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Chromosome mis-segregation and cytokinesis failure in trisomic human cells

Joshua M Nicholson, Joana C Macedo, Aaron J Mattingly, Darawalee Wangsa, Jordi Camps, Vera Lima, Ana M Gomes, Sofia Dória, Thomas Ried

Virginia Tech, United States; Universidade do Porto, Portugal; National Institutes of Health, United States

Research Article · May 5, 2015

Cited 46 Views 7,761 Annotations 0

Cite as: eLife 2015;4:e05068 DOI: 10.7554/eLife.05068

Abstract

Cancer cells display aneuploid karyotypes and typically mis-segregate chromosomes at high rates, a phenotype referred to as chromosomal instability (CIN). To test the effects of aneuploidy on chromosome segregation and other mitotic phenotypes we used the colorectal cancer cell line DLD1 (2n = 46) and two variants with trisomy 7 or 13 (DLD1+7 and DLD1+13), as well as euploid and trisomy 13 amniocytes (AF and AF+13). We found that trisomic cells displayed higher rates of chromosome mis-segregation compared to their euploid counterparts. Furthermore, cells with trisomy 13 displayed a distinctive

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Chromosome mis-segregation and cytokinesis failure in trisomic human cells

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Abstract: Cancer cells display aneuploid karyotypes and typically mis-segregate chromosomes at high rates, a phenotype referred to as chromosomal instability (CIN). To test the effects of aneuploidy on chromosome segregation and other mitotic phenotypes we used the colorectal cancer cell line DLD1 (2n = 46) and two variants with trisomy 7 or 13 (DLD1+7 and DLD1+13), as well as euploid and trisomy 13 amniocytes (AF and AF+13). We found that trisomic cells displayed higher rates of chromosome mis-segregation compared to t...

Cited by 34 papers (61 citation statements)

- "...Compared to the diploid parental line, the frequencies of chromosome missegregation and micronuclei formation were significantly elevated in most PTA clones (Figure 2A) but not in the tetraploid line (Figure 2A). In agreement with previous work (Nicholson et al., 2015), the trisomic clones showed similar aberrations, albeit to a lesser extent (Supplemental Figure S2B). Furthermore, we observed an increase of structural aberrations in PTA lines and, consistent with earlier work (Kuznetsova et al., 2015; Passerini et al., 2016), also in trisomic clones (Figure 2B)...."


- "...To independently confirm the observed chromosome instability, RPE +18+18 aneuploid cells were treated with..."
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**Epigenetic silencing of miR-483-3p promotes acquired gefitinib resistance and EMT in EGFR-mutant NSCLC by targeting integrin β3**

**Jinnan Yue, Dacheng Lv, Caiyun Wang, Ling Li, Qingnan Zhao, Hongzhuan Chen, Lu Xu**

Abstract: All lung cancer patients with epidermal growth factor receptor (EGFR) mutation inevitably develop acquired resistance to EGFR tyrosine kinase inhibitors (TKI). In up to 30% of cases, the mechanism underlying acquired resistance remains unknown. MicroRNAs (miRNAs) are a group of small non-coding RNAs commonly dysregulated in human cancers and have been implicated in therapy resistance. The aim of this study was to understand the roles of novel miRNAs in acquired EGFR TKI resistance in EGFR-mutant non-small cell lung cancer (NSCLC). Here, we reported the evidence of miR-483-3p silencing and epithelial-to-mesenchymal transition (EMT) phenotype in both in vitro and in vivo EGFR-mutant NSCLC models with acquired resistance to gefitinib. In these tumor models, forced expression of miR-483-3p efficiently increased sensitivity of gefitinib-resistant lung cancer cells to gefitinib by inhibiting proliferation and promoting apoptosis. Moreover, miR-483-3p reversed EMT and inhibited migration, invasion, and metastasis of gefitinib-resistant lung cancer cells. Mechanistically, miR-483-3p directly targeted integrin β3, and thus repressed downstream FAK/Erk signaling pathway. Furthermore, the silencing of miR-483-3p in gefitinib-resistant lung cancer cells was due to hypermethylation of its own promoter. Taken together, our data identify miR-483-3p as a promising target for combination therapy to overcome acquired EGFR TKI resistance in EGFR-mutant NSCLC.

**44 references detected 15,871 total citations 1 references with a retraction**

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"...These discrepancies would be explained that miR-483-3p has opposite functions depending on its cellular context. It is not unusual of a miRNA that exerts distinct function in cancer depending on tumor type, tumor state and/or genetic background, such as miR-31 [38,39]. In order to understand the roles of miR-483-3p in the tumorigenesis of lung adenocarcinoma, we examined the miR-483-3p level in lung adenocarcinoma compared with matching adjacent non-tumor tissues using multiple miRNA expression profiling data sets from TCGA and GEO database ( Fig...."

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Stevens, M.E., Giuliano, V.E., & Garfield, E. (1964). Can Citation Indexing Be Automated?
Questions?

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