Covered Article: “Resistance Exercise-Induced Hypertrophy: A Potential Role for Rapamycin-Insensitive mTOR”
Authors: Riki Ogasawara, Thomas E. Jensen, Craig A. Goodman, and Troy A. Hornberger

1) Explain the differences between mTORC1 versus mTORC2.

2) Explain the differences between rapamycin-sensitive versus rapamycin-insensitive components of mTOR signaling.

3) Resistance exercise can stimulate both mTOR signaling and muscle protein synthesis. Explain the association between exercise-induced activation of mTOR and protein synthesis.

4) From a structural perspective, explain how rapamycin can inhibit some, but not all, mTORC1-dependent signaling events.

5) From a structural perspective, explain why rapamycin does not exert an acute inhibitory effect on mTORC2 signaling.

6) What evidence supports the conclusion that signaling by mTORC1, within the muscle cells themselves, is necessary for chronic mechanical overload-induced hypertrophy?

7) Why do the data obtained with pharmacological inhibitors AZD-8055 and rapamycin suggest that mTORC2 may play a role in resistance exercise-induced hypertrophy?

8) What is the molecular contribution of mTORC2 to the hypertrophy observed in muscle after training with resistance exercises?

9) What are the methodological strengths and weaknesses of using pharmacological inhibitors for mechanistic studies?

10) What are the strengths and weaknesses of using rat in situ contractions to study the resistance-exercise adaptations observed in humans?