# CURRENT POSTGRADUATE STUDENT

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| **NAME** | Zhongqin | |
| **PROGRAM** | PhD in Surgery | |
| **DATE OF REGISTRATION** | 1/9/2017 | |
| **SUPERVISOR** | Professor CHEN Gong George | |
| **FIELD OF RESEARCH / INTENDED THESIS TITLE** | Liver cancer | |
| **KEYWORDS FOR RESEARCH** | Hepatocellular carcinoma, Transcription factor, FOXP3, Isoform | |

**RESEARCH STUDY:**

Hepatocellular carcinoma (HCC) still is a big health problem worldwide. Understanding its underlying molecular biology can lead to a better diagnosis and treatment of this lethal cancer. Forkhead box P3 (FOXP3) is a transcription factor that acts as both a transcriptional activator and repressor of specific genes. Four main domains, the repressor domain, zinc finger domain, leucine zipper and FKH domain make up FOXP3 with the function of each domain contributing differently to the overall function of FOXP3. Abnormal transcript or protein level of FOXP3 may correlate with either aggressive property or better survival rate in different cancers, indicating that FOXP3 plays a dual role in carcinomas. Besides, mutations in function domain of FOXP3 also affect its role in cancers. Hence, the activities of FOXP3 in cancers will be affected by the changes in its expression level or function domains.

Alternative splicing is another factor that impairs the function domains of FOXP3, which encoding a shorter function domain because lacks one or more exons. Our preliminary results showed that the 10 HCC patient samples and paired peritumour samples mainly expressed FOXP3 full length (FOXP3fl) and FOXP3 exon 3 deleted isoform (FOXP3Δ3). Furthermore, we found that both FOXP3fl and FOXP3Δ3 could inhibit proliferation and decrease colony formation in Hep3B cells. However, the underlying mechanism remains largely unknown. We aim to explore the roles of FOXP3fl and FOXP3Δ3 in HCC and to elucidate the underlying mechanism in this study.

**CONFERENCE TITLE / ABSTRACT / POSTER:**