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Ultrastructural Observation of Mitochondria in Human Breast Carcinoma Cells

R. L. Elliott and B. G. Barnett

Elliott-Elliott-Head Breast Cancer Research and Treatment Center
17050 Medical Center Drive, 4th Floor, Plaza II, Baton Rouge, LA 70816

Tissue from (778) patients with breast carcinoma was fixed in Zamboni's; post fixed in 1% OSO_4; embedded in Med Cast Resin; and thin sections were cut on an ultramicrotome. Sections were stained with uranyl acetate and lead citrate and examined in a Hitachi H-600 electron microscope [1].

We have previously reported on tissue ferritin concentration and electron microscopic evaluation of ferritin in breast carcinoma [2], and also have presented the ultrastructural observations of ductal carcinoma in 346 patients [3]. The conversion of glucose to lactic acid in the presence of oxygen is known as aerobic glycolysis or "Warburg effect". Aerobic glycolysis is increased and observed uniquely in cancer and was first reported by Warburg in the 1920s [4]. This caused him to suspect that cancer results from impaired mitochondrial metabolism. This was later though incorrect, but renewed interest in tumor metabolism has been rekindled because of the clinical application of positron emission tomography (PET) imaging. Primary and metastatic human cancers show marked increased glucose uptake.

In 2005, Gottlieb and Tomlinson reported on a genetic and biochemical uptake of mitochondrial tumor suppressors [5]. This has prompted us to review and concentrate on the ultrastructure of mitochondria in our breast carcinoma patients. Ultrastructural observation revealed these three groups: (1) mitochondria absent (458), (2) mitochondria present (145), and (3) mitochondria present but sparse (175) (Figs. 1,2,3). The absent mitochondria groups were ultrastructurally much more anaplastic and aggressive (Fig. 1); while the mitochondria present group were more differentiated with normal appearing mitochondria (Fig. 2). The mitochondria in the present but sparse group were frequently very abnormal. They were swollen, ovoid, vacuolated, less dense with fractured cristae (Figs. 3,4).

These findings support that mitochondrial dysfunction in breast carcinoma is definitely involved in tumor metabolism. It suggests that the absence of mitochondria in breast carcinoma cells might be an indication of treatment resistant more aggressive disease, and could be a therapeutic target.

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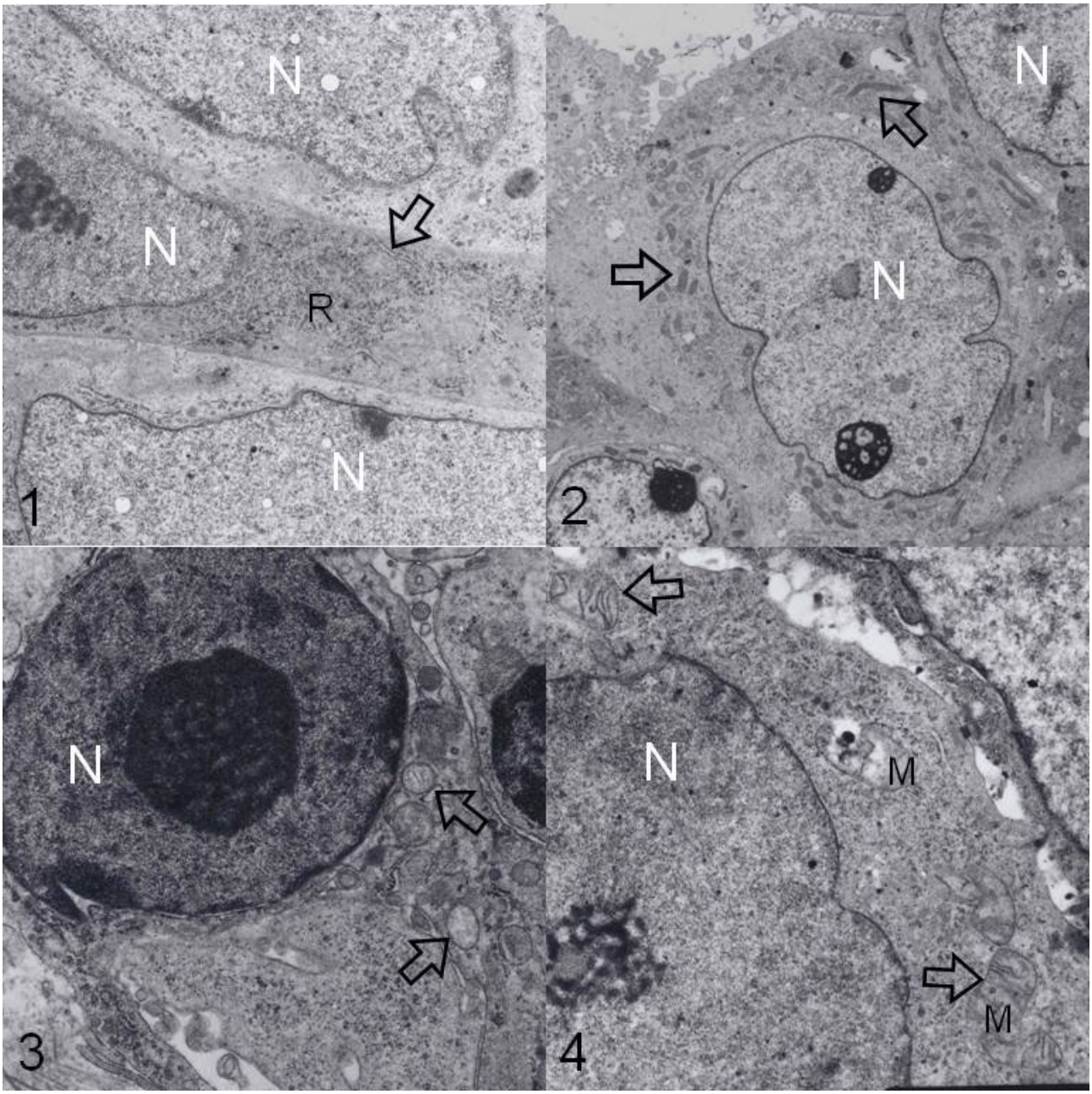

Fig.1. Group I Carcinoma Cells. Compact cytoplasm with granular endoplasmic reticulum (arrow), free ribosomes (R) and no mitochondria, nuclei (N). x 14,000.

Fig. 2. Group II Carcinoma Cells. Compact cytoplasm with abundant rod-shaped dense mitochondria (arrows), nuclei (N). x 8,000.

Fig. 3. Group III Carcinoma Cells. Electron dense cytoplasm with a few swollen mitochondria (arrows), nuclei (N). x 20,000.

Fig. 4. Group III Carcinoma Cell with a few large swollen mitochondria (M) and disrupted cristae (arrows), nucleus (N). x 25,000.

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