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*
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


   • 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


134 Cited literature


   • 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


   • 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


  • 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 an overexpressed in laryngeal carcinoma


• 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


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**Using FISH and immunohistochemistry the authors link increased cyclin D1 protein expression in premalignant tissue to the occurrence of 11q13 amplification in tumors**


**This manuscript defines replication transition regions at the 11q13 region, providing a possible structural cause for chromosomal breakage and subsequent amplification**


**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


148 Cited literature
   - 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-κappaB activity

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


