Leprosy is a neglected disease caused by *Mycobacterium leprae*, that affects the skin and peripheral nerves. *M. leprae* hosts in macrophages and modulate innate responses to maintain the infection. The world health organization (WHO) classifies the disease as paucibacillary - PB (<5 cutaneous lesions) or multibacillary - MB (> 5 lesions). Immunopathologically, Leprosy presents as a spectrum of clinical forms, Tuberculoid (TT), Boderline (BL) and Lepromatous (LL). The immune response in HT is associated to a predominant Th1 response and in LL to a Th2 and T regulatory response. There are strong evidences of an influence of host genetics on leprosy susceptibility and clinical outcome. Overexpression of IL-17A member has been associated with efficient immune response in Leprosy. The chemokine, CC Motif, Ligand 2 (CCL2) is produced by lymphocytes and monocytes and plays a role in cellular immune responses to acute tissue injury. The rs2275913 SNP of IL-17A is the located in promoter region of the gene. We collected phenotype and genotype information of 200 patients, with confirmed diagnosed of leprosy from Sergipe, Brazil, and 100 genetically unrelated control individuals with prolonged and intense contact with the leprosy patients. Genomic DNA was genotyped by TaqMan assays. We compare the allele and genotype frequencies of the IL-17A rs2275913 and analyzed the association of these SNP with Leprosy per se (patients versus controls). The preliminary data, Indicate that there is an association of the G allele of IL-17A SNP with leprosy susceptibility. We are analyzing the family data from these patients and collecting more patients to confirm these associations.