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Influence of exercise on linear growth

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The effects of exercise on linear growth were investigated in malnourished rats and in preschool children recovering from protein-energy malnutrition (PEM). Physical activity was either restricted or forced upon weanling rats that were pair-fed with 73 or 49% of the food eaten *ad libitum* by rats of the same age. Physically active animals grew more in length and weight than their inactive counterparts. In a subsequent study, 20 children, 24–48 months old, under treatment for severe PEM, were assigned to either an Active group (stimulated, but not forced, to participate in games and activities that involved walking uphill, climbing a ramp, running, tumbling and climbing stairs) or a Control group (with the *ad libitum* pattern of physical activity and rest commonly observed in child-care and nutrition rehabilitation centers). Dietary intake was controlled and almost identical in both groups (2.5 ± 0.07 g protein and 117 ± 7 kcal/kg/day). Mean energy expenditure during daytime, assessed by heart rate monitoring and its relationship with oxygen consumption, were 2.0 and 1.7 times basal metabolic rate in the Active and Control groups, respectively ($P < 0.01$). In 6 weeks, both groups gained an average of 1.98 kg. However, the Active group grew more in length (22 ± 8 vs 14 ± 6 mm, $P < 0.05$) and lean body mass (final creatinine-height index: 0.97 ± 0.12 vs 0.89 ± 0.09, $P < 0.05$). The inactivity that accompanies severe malnutrition may contribute to stunting, whereas mild-to-moderate exercise combined with a good diet enhances linear growth. This may be mediated by endocrine growth factors, whose synthesis is prompted by exercise.

1. Introduction

Protein-energy malnutrition leads to growth retardation in children, affecting both body mass and stature. Dietary energy deficit has been considered one of the main causes of this growth retardation. However, the amount of dietary energy needed for growth between 1 and 5 years of age is only about 2–3% of the total daily requirement (WHO, 1985). Furthermore, although it has been shown that children of that age with dietary intakes 20–30% below requirement remain in energy balance by decreasing physical activity, they cease to grow, at least in terms of body mass (Rutishauser & Whitehead, 1972; Torun & Viteri, 1981). This occurred even when dietary intake of all nutrients, except energy, was adequate (Torun & Viteri, 1981).

This suggests that factors, other than dietary, may play a role in the growth retardation seen in malnutrition.

2. Studies with weanling rats

Based on the known anabolic effect of exercise on muscle growth, and on the fact that severely malnourished animals and children are very inactive, Viteri (1973) explored the role that exercise played on the growth of malnourished weanling rats.

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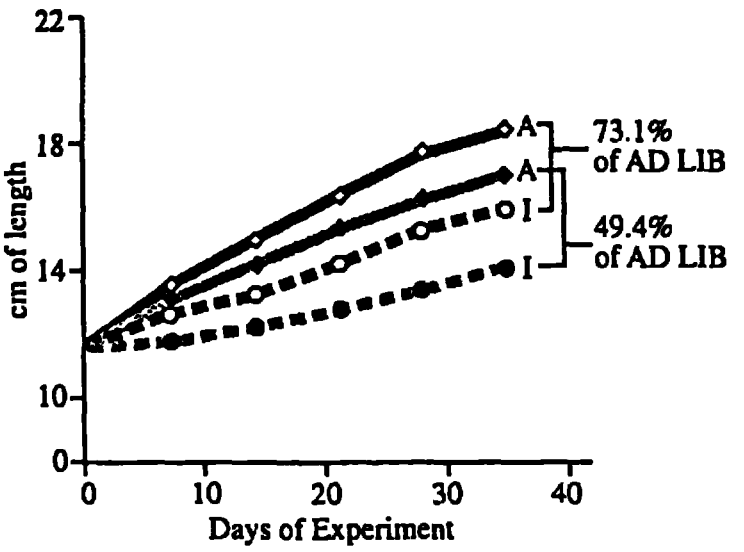

Fig. 1. Length of active (A) and inactive (I) rats fed either 73 or 49% of the diet normally eaten by well-fed rats (Viteri, 1973).

Male Wistar rats were weaned at 21 days of age and fed either 73 or 49% of the food eaten *ad libitum* by rats of the same age. Within each dietary regimen, one group of animals was housed in small individual metabolic cages that did not allow moving around ('Inactive' group), whereas another group ('Active') lived in larger individual metabolic cages and ran for 25 minutes in a revolving drum, twice daily, at a speed of 12.5 m/min. Both groups of rats were pair-fed at the prescribed level of food restriction.

As Fig. 1 shows, active rats grew more in terms of length than their inactive counterparts. Similar results were obtained in another study where a cross-over design was followed and special care taken to avoid coprophagia (Viteri & Torun, 1981).

3. Studies with children recovering from malnutrition

The studies in rats and observations in Nutrition Rehabilitation Centers, indicating that malnourished children recovered faster in centers with playgrounds than in smaller centers where the patients played indoors and were more sedentary (Viteri & Torun, 1981), led to a study designed to assess the effect of exercise on the growth of children during recovery from protein-energy malnutrition.

Twenty boys, 24–48 months old, with edematous protein-energy malnutrition, were admitted to INCAP's Clinical Research Center. All were wasted and stunted. The degree of stunting resulted in heights corresponding to those of healthy children 17 ± 3 months old, 19 months younger than the patients' chronological ages (Table 1). After initial recovery, when edema, apathy and irritability had disappeared, and the children were willing to participate in the games and activities that were part of this study, they were randomly assigned to one of two groups (Active and Control), paired according to age, height, and nutritional status (Table 1). Ten children were originally assigned to each group, but one of them was transferred from the Control to the Active group, as described below.

Nutritional and medical treatment, emotional support and other routine activities were the same for both groups. During the 6 weeks of study, they received a diet similar to that offered at that time in most nutrition rehabilitation centers in Guatemala. It was designed to provide 2.5 g protein/kg/day (1 g from milk, 1 g from egg and 0.5 g from corn) and 120 kcal/kg/day (30% from lipids). The diet was supplemented with vitamins and minerals to satisfy the needs of children recovering from malnutrition. All patients adhered strictly to this dietary protocol, and small amounts of fruit were given when a child requested additional food.

The two groups of patients differed only in relation to physical activity. Children in the Control group followed the pattern of physical activity and rest periods most commonly observed in rehabilitation and child-care centers dealing with malnourished patients. While out of bed they walked and moved around in their room and in the playing areas, and they were entertained by the staff with toys and games that did not require running or jumping, although such activities were not restricted. Children in

Table 1. Characteristics of patients on admission to the study

	Active group $n = 11$	*Control group* $n = 9$
Age (months)	39 ± 8	34 ± 8
Height-age (months)	16 ± 3	17 ± 3
Weight (kg)	9.12 ± 0.94	8.90 ± 0.87
Height (cm)	79.9 ± 3.3	80.5 ± 3.0
Weight-for-height (% of standard)	83 ± 5	80 ± 6
Creatinine-height index	0.76 ± 0.11	0.72 ± 0.08
Serum proteins (g/dl)	7.2 ± 0.6	6.8 ± 0.9

the Active group were taken out of bed one hour earlier in the morning and 1/2 hour earlier after the mid-day nap. In addition to freedom of movement in their room and play areas, these children were encouraged to participate in games and activities that involved walking on a grade, running, tumbling and climbing stairs. Those activities alternated with periods of rest or sedentary play, similar to the Control group, to avoid tiredness and boredom.

No child was forced to remain inactive nor to participate in the game scheme. One child who was initially assigned to the Control group was quite restless and insisted in participating in the more active games. After one week, he was therefore transferred to the Active group.

Physical activity in both groups was examined by continuous heart-rate monitoring, and energy expenditure was calculated from individual heart rate:energy expenditure relationships and periodic measurements of basal metabolic rate. Growth was evaluated through standardized daily measurements of weight, and weekly measurements of length on two consecutive days. The latter were always measured by the same person.

Dietary intake was almost identical in both groups, averaging during the 6 weeks 2.50 ± 0.07 g protein and 117 ± 7 kcal/kg/day (mean ± standard deviation).

Physical activity raised the daytime heart rate an average of 23 and 17% above the night-time heart rate in the Active and Control groups, respectively ($P < 0.01$). Mean energy expenditures during the day were 2.0 and 1.7 times the basal metabolic rate ($P < 0.01$).

The two groups of children showed similar clinical improvement and average weight gains of 1.98 kg over the 6-week period. Nutritional recovery assessed by weight-for-length was equally satisfactory, 96 ± 7 and 95 ± 6% of expected. However, the Active group reached a higher creatinine-height index (0.97 ± 0.12, vs 0.89 ± 0.09, $P < 0.05$). indicating greater muscle mass and protein repletion.

The Active group also had a greater linear growth of 22± 8 mm, compared with 14 ± 6 mm in the Control group ($P < 0.05$). The difference in linear growth became evident by the fifth week of the study (Fig. 2). The Active group's increment of 22 mm in 6 weeks was two times greater than the linear growth of about 10 mm expected in normal children of the same chronological age (WHO, 1983), and 60% greater than the 13–14 mm expected in normal children of the same height-age (i.e., 17 months).

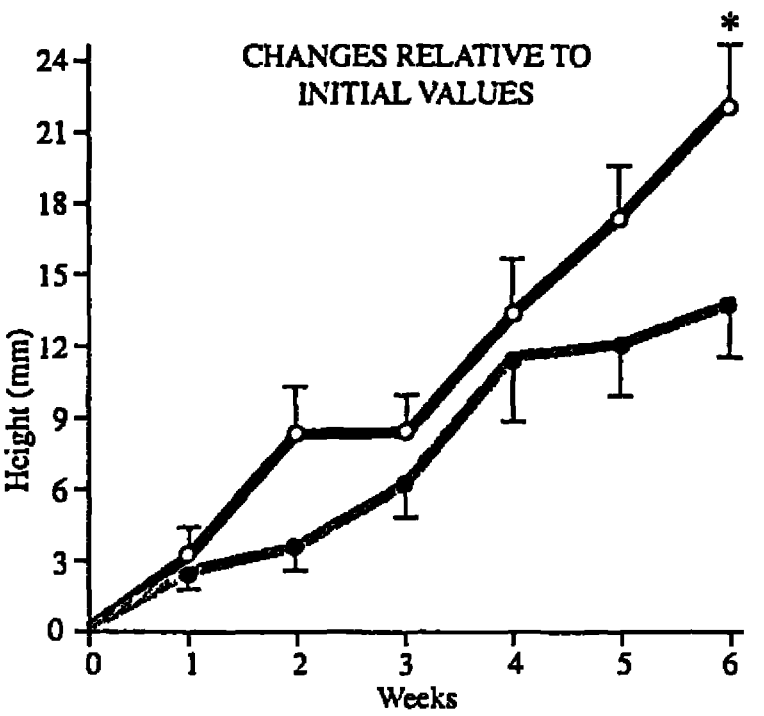

Fig. 2. Cumulative linear growth of Active (black circles, $n = 11$) and Control (open circles, $n = 9$) groups of children recovering from protein-energy malnutrition.

Mean ± SEM. * = Groups differ; $P < 0.05$.

4. Conclusions

The studies in malnourished rats and in children recovering from protein-energy malnutrition, indicate that moderate but consistent physical exercise enhances linear growth and promotes catch-up from stunting related to malnutrition.

This may not be the case with well nourished children who are not stunted, as suggested by the results of Viteri's studies (1973) on weanling rats without dietary restrictions (i.e., fed *ad libitum*). Using the activity-inactivity protocol described above, plus a Control group that was not forced to exercise in the revolving drum nor to remain inactive, it was found that the Active group had a small, marginally significant, increase in linear growth compared with the Inactive group, but there was no difference compared with the growth of the Control rats. On the other hand, inactivity might impair linear growth among well-fed individuals, since the inactive rats also grew significantly less than the Control group.

The mechanisms by which exercise enhances linear growth – at least among children who need to catch up – may be mediated by

endocrine factors that promote the growth of long bones, such as growth hormone and insulin-like growth factors (somatomedins). These hormones, as well as insulin, also enhance the muscle synthesis that seemed to occur in the studies on children and rats. The fact that physical exercise is an important stimulus for the secretion of growth hormone and the synthesis of insulin-like growth factor I, supports this concept.

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Discussion of the papers by Golding and Torun & Viteri

Skerry summarized briefly some results of experiments in animal models. Bone formation and remodelling is stimulated by cyclical but not static loads. Characteristics of the load that are osteogenic include its nature (twisting, compression, stretching), its magnitude, but probably not its duration. The amount of time that one needs to spend exercising to maintain or increase bone tissue seems very short; 36 cycles of dynamic loading in 24 hours, which takes no more than a minute, is enough to switch on maximal bone formation, and four cycles in 24 hours are enough to maintain bone tissue. If the same applies to children, then it is difficult to imagine how physical exercise could affect bone growth, except in extreme cases such as in children who are recumbent all day long, and in these only a small amount of the right exercise should suffice to maintain normal bone growth and remodelling.

In some tropical countries the mothers exercise their children's legs and encourage infants to stand and walk as early as they possibly can. Not wearing clothes and shoes also facilitates ambulation. The question is raised if that could be an explanation why Africans generally have relatively longer legs than Europeans.

Skerry commented that we should not think that the legs are only loaded when they are weight-bearing and the arms when heavy loads are lifted. There is some strain on all the bones most of the time, with the exception of the skull. The fact that the skull is maintained even though it is loaded very little would suggest that there are probably regional differences in the perception and responsiveness of bone to strain.

Nevertheless, from the surgeon's perspective there is a great difference between the upper and lower limbs. Deformities in the lower limb have a tendency to correct themselves, whereas those in the upper limb do not. What it comes down to is this: in so far as mechanical factors are concerned, their effects should be site-specific. We know, for example, that denervation, as in polio, causes shortening of the affected leg. In scoliosis one leg and the contralateral arm grow disproportionately. On the other hand, if the cause is nutritional or hormonal, one would expect the effects to be proportional. Martorell reported that in the Guatemala Oriente Study food supplementation affected total height and arm- and leg-length proportionally. In Pakistan the secular growth in height has involved both arms and legs.

However, disproportionate growth does not rule out nutritional and endocrine effects. Tanner showed that in puberty different segments of the body are growing at different rates. The experiments of McCance and Widdowson on pigs provided an example of different nutritional conditions – protein deficiency and energy deficiency – having different effects on growth of the jaw.