Psoriasis is a chronic, immune-mediated skin disease characterized by production of reactive oxygen species due to the activation of tumor necrosis factor alpha (TNF-α), which is thought to be an important factor in inducing and maintaining psoriatic lesions. As an external factor, ultraviolet B (UVB) radiation stimulates TNF-α production and secretion by human keratinocytes in vitro and can also reach the upper dermis and suppress endothelial cells in vitro. The selenium level in psoriatic patients has been found to be lower than expected, but studies on its role in the pathogenesis of the disease are scarce. Selenium can influence immune response by changing the expression of cytokines and their receptors or by making immune cells more resistant to oxidative stress. It was reported that selenium supplementation had inhibitory effects on TNF-α levels in patients with psoriasis, but the details are not completely elucidated. Selenium compounds are also known to prevent the in vitro release of UVB-induced proinflammatory cytokines by inhibition of mRNA in human keratinocytes. In the present review, the protective role of selenium in oxidative stress, lesions, and immune system regulation in patients with psoriasis is summarized.