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GATA2 directly represses cardiac fates to promote hematopoietic specification of human mesoderm

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In vertebrates, GATA2 is a master regulator of hematopoiesis, repeatedly used throughout embryo development and the adult life. Although it is well established that GATA2 is essential for the onset of mouse hematopoiesis, its role during early human hematopoietic development remains elusive. By combining time-controlled overexpression of GATA2 with genetic knockout experiments, we found that GATA2, at the mesoderm specification stage, promotes the generation of hemogenic progenitors and their further differentiation to hematopoietic progenitor cells, while negatively regulating cardiac differentiation. Surprisingly, genome-wide transcriptional and chromatin immune-precipitation analysis showed that GATA2 bound preferentially to regulatory regions, and repressed expression, of cardiac development-related genes. In contrast, genes important for hematopoietic differentiation were up-regulated by GATA2 in a mostly indirect manner. Collectively, our data reveal a previously unsuspected role of GATA2 as a direct repressor of cardiac fates, and highlight the importance of coordinating the specification and repression of alternative cell fates.

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