"A good gene in one time and place may be a bad gene in another time or place"
This quotation proposed by Woolhouse et al (2002) in the context of spread of viruses, may actually be well applied to conclude the riddle of cancer. In one or another way this may explain the main questions; why does cancer arise? How can it be prevented? And, when developed, how can it be treated? No doubt, cancer arises as a result of a sustained long-term complex interplay between the outer and inner environment and an extensive number of genetic and epigenetic factors, throughout all tissues and cells of the body. To distinguish the underlying patterns and mechanisms explaining which gene is good or bad in which time and place would possibly be essential to find the answers.

The work presented in this thesis focuses on the genes S6K1, S6K2 and 4EBP1 which are the main effectors of the intracellular mTOR signalling pathway and thereby secondary targets of the mTOR inhibitor Everolimus, a recently clinically approved drug for treatment of advanced breast cancers. Our results suggest that the gene amplification status, expression levels of the corresponding mRNA and protein of S6K1, S6K2 and 4EBP1 as well as their cellular localisation may be used to predict outcome and the benefit from antioestrogen treatments in breast cancer. These factors are indicated to play separate roles in different subtypes of breast cancer, and specific targeting of S6K1 and S6K2 may be valuable in different tumour subtypes, and in comparison to present day’s mTOR inhibitors, further promote individualised therapies and thereby increase breast cancer survival.

Our hope is, that in the light of other findings, this present study may be able to be a contribution in some way to the comprehensive puzzle regarding which gene is good or bad in which time and place, and its future solution.

Elin Karlsson, 2014