MiR-10a and HOXB4 are overexpressed in atypical myeloproliferative neoplasms

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Résumé en anglais	BACKGROUND: Atypical Myeloproliferative Neoplasms (aMPN) share characteristics of MPN and Myelodysplastic Syndromes. Although abnormalities in cytokine signaling are common in MPN, the pathophysiology of atypical MPN still remains elusive. Since deregulation of microRNAs is involved in the biology of various cancers, we studied the miRNome of aMPN patients. METHODS: MiRNome and mutations in epigenetic regulator genes ASXL1, TET2, DNMT3A, EZH2 and IDH1/2 were explored in aMPN patients. Epigenetic regulation of miR-10a and HOXB4 expression was investigated by treating hematopoietic cell lines with 5-aza-2'deoxycytidine, valproic acid and retinoic acid. Functional effects of miR-10a overexpression on cell proliferation, differentiation and self-renewal were studied by transducing CD34 cells with lentiviral vectors encoding the pri-miR-10a precursor. RESULTS: MiR-10a was identified as the most significantly up-regulated microRNA in aMPN. MiR-10a expression correlated with that of HOXB4, sitting in the same genomic locus. The transcription of these two genes was increased by DNA demethylation and histone acetylation, both necessary for optimal expression induction by retinoic acid. Moreover, miR-10a and HOXB4 overexpression seemed associated with DNMT3A mutation in hematological malignancies. However, overexpression of miR-10a had no effect on proliferation, differentiation or self-renewal of normal hematopoietic progenitors. CONCLUSIONS: MiR-10a and HOXB4 are overexpressed in aMPN. This overexpression seems to be the result of abnormalities in epigenetic regulation mechanisms. Our data suggest that miR-10a could represent a simple marker of transcription at this genomic locus including HOXB4, widely recognized as involved in stem cell expansion.
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