CBX7 gene expression plays a negative role in adipocyte cell growth and differentiation.

 $\frac{\text{Forzati } F^1, \text{Federico } A^1, \text{Pallante } P^1, \text{Colamaio } M^1, \text{Esposito } F^1, \text{Sepe } R^1, \text{Gargiulo } S^2, \text{Luciano } A^3, \text{Arra } C^3, \text{Palma } G^4, \text{Bon } G^5, \text{Bucher } S^6, \text{Falcioni } R^5, \text{Brunetti } A^7, \text{Battista } S^1, \text{Fedele } M^1, \text{Fusco } A^8.$

Author information

Abstract

We have recently generated knockout mice for the Cbx7 gene, coding for a polycomb group protein that is downregulated in human malignant neoplasias. These mice develop liver and lung adenomas and carcinomas, which confirms a tumour suppressor role for CBX7. The CBX7 ability to downregulate CCNE1 expression likely accounts for the phenotype of the Cbx7-null mice. Unexpectedly, Cbx7-knockout mice had a higher fat tissue mass than wild-type, suggesting a role of CBX7 in adipogenesis. Consistently, we demonstrate that Cbx7-null mouse embryonic fibroblasts go towards adipocyte differentiation more efficiently than their wild-type counterparts, and this effect is Cbx7 dose-dependent. Similar results were obtained when Cbx7-null embryonic stem cells were induced to differentiate into adipocytes. Conversely, mouse embryonic fibroblasts and human adipose-derived stem cells overexpressing CBX7 show an opposite behaviour. These findings support a negative role of CBX7 in the control of adipocyte cell growth and differentiation.