Bonus: Using the above comparison try to find out the genetic mutation that lead to Psoriasis and why the mutation at one of the nine loci may lead to Psoriasis. Do we have any relation between the nine loci as mutation among them leads to the same disease?

Scientists have now identified about 25 genetic variants that make a person more likely to develop psoriatic disease. Scientists now believe that at least 10 percent of the general population inherits one or more of the genes that create a predisposition to psoriasis. However, only two percent to three percent of the population develops the disease.

The research shows that rare mutations in the *CARD14* gene, when activated by an environmental trigger, can lead to plaque psoriasis. This type of psoriasis accounts for 80 percent of all cases and is characterized by dry, raised, red patches covered with silvery scales that can be itchy and painful. The new findings also indicate that mutations in *CARD14* can be involved in the pustular form of psoriasis and in a debilitating arthritis linked to the psoriasis. The discovery may lead to more effective, targeted therapies for plaque psoriasis and other forms of the disease.

Loci at chromosome 17q25 and 4q have been identified following genome-wide linkage scans of large, multiply affected families

Psoriasis in some families is inherited as an autosomal dominant trait with high penetrance. Loci at chromosome 17q25 and 4q have been identified following genome-wide linkage scans of large, multiply affected families. In the case of at least the susceptibility locus at 17q25, the development of psoriasis does not require the presence of HLA-Cw6. Sib-pair analyses have confirmed the association with HLA-Cw6, confirmed the existence of a locus at 17q25 and identified other possible susceptibility loci.Two independent groups have reported a third region on chromosome 20p

Mutations in *IL36RN* were first described in 2009 in two families with severe pustular psoriasis. This gene, also called *IL-1F5*, encodes for an anti-inflammatory protein IL-36Ra, which is a natural antagonist of IL-1F9. Hence, a mutation in this gene leads to an altered protein with decreased effect and to unopposed IL-1F9 effects of NF-κB and MAPK activation through IL-1Rrp2 and IL-1RacP.