Alzheimer's disease is the most common cause of dementia where the loss of intellectual and social abilities is severe enough to interfere with daily functioning. This may be a warning sign to our economy as unfortunately, the disease is growing up especially in the developing countries . Deposition of â-amyloid in brain is one of the pathological hallmarks of AD that is often associated with oxidative stress response. In this study, we investigated the possible mechanisms of tempol (superoxide scavenger), in a lipopolysaccharide model of Alzheimer's disease. Mice were randomly divided into three groups each group consisted of 8 mice. Mice were injected with lipopolysaccharide (0.8 mg/kg, i.p.) once then divided into two groups: the first was remained injected with lipopolysaccharide only serving as Alzheimer's disease control and the second was injected with tempol (100 mg/kg/day, i.p.), in addition to the normal control mice injected with 1% tween 80. Brain oxidative stress markers namely malondialdehyde, reduced glutathione and superoxide dismutase in addition to â-amyloid levels were measured. Lipopolysaccharide increased oxidative stress burden and finally caused â-amyloid deposition in brain. These effects were reversed by using tempol. Tempol decreased parameters of Alzheimer's disease and oxidative markers serving as a good protective a gaint against Alzheimer's disease.