I would like to thank Omar and colleagues for drawing our attention to genetics of androgenetic alopecia (AGA). Although it is one of the most common dermatological problems, current treatment strategies are limited and their effectiveness remains modest at best.

In V89L polymorphism, leucin is inserted in the 5 alpha reductase enzyme type II instead of valine amino acid. Carrying leucine allele predicts about 3.7 higher risk of having AGA according to the study. The statistically significant positive family history of AGA in patients confirms this association. In addition, it should have been better to see the difference in family history of AGA as well as in controls.

Superoxide dismutase (SOD) and catalase enzymes are important antioxidant mechanisms in the body. They function cooperatively. Superoxide dismutase converted superoxide into hydrogen peroxide and oxygen. To complete the antioxidant process this hydrogen peroxide must be converted to water and oxygen by catalase. The authors found higher levels of lysate SOD and plasma catalase in patients than controls. The significantly difference of age between patients and controls may also contribute to this result in the study.

Regarding to catalase, there was a statistically significant difference between the homozygote (LL) genotype and the heterozygote (VL) genotype. On the contrary, the mean of SOD enzyme was lower in patients carrying the mutant (LL) genotype than those who are carrying the heterozygote (VL) genotypes, but the difference was statistically insignificant.

Mutations do not always cause the lack of expressing or translating related protein as we have seen in V89L polymorphism. Like this non-synonymous mutation, amino acid can be replaced by another amino acid. Although valine and leucine are physically and chemically similar, the effect on the enzyme activity of this polymorphism is still not known properly. The association between this polymorphism and antioxidant enzymes mentioned in the study may be one of these effects.

Further studies are needed to reveal the clinical and therapeutic implications of genetic polymorphisms in androgenetic alopecia.