Detection of CCNE1/URI (19q12) amplification by in situ hybridisation is common in high grade and type II endometrial cancer.

Abstract:
One TCGA subgroup of endometrial cancer (EC) is characterised by extensive genomic DNA copy number alterations. CCNE1 located at 19q12 is frequently amplified in EC and a target for anti-cancer therapy. The relevance of URI, also located at 19q12, is unknown. To evaluate the prevalence of 19q12 (CCNE1/URI) in EC, we investigated different histologic types by in situ hybridisation (ISH) and copy number assay. We applied a previously established 19q12 ISH for the detection of CCNE1/URI copy numbers in EC (n = 270) using conventional bright field microscopy. In a subset (n = 21), 19q12 amplification status was validated by OncoScan assay. Manual ISH was controlled by a recently developed computational ISHProfiler algorithm. Associations of 19q12 status with Cyclin E1, URI and p53 expression, and clinico-pathological parameters were tested. Amplification of 19q12 (CCNE1/URI) was found in 10.4% (28/270) and was significantly associated with type II EC (high grade and non-endometrioid; p < 0.0001), advanced FIGO stage (p = 0.001), high Cyclin E1 expression (p = 0.008) and aberrant p53 expression (p = 0.04). 19q12 ISH data were confirmed by OncoScan and computational ISHProfiler techniques. The 19q12 in situ hybridisation is a feasible and robust biomarker assay in molecular
pathology. Amplification of CCNE1/URI predominantly occurred in type II endometrial cancer. Prospective clinical trials are warranted to assess the utility of combined 19q12 amplification and Cyclin E1/URI protein expression analysis for the prediction of therapeutic response to chemotherapy and/or cyclin-dependent kinase inhibitors in patients with endometrial cancer.

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