Activated carbon diets and early vitamin A deficiency. Application to the preparation of diets without vitamin A

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By studying various intestinal antiseptics [1] on the rat, we noted that the animals whose diet contained 1% activated carbon consistently showed the classic signs of vitamin A deficiency.

The theoretical interest of this fact was in itself sufficient to encourage us to study in a more precise way the modes of action of carbon. Is it the disappearance of vitamin A in the food mixture during its preparation and storage, a similar action during digestion, or even physiological reasons that we do not don't know?

The adsorbent and destructive action (by oxidation) of activated carbon is well known, but we also know from HOLMES [2] that vitamin A can be treated with carbon without appreciable destruction if care is taken to de-aerate it completely in advance.

BUXTON [3] has however shown that, under the same conditions, there is a disappearance of the natural antioxidants contained in the oils, which leads to a subsequent destruction of vitamin A.

But a question of a purely practical order and of no less decisive interest was grafted onto this problem. It was important to know whether the mere addition of activated carbon to food substances did not allow a practical, simple and economical realization of diets deprived of vitamin A, but otherwise complete, a preparation which all the specialists know is usually long, onerous and delicate.

To solve this problem we asked ourselves the following questions:

— Does the addition of activated carbon to diets devoid of vitamin A or poor in vitamin A increase the speed of appearance and the intensity of the signs of vitamin A deficiency?

— Does carbon act specifically on vitamin A?

To answer this set of questions we instituted a series of experiments that we will describe.

I. — ACTIVATED CARBON AND VITAMIN A DEFICIENT DIETS.

24 male rats (*), with an average weight of 40 g. are divided into 2 groups, 357 and 398, receiving as exclusive food the following basic diet, which is added with 2% of activated carbon (**) for animals of group 338.

Dextrine	670 g.
Casein without vitamin A	140 g.
Wheat germ without vitamin A	50 g.
Oil without vitamin A (cotton, flax, peanut)	50 g.
Dried yeast	50 g.
Mineral blend (McCollum and Davis)	40 g.
Vitamin D	1 500 IU

To obtain products without vitamin A, the casein had been extracted with boiling alcohol, then with ether; wheat germs extracted for a long time with ether and oils chromatographed on an adsorbent montmorillonite destroying vitamin A [4].

Once the vitamin deficiency had set in, we administered vitamin A to certain animals in both groups, after removing the carbon in group 338. The animals in both groups reacted normally to this administration.

Those animals that did not receive vitamin A were followed until death. Figure 1 reproduces the growth curve of these latter animals.

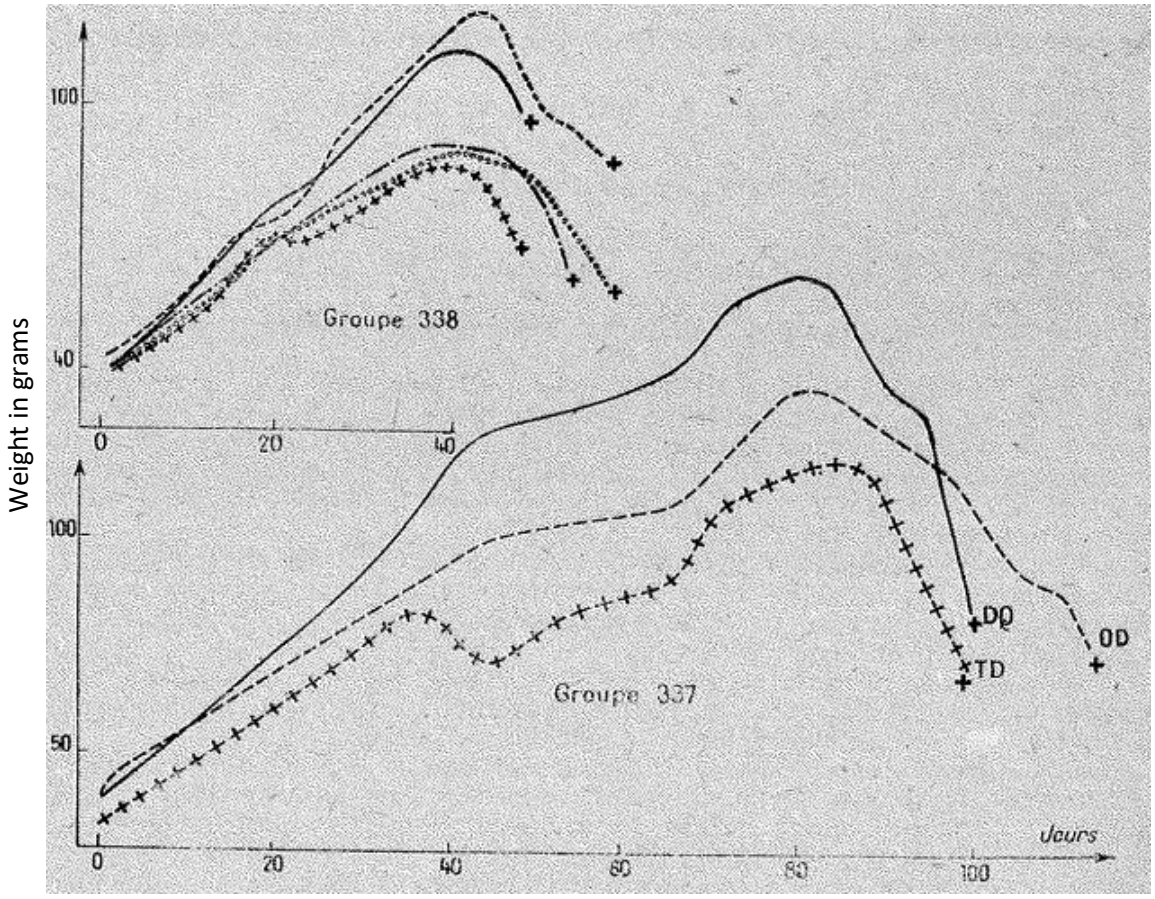

Fig. 1 —Individual weight curves of rats receiving a diet without vitamin A, supplemented (group 338) or not (group 337) with 2% of activated carbon. [Jours = Days].

(*) All the animals used in these tests come from a strain selected for five years by us. Strain quite widespread in France at the present time.

(**) Pure vegetable carbon Prolabo n° 22632

It clearly appears that the vitamin deficiency is revealed much more quickly in animals managed with carbon: the plateau sets in on the 30:th day, the weight drop takes place around the 47:th day and death around the 50th day, whereas in the controls, the animals reach the plateau on the 40th day, progress slowly until the 80:th day, only to die around the 100-120:th day.

Along with the arrest of growth, the other signs of deficiency are also much clearer: the first signs of xerophthalmia appear around 40 days for the second group, around 60 for the control group.

We have observed in both groups a whole series of varied pathological symptoms: some of them classic for vitamin A, various defects, in particular of the paw, tail, base of the tongue, presence of pus in the nostrils; others are superimposed on the clinical picture of vitamin A, such as hemorrhages in the hind legs, and that the work of JONSSON et al. [5] make it possible to attribute a vitamin C deficiency consecutive to a vitamin A deficiency.

At the autopsy: very diverse lesions, enteritis, abyss of the lung, arthritis, pericarditis, hemorrhagic suffusions at the level of the joints of the hind legs, which we have linked to the same cause as the hemorrhages mentioned above.

II. — ACTIVATED CARBON AND NATURAL DIETS THAT CONTAIN LITTLE VITAMIN A.

We then compared two batches of four male animals weighing approximately 35 g. receiving the following natural mixture R 349 [written in g per 1000 g]:

Brewer's yeast	20
Fish flour	100
Wheat germ	150
Peanut flour	150
Cassava flour	180
Whole barley flour	130
Whole oat flour	220
Mineral mix with vitamins (except vitamin A)	50

The control group (n° 474) received only the basal diet and the second one (473) the basal diet supplemented with 2% activated carbon.

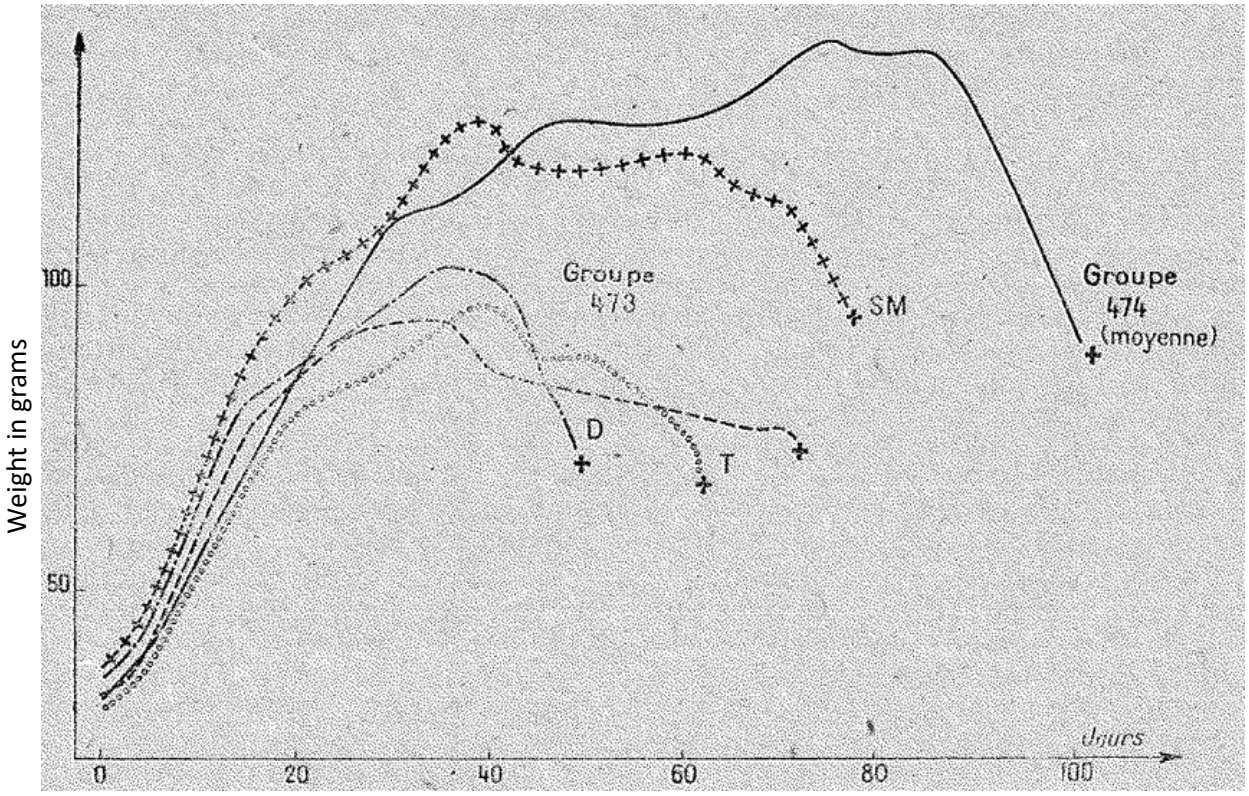

Fig. 2 — Individual (group 473) or average (group 474) weight curves of rats receiving a natural diet with the addition (group 473) or not (group 474) of 2% of activated carbon. [Jours = Days; moyenne = average]

Figure 2 reproduces the individual growth curve of the second batch and the average of the control batch. Again, same observations as in the previous experience and same evolution of events, fast for the 473, slow for the 474.

III. — ACTIVATED CARBON AND DIETS FORTIFIED WITH VITAMIN A.

A third experiment, modeled on the previous one, involved the same R 349 diet, deliberately enriched with the normal amount of vitamin A, that is eight units per gram. We compared two batches of four 40 g. male animals. approximately, the first group receiving in addition in its mode 2% of activated carbon. No difference was found between the two groups, during the three months of the experiment.

IV. — ACTIVATED CARBON AND BREASTFEEDING.

During a fourth experiment, we caused a particularly early vitamin A deficiency in young female rats whose mothers were fed from birth on the R 349 diet supplemented with 2% activated carbon.

On the 18th day, even before being weaned, almost all the little ones showed signs of xerophthalmia, their average weight was already below normal, and we then witnessed the development of a particularly severe and rapid vitamin A deficiency, ending in death at 35 to 40 days of age.

The conclusion of these four experiments is very clear: in the case of diets artificially deprived of vitamin A, or containing only very little in the natural state, an addition of 2% of activated carbon hastens the appearance of the signs of severe vitamin A deficiency. In the case of diets enriched with vitamin A, and, at the dose used, activated carbon appears to have no action for the duration of our experiment.

Although this last fact is already a proof in favor of the specificity of the action of carbon in the case of diets containing small amounts of vitamin A, it was necessary to verify it in a more precise and narrow way, and to know if the quickness and severity of the events observed were due to the simple destruction of vitamin A in these diets, or, on the contrary, to any secondary action such as toxicity, secondary deficiency, etc...

We turned to animals with a high hepatic reserve of vitamin A, to see if the action of carbon appeared under these conditions.

V. — ACTIVATED CARBON AND HEPATIC RESERVE OF VITAMIN A.

To two groups of 12 rats, we gave 5 500 IU of vitamin A per animal over four days, so as to build up a good liver reserve.

According to previous experiments which will be published soon, we estimate at 25 to 30% of the vitamin A thus administered, the quantity stored in the liver, that is, in the present case, a hepatic reserve in the order of 1400 to 1600 units per rat.

The two groups of animals were then fed: one (n° 384) with the R 349 diet; the other (n° 383) with the same diet supplemented with 2% activated carbon. Figure 9 reproduces the growth curve of these two batches.

For 3.5 months, the two groups behaved in an absolutely comparable way: the animals were very beautiful, their behavior quite normal, the weight curves identical, and we noted in both groups, signs onset of xerophthalmia (crusts, then abscess) while the weight curves became stationary. The search for Vitamin A in the liver, in half of the rats of each batch, showed the complete depletion of the reserve initially constituted, and thus confirmed the vitamin A deficiency. From this moment, the two curves of growth, so far very close to each other, separate quickly. The carbon-free group continued to maintain a plateau, while their ocular lesions progressed very slowly thanks to the retarding action of minimal amounts of vitamin A present in the diet. The group with carbon sees, on the contrary, its lesions worsen, its growth curve bend sharply and end quickly with the death of all the animals.

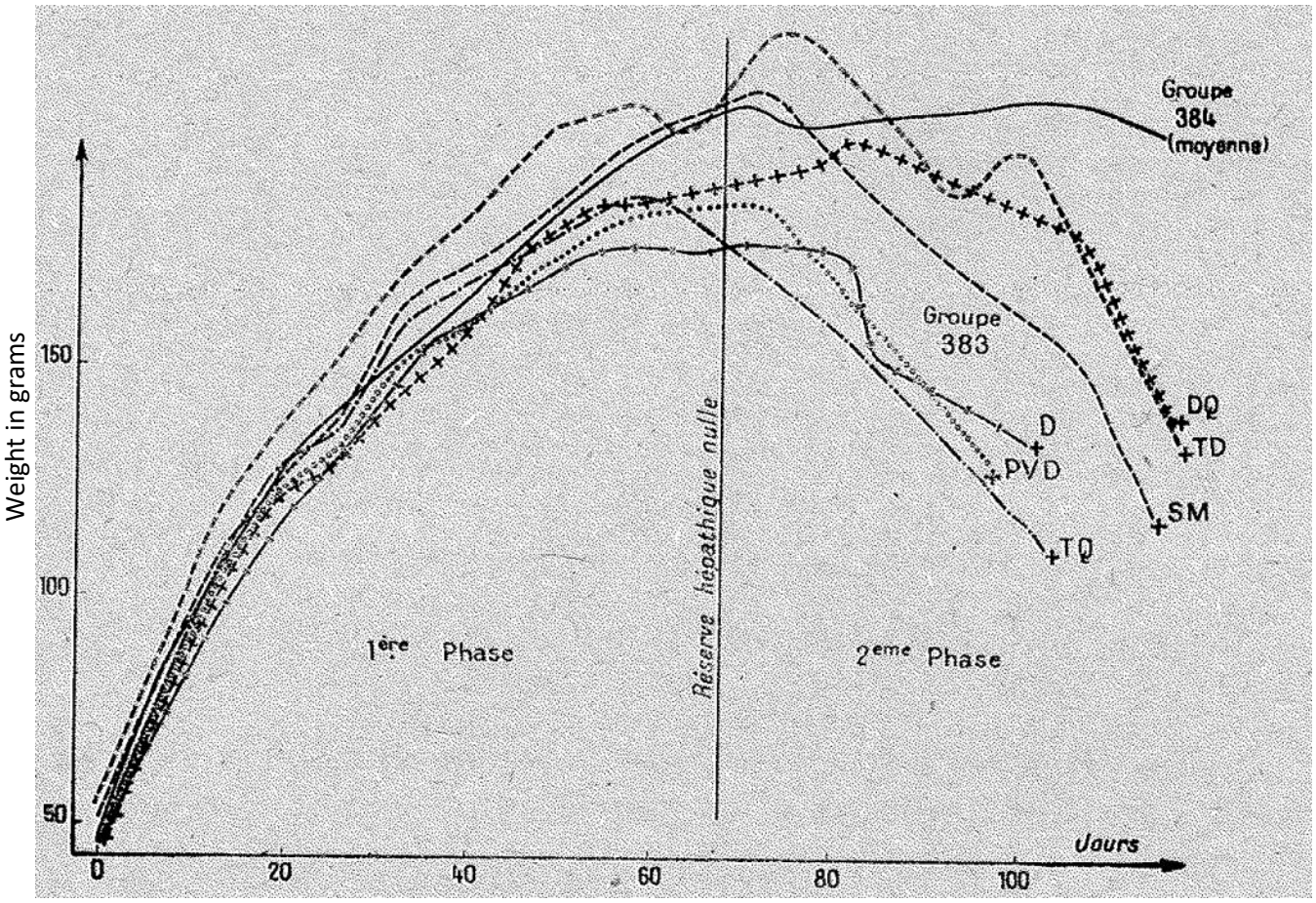

Fig. 3 — Individual (group 383) or average (group 384) weight curves of rats having initially received a large dose of vitamin A and receiving a natural diet supplemented (group 383) or not (group 384) with 2% of activated carbon.
[Jours = Days; Réserve hépathique nulle = Exhausted hepatic reserve; 1ère Phase = 1:st Phase; 2eme = 2:nd Phase]

Thus, it is clear that during the first phase of the experiment, the carbon exerted no harmful influence on the health of the animals. This phase ended with the depletion of the initial hepatic reserve of vitamin A.

In the second phase, on the contrary, the carbon played the same role as in the experiments of the first group: by destroying the small quantity of dietary vitamin A, it precipitated the signs of deficiency.

CONCLUSIONS.

It therefore seems certain that the addition of 2% activated carbon (*) in diets without vitamin A makes it possible to use for their preparation natural raw materials that have not undergone any purification.

The «carbon» diets apparently do not have any secondary action likely to falsify any subsequent studies on animals (rats) that can be made deficient in this way.

They also make it possible to obtain extremely early deficiencies which can be valuable in certain cases.

(*Laboratoire de Recherches de l'Alimentation Equilibrée, Commentary*).

(*) We only studied the activated carbon mentioned above. It is likely that other commercial activated carbon will have the same results, but this would obviously have to be verified.

Note. — It was only at the end of this study that we were able to become acquainted with the Anglo-Saxon literature of the years 1940-1945, in particular the work of H. J. ALMQUIST and D. ZANDER [6] on the role of adsorbent carbons in diets for chicks. The observations of these authors concerning vitamin A deficiency and vitamin K deficiency produced by the ingestion of carbon agree fairly well with ours. It seems that the hemorrhagic accidents that we have related above (exp. 338) are connected with this last action of carbon on vitamin K.

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