Introduction to LaTeX

Author's Name

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Abstract

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1 Introduction

- Time course of pS6K in AA and AA + rapamycin conditions [1]
- Rheb activates AMPK and reduces p27 in TSC2 null cells which in turn reduces cdk2 [2]
- Rheb is constitutively active in TSC2 knockout cells [2]
- In TSC2 null cells, down regulating Rheb down regulated mTORC1 and s6k
- TSC2 is a GAP for Rheb [3]
- The more TSC2 in the system the more Rheb that is hydrolysed [3]
- Rheb-GTP is an activator of mTORC1, measured by an increase in S6K and 4EBP phos
- The more RhebGTP present the more mTORC1 activation and S6K/4EBP phos [3]

1.1 [?]

- mTORC1 phosphorylates Akt at S473

1.2 [?]

This is a review

- Amino acids inhibit TSC2

1.3 [?]

- Insulin and amino acids both stimulate mTORC1 individually and synergize together
- Wortmannin inhibits these reactions

1.4 [?]

- RAG proteins bind TSC2. Therefore, RAGs not only activate mTORC1 by inducing their recruitment to the lysosome, they also actively repress mTORC1 in the absence of AA

References

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[2] MD Lacher, R Pincheira, Z Zhu, B Camoretti-Mercado, M Matli, RS Warren, and AF Castro. Rheb activates ampk and reduces p27kip1 levels in tsc2-null cells via mtorc1-independent mechanisms: implications ffile:///home/ncw135/downloads/10.1038$_n$*cb*839.*risorcellproliferationandtumorigenesis.Oncogene*, 29(5
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[3] Ken Inoki, Yong Li, Tian Xu, and Kun-Liang Guan. Rheb gtpase is a direct target of tsc2 gap activity and regulates mtor signaling. *Genes & development*, 17(15):1829–1834, 2003.