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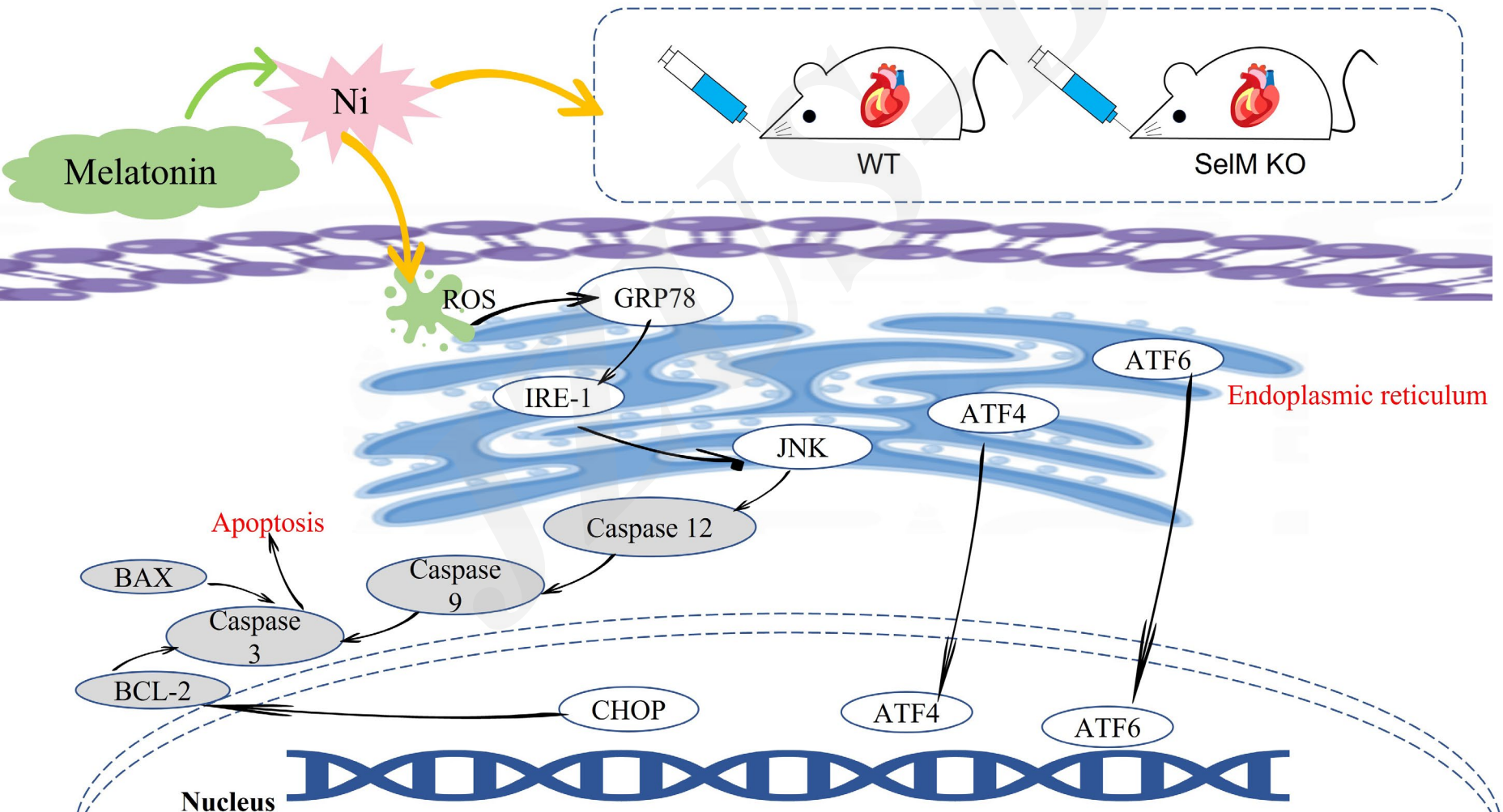
Role of selenoprotein M knockdown in the melatonin antagonism of nickel-induced apoptosis and endoplasmic reticulum stress in mouse heart

Key words: Selenoprotein M (SelM); $NiCl_2$; Melatonin; Apoptosis; Heart; Mouse

Research Summary

Our findings suggested that the antioxidant capacity of SelM, as well as its modulation of endoplasmic reticulum stress and apoptosis, play important roles in nickel-induced heart injury.



Innovation points

- **$NiCl_2$** induced oxidative stress, apoptosis and endoplasmic reticulum stress in male SELENOM wild-type (WT) and knockout (SELENOM$^{-/-}$) C57BL/6 J mice hearts.

- **Melatonin** could antagonize the cardiotoxic effects of $NiCl_2$ by alleviating apoptosis and endoplasmic reticulum stress in male SELENOM wild-type (WT) mice hearts.

- **After** exposed to $NiCl_2$, melatonin could not exert an antagonistic effect in SelM$^{-/-}$ mice, indicating that SelM played an important role in nickel-induced cardiac apoptosis and endoplasmic reticulum stress of mice.

Innovation points

A series of images were generated to elucidate the role of SelM in melatonin antagonism of $NiCl_2$ –induced the injury of mouse heart

Figure 1 | H&E staining of the hearts of WT and SelM KO mice after exposure to $NiCl_2$ chloride and/or melatonin (MT).

Figure 2 | Transmission electron microscopy results of cardiac ultrastructure in WT and SelM KO mice after exposure to $NiCl_2$.

Figure 3 | MDA content and T-AOC activity.

Figure 4 | mRNA and protein expression results of endoplasmic reticulum stress-related genes.

Figure 5 | mRNA and protein expression results of apoptosis-related genes.

Figure 6 | Correlation heatmap indicating the correlation of detected gene expression in mouse hearts exposed to $NiCl_2$ and/or melatonin (MT). Protein-protein interaction (PPI) network regulated between apoptosis and endoplasmic reticulum stress based on the String database in mouse hearts exposed to $NiCl_2$ and/or MT.