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Morphological Aspects of Exocytosis in the Adrenal Medulla

Odile Grynszpan-Winograd

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Morphological aspects of exocytosis in the adrenal medulla

By Odile Grynszpan-Winograd
Laboratoire de Cytologie, Faculté des Sciences, 7, Quai Saint Bernard, Paris 5e, France

[Plates 47–50]

In the adrenal medulla of the hamster, we have clearly observed profiles which show exocytosis. Such profiles have been seen on all surfaces of the chromaffin cells, near the large extracellular spaces which are mainly occupied by the blood vessels and connective tissue and also near the narrow intercellular cleft of about 20 to 25 nm which separates neighbouring chromaffin cells.

(1) Near the large extracellular spaces, the plasma membrane shows granule-containing invaginations. An electron-translucent space separates the plasma membrane and the granule which is alone in the invagination. The same diversity in size and shape may be seen among the expelled granules and the intracellular granules (see figures 1 to 7 and, plates 47 to 50). These figures look like the exocytosis aspects which have been already described in other types of cells, in particular adenohypophysis (Farquhar 1961).

In the adrenaline cells (A-cells), the granules of which are moderately electron-dense, the expelled granules have the same degree of electron density as those within the cell (figures 1 to 4). In the noradrenaline cells (NA-cells), the granules of which are strongly electron-dense, the expelled granules are much less electron-dense than the intracellular granules and look like the A-cell granules (figure 7). According to Coupland & Hopwood (1966), the low electron density of the A-cell granules seems to be mainly due to the non-catecholamine components—in particular the proteins—because adrenaline is not kept in the tissue during the fixation and dehydration procedures. Noradrenaline, which gives an insoluble complex with glutaraldehyde, is kept in the cell during the same procedures. This complex seems to be responsible for the strong electron density of the NA-cell granules. If this is so, noradrenaline is no longer present in the expelled granules observed in the electron micrographs. We may think that noradrenaline has become more labile than it was in the intracellular, membrane-protected, granules.

(2) Between neighbouring chromaffin cells, the intercellular cleft shows dilatations, which are generally situated near attachment plates and interdigitations (figures 5 and 8). These dilatations contain an electron-dense substance and granules (figures 5, 6 and 8 to 10). This substance does not appear elsewhere in the intercellular cleft. Some granules have about the same size as the intracellular granules; others are much smaller and more electron-translucent than the intracellular granules, not only in the NA-cells but also in the A-cells. They are often numerous in the same dilatation. These figures suggest that the expelled granules have been altered. The conditions of fixation are probably not the same at cell surfaces close to the capillaries by which the perfusion of glutaraldehyde is achieved, as at the large extracellular spaces and in the region of the narrow intercellular cleft.

(3) In about 30 to 40 % of the cases, the invaginated membrane, at the site of exocytosis, shows one or two bristle-coated pits, more or less opened towards the extracellular space (figures 2 to 4, 7 and 10). These bristle-coated pits associated with exocytosis represent about a quarter or a fifth of all the bristle-coated pits of the plasma membrane. This association has

been already mentioned in a previous publication (Diner 1967), and a similar observation has been made in the crustacean sinus gland (Bunt 1969). It has been demonstrated in many cells that such coated pits and derived vesicles are involved in the cellular uptake of proteins; this has also been reported for the chromaffin cells (Holtzman & Dominitz 1968). As exocytosis increases the surface of the plasma membrane by the incorporation of the granule membranes, the presence of pinocytotic pits, at the same location, would explain why the surface of the cell does not increase indefinitely. Further investigations are necessary to support this hypothesis.

References (Grynszpan-Winograd)

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Diner (Grynszpan-Winograd), O. 1967 *C. r. hebd. Séanc. Acad. Sci., Paris* **265**, 616.
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Holtzman, E. & Dominitz, R. 1968 *J. Histochem. Cytochem.* **16**, 320.

Explanation of plates 47 to 50

Fixation: perfusion of glutaraldehyde, followed by immersion of small pieces in osmium tetroxide. Coloration: uranyl acetate and lead citrate or lead citrate alone. The scale on the photographs represents 0.2 μm.

Figures 1 to 4 (A-cells) and 7(NA-cell): Exocytosis aspects at the level of the plasma membrane which borders capillaries (*c*) or connective tissue. The expelled granules and the intracellular granules are alike in the A-cells; in the NA-cell, the expelled granule has not the bright black appearance of the intracellular granules (see text). Note the bristle-coated pits associated with the location of exocytosis.

Figures 5, 6 (A-cells) and 8 to 10 (NA-cells): exocytosis aspects at the level of cell-to-cell apposed plasma membranes; the expelled granules seem to have been altered: smaller and lighter units as well as a moderately electron-dense substance appear in the dilatation of the intercellular cleft. Note, especially in figure 10, the bristle-coated pits and vesicles just at the level of the dilatation.

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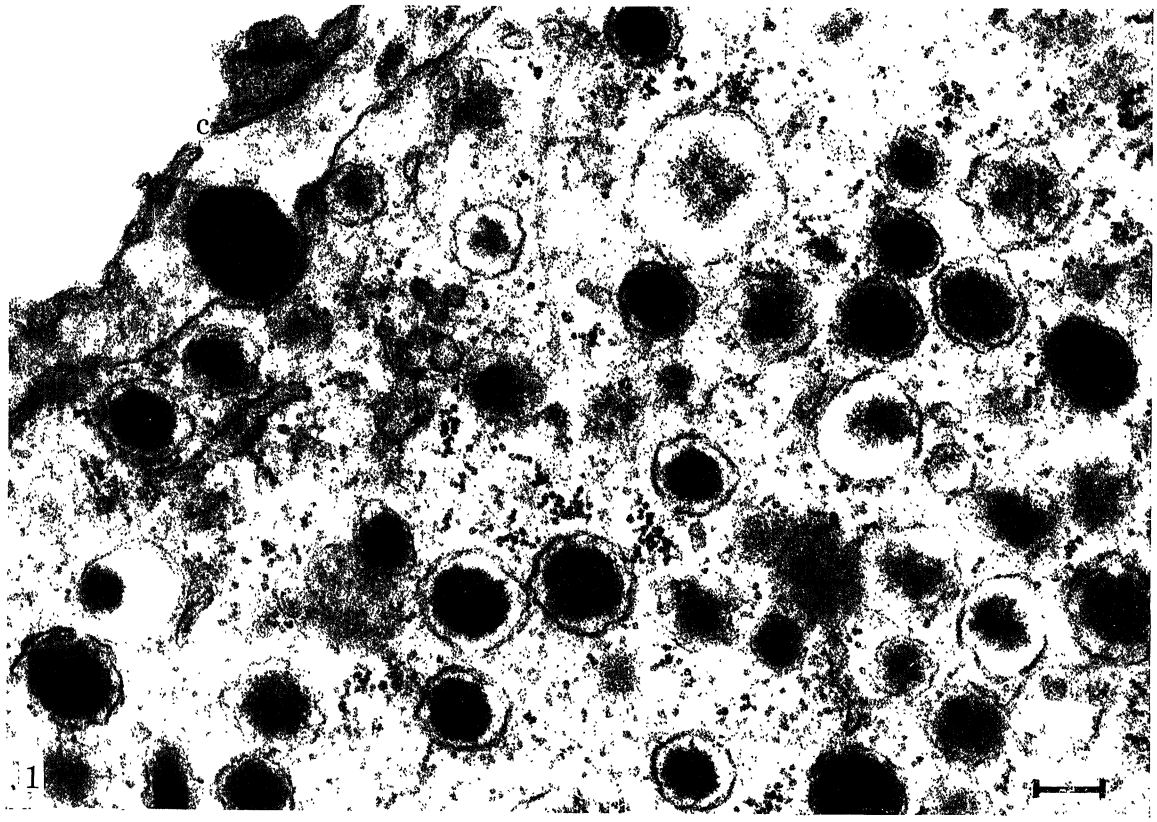

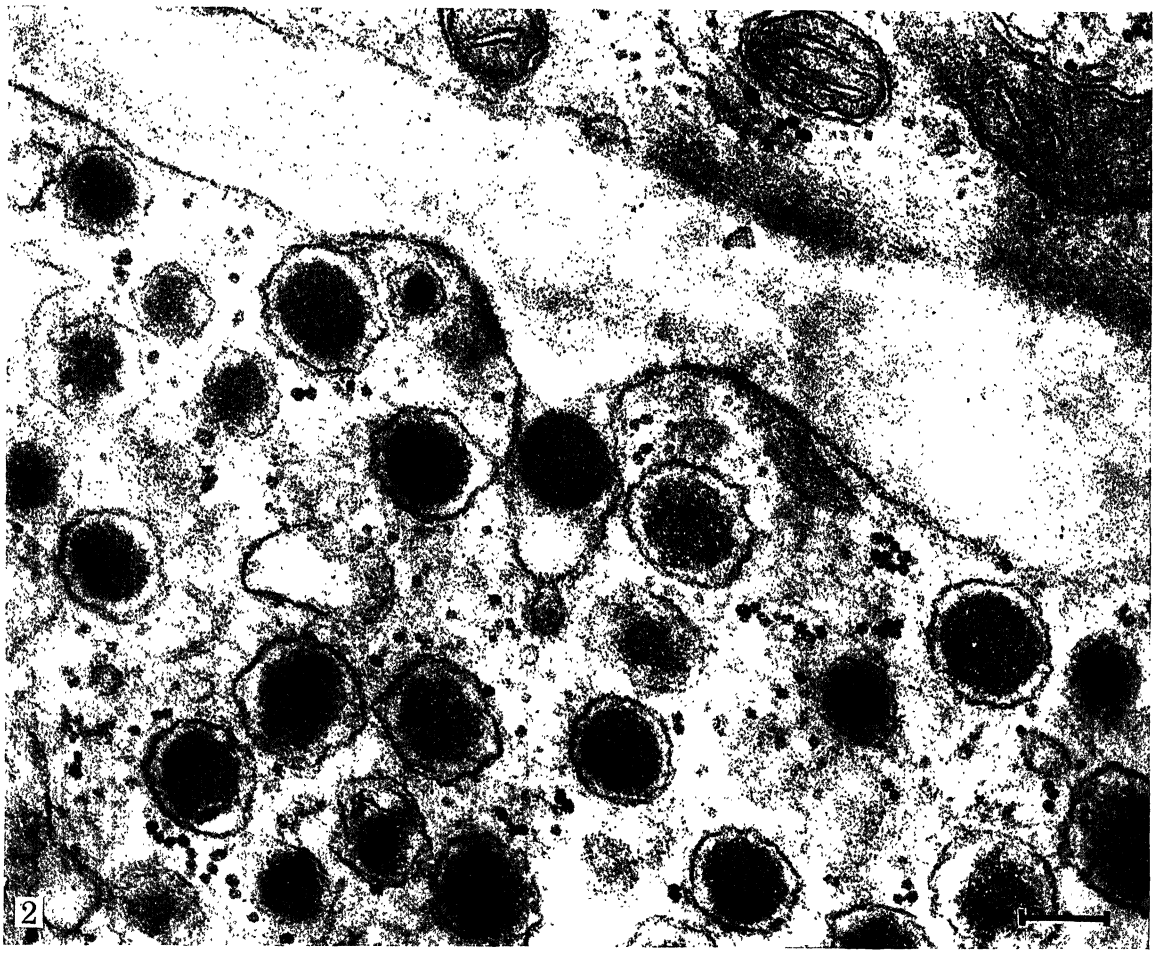

FIGURES 1 TO 10. For legends see facing page.

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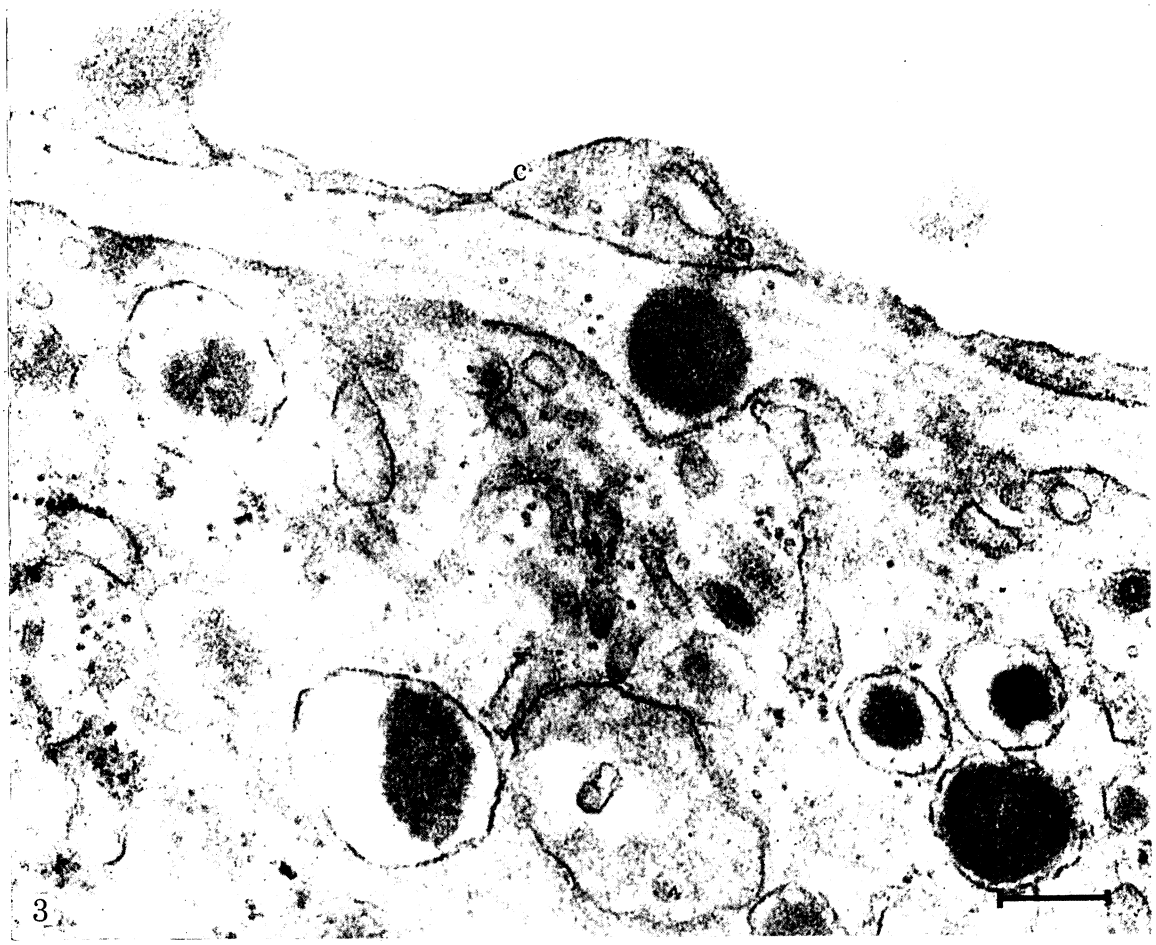

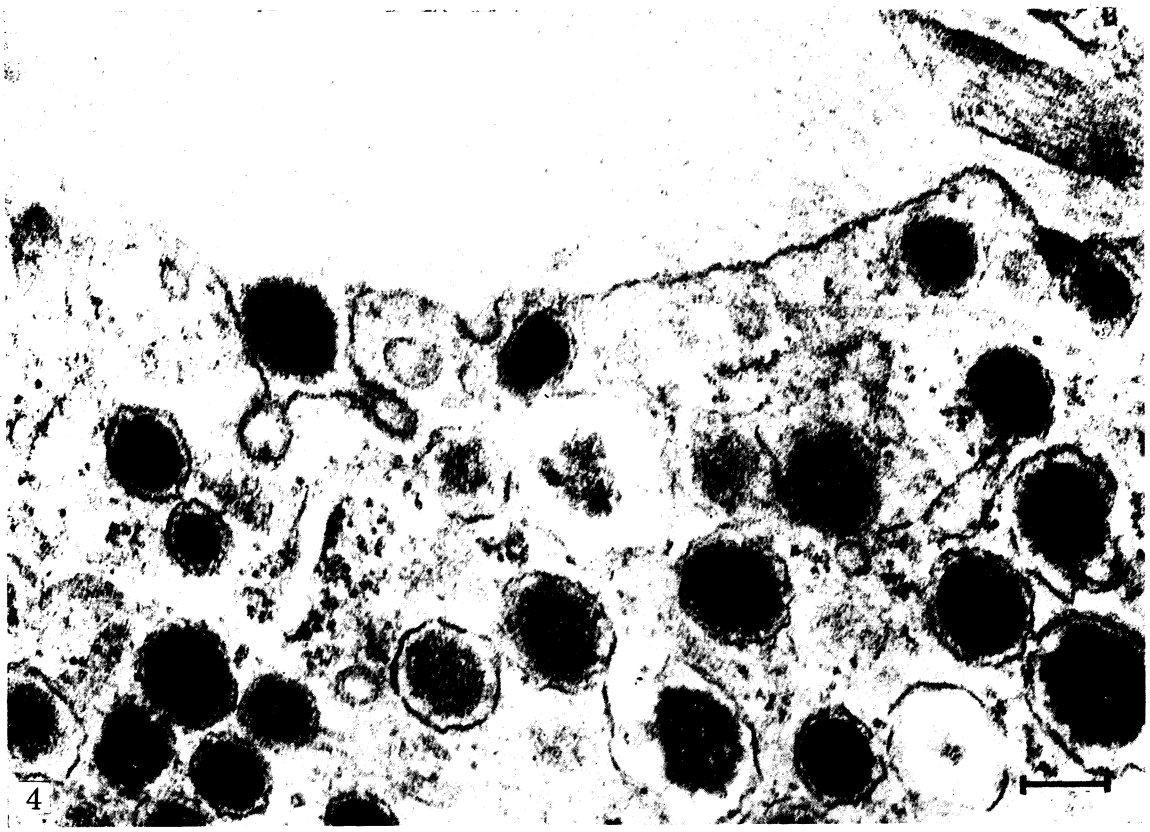

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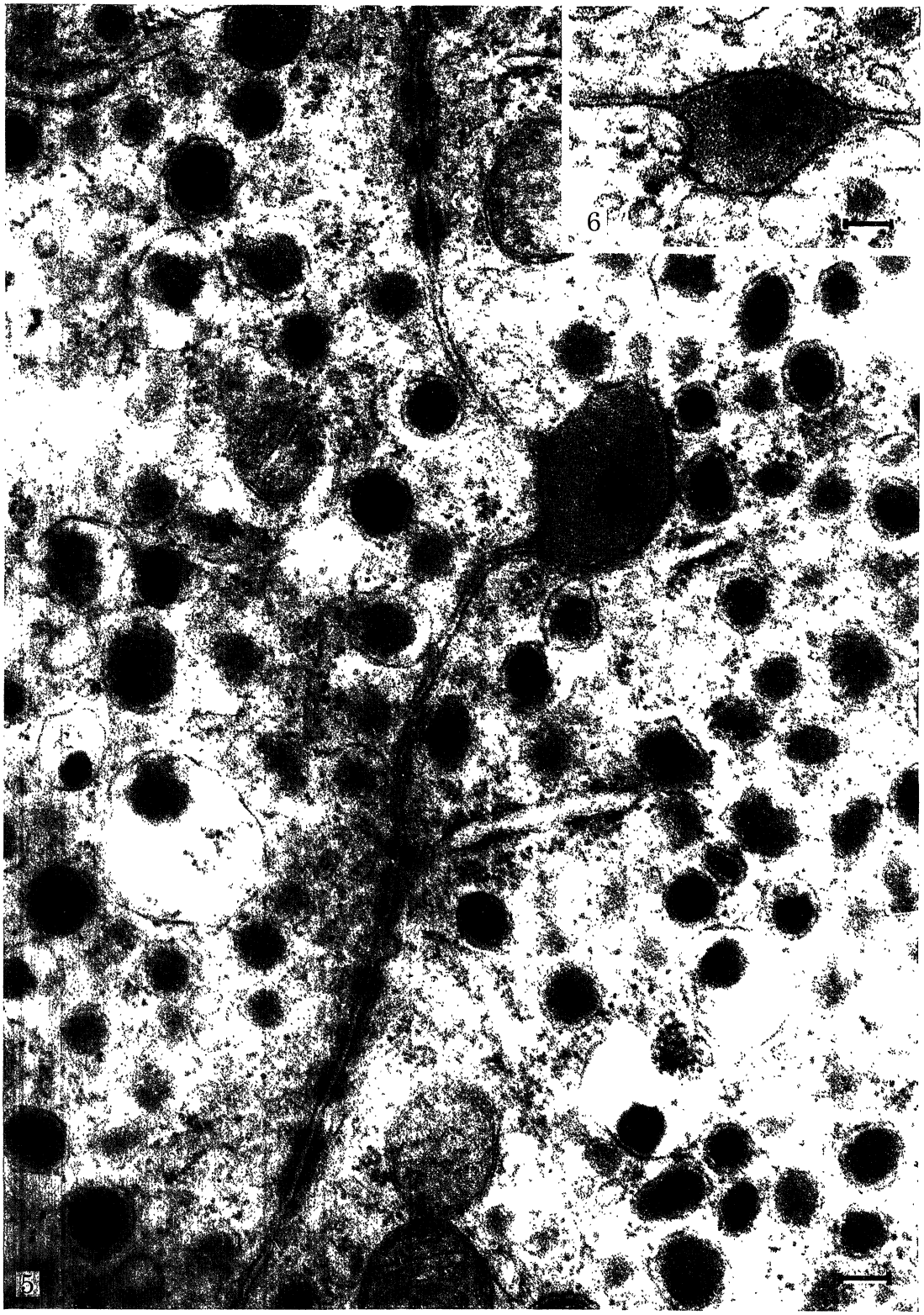

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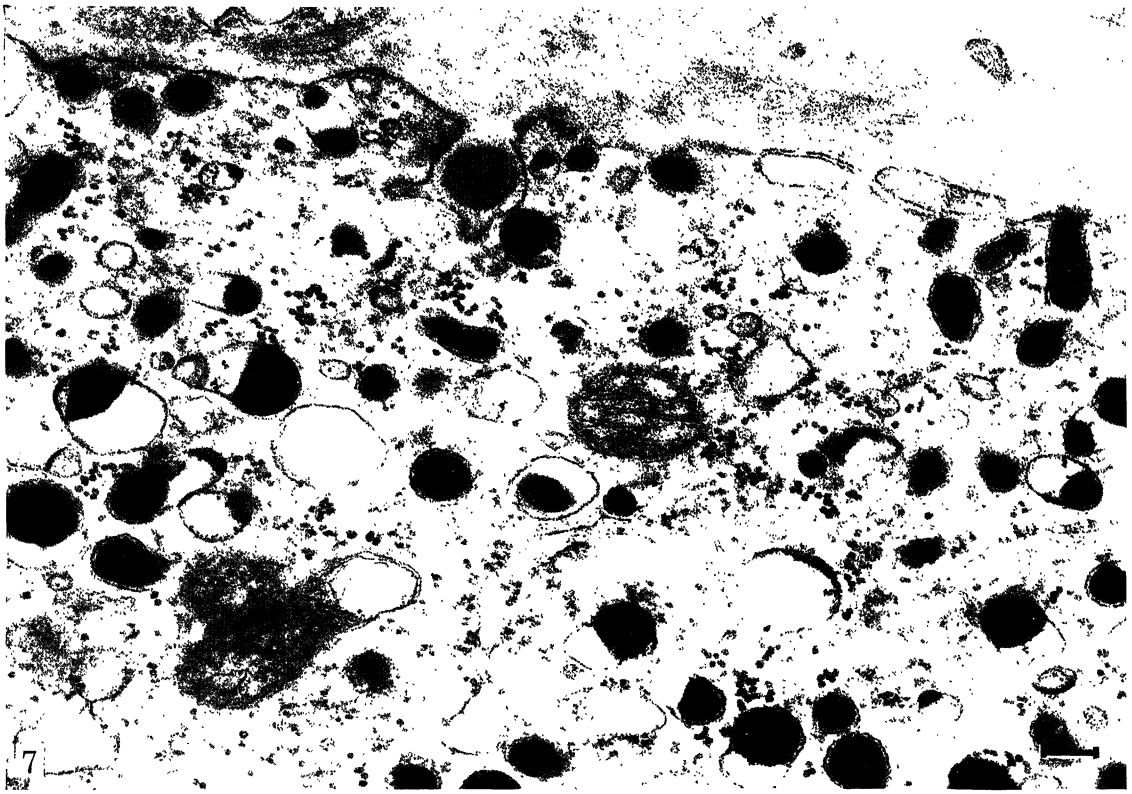

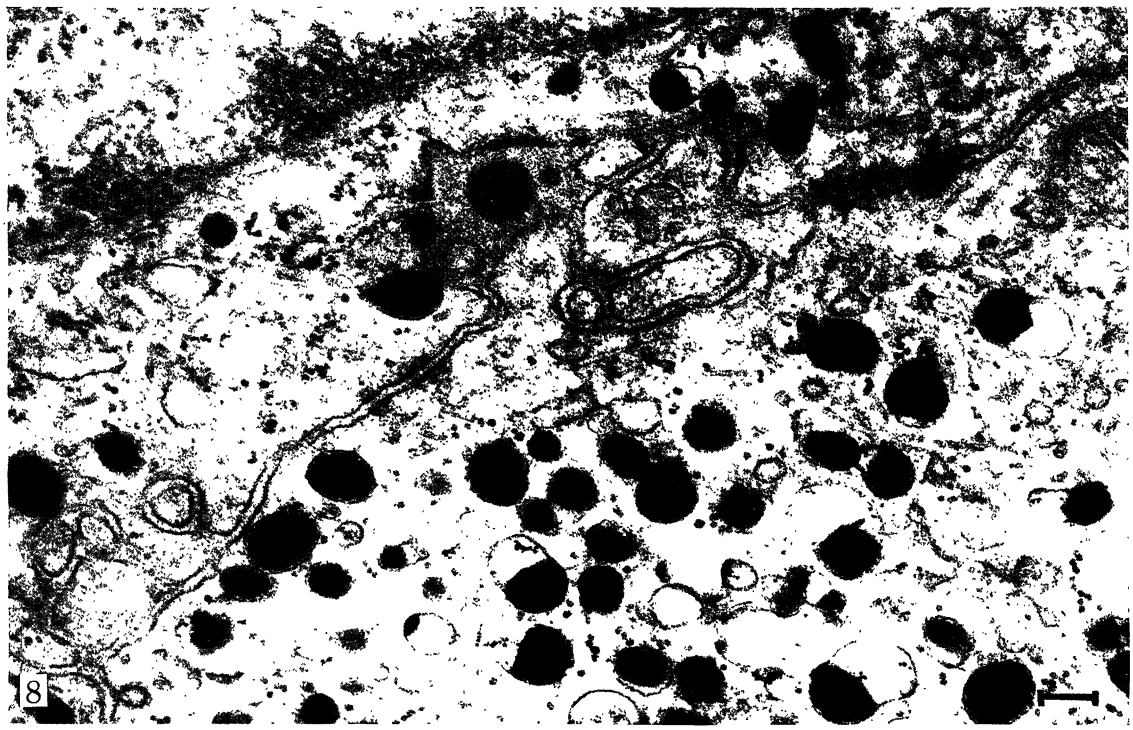

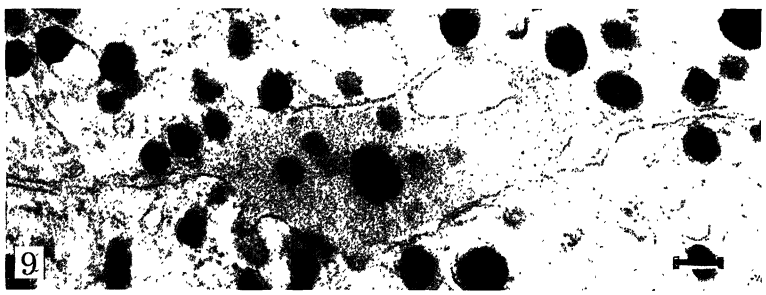

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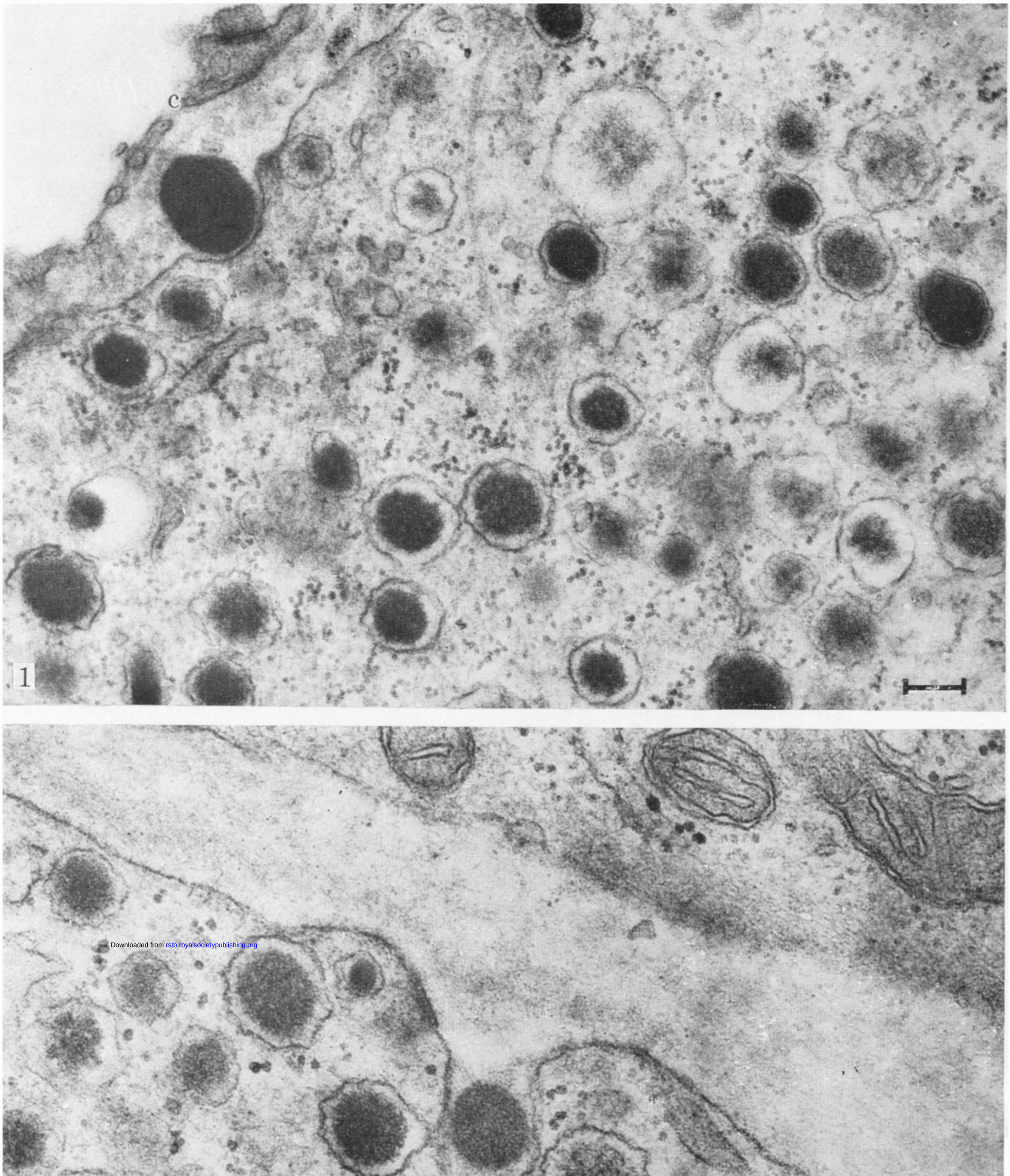

FIGURES 1 TO 10. For legends see facing page.

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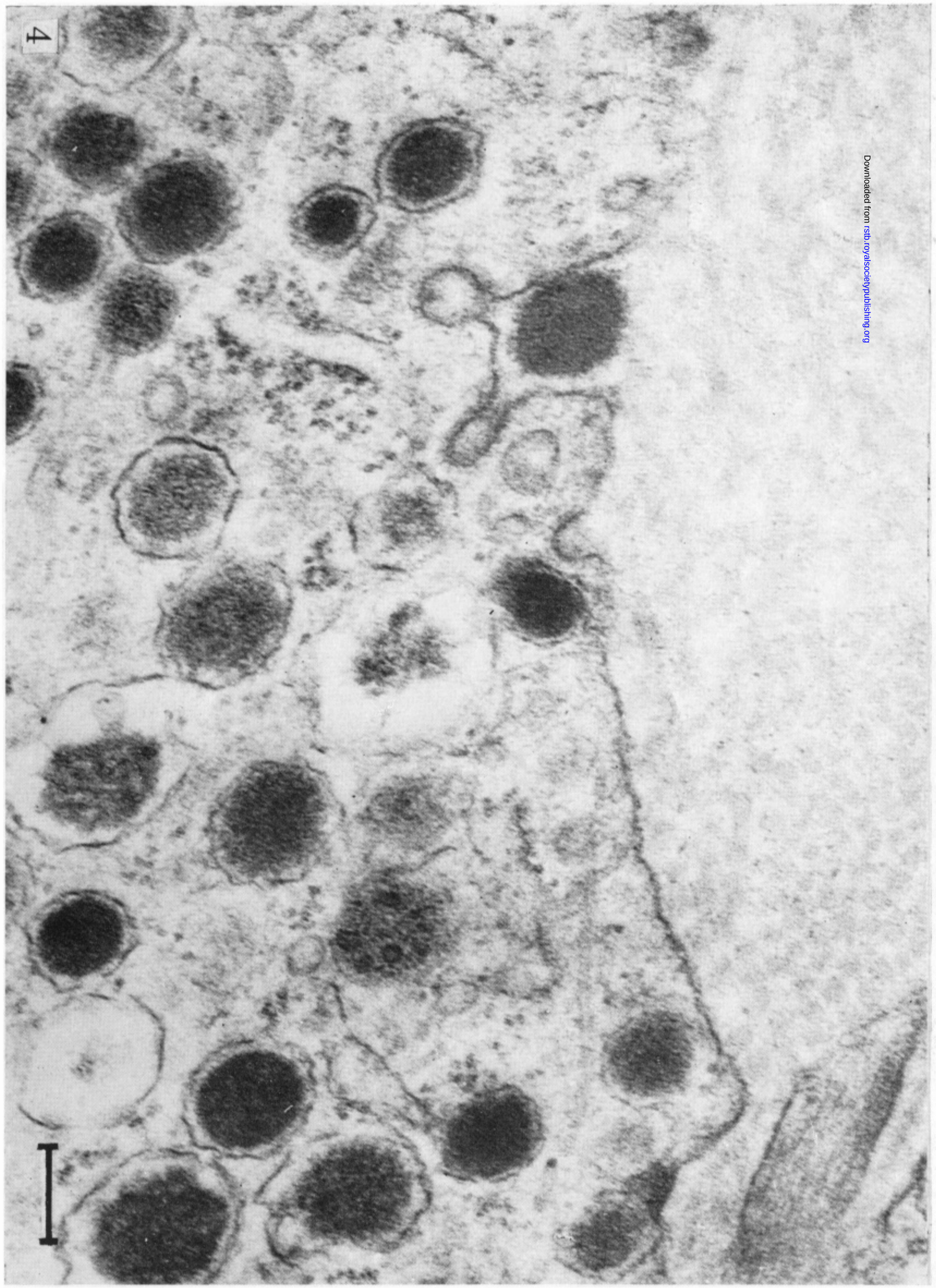

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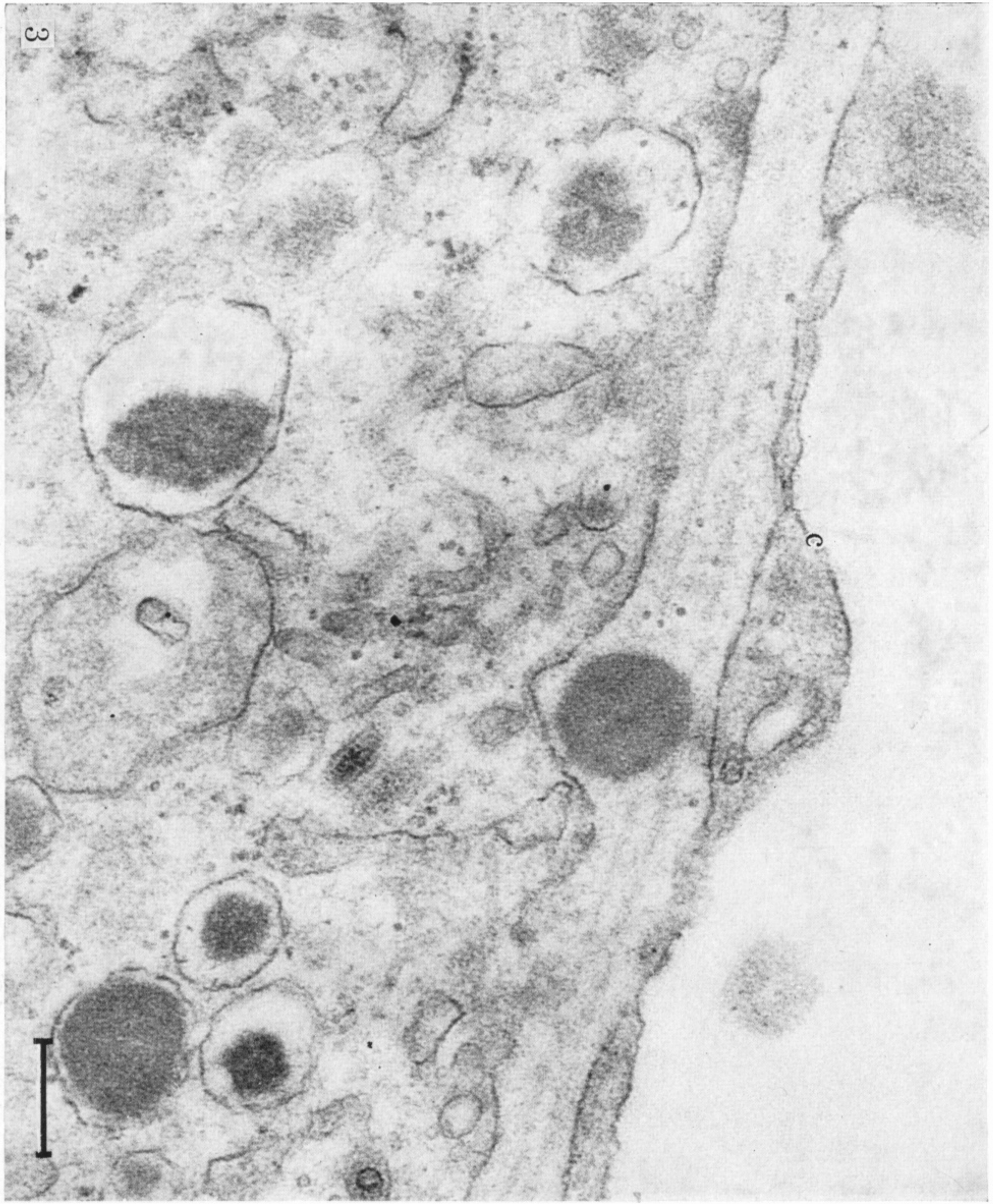

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