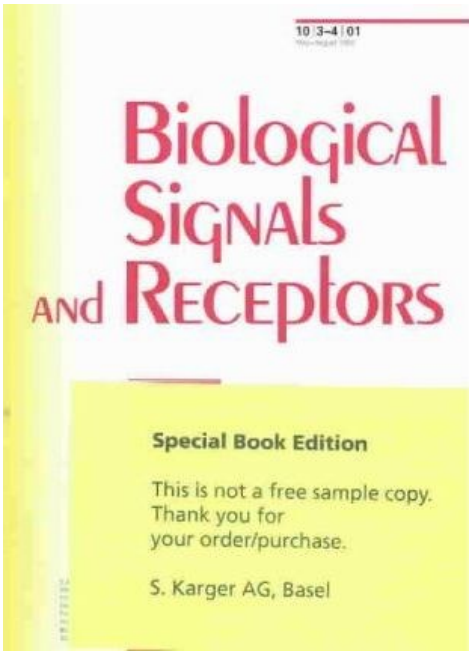

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Neurons are particularly vulnerable to age-associated decline in mitochondrial function due to their high energy demand. Thus, several models are proposed implicating mtDNA damage and mutation, and subsequent mitochondrial dysfunction, as an underlying factor in a substantial number of human pathologies including aging, neurodegenerative and neuromuscular diseases, and ischemia-reperfusion injury. Furthermore, the results of clinical, biochemical and molecular biology studies suggest that defective mitochondria contribute to neurodegenerative diseases in humans. For example, Friedreichs ataxia exhibits mitochondrial iron overload, Huntingtons disease is characterized by defects in mitochondrial oxidative phosphorylation and aconitase deficiency, Parkinsons disease is associated with a deficiency in mitochondrial complex I activity, and Alzheimers disease exhibits cytochrome oxidase deficiency. Neurologists, psychiatrists and biochemists will profit most from this special issue.