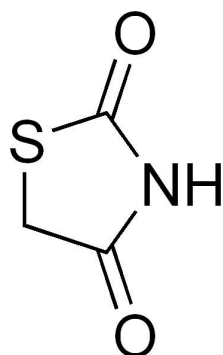


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By: Tudor Vieru, Science Editor



The basic chemical structure of thiazolidinedione  
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## [New Find Explains Why Diabetes Drugs Affect the Heart](#)

*Experts studying heart tumors have made the find*

Some time ago, researchers figured out that a specific class of anti-diabetes prescription drugs, known as thiazolidinediones (TZD), came with the risk of patients developing heart complications after use. The reason why this happened remained a mystery until recently, when a team of scientists managed to understand exactly how heart enlargement led to heart failure. The find, which was mostly the result of a study done on mice, may hold the clues experts need to prevent the side-effects of future generations of TZD.

In a famous 2007 scandal, the drug Avandia (rosiglitazone), produced by GlaxoSmithKline, part of the TZD class, was proven to increase the chance of users taking it having a heart attack. While this set of pieces of evidence was highly controversial, another one holding that the compound was also associated with an increased frequency of heart failures was much less so. In heart failure, which is a fairly common condition, the heart becomes unable to pump sufficient blood through the body, which fails, [Nature News](#) reports.

"We already knew if you had heart failure you probably should not be taking these drugs, but this paper provides an additional explanation why," Washington University School of Medicine (WUSM) Endocrinologist Clay Semenkovich said. According to the mouse study, the PPAR- $\gamma$ , a TZD-activated molecule, is the main reason why heart failures appear. However, the investigation, which was led by Swiss Federal Institute of Technology Cancer Biologist Wilhelm Krek, was not focused on diabetes, but on studying what happened when the tissue of the heart enlarged, and how it affected the organ's functions.

When the hearts increase, they switch from burning fat to burning glucose, which means that they have to get less oxygen, which saves energy. However, the latter fuel is not the optimum one, and the heart cells eventually become clogged with fat and commit suicide. The new study found that PPAR- $\gamma$  was actually responsible for speeding up this transition. Further results of the study are available in the June issue of the scientific journal Cell Metabolism.