**Genetic and epigenetic pathogenesis and mechanisms of thyroid cancer.**

Shicheng Guo

Thyroid cancer is a common endocrine malignancy. There has been exciting progress in understanding its molecular pathogenesis in recent years, as best exemplified by the elucidation of the fundamental role of several major signalling pathways and related molecular derangements. Central to these mechanisms are the genetic and epigenetic alterations in these pathways, such as mutation, gene copy-number gain and aberrant gene methylation. Many of these molecular alterations represent novel diagnostic and prognostic molecular markers and therapeutic targets for thyroid cancer, which provide unprecedented opportunities for further research and clinical development of novel treatment strategies for this cancer

## **Key Points**

### **Thyroid cancer risk and protective genetic variants identified by GWAS study**

### Genetic and epigenetic of RA

Genetic alterations in thyroid cancer

### Functional implications of risk alleles

## **Epigenetics of thyroid cancer**

Genetic and epigenetic complementary effects were frequently observed in the pathogenesis of the diseases. For example, KLLN was hyper-methylated and transcriptional repressed in non-PTEN mutation Cowden and Cowden-like syndrome characterized by high risks of thyroid cancers1.

### Epigenetic mechanisms

### Environmental influences

### Epigenetic studies in RA

## **Future directions**

## **Conclusions**

## **Review criteria**

## **References**

Table 1 | Copy-number gains in thyroid cancer\*

Table 2 | Copy-number gains in thyroid cancer\*

Table 3 | Copy-number gains in thyroid cancer\*

1. Bennett, K.L., Mester, J. & Eng, C. Germline epigenetic regulation of KILLIN in Cowden and Cowden-like syndrome. *JAMA* **304**, 2724-31 (2010).