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Persorption of Raw Starch: A Cause of Senile Dementia?

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Abstract — Intact starch granules in food can pass through the intestinal wall and enter the circulation. They remain intact if they have not been cooked for long enough in the presence of water. Some of these granules embolise arterioles and capillaries. In most organs the collateral circulation suffices for continued function. In the brain, however, neurones may be lost. Over many decades the neuronal loss could be of clinical importance. To test this hypothesis, there is a need to examine brains for the presence of embolised starch granules. Examining tissues polariscopically clearly distinguishes starch granules from other objects of similar appearance.

Introduction

'Persorption' is the term used to describe the passage of particles, in the range 3–130 μ m, from the intestinal lumen through gaps in the mucosa and into the general circulation, whence a proportion is excreted in the urine. This phenomenon was described by Hirsch in 1906 (1). Volkheimer has used a variety of particulate materials but found raw starch especially convenient because of its easy identifiability in tissues and blood. When viewed polariscopically, starch granules exhibit a characteristic dark cross on a light ground (Fig. 1) (2). The majority have a diameter in the range $10-100~\mu$ m.

Experimental data

Volkheimer reported on young adult volunteers who ingested 200g starch suspended in milk or water. Venous blood, withdrawn at frequent intervals after in-

gestion, contained starch granules. There were two peaks, averaging 70 granules per 10ml blood at 10 min, and about 90 granules at 110 min (2).

Fate of persorbed starch granules in humans and animals

Starch granules were found in the urine, bile (after cholecystectomy), cerebrospinal fluid, peritoneal fluid (in ascites) and in breast milk, after ingestion by human subjects of a suspension of 200g raw starch (3). When ingested by dogs, persorbed starch was found in the portal venous system (4) and in the intestinal lacteals (5). Histological examination of tissues after ingestion by dogs and pigs showed embolisation of arterioles in lung, muscle, spinal cord and brain (6). Retained starch granules appear to be slowly destroyed either by enzymic breakdown or by phagocytosis.

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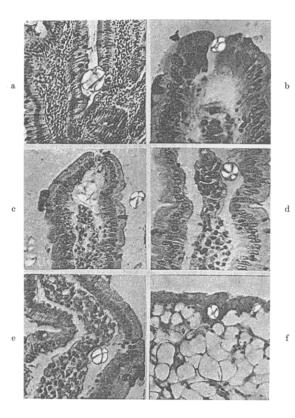


Fig. 1 Mechanism of the process of persorption, reconstructed in the rat intestine. Starch granules after feeding, a between two villi in the small intestine, b. between epithelial cells in the area of desquamation, c, d, e. in the subepithelial layer, f. in the lumen of a mesenteric lymph vessel.

Of special interest in the context of this article is the work of Volkheimer's team on the embolisation of small cerebral vessels. They infused very slowly an aqueous suspension of potato starch, via femoral artery catheterisation, into the left ventricular cavity of dogs. The brains were examined after infusion at various intervals of 5 min-200 days. Initially the starch granule emboli were surrounded by cellular infiltrate. Later, there were foci of cerebral softening followed by 'micronecroses' and scarring. After several weeks the granules were in the process of destruction. At 200 days focal reductions in neurones were seen, but scars were found with difficulty (6).

Discussion

What has this to do with the human condition? Who eats raw starch? The answer is 'Most of us do'. When starch granules are heated in the presence of water, they swell and undergo 'gelatinisation'. This is the

normal process of cooking. On being ingested, they are then broken down into sugars by digestive amylases. If starch granules are heated in a deficiency or absence of water, many do not gelatinise. They remain intact and resist digestion, and some of these will be persorbed. Factory-made wheat flakes, shredded wheat, rolled oats, shortcakes and crispbreads contain many intact starch granules (Fig. 2) (2). Ingestion of 200g of wheat flakes or rolled oats by volunteers yielded about 60 starch granules in 10 ml of venous blood (3).

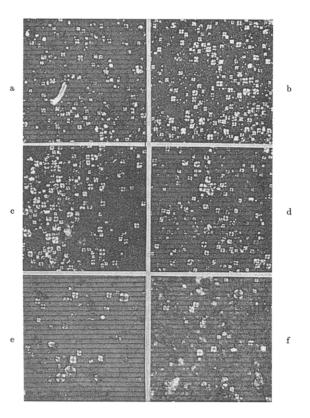


Fig. 2 Polarisable starch granules in baked foodstuffs (distance between lines 20um). a. Shredded wheat, b. Shredded wheat steeped for 16 h at room temperature, c. Biscuits, d. 'Hard tack', e. crispbread, f. Shortcake. The materials were suspended in a little water and photographed in polarized light with half-crossed polarizing filters.

Hypothesis

If we assume an average serving weighs 100g, and a total blood volume of 4l, this represents 12,000 granules in the circulation, of which 15% go to the brain. A daily intake of this amount would constitute a calculable cumulative load after several decades.

It is conceivable that a proportion of ingested raw starch granules contained in the above mentioned foods would embolise the human brain in numbers that are clinically important.

Volkheimer's intraventricular infusion experiments differed from what might occur with prolonged but less practicable feeding experiments. They cannot, however, be cast aside as irrelevant. There are, for example, analogous and acceptable industrial tests in which weathering of exposed materials, and wear and tear in the moving parts of mechanisms are speeded up in order to provide quick answers to questions regarding long-term durability.

Conclusions

I submit the hypothesis that the decline in mental function with advancing years that afflicts a proportion of the human population (amounting in severely affected cases to dementia) and the associated neuronal loss, may be partly, in some persons perhaps wholly, due to the cumulative embolisation, over a period of many years, of small cerebral vessels by persorbed food particles in the range of $20-130~\mu m$. It should be unnecessary to add that this hypothesis in no way conflicts with the several other established causes of senile brain failure; nor are we here concerned with so-called corpora amylacea, which were in the past mistaken for starch when found in the brains of older persons.

If the disappearance of embolised starch granules in the human brain resembles that seen in dogs, then one would expect to find only those granules that were deposited in the six months before death. The only evidence for previous decades of embolisation would probably be focal neuronal loss; possibly also

microcicatrices. It is a fact that there is cerebral neuronal loss with aging. This has been estimated at a loss of 100,000 cells per day (Corsellis, personal communication).

The consumption of ready-made foods, such as crispbreads and breakfast cereals, and their popularity in Western dietaries, has greatly increased in the past 70 years. Previously, most food starch was cooked in the presence of water before ingestion.

The purpose of this article is to prompt morbid anatomists to test this hypothesis. A first step, surely, would be to examine the brain polariscopically. Embolised intact starch granules would be manifest by their characteristic dark cross on a white ground. A dietary history of ingestion of ungelatinised starch during the previous six months might yield corresponding relevancy. To the list of foods already given one might add cocktail savouries and potato crisps. An adult brain of any age would qualify for examination.

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