'Fibrils' in Human Blood Platelets

During investigations concerning the storage of serotonin in human platelets, which have included a study of the ultrastructure of platelets from normal subjects and from patients with the carcinoid syndrome, we have frequently observed within the cytoplasm of normal platelets a system of filaments or fibrils measuring approximatley 200 Å in diameter. These filaments appear to consist of a single agranular membrane enclosing a less electron dense core and they characteristically form parallel bundles, often extending throughout the cell and into the processes (Figure 1). Occasionally they are arranged circumferentially around the periphery of the cell, but more frequently they have their origin in the centre of the platelet where they may be so tightly packed that at low magnifications the area appears to be of uniform density.

It is important that this cytoplasmic feature is distinguished from the tubular structure (Figure 2) (approx. 400 Å in diameter) which is also seen in human blood platelets. These larger tubules, which resemble a smooth endoplasmic reticulum, clearly communicate with the small vacuoles seen in normal platelets and it is these tubules and vacuoles which are more prominent in platelets from patients with the carcinoid syndrome when they contain high concentrations of serotonin.

The smaller fibrils have been observed in platelets from 4 normal subjects and also in normal platelets after incubation with serotonin (2000–2500 ng serotonin/10^9 platelets). They have only occasionally been observed in platelets from patients with the carcinoid syndrome (4000–8000 ng serotonin/10^9 platelets), and are not considered to be associated with increased serotonin storage.

Since they are more frequently seen in cells in which the cytoplasmic processes are more prominent, it is possible that these fibrillary elements may be associated with the 'contractile protein' known to be present in blood platelets (BETTEX-GALLAND and LUSCHER[1,2] and GRETTE[3]). This protein, which represents about 15% of the total platelet protein in human platelets and slightly more in pig platelets, can be extracted with KCl at high ionic strengths, and at lower ionic strengths it has been shown to have an ATP-splitting activity (BETTEX-GALLAND and LUSCHER[2]). Its role in viscous metamorphosis, clot retraction and similar mechanisms which result in asymmetry of the platelet is by no means clear, but the formation of ADP by intracellular ATP-ase activity may be of considerable significance in platelet adhesiveness, aggregation and other less severe alterations in their physical properties. The parallel orientation of these fibrils, considered together with the known solubility characteristic of the platelet contractile protein (BETTEX-GALLAND and LUSCHER[2]), would suggest that their degree of differentiation may be dependent upon changes in local ionic concentrations within the cell cytoplasm.

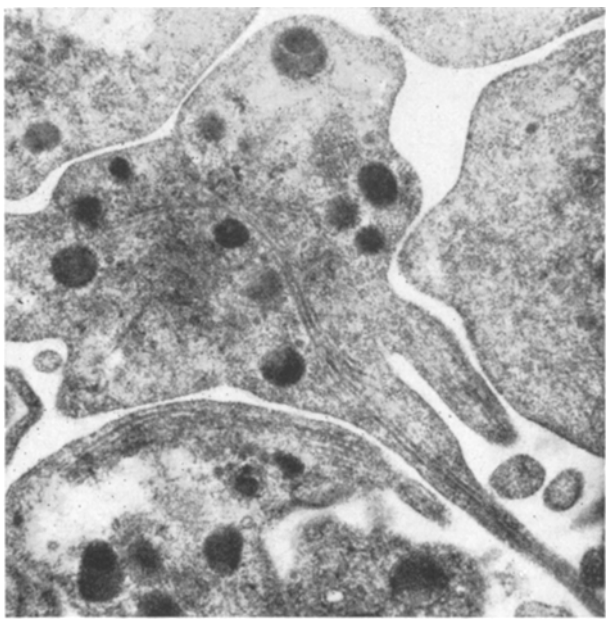

Fig. 1. Bundles of fine filaments extend across the central platelet into a cytoplasmic process. Similar filaments in an adjacent platelet lie parallel to the boundary membrane. Uranyl acetate stain. × 33,000.

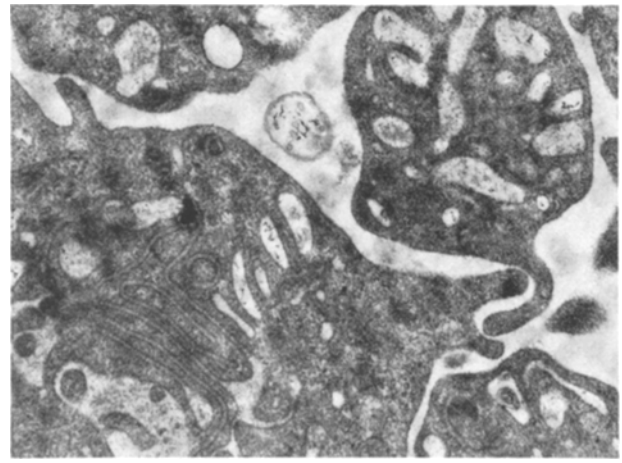

Fig. 2. Platelets from a patient with the carcinoid syndrome showing prominent tubular structure communicating with the larger vacuoles. Uranyl acetate stain. × 33,000.

Zusammenfassung. Im Verlauf elektronenmikroskopischer Untersuchungen der Thrombocyten des Menschen wurden Fibrillen von etwa 200 Å im Durchmesser beobachtet, die Bündel bilden, welche sich durch die ganze Zelle und in die cytoplasmatischen Fortsätze erstrecken können. Diese Struktur steht mit dem Vorhandensein eines kontraktilen Proteins im Zusammenhang.

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