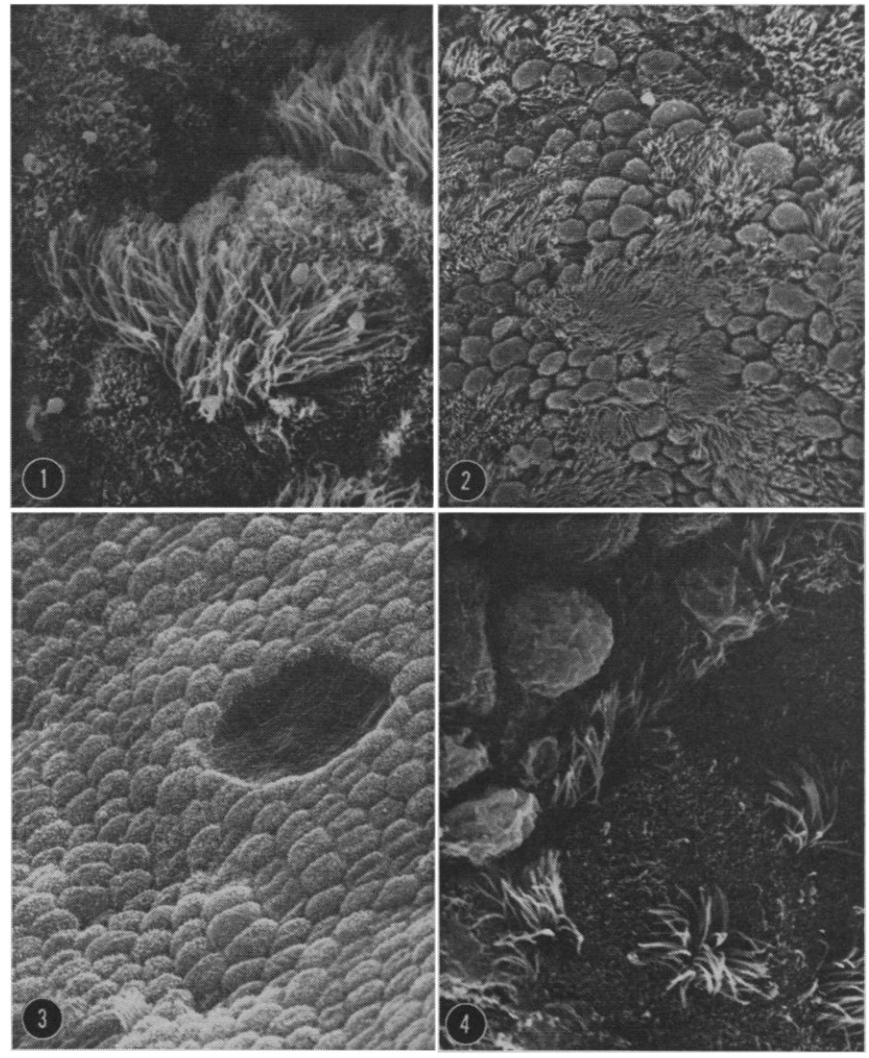
# Scanning Electron Microscopy of the Human Fallopian Tube

Abstract. *Fallopian tubes from women of reproductive and postmenopausal ages were examined by scanning electron microscopy. The surface topography of these Fallopian tubes is described and illustrated.*

Several studies of the human Fallopian tube by light microscopy and by transmission electron microscopy, along with the use of special stains and biochemical techniques, have been reported (*1–4*). However, many features of the cyclic morphologic changes in the tubal epithelium and of the events that occur during ovum and sperm transport, fertilization, cleavage, disturbed endocrine states, infertility, and contraception remain obscure. To extend our knowledge of the epithelium of the human oviduct and its responses to various physiological and pathological states, we have used the scanning electron microscope (SEM) to examine topographical relations of the epithelial surfaces of normal and atrophic human Fallopian tubes.

Intact Fallopian tubes were obtained at hysterectomy from patients aged 30 to 38 years, with normal menstrual cycles, and from postmenopausal patients aged 30 years and more. Samples were taken from the isthmus, ampulla, and fimbria, and immediately fixed in 2.5 percent repurified glutaraldehyde, frozen in liquid Freon, chilled with liquid nitrogen, and dried while frozen in a Pearse-Edwards tissue drier. They were then cemented onto specimen holders with a conductive silver paint and coated with gold-palladium in a vacuum evaporator. The specimens were examined with a Cambridge Stereoscan SEM operating at 20 kv, with secondary electron emission. The stage of the menstrual cycle was determined by histological examinations of the endometrium in the hysterectomy specimens.

In the late proliferative phase (12 to 14 days), the luminal surface contained clusters of ciliated and nonciliated cells in approximately equal numbers and similar proportions within the isthmic, ampullary, and fimbriated sections, but the ciliated cells were more prominent on the plical summits than in the crypts (Fig. 1). Although the surface of both cell types often appeared to be at about the same level, masses of cilia, sometimes adherent to each other and sometimes having a wavy appearance, projected well above the secretory cells and resulted in an irregular surface pattern. Groups of nonciliated secretory cells were arranged in a cobblestone fashion and covered by conspicuous, fine microvilli (Fig. 1). Numerous droplets of varying size, presumably secretory in nature, were found on the tips of cilia and on nonciliated cells in all segments of the oviduct (Fig. 1). Occasionally, several small cytoplasmic buds were noted on the secretory cell surfaces; perhaps this represented apocrine secretory activity (*3*). Small surface, or peg cells found between the secretory cells in light and transmission microscopy were visible in our preparations. They often ex-




Figs. 1–4. The figures all are scanning electron micrographs from the ampullae of human oviducts obtained by hysterectomy at various stages of the reproductive cycle. Fig. 1. Proliferation phase. Tufts of cilia sprout from cells interspersed among nonciliated secretory cells. The domes of the latter are covered with profuse small microvilli. Small droplets of secretory material are present on the surfaces of these cells and also on the tips of the villi (× 2600). Fig. 2. Secretory phase. The cobblestone-like secretory cells bulge into the lumen. The microvilli are more sparse on the swollen domes of these cells, which seem to project above the level of the ciliated cells, but not of the cilia themselves (× 810). Fig. 3. Atrophic, postmenopausal phase. The surface is dominated by the cobblestone-like secretory cells. At the lower left corner a single tuft of cilia is seen. Here and there, circular or ellipsoidal depressions occur. These may represent atrophic secretory cells (× 620). Fig. 4. Secretory phase. The tufts contain fewer cilia and there are areas apparently representing denuded surfaces of the ciliated cells. The cilia themselves are bent and have irregular contours. Bulging secretory cells are seen in the upper left of the figure. At about the center left, a small cell, presumably a peg cell, is seen (× 1200).

hibited indentations of their luminal surfaces, probably as representations of their discharged secretion (Fig. 4).

In the early luteal phase (Fig. 2) the tubal epithelium had a surface topography similar to that found in the proliferative phase. The ciliated cells were more prominent on the summits of the tubal plicae, and this suggested that cilial transport occurs principally in the central tubal lumen with secretory activity being more concentrated in the plical crypts. The finding of a relatively uniform distribution of the various cell types throughout the entire length of the tube is in agreement with the report of Hashimoto *et al.* (*3*) and in contrast to those of Clyman (*2*) and of Woodruff and Paurstein (*1*) who reported more ciliated cells in the fimbria. The smaller peg cells, found by transmission microscopy to be compressed between the larger ciliated and secretory cells, have been identified with a reserve or precursor function. It may be that this is not so and that the peg cells represent exhausted secretory cells.

Secretory activity occurred in all portions of the oviduct. Small cytoplasmic buddings were occasionally seen on the surface of secretory cells. The secreted material, once detached from the cell, resembled mucus droplets and most likely correspond to the periodic acid Schiff test-positive, diastase-resistant (*5*) secretory substances seen in the secretory cells at the fine structural level (*2*, *3*).

Atrophy occurring after the menopause apparently affects both the ciliated and nonciliated cells of the oviduct (Fig. 3). Only rarely are tufts of cilia encountered. The greater part of the oviduct's surface is lined by cells having the domed apices with fine microvilli characteristic of the secretory cells. These rarely exhibit the bulging contours or collapsed appearances identified during the cycle as evidences of secretion. In scattered locations, this cobblestone pattern is interrupted by areas of flattened cells. The hexagonal pattern and prominent terminal bar attachments of these flattened cells resemble the arrangements of mesothelia (Fig. 3). The flattened cells probably represent atrophied tubal cells.

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**References and Notes**

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## Inhibition of Antibodies to Nuclear Antigen and to DNA in New Zealand Mice Infected with Lactate Dehydrogenase Virus

Abstract. *New Zealand mice developed antibodies to nuclear antigen leading to immune-complex nephritis. Humoral antibody was directed primarily against denatured DNA, although antibody to native DNA was also found. Persistent infection with lactate dehydrogenase virus significantly lowered antibodies both to nuclear antigen and to DNA in these mice. In addition, female (NZB × W)$F_1$ mice infected with lactate dehydrogenase virus were protected from the usual nephritic death occurring after the trapping of complexes of nuclear antigen and its antibody and of DNA and its antibody in the glomerular filter.*

New Zealand (NZ) mice serve as a model of human systemic lupus erythematosus. A certain proportion of these mice [NZB, NZW, (NZB × W)$F_1$] develop autoimmune disease manifested mainly by antibodies to nuclear antigen (ANA) and antibodies to red blood cells, subsequently accompanied by immune-complex glomerulonephritis and autoimmune hemolytic anemia, respectively (*1–3*). While the etiology of the development of the ANA response is not clear, it appears most likely that immunologic hyperresponsiveness of NZ mice to nucleic acid antigens is an important predisposing factor. In our laboratory we have shown that persistent infection with either of two dissimilar viruses, lymphocytic choriomeningitis virus (an RNA virus) and polyoma virus (a DNA virus), enhances ANA formation and aggravates the immune-complex glomerulonephritis (*1*, *4*). Others have reported that immunization with DNA or synthetic polynucleotides enhances both ANA and glomerulonephritis (*3*, *5*, *6*).

We report now that persistent infection with lactate dehydrogenase virus (LDV), a relatively noncytopathic RNA virus, significantly decreased the production of both ANA and antibody to DNA in NZ mice. In addition, 9-month-old (NZB × W)$F_1$ females infected with LDV over a long period were protected from the nephritis and death that usually occurs as a result of nuclear antigen-ANA complexes or of DNA–antibody to DNA complexes being trapped in the glomerular filter.

The NZB and NZW mice were originally obtained from the Laboratory Animal Center, Medical Research Council, Surrey, England, and were inbred in our laboratory by brother-sister mating. The NZB females were crossed (at random) with NZW males and vice versa. Periodic testing indicated that the mice were free of infection with lymphocytic choriomeningitis virus, polyoma virus, and LDV. The persisting LDV infection was induced by inoculating 4-, 10-, or 12-week-old mice intraperitoneally with $10^5$ infectious doses ($ID_{50}$) of LDV prepared from pools of isologous serums. Details of virus passage, inoculation procedures, and development and testing for LDV infection have been described (*7*, *8*). Tests for ANA and for clinical and immunopathologic changes associated with nephritis have been reported (*3*, *4*, *9*). Antibodies directed against denatured and native DNA were measured in samples of plasma from noninfected and LDV-infected NZ mice by means of a modified Farr technique (*10*). Briefly, the ability of various dilutions of NZ plasma to bind immunospecifically with $H^3$-labeled DNA prepared from either SV20 virus, adenoviruses, or mouse L cells was determined. By this procedure, it is possible to detect as little as 20 to 30 ng of antibody to DNA.

NZW mice have ANA and glomerulonephritis, but the incidence and severity are lower than in the NZB × W hybrid. Normally, in NZW mice the incidence of ANA increases with age. In contrast, NZW littermates infected with LDV over a long period show no increased incidence of ANA (Fig. 1).