(1) In peeling the eggs. Repeated experience showed that an excellent criterion for judging the injured eggs was the presence of granules, in Brownian movement, within the micropyle. It was often found that, although there was no other apparent injury, when the micropyle contained granules the egg did not develop in a normal manner. On the other hand, when there was no visible injury and these granules were absent, the egg invariably developed normally.

(2) In the application of amyl acetate. Care was taken that the amyl acetate should not wash ambroid over the egg, for, on drying, a film of ambroid covering the egg would exert great pressure and thus injure it.

(3) By excessive desiccation. This was avoided by preparing the egg for observation very rapidly and by sealing the coverslip to the depression slide with vaseline. Another precaution that was taken was to place a drop of the medium on the bottom of the depression.

Only after proper orientation can the pole cells be observed to best advantage. Since they are more concentrated dorsally, the best view of them was obtained when the egg was placed with its dorsal (more or less concave) surface against the coverslip. The pole cells begin to bud off at the posterior end of the egg at the time of the 8th or 9th cleavage. As soon as they push off, they divide, so that by the time the egg is in the 11th cleavage, 10 to 12 pole cells are present. The pole cells are particularly advantageous for the observations of central bodies since they are large, since they are free from yolk spheres and since the surface of the nucleus of the pole cell is free of any extraneous granules which are found scattered through the cell.

With a magnification of 1,500 times, in many of the pole cells on the upper periphery of the pale, hyaline nucleus, two spherical bodies opposed to each other were observed. The distance between these bodies varied. When they were close together, they seemed to lie within a single vacuole. When they were some distance apart, each one appeared to be enclosed in its own smaller vacuole. (Wilson and Huettner[1] described such a small clear area about the central body, in fixed preparations, at the beginning of prophase). These bodies were identified as the central bodies by their behavior during division of the cell, by their position in the cell and by their distinctive appearance. They possessed a slight vibratory motion so that one could get both of them in focus at the same time for only a very short interval. The central bodies in each pair were of the same size. On the whole they seemed to be less transparent than the ordinary granules which are present in the cytoplasm of the pole cell.

The central bodies may also be identified near the nuclei of blastodermal cells. These cells are not as favorable for observation as the pole cells, since they are much smaller. Moreover, it is easy for one to confuse the central bodies with the mitochondria and other cell inclusions which swarm about the nuclei. For the same reason, the behavior of the central bodies, during cell division, can not be followed in the blastodermal cells.

During divisions of the pole cells the central bodies were seen to move apart along the periphery of the nucleus. This movement was gradual at first, but as the nucleus lengthened, they moved apart more rapidly. Although they were not actually seen to divide, shortly after this, the nucleus still being elongate, 2 pairs of central bodies were observed, one pair near each end of the nucleus. The central bodies of each pair kept shifting their positions with respect to each other. This accounts for certain conditions found in fixed preparations. For example, in telophases of the nuclear cleavages, at each end of the cleavage figure, the two pairs of central bodies are not placed similarly but may occupy any conceivable position with respect to each other.

The central bodies were observed best at the stage in the development of the egg where there are about 10 to 12 pole cells. At this time they are undergoing division and are not crowded to such an extent that they overlap each other. They were seen in the pole cells of eggs developing in various external media, namely, 33 per cent. sea water, 0.5 M. glycerine and pure egg albumin. In each medium, the eggs developed quite normally, *i.e.,* larvae hatched out in the normal length of time.

These observations, though they are of a preliminary nature, demonstrate that the central bodies have an actual existence in the living cell. They have a distinctive appearance and behavior in the pole cells of the normally developing egg of *Drosophila melanogaster*. One can also observe them in the cells of the blastoderm. They are not as obvious in these cells, however, for cell inclusions such as mitochondria, which are very numerous, fill the cells.

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A STUDY OF CANNED SHRIMP WITH REFERENCE TO THE PRESENCE OF VITAMINS A, B AND D

QUALITATIVE tests on wet-pack and dry-pack canned shrimp[1] show that vitamins A[2] and D are present in

[1] After shrimp are cooked and weighed into cans, wet-

shrimp fat, but that B is either absent or present in very small quantities. There was no evidence of any appreciable difference in the vitamin potency of the two kinds of shrimp.

The vitamin A determinations were made on 16 rats,[3] four receiving a normal diet,[4, 5, 6] twelve receiving an A-deficient diet.[7] At the end of the fifty-day depletion period all rats on the deficient diet showed a flattened growth curve (daily weighings plotted); all had scraggy, rough coats and eyelids showing extreme redness and varying degrees of incrustation. Shrimp fat[8] then was added to the diet of all but four of the A-deficient rats. These four rats were continued on the deficient diet to serve as negative controls. The worst of the group had snuffles and was almost blind; it was included in the group fed with shrimp fat, and while it made some improvement as to weight and eye condition, it died a few days after the curative feeding was begun. With this exception, every rat receiving shrimp fat (0.2–0.3 gm daily) began to show a sudden and continuous rise in the growth curve, the curve paralleling that of the rats on the normal diets. The general appearance improved and the eye condition healed. Of the negative control group, one died; one went blind in both eyes; one went blind in one eye; and one had eyes almost incrusted at the end of the test period. Vitamin A was considered to be present in the fatty extract of the canned shrimp, as evidenced by its effect on the growth curves of the A-deficient rats and its curative effect on ophthalmia.

In the vitamin B set-up, all rats were put on a B-deficient diet.[9] One half of the group received in addition the canned shrimp *ad libitum*. One half a shrimp, about 3.5 gm, was the average daily intake. Both groups showed increasing signs of B-deficiency; and when spastic paralysis of the hind quarters set in, the group that had been receiving shrimp had yeast added to the diet. All except one rat recovered, and it died a few hours after paralysis was noted. The group that had received no shrimp had shrimp added to the diet when paralysis was manifest. They ate avidly of it (8–10 gm daily), but no improvement followed. Vitamin B appears to be absent in shrimp or to be present in such small amounts that 3.5 gm did not protect the rats from peripheral neuritis and 8–10 gm exerted no beneficial effects.

The vitamin D group consisted of 16 rats, four on the normal diet, twelve on Steenbock's D-deficient diet.[10] All were x-rayed[11] after the depletion period. The four normals had no evidence of rickets; the twelve on the deficient diet were rachitic. Four rats were chosen to be continued on the rachitic diet, and these showed rickets throughout the test period. An attempt was made to feed whole shrimp as the source of vitamin D, but the rats would not average over a 1.4–4.1 gm daily intake; so at the end of ten days, shrimp fat which they relished was added to the D-deficient diet in amounts representing 10–15 gm canned shrimp (0.2–0.3 gm fatty extract). On the thirteenth day of this new feeding régime, the x-rays showed that the rachitic condition of five of the eight rats receiving shrimp fat was healed and the other three showed improvement. At the end of the next week, one of these three was healed, one almost healed, and the third not much improved over the condition of the previous week, but better than at the beginning of the shrimp-feeding period. The negative controls were still rachitic and the normals free from rickets. Vitamin D, as evidenced by the beneficial effect on bone calcification of the feeding of shrimp fat with an otherwise D-deficient diet, was judged to be present in canned shrimp.

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pack shrimp are covered with a mild brine solution before sealing.

[2] Hjort, *Proc. Roy. Soc.*, London, B 93, 440, 1922, and Jansen and Donath, *Meded. Begerl. Geneesk. Dienst Nederland. Indie*, 46–48, 1924.

[3] The rats used in these experiments were obtained through the kindness of Dr. Henry Laurens and Dr. H. S. Mayerson, department of physiology, Tulane University, and were from the breeding stock which had been kept five years on a normal diet. They represent entire litters approximately 28 days old when taken from the mothers' cages.

[4] Casein, 20 parts; salt mixture,[5] 5 parts; butter, 15 parts; cornstarch, 60 parts.[6] Yeast and Viosterol were given daily.

[5] Osborne and Mendel, "Salt Mixture IV," *Jour. Biol. Chem.*, 37, 572, 1919.

[6] Jones, Murphy, Nelson, *Jour. Ind. and Eng. Chem.*, 20, 205, 1928.

[7] The A-deficient diet was the same as the normal, except that the casein was purified and Crisco was substituted for butter.

[8] Shrimp fat was made fresh every week or ten days. Six cans of shrimp—800 grams—were ground fine; allowed to stand 48 hours in contact with petroleum ether (b.p. below 50° C.); the ether expressed with pressure; the extract filtered and evaporated at room temperature before a fan. The extract was approximately 2 per cent. of the original weight and approximately 50 per cent. fat; but no further effort was made to purify the fat as it was thought that a minimum of handling would conserve the vitamin content.

[9] The B-deficient diet was the same as the normal, except that no yeast was given.

[10] Steenbock and Black, *Jour. Biol. Chem.*, 64, 263; Nelson and Steenbock, *ibid.*, 64, 299, 1925.

[11] "The Quantitative Estimation of Vitamin D by Radiography," Medical Research Council, London, 1931. The radiography was done by Dr. J. N. Ané, department of roentgenology, Tulane University, School of Medicine.