

# Role for phosphatidylinositol 4-kinase III $\beta$ in cardiac metabolic diseases

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## Propositions

accompanying the dissertation

### Role for phosphatidylinositol 4-kinase III $\beta$ in cardiac metabolic diseases

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Maastricht, September 8, 2020

1. In cardiac myocytes PI4KIII $\beta$  is a key component of contraction-induced glucose uptake but is not involved in contraction-induced fatty acid uptake (*this thesis*).
2. Unlike protein kinase D1 (PKD1), PI4KIII $\beta$  is not participating in common pathways associated with cardiac hypertrophy (*this thesis*).
3. Overexpression of PI4KIII $\beta$  increases basal glucose uptake in cardiomyocytes and preserves insulin sensitivity in lipid-overloaded cardiomyocytes (*this thesis*).
4. Inhibition of PI4KIII $\beta$  by MI14 decreases enhanced glucose uptake in the hypertrophic cardiomyocytes (*this thesis*).
5. As an inhibitor of PI4KIII $\beta$ , MI14 prevents contractile dysfunction in the hypertrophic heart (*this thesis*).
6. PI4KIII $\beta$  is an essential host factor for severe acute respiratory syndrome coronavirus (SARS-Cov).
7. Patient-specific human induced pluripotent stem cell-derived cardiomyocytes offer an experimental platform to model cardiovascular diseases.
8. By virtue of its contribution to the development of pathological cardiac hypertrophy, store-operated Ca<sup>2+</sup> entry (SOCE) provides a suitable therapeutic strategy for maintaining contractility reserve after hypertrophic stress.
9. Science gathers knowledge faster than society gathers wisdom, which is the saddest aspect of life right now (*Isaac Asimov*).
10. The science of today is the technology of tomorrow (*Edward Teller*).
11. Bad times have a scientific value. These are occasions a good learner would not miss (*Ralph Waldo Emerson*).