant negative correlation was found between the DNA content of adipose tissue and total body fat ratio, the higher the adipose tissue in the organism, the lower the DNA content. When the released FFA were recalculated in relation to DNA content in adipose tissue no difference between groups according to physical activity could be demonstrated. Further, released FFA per DNA content in adipose tissue had no relationship to the total body fat ratio (Pařízková *et al.* 1966). All these results led us to the conclusion that physical activity probably inhibits the hypertrophy of fat cells which occurs regularly during ageing (especially in the case of nutrition with *ad lib.* feeding) and keeps the adipose tissue from some points of view, i.e. total body ratio, cellularity and ability to react more promptly to adrenaline by releasing FFA, in a state corresponding to that found at an earlier stage of development.

We were further interested to know whether the difference between adipose tissue of animals with different physical activity could be proved also in the basal state; the spontaneous FFA release and lipolytic activity of adipose tissue (Cherkes & Gordon, 1959-60) was also studied in one of our groups (Pařízková *et al.* 1966). The results did not differ significantly, the difference in adipose tissue in groups varying in physical activity manifested itself only in the presence of adrenaline which further related to the content of DNA (Pařízková *et al.* 1966). Different proportions of some FFA in adipose tissue of running rats were also found. The proportion of palmitoleic acid was significantly lower in comparison with controls. There was a close relationship between total body fat and the content of palmitoleic acid (% of total FFA) $r=0{\cdot}623$; $0{\cdot}01<P<0{\cdot}02$ (Kohout, Braun & Pařízková, 1965) and the release of FFA in vitro after adrenaline and the content of palmitoleic acid $r=0{\cdot}682$; $0{\cdot}01<P<0{\cdot}02$ (Pařízková & Staňková, 1966).

Observed changes in body composition, ratio of body fat and lean body mass in relation to physical activity, could be caused not only by the intensity of FFA release but also the ability of adipose tissue to incorporate FFA into triglycerides. The uptake by epididymal adipose tissue of FFA added to the incubation medium with insulin and glucose was therefore studied in one of our groups (195 days). The same uptake of FFA after addition of insulin or insulin and glucose in all groups was observed; only after addition of glucose alone was a significantly lower uptake of FFA from the medium found in the adipose tissue of running animals in comparison with controls and animals with limited physical activity. In relation to the DNA content of adipose tissue the uptake was however not different in any instance. In our experimental conditions physical activity did not apparently influence the ability of adipose tissue to take up the FFA from the medium in the presence of insulin. It seems that increased amount of glucose alone was in this instance a less significant stimulus for the adipose tissue of running animals to take up the FFA from the medium and probably also for triglyceride formation than in other groups. The increased ability to release FFA from adipose tissue in the presence of adrenaline and the lower uptake of FFA from the medium with glucose, which is probably also utilized in greater quantity in running animals and is eventually stored in higher amounts in the form of glycogen, was shown in trained subjects. This seems to prove that the adipose tissue in running animals adapted for higher physical activity is obviously adapted for high turnover rate and rapid mobilization of FFA according to energetic needs of the working mechanism. This conclusion is in agreement with the results of Issekutz, Miller, Paul & Rodahl (1965) who found markedly enhanced turnover of FFA during exercise in trained dogs in comparison with untrained controls.

The results presented show that adaptation for intensive muscular work changes not only the need for quantity and quality of ingested calories, but also influences markedly all metabolism and utilization of nutrients, especially of fat, which is further reflected in changes in the ratio and reactability of adipose tissue.

REFERENCES

Altschuler, H., Lieberson, M. & Spitzer, J. J. (1962). *Experientia* **18**, 91.
Behnke, A. R., Feen, G. B. & Welham, W. C. (1942). *J. Am. med. Ass.* **118**, 495.
Brožek, J. (1956). In *Body Measurement and Human Nutrition*, p.14. [J. Brožek, editor.] Detroit, Michigan: Wayne University Press.
Carlson, L. A. & Pernow, B. (1959). *J. Lab. clin. Med.* **53**, 833.
Carlson, L. A. & Pernow, B. (1961). *J. Lab. clin. Med.* **58**, 673.
Cherkes, A. & Gordon, R. S. Jr. (1959-60). *J. Lipid. Res.* **1**, 97.
Cohn, C. (1963). *Ann. N.Y. Acad. Sci.* **110**, 395.
Dole, V. P. (1956). *J. clin. Invest.* **35**, 150.
Drobny, Z. (1958). *Wychowanie fyziczne i sport.* **2**, 473.
Eiselt, E., Pařízková, J. & Zbuzek, V. (1961). *Vnitřní Lék.* **7**, 778.
Engel, F. L. (1962). In *Adipose Tissue as an Organ. Proceedings of the Deuel Conference on Lipids*, p. 126. [L. W. Kinsell, editor.] Springfield, Ill.: Ch. Thomas.
Fábry, P., Petrásek, R., Kujalová, V. & Holečková, E. (1962). *Adaptace na změněný příjem potravy.* Prague: Státní zdravotnické nakladatelství.
George, J. C. & Naik, R. M. (1957). *J. Anim. Morph. Physiol.* **4**, 23.
George, J. C. & Vallyathan, N. V. (1963). *Can. J. Physiol. Pharmac.* **42**, 447.
Havel, R. J., Naimark, A. & Borchgrevink, C. F. (1963). *J. clin. Invest.* **42**, 1054.
Issekutz, B. Jr, Miller, H. I., Paul, P. & Rodahl, K. (1965). *J. appl. Physiol.* **20**, 293.
Jelinková, M., Myslivečková, A. & Hrůza, Z. (1965). *Physiologia bohemoslov.* **14**, 146.
Keys, A. & Brožek, J. (1957). *Metabolism* **6**, 425.
Khanina, K. P. & Chagovetz, P. V. (1954). *Dopov. Akad. Nauk ukr. RSR* no. 2, p. 94.
Kohout, M., Braun, T. & Pařízková, J. (1965). *Physiologia bohemoslov.* **14**, 276.
Koldovský, O., Pařízková, J., Špaček, M. & Hahn, P. (1959). *Vojen. Zdrav. Listy* **28**, 190.
Kuta, I., Pařízková, J. & Eiselt, E. (1964). *Teorie Praxe tělesné Výcho Sportu* **12**, 348.
Marshall, N. B. & Engel, F. L. (1959–60). *J. Lipid. Res.* **1**, 339.
Matiegka, J. (1921). *Am. J. phys. Anthrop.* **4**, 223.
Pařízková, J. (1959). *Physiologia bohemoslov.* **7**, 112.
Pařízková, J. (1962). *Rozvoj aktivní hmoty a tuku u dětí a mládeže.* Prague: Státní zdravotnické nakladatelství.
Pařízková, J. (1963). *Ann. N.Y. Acad. Sci.* **110**, 661.
Pařízková, J. (1965). *Body Composition*, p. 161. Oxford: Pergamon Press.
Pařízková, J. & Poupa, O. (1963). *Br. J. Nutr.* **17**, 341.
Pařízková, J. & Staňková, L. (1964). *Br. J. Nutr.* **18**, 325.
Pařízková, J. & Staňková, L. (1966). *Nutritio Dieta.* (In the Press.)
Pařízková, J., Staňková, L., Fábry, P. & Koutecký, Z. (1966). *Physiologia bohemoslov.* **15**, 31.
Pařízková, J., Staňková, L., Šprynarová, S. & Vamberová, M. (1965). *Nutritio Dieta* **7**, 21.
Pařízková, J., Zbuzek, V. & Bartosová, D. (1962). *Čslka Fysiol.* **11**, 466.
Pařízková - Čaphová, J. (1957). *Čslka Pediat.* **12**, 310.
Slabochová, Z. & Placer, Z. (1962). *Nahrung* **6**, 525.
Šprynarová, Š. & Pařízková, J. (1965). *J. appl. Physiol.* **20**, 934.
Tanner, J. (1952). *Am. J. phys. Anthrop.* NS **10**, 427.
Tenorová, M. & Hrůza, Z. (1962). *Čslka Fysiol.* **11**, 485.
Teppermann, J., Brobeck, J. R. & Long, N. H. (1942). *Yale J. Biol. Med.* **15**, 855.
Welham, W. C. & Behnke, A. R. (1942). *J. Am. med. Ass.* **118**, 498.
Zhdanova, A. G. (1962). *Proceedings of an International Scientific Methods Conference on the Problems of Sport Training, Moscow*, p. 94.