

The History of Thalidomide

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What is Thalidomide?

Thalidomide is a sedative that used to be prescribed to treat anxiety, tension, gastritis and insomnia. It was also used to relieve morning sickness in pregnant women.



Where and when did this started?

Thalidomide was first marketed in 1957 in West Germany under the trade-name Contergan. The German drug company Chemie Grünenthal developed and sold the drug. Primarily prescribed as a sedative or hypnotic, thalidomide also claimed to cure anxiety, insomnia, gastritis, and tension.



What happen if Thalidomide go wrong?

In 1961, several physicians linked thalidomide with birth defects observed in their patients currently taking it. Almost immediately afterwards, physicians worldwide began confirming these results. Soon after the discovery of the teratogenic effects became known, thalidomide was taken off the market, but 15,000 children suffered disfiguring birth defects and many died.



What problem does thalidomide affect babies?

About 10,000 cases were reported of infants with phocomelia due to thalidomide, only 50% were survived. For babies, the effects included deformed eyes and hearts, deformed alimentary and urinary tracts, blindness and deafness.



After they stop producing thalidomide, does anyone still using?

Yes, an Israeli doctor discovered thalidomide autoimmune symptoms of leprosy have a therapeutic effect. They discovered that thalidomide for Kaposi's sarcoma, systemic lupus erythematosus, multiple myeloma have a therapeutic effect. So in 1998, the US Federal Food and Drug Administration approved thalidomide as a treatment for erythema nodosum leprosy of marketed drugs.



Does thalidomide has any benefit for us?

This review considers the recent clinical and relevant preclinical evidence that thalidomide may have therapeutic benefit in chronic heart failure (HF), and considers some of the mechanisms by which thalidomide may elicit potentially beneficial effects. Persistent inflammation, involving increased levels of inflammatory cytokines, seems to play a pathogenic role in chronic HF by influencing heart contractility, inducing matrix degradation and fibrosis, and promoting apoptosis, contributing to myocardial remodeling.

