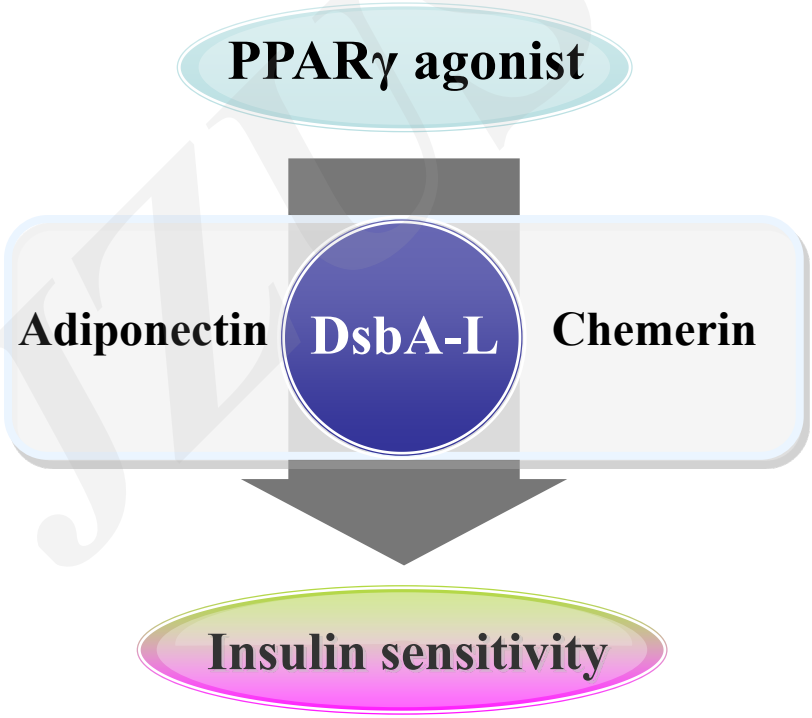
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Silencing of *DsbA-L* gene impairs the PPARγ agonist function of improving insulin resistance in a high glucose cell model

Key words: **Disulfide-bond A oxidoreductase-like protein (DsbA-L), Peroxisome proliferator-activated receptor γ (PPARγ), Chemerin, Insulin signaling pathway, Gestational diabetes mellitus**

Research Summary

This article mainly focused on the necessity of DsbA-L in the function of PPARγ agonists, as well as the potential relationship between DsbA-L and chemerin in high glucose treated HTR-8/SVneo cells.



Innovation points

- **Silencing DsbA-L affected the function of PPARγ agonist in promoting the phosphatidylinositol 3-kinase (PI3K) - protein kinase B (PKB) / AKT pathway.**

- **We demonstrated that chemerin enhances DsbA-L expression for the first time.**

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