Characterization of the 11q13.3 amplicon in head and neck squamous cell carcinoma

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Cited literature
CITED LITERATURE


   • This elegant review shows that cancer is a multistep genetic process that progresses over time


   • This interesting review discusses cancer development in the light of evolution and sheds new light on central controversies in cancer research


   • This review covers all aspects of amplification, including detection methods, appearance and clinical implications


- *Anaphase bridges indicate that amplification of 11q13.3 is likely to be caused by breakage–fusion–bridge cycles*


- *The methotrexate model system of amplification is used to show that there are site specific differences in the organisation of amplicons and their propensity to amplify*

- *This paper describes the influence of increased gene copy number on gene expression*


   • Publication of the first tiling whole genome array CGH consisting of more than 32,000 probes
100. van Wieringen WN, van de Wiel MA and Ylstra B. Normalized, Segmented or Called aCGH Data? Cancer Informatics 2007, 3:331–337
103. Venkatraman ES and Olshen AB. A faster circular binary segmentation algorithm for the analysis of array CGH data. Bioinformatics. 2007,
   • Description of a user friendly breakpoint detection algorithm
   • This review covers amplification frequency for the 11q13 region in different tumor types; the most likely candidate genes for driving the amplification are discussed
   • The first report on EMS1


- FISH analysis on microdissected epithelium shows that 11q13.3 amplification is present in the hyperplasia to dysplasia transition preceding HNSCC development


- Using comparative genomic hybridization, this paper defines the common aberrations in head and neck squamous cell carcinoma


   • This report shows that cortactin amplification and lymph node metastasis are independent prognostic factors for reduced survival in HNSCC
• Cortactin overexpression correlates with amplification and might serve as a prognostic marker for invasion and (lymph node) metastasis


- A high resolution FISH approach to determine the amplified region at 11q13.3 in HNSCC


• FADD is proposed as a new driver gene that is amplified and overexpressed. FADD expression correlates with decreased disease specific survival
• Cortactin expression is a better prognostic factor than cyclin D1 and FADD, which are generally co-amplified

Cited literature


273. Trask BJ and Hamlin JL. Early dihydrofolate reductase gene amplification events in CHO cells usually occur on the same chromosome arm as the original locus. *Genes Dev.* 1989, 3:1913–1925


281. Maser RS and DePinho RA. Telomeres and the DNA damage response: why the fox is guarding the henhouse. DNA Repair (Amst) 2004, 3:979–988


288. Woodfine K, Fiegler H, Beare DM, Collins JE, McCann OT, Young BD and others. Replication timing of the human genome. Hum. Mol. Genet. 2004, 13:575. • This paper uses array CGH to determine the replication timing within the human genome; chromosome 11q is a highlighted example


- This review discusses the possible role of low copy repeats in both evolution and disease


- Founding paper linking a cytogenetic BAC clone map to the human genome sequence


- Local control of laryngeal carcinoma is significantly increased when radiotherapy follows induction chemotherapy


- This paper shows that cortactin potentiates migration and influences cell invasion via anoikis resistance


- This paper shows that FADD overexpression is related to cyclin D1 overexpression and that p–FADD expression, correlating with adverse outcome, enhances NF–kappaB activity


150 | Cited literature


• This report shows the oncogenic potential of cyclin D1 in normal keratinocytes


382. Hui R, Campbell DH, Lee CS, McCaul K, Horsfall DJ, Musgrove EA and others. EMS1 amplification can occur independently of CCND1 or INT–2 amplification at 11q13 and may identify different phenotypes in primary breast cancer. *Oncogene* 1997, 15:1617–1623


   • *This paper shows that DNA double strand breaks at a fragile site lead to MET amplification by breakage–fusion–bridge cycles*


408. Bates S, Bonetta L, MacAllan D, Parry D, Holder A, Dickson C and others. CDK6 (PLSTIRE) and CDK4 (PSK–J3) are a distinct subset of the cyclin–dependent kinases that associate with cyclin D1. Oncogene 1994, 9:71–79


422. Kratochwil K, Galceran J, Tontsch S, Roth W and Grosschedl R. FGF4, a direct target of LEF1 and Wnt signaling, can rescue the arrest of tooth organogenesis in Lef1(−/−) mice. Genes Dev. 2002, 16:3173–3185


*This publication underscores the importance of validating the expression of seemingly unimportant genes that are coamplified with known oncogenes*


