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

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Cited literature
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   - *This elegant review shows that cancer is a multistep genetic process that progresses over time*


5. **Hanahan D and Weinberg RA.** The hallmarks of cancer. *Cell* 2000, 100:57–70


8. **Loeb LA, Springgate CF and Battula N.** Errors in DNA replication as a basis of malignant changes. *Cancer Res* 1974, 34:2311–2321


    - *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*


33. Bassing CH and Alt FW. The cellular response to general and programmed DNA double strand breaks. DNA Repair (Amst) 2004, 3:781–796
   • This paper underlines the importance of chromosomal double strand breaks in amplicon formation
   • Double minutes and homogeneously staining chromosome regions are both initiated by chromosome breaks

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43. Futcher AB. Copy number amplification of the 2 micron circle plasmid of *Saccharomyces cerevisiae*. *J.Theor.Biol.* 1986, 119:197–204


**This is the first description of the breakage–fusion–bridge model**


**Chromosomal breakage at fragile sites is found to induce amplification via the breakage–fusion–bridge mechanism**

Cited literature | 133

   - 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*


• This paper shows that the duplication of RIN1 at the border of the amplified 11q13 region fits the breakage–fusion–bridge model


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


   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.


Cited literature


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


• Using fluorescent in situ hybridization, this article accurately describes the relation between 11q13 amplification and deletion of distal 11q


209. Combined analysis of aCGH and expression array reveals that FADD and PPFIA1 are amplified in laryngeal carcinoma


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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


- 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


• Using gene expression arrays this report shows genetic alterations in HNSCC can be identified by mapping altered gene expression to chromosomal position


• Phosphorylated FADD lowers the threshold for G2/M transition and hereby increases proliferation


• The function of FADD is associated with chemosensitivity to paclitaxel in prostate cancer cells


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


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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*


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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.


