

ARTICLE

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# Identification of four novel associations for B-cell acute lymphoblastic leukaemia risk

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There is increasing evidence for a strong inherited genetic basis of susceptibility to acute lymphoblastic leukaemia (ALL) in children. To identify new risk variants for B-cell ALL (B-ALL) we conducted a meta-analysis with four GWAS (genome-wide association studies), totalling 5321 cases and 16,666 controls of European descent. We herein describe novel risk loci for B-ALL at 9q21.31 (*rs76925697*,  $P = 2.11 \times 10^{-8}$ ), for high-hyperdiploid ALL at 5q31.1 (*rs886285*,  $P = 1.56 \times 10^{-8}$ ) and 6p21.31 (*rs210143* in *BAK1*,  $P = 2.21 \times 10^{-8}$ ), and *ETV6-RUNX1* ALL at 17q21.32 (*rs10853104* in *IGF2BP1*,  $P = 1.82 \times 10^{-8}$ ). Particularly notable are the pleiotropic effects of the *BAK1* variant on multiple haematological malignancies and specific effects of *IGF2BP1* on *ETV6-RUNX1* ALL evidenced by both germline and somatic genomic analyses. Integration of GWAS signals with transcriptomic/epigenomic profiling and 3D chromatin interaction data for these leukaemia risk loci suggests deregulation of B-cell development and the cell cycle as central mechanisms governing genetic susceptibility to ALL.

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