Mitochondrial Control of TGFβ-Induced Cellular Plasticity

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Mitochondrial dysfunction is increasingly recognized as a key contributor to fibrotic disease, yet how it regulates cellular plasticity remains unclear. Here, we examined mitochondrial changes during TGFβ-induced epithelial–mesenchymal transition (EMT). TGFβ impaired mitochondrial function and altered mitochondrial membrane potential, accompanied by disrupted ATP synthase activity linked to reduced levels of its assembly factor, ATPAF1. Modulation of ATPAF1 influenced mitochondrial function and the extent of EMT. Consistent alterations were observed in experimental models and human samples. These findings suggest that mitochondrial regulation, partly through ATPAF1, plays an important role in TGFβ-driven cellular plasticity and may represent a potential target for fibrotic disease.