mutation of the *FLG* gene (Chervet et al 2010) which is corresponding to the loss-of-function FLG mutation in HAD (Palmer et al 2006). The mutations of FLG gene in HAD may not be related to the decrease in mRNA expression because the mutated full-length mRNA transcript was found (Marenholz et al 2006; Palmer et al 2006; Sandilands et al 2007). However, a recent study in West Highland White Terriers demonstrated that atopic and normal dogs did not display significantly difference of FLG haplotype frequencies (Barros Roque et al 2009). Thus, the association of AD with the gene expression and/or mutation was needed to be further investigated.

6. Conclusion

In conclusion, this study reveals, at least in part, the pathogenesis of CAD. Dogs with AD are predisposed to develop hyperproliferation and alterations in differentiation of their skin keratinocytes, leading to epidermal hyperplasia. This was supported by histopathologically increased Ki-67 positive basal cells and reduced cytokeratins and CE protein expression although the dissociation between gene and protein expression was observed. Since IVL and Ki-67 protein expression was shown to be associated with the AD clinical severity scores in the present study and some topical drugs demonstrated keratinocyte antiproliferation and enhanced IVL expression (Hong et al 2008b; Kim et al 2010), IVL and Ki-67 could probably be used as protein markers in AD therapy.

7. Suggestion for Future Work

For the future work, the correlation of epidermal proliferation and differentiation to dry skin should be further investigated in CAD since skin dehydration were shown to be increased together with epidermal proliferation in human AD skin (Jensen et al 2004) and increased transepidermal water loss (TEWL) and decreased skin hydration and ceramides, lipid contents of the skin, was found in lesional CAD skin compared to the normal controls (Shimada et al 2009).