

## **ERBB2 as a Novel Target to Treat Heart Failure by Inducing Cardiomyocyte Proliferation (Yeda)**

**code:** T4-1752

[Eldad Tzahor](#), Biology, Biological Regulation

### **Summary**

Existing treatments fail to compensate the irreversible loss of functional cardiomyocytes (CM), thus presenting a major medical unmet need. Inducing native CM replacement is one approach being tested as regenerative treatment, with the advantage of a more straightforward methodology over cell transplantation approaches. In a multidisciplinary study, headed by Prof. Eldad Tzahor from the Weizmann Institute of Science, the tyrosine kinase ERBB2 was shown to be both necessary for CM proliferation and sufficient to reactivate postnatal CM proliferative and regenerative potentials. Thus, potentiation of ERBB2 signalling in adult CMs might represent a promising therapeutic approach for CM replacement in heart failure.

### **Applications**

Induction of cardiomyocytes replacement therapy following heart injury.

### **Advantages**


Straightforward methodology - Avoids complications associated with the requirement for cell transplantation.

Include several optional targets - both ERBB2 and its downstream effectors serve as potential targets for therapeutic agents, which may be administered in combination, to increase chances for successes.

### **Technology's Essence**

In a multidisciplinary study, headed by Prof. Eldad Tzahor from the Weizmann Institute of Science, ERBB2 was shown to be necessary and limiting for NRG1-induced CM proliferation in the neonate. Inspired by this finding, the team examined the possibility to use ERBB2 as a target for induced cell proliferation and regeneration in adult hearts. Using loss- and gain-of-function genetic experiments in mice, they reveal that NRG1/ERBB2 signalling is both essential for CM proliferation and heart integrity in the neonatal period, and sufficient to prolong the postnatal proliferative and regenerative windows. Regeneration was shown to be a result of increased CM dedifferentiation and proliferation accompanied by neovascularization and followed by redifferentiation, tissue replacement with reduced scar formation and restoration of function. Thus, these findings highlight ERBB2 as a strong target for heart regeneration treatments as well as its downstream effectors.

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