GOLGI ORIGIN OF TUBULAR INCLUSIONS IN ENDOTHELIAL CELLS

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INTRODUCTION

In 1964 Weibel and Palade described in endothelial cells peculiar rod shaped inclusions, measuring 0.1–0.3 μ in diameter for a length reaching as much as 3 μ. These inclusions are bounded by a unit membrane and contain fine tubular elements of about 150 A in diameter, which are parallel to themselves or have multiple orientations. The density of the intertubular substance is variable and is generally more osmiophilic than the contents of the fine tubular elements (1, 2).

These tubular inclusions are present in all the studied vessels, whatever their animal and tissue origin; but they are especially numerous in vessels whose diameters are over 30 μ (3). They have been thought to play a role in coagulation (4), but neither the place of their synthesis, the mechanism of their degradation, nor their chemical nature has been elucidated. The purpose of this note is to show that these bodies are formed in the Golgi complex.

MATERIAL AND METHODS

Our observations were made on human blood vessels in the course of other studies. Small blocks of muscular, endometrial, pleural, and nervous tissue were fixed in 1% osmium tetroxide, buffered according to Palade, or in 3.8% glutaraldehyde pH 7.2 with, in this case, osmium tetroxide postfixation. The blocks were dehydrated in ethanol, embedded in Araldite, and sectioned on a Porter-Blum microtome (Ivan Sorvall Inc., Norwalk, Conn.). The thin sections were contrasted with uranyl acetate and lead citrate and then examined with a Philips EM 200 microscope.

RESULTS

In the endothelial cells we have observed that the endoplasmic reticulum is sparse. The Golgi complex consistes of 5–6 flattened cisternae curved toward the vascular lumen. Around the cisternae there are numerous vesicles; they are either smooth vesicles of variable size, which are derived by pinocytosis and/or by budding of the endoplasmic reticulum, or small coated vesicles 500 A in diameter that supposedly serve to transport hydrolytic enzymes (5).

In some cases the distended extremity of the innermost cisternae contains fine tubular elements 150 A in diameter (Fig. 1). The intertubular substance is of low density in the tubular inclusions adjacent to the Golgi complex (Fig. 2), whereas it is more osmiophilic in the other cytoplasmic localizations (Fig. 5).

The membrane of most of the tubular inclusions is regular and even, but it may be irregular with semicircular protrusions that suggest vesicular remnants (Figs. 3 and 4).

Contiguous relations exist between the tubular inclusions and the plasma membrane and more

especially, the pinocytotic vesicles; these contacts occur on the basal side as well as on the luminal side of the endothelial cell; fusion between the tubular-inclusion membrane and pinocytotic vesicles was not observed (Fig. 5).

Like all cells, the endothelial cell contains cytoplasmic microtubules (6); with respect to their diameter (250–300 A) the microtubules are different from the fine tubular elements of the tubular inclusions (Fig. 3).

DISCUSSION

These observations suggest that the tubular inclusions of the endothelial cell are assembled and possibly synthesized in the Golgi complex. The extremity of the innermost cisternae expands, accumulates fine tubular elements, and then is released into the cytoplasm mechanisms that are common in a wide variety of secretory cells (7, 8).

The irregular outline of some tubular inclusions and their contacts with pinocytotic vesicles lead us to consider the following two directions of material transport: a budding of the inclusion with formation of vesicles containing intertubular substance; a secondary incorporation of substance in the tubular inclusions with fusion of vesicles. The latter mechanism seems most probable; it is comparable to the intracellular transport of secretory proteins in the Golgi condensing vacuoles (7, 9).

What happens to the mature tubular inclusions is not certain. Lysosomal degradation with transformation to heterogeneous dense bodies does not occur (10). But an elimination into the vascular lumen seems to exist; indeed, when the aorta is perfused with adrenalin solutions, the appearance of a coagulating substance (11) is accompanied by a quantitative reduction of the tubular inclusions (4). However, neither these tubular inclusions nor their tubular elements was seen in the vascular lumen, and the discharge of the tubular inclusions outside the cell was not observed.

Though a Golgi origin of the tubular inclusions of the endothelial cell seems to be evident, the mechanism of their elimination and the chemical nature of these inclusions need further investigation.

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The endothelial cell of the first figure is from a pleural arteriolar vessel during a serofibrinous pleurisy; the endothelial cells of the other figures are from vessels of human endometrium.

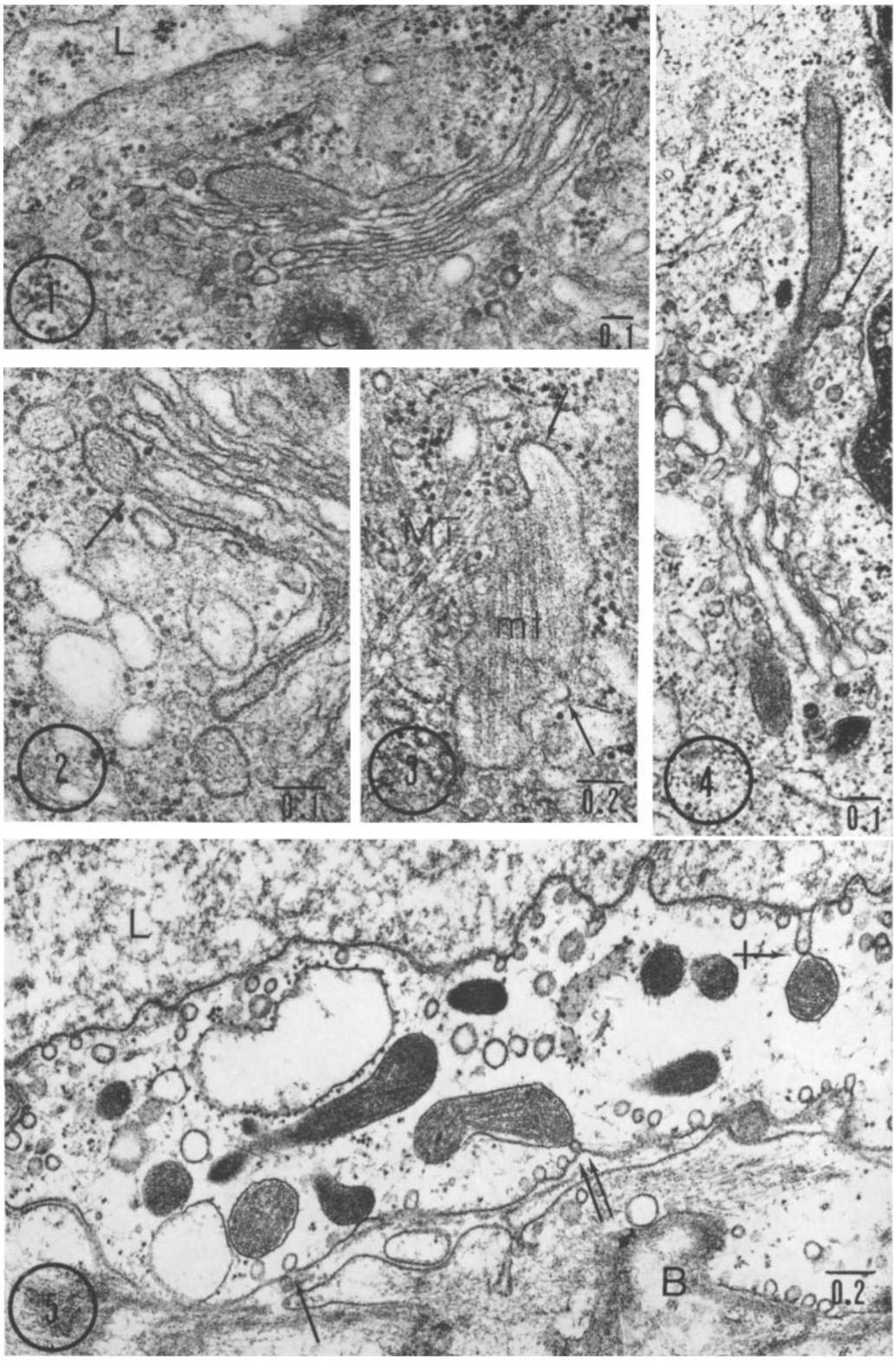
FIGURE 1 The concavity of the Golgi complex is turned toward the vascular lumen. The distended extremity of the inner cisternae contains fine tubular elements. Glutaraldehyde fixation. *L*, vascular lumen; *C*, centriole. × 51,000.

FIGURE 2 Three tubular inclusions of the Golgi zone; one seems continuous with the inner Golgi cisterna (arrow). Glutaraldehyde fixation. × 80,000.

FIGURE 3 Tubular inclusion of irregular shape, with semicircular outlines (vesicular remnants ?) (arrows). Note the difference in diameter between the fine tubular elements in the tubular inclusion and the microtubules (*MT*) in the cytoplasm. Glutaraldehyde fixation. × 37,000.

FIGURE 4 Two tubular inclusions in the Golgi zone, one with a well-recognizable vesicle (arrow). Glutaraldehyde fixation. × 44,000.

FIGURE 5 The tubular inclusions are numerous. Note the variable density of the intertubular substance, the multiple directions of the fine tubular elements, the contacts with the plasma membrane (→), with a pinocytotic vesicle of the basal face (⇒), and of the luminal face (↔). Palade fixation; L, vascular lumen; B, basal lamina. × 37,000.


L
1
0.1
2
0.1
MT
mi
3
0.2
4
0.1
L
5
B
0.2

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