Gene expression of selenoproteins can be regulated by thioredoxin(Txn) silence in chicken cardiomyocytes.


Abstract

Thioredoxin (Txn) system is the most crucial antioxidant defense mechanism in myocardium. The aim of this study was to clarify the effect of Txn low expression on 25 selenoproteins in chicken cardiomyocytes. We developed a Se-deficient model (0.033mg/kg) and Txn knock down cardiomyocytes model (siRNA) studies. Western Blot, Quantitative Real-time PCR (qPCR) were performed, and correlation analysis, heat map were used for further analysis. Both low expression of Txn models are significantly decreased (P<0.05) the mRNA levels of Deiodinase 1, 2 (Dio 1, 2), Glutathione Peroxidase 1, 2, 3, 4 (Gpx 1, 2, 3, 4), Thioredoxin Reductase 1, 2, 3 (TR 1, 2, 3), Selenoprotein t (Selt), Selenoprotein w (Selw), Selenoprotein k (Selk), selenoprotein x1 (Sepx1), and significantly increased (P<0.05) the mRNA levels of the rest of selenoproteins. Correlation analysis showed that Deiodinase 3 (Dio 3), Selenoprotein m (Selm), 15-kDa Selenoprotein (Selp15), Selenoprotein h (Selh), Selenoprotein u (Selu), Selenoprotein i (Seli), Selenoprotein n (Seln), Selenoprotein p1 (Sepp1), Selenoprotein o (Selo), Selenoprotein s (Sels), Selenoprotein synthetase 2 (Sels2) and Selenoprotein p (Selp) had a negative correlation with Txn, while the rest of selenoproteins had a positive correlation with Txn. Combined in vivo and in vitro we can know that hamper Txn expression can inhibit Gpx 1, 2, 3, 4, TR 1, 2, 3, Dio 1, 2, Selt, Selw, Selk, Sepx1, meanwhile, over expression the rest of selenoproteins. In conclusion, the different selenoproteins possess and exhibit distinct responses to silence of Txn in chicken cardiomyocytes.

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