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RealmlP Color 101+ guide pdf - Full deck review - Actioncard - No download - League of legends 68877 Panda 3 bedroom full house - water house, corner house, home theater and u plan 568, floor plan,Â .Homeostasis of the cellular redox state, NADH/NAD+ and NADPH/NADP+ ratio, determines the cell fate decision of cells responding to oxidative stress, mitochondria are the main source of reactive oxygen species. Mitochondria form a dynamic network that is shaped by the ongoing fusion and fission processes. Both fission and fusion are important for their function and maintenance, but whether the balance between fission and fusion is important for cellular homeostasis and whether this balance is a cellular decision node is unknown. Recently, several labs have shown that fission is an active process, driven by mitochondrial dynamics proteins in a GTPase Rheb-dependent manner. We have shown that Rheb is a metabolic sensor that regulates the mitochondrial fission protein Drp1. Thus, Drp1 activity is not only regulated by the balance between fission and fusion, but also by mitochondrial metabolism. Therefore, in this proposal we will test whether the mitochondrial metabolism is a major node in the decision of cells responding to oxidative stress, and if so, what are the mechanisms of this decision? To test this hypothesis, we propose to use mouse embryonic fibroblasts (MEFs) as a model system to study the role of mitochondrial metabolism in the decision of cells to undergo apoptosis, and in particular, if changes in the metabolic state alter the threshold of oxidative stress for this decision. We propose three Aims: 1) To study the role of mitochondrial metabolism in the decision of cells to undergo apoptosis. We will determine if changes in mitochondrial metabolism alter the threshold of oxidative stress for apoptosis in MEFs in response to stress. 2) To study the molecular basis of the mechanisms of Rheb-dependent Drp1 activation in stress. We will determine how Rheb activity is regulated by mitochondrial metabolism and study the pathways that link changes in mitochondrial metabolism to Drp1 activation in cells responding to oxidative stress. 3) To determine the role of Drp1, mitochondrial metabolism, and the decision of cells to undergo apoptosis in the pathogenesis of mitochondrial disease. We will study the mechanisms of the pathogenesis of mitochondrial diseases in MEFs carrying mutations in mitochondrial fusion or fission proteins. This work will elucidate how the balance between

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