By any measure, malaria is one of the most ruthless threats to global human health. It has been estimated that the parasite a protozoan called Plasmodium kills one child per minute in Africa alone. While it used to take the lives of over one-million people each year, mostly sub-Saharan African infants and children, the number has been reduced substantially thanks to modern public-health efforts, to approximately 650,000. But this number is still unacceptable, and twenty-times that number are chronically ill from malaria.

The infection can be treated or prevented, although there are significant problems with both strategies.

Any hope of effective prevention was pretty much tossed in the trash by the misguided ban on DDT the most effective weapon against the Anopheles mosquito, which is the vector for the infection initiated by our EPA in 1972.

It would be difficult to find a better example of the consequences of the folly illustrated by this ban than the 2005 article [1] by Paul Driessen entitled The Killer Elite: Anti-pesticide activists perpetuate diseases that kill millions.

Driessen, writing for the Capital Research Center [2] pulls no punches: Malaria is a disease that kills three times more African children than AIDS. Hundreds of millions are infected and up to two million die annually. But as the body count continues to mount, environmental activists and international aid agencies continue a deadly campaign against DDT.

Therapeutic options always inferior to prevention are limited. There is no vaccine against malaria, and the drugs used to treat the infection are far from optimal.

But, thanks to some ground-breaking research from scientists at St. Jude’s Research Hospital in Memphis, the malaria landscape could change significantly.
In the latest online early edition [3] of the Proceedings of the National Academy of Sciences (PNAS), a potential new drug called (+)-SJ733 has been shown to rapidly destroy red blood cells that are infected by malaria while leaving uninfected cells unharmed. This effect has been demonstrated in cellular assays, and in infected mice, where the results were astounding: In a mouse model of malaria, a single dose of the drug killed 80 percent of malaria parasites within one day. And, after two days hours the Plasmodium was undetectable.

Lead author Dr. R. Kiplin Guy, chair of the St. Jude Department of Chemical Biology and Therapeutics, explained: "Our goal is to develop an affordable, fast-acting combination therapy that cures malaria with a single dose."

Have they met this goal? In mice, the answer would seem to be a resounding yes. But will this translate into an effect in humans?

At ACSH, we frequently point out that rodent assays, although they certainly have a place in research, may or may not have relevance to what will happen in people. ACSH’s Dr. Josh Bloom explains, Rodent tests should be used as a rough guide to determine the toxicity or efficacy of a potential drug in humans. Although imperfect, they are an essential research tool for giving direction to a discovery program, or getting an idea of the toxicity of a compound. Sometimes the rodent models are relevant and sometimes they are not.

Fortunately, in the development of anti-infective drugs, especially antibiotics, rodent models are rather good predictors of the effect in people. Infectious disease expert, and ACSH advisor Dr. David Shlaes explains, In bacterial infections, certain animal models predict both in vivo activity in humans and even dosing required for best efficacy in humans. Outside of bacterial infections, the models are less robust.

Dr. Bloom elaborates, Cancer would have been cured hundreds of times by now if mouse models were predictive of anticancer effects in people. But they are far from it. At best they are an imperfect tool in oncology research.

Perhaps the most interesting aspect of (+)-SJ733 is that it operates by an entirely new mechanism. It targets an enzyme called ATP4, which maintains the proper concentration of sodium within the Plasmodium parasite. When this process is inhibited, the infected erythrocytes (red blood cells) commit suicide, but for uninfected cells, it is business as usual.

Dr. Bloom concludes, Drug discovery is impossibly difficult. There is so much that can go wrong, and the lack of reliable, predictive animal models contributes greatly to the difficulty. Having worked on a variety of failed projects over my career, my sense is that this one looks very good by comparison. We cannot undo millions of preventable deaths caused by stupid policy, but if this drug works as well in people as it does in mice, the entire landscape of malaria will change very quickly.