

Gene Section

Review

GATA6 (GATA binding protein 6)

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Identity

Other names: GATA-6

HGNC (Hugo): GATA6

Location: 18q11.2

Local order: GATA-6 is flanked in the direction of the centromere by:

LOC100128893, hypothetical protein LOC100128893 - RNU7-17P, RNA U7 small nuclear 17 pseudogene - LOC100287318 - RPL34P32, ribosomal protein L34 pseudogene 32 - MIB1, mindbomb homolog 1 - MIR1-2 - MIR133A1.

GATA6 is flanked in the direction of the telomere by: CTAGE1, cutaneous T-cell lymphoma-associated antigen 1 - RPS4P18, ribosomal protein S4X pseudogene 18 - RBBP8, retinoblastoma binding protein 8 - CABLES1, Cdk5 and Abl enzyme substrate 1 - C18orf45, chromosome 18 open reading frame 45 - RIOK3, RIO kinase 3.

Note: GATA6 is one of a family of 6 related GATA binding proteins. All six proteins possess zinc finger-type DNA binding domains and act as transcription factors.

DNA/RNA

Description

Genomic DNA encoding GATA6 encompasses 33088 bp on the long arm of chromosome 18. The gene is encoded on the plus (forward) strand.

Transcription

The pre-mRNA comprises 7 exons, one of which is non-coding, and 6 introns. The mouse and human GATA6 genes contain two alternative non-coding upstream exons, transcribed from two distinct promoters (Brewer et al., 1999), similar to other GATA

family members. The non-coding exons possess regulatory capability and may act to promote transcription. Two isoforms of GATA6 are expressed from two distinct open reading frames and distinct initiator Met codons as a result of leaky ribosome scanning.

There are no apparent differences in the amounts or sites of expression of the two transcripts that result from initiation at different Met codons.

Protein

Description

The GATA6 protein products that result from different initiation codons comprise a long isoform of 595 aa (64 kDa) and a short isoform of 449 aa (52 kDa).

Both isoforms possess an N-terminal transactivation domain and two zinc finger domains, all of which are essential for activity (Takeda et al., 2004). The two isoforms display different transactivation potential on GATA6-dependent promoters with long GATA6 showing higher activity than short GATA6.

Expression

GATA6 is expressed predominantly in tissues of mesodermal and endodermal origin. In early development, high levels are detected in the precardiac mesoderm, embryonic heart tube and primitive gut. As development proceeds GATA6 expression is observed in vascular smooth muscle cells, the developing airways, urogenital ridge and bladder (Morrissey et al., 1996).

Localisation

Nuclear.

Function

GATA6 binds to a 5'-(T/A)GATA(A/G)-3' consensus sequence in the promoters of target genes to regulate

their transcription. GATA6 is post-translationally modified by MEK/Erk-dependent phosphorylation at Ser120 (S266 in long GATA6). Ser120Ala mutation abolished GATA6 DNA-binding activity and GATA6-mediated Nox1 promoter activation, and also suppressed growth of CaCo-2 colon carcinoma cells (Adachi et al., 2008). GATA6 activity is also regulated through interaction with members of the Friend of GATA (FOG) family of proteins. Two FOG proteins have been identified in mice and humans, FOG-1 and FOG-2, and their interaction with GATA factors can promote or inhibit GATA activity, depending on context (Cantor and Orkin, 2005).

GATA6 is essential for normal development, since genetic knockout in mice leads to embryonic lethality as early as E6.5. The underlying defect in GATA6-null mice was determined to be a failure of endoderm differentiation resulting in attenuated expression of GATA6 target genes including GATA4, HNF3beta and HNF4 (Morrisey et al., 1998). GATA6 was shown subsequently to be essential for early extraembryonic development (Koutsourakis et al., 1999). Partial rescue of GATA6-deficient embryos by tetraploid embryo complementation demonstrated additional functions for GATA6 in liver development. The early lethality in GATA6-null embryos could be overcome by providing wild type extraembryonic endoderm and allowed embryos to proceed through gastrulation. However, although hepatic specification occurred normally in rescued GATA6^{-/-} embryos, normal differentiation did not occur and hepatic development arrested at E10.5 (Zhao et al., 2005).

Early development of other organ systems was unaffected in rescued GATA6-null embryos, including the heart and vasculature. Interestingly, conditional deletion of GATA6 using SM22alpha promoter-driven Cre recombinase led to perinatal lethality as a result of cardiovascular defects emerging later in embryonic development. In that analysis, the underlying mechanism was determined to be diminished expression of the vascular and neuronal guidance molecule semaphorin 3C, a direct target of GATA6 (Lepore et al., 2006). Consistent with GATA6-dependent regulation of Sema3C in mice, mutations in GATA6 were found to cause cardiac outflow tract defects in humans by dysregulating semaphorin-dependent signaling (Kodo et al., 2009). In general, GATA6 does not act alone in regulating developmental processes, but rather achieves its effects through physical and functional interaction with other transcription factors and signaling molecules, including FOG factors, GATA4 (Xin et al., 2006; Zhao et al., 2008), Tbx5 (Maitra et al., 2009), members of the Nkx2 family (Peterkin et al., 2003) and Wnt family proteins. The complexity of these interactions is exemplified by the functional cooperation of Wnt2 and GATA6 in regulating heart development. In this case, GATA6 not only regulates Wnt2 transcription during

heart development through direct binding to the Wnt2 promoter (Alexandrovich et al., 2006), but is itself regulated by a Wnt2-dependent mechanism, since GATA6 expression is markedly reduced in Wnt2-null mice (Tian et al., 2010).

GATA6 has also been implicated in regulating development of other organs including the lung and pancreas. In the lung, GATA6 has been shown to regulate specification, differentiation and maturation of the pulmonary epithelium as well as branching morphogenesis (Keijzer et al., 2001; Yang et al., 2002; Liu et al., 2002; Zhang et al., 2008). Inhibition of GATA6 at E6.0 prevented alveolar maturation and also diminished expression of surfactant proteins required for normal pulmonary function. In the pancreas, GATA6 is co-expressed with GATA4 in the epithelium early in development, but as development progresses is expressed only in endocrine cells. Ablation of GATA6 function using a dominant inhibitory engrailed fusion protein strategy led to a reduction or complete loss of pancreatic tissue, consistent with a critical role for GATA6 in pancreatic development (Decker et al., 2006).

GATA6 has also been implicated in postnatal maintenance of the differentiated phenotype in various tissues including bladder smooth muscle (Kanematsu, 2007), gut mucosa (Fang, 2006) and airway epithelium (Zhang, 2008).

Homology

GATA6 shares homology with the other 5 GATA factors, all of which are evolutionarily conserved across multiple species. All 6 GATA factors possess two zinc fingers of the Cys-X₂-Cys-X₁₇-Cys-X₂-Cys configuration. The C-terminal zinc finger mediates high affinity DNA binding and the N-terminal zinc finger stabilizes the interaction with DNA.

Mutations

Germinal

None known.

Somatic

Two mutations in GATA6 were identified in patients with persistent truncus arteriosus, as follows (Kodo et al., 2009).

GATA6-E486del resulted in conversion of P489 to a stop codon, disruption of the nuclear localization signal and truncation of the C-terminus by 100 aa. The encoded protein showed abnormal nuclear localization, no transcriptional activity against atrial natriuretic factor and WNT2 promoters and was dominant negative.

GATA6-N466H contained a point mutation in the C-terminal zinc finger domain. Despite normal nuclear localization, the encoded protein had no transcriptional activity against atrial natriuretic factor and WNT2 promoters.

Implicated in

Pancreatic cancer, pancreatobiliary cancer

Disease

Genomic profiling of pancreatic and bile duct cancers revealed focal amplification at 18q11.2 that encoded GATA6. Amplification led to overexpression of GATA6 at both mRNA and protein levels in nearly 50% of tumor samples, whereas no normal pancreatic tissues showed overexpression (Kwei et al., 2008; Fu et al., 2008). Consistent with an oncogenic role for GATA6 in pancreatic cancer, RNAi-mediated silencing in pancreatic cancer cell lines in which GATA6 was amplified decreased cell cycle transit, growth and clonogenic ability (Kwei et al., 2008). Conversely forced expression of GATA6 in a pancreatic cancer cell line stimulated anchorage-independent growth and proliferation (Fu et al., 2008).

Prognosis

GATA6 silencing by RNAi in pancreatic cancer cells in vitro reduced proliferation, cell cycle transit and colony formation, whereas forced overexpression promoted colony formation in soft agar and enhanced proliferation, consistent with a role for GATA6 in driving the tumorigenic phenotype.

Cytogenetics

Focal amplification of the locus encoding GATA6 at 18q11.2 was identified by array-based genomic profiling and validated by fluorescence in situ hybridization, quantitative PCR, immunohistochemical analysis and immunoblotting.

Ovarian cancer

Disease

Consistent with their expression in the mouse ovary, GATA6, GATA4 and FOG2 are also expressed in human ovary and in tumors derived from granulosa and thecal cells (Laitinen, 2000). Under normal conditions, both GATA4 and GATA6 are robustly expressed in ovarian surface epithelial cells. However, in a majority of ovarian carcinomas, GATA6 is lost or mislocalized to the cytoplasm (Capo-chichi et al., 2003; McEachin et al., 2008), leading to irreversible epithelial dedifferentiation (Capo-chichi et al., 2003). Expression of GATA4 and GATA6 was shown to correlate with specific histological subtypes of ovarian cancer. In particular, although expression of both factors was lost in the over 80% of endometrioid, clear cell and serous tumors, GATA4 and GATA6 expression persisted in mucinous carcinomas (Cai et al., 2009). Loss of GATA factor expression preceded neoplastic transformation, consistent with an important role for these proteins in tumor development. The mechanism underlying loss of GATA6 and GATA4 expression in ovarian cancer cell lines was demonstrated to be histone deacetylation at the GATA factor promoter regions. Inhibition of

histone deacetylase activity with trichostatin A restored GATA6 and GATA4 expression in cell lines (Caslini et al., 2006).

Prognosis

Loss of GATA6 expression precedes neoplastic transformation in ovarian surface epithelia (Cai et al., 2009) and is correlated with loss of markers of differentiated epithelia (Capo-chichi et al., 2003). Although a majority of ovarian carcinomas retained GATA4 expression, most had either aberrantly localized or absent GATA6 expression. Cytoplasmic expression of GATA6 showed a correlation with overall survival, but this association did not reach statistical significance (McEachin, 2008).

Gastrointestinal cancer

Disease

Expression of GATA6 has been linked, both positively and negatively, to development of gastrointestinal tract tumors.

Prognosis

GATA6 expression was found to be decreased in colon carcinoma compared to normal intestinal tissue or benign intestinal lesions (Haveri et al., 2008), which showed robust expression, especially in cells with proliferative capacity. Conversely GATA6 was reported to be overexpressed in human colon cancer cells, where it contributes to silencing of 15-lipoxygenase-1 (Shureiqi et al., 2007). The biological significance of this discrepancy in GATA6 expression between colon cancer cells and tissues has not been determined. Expression profiling of Barrett's esophagus and adenocarcinoma to identify genes whose expression correlated with disease progression revealed changes in GATA6 expression among other genes, consistent with upregulation of GATA6 in the transition from normal esophageal epithelium to carcinoma (Kimchi et al., 2005).

Lung cancer

Disease

Despite substantial evidence linking GATA6 to pulmonary development, only one study has investigated the potential role of GATA6 in lung cancer. Specifically, expression of GATA6 was evaluated in malignant mesothelioma and pleural metastases of lung adenocarcinomas and staining patterns correlated with biological and clinical outcomes. Nuclear immunoreactivity for GATA-6 was stronger and more frequent in malignant mesothelioma than in metastatic lung adenocarcinoma (Lindholm et al., 2009). However, no relationship was found between GATA6 expression and growth or apoptotic endpoints.

Prognosis

Prognosis was better in malignant mesothelioma patients whose tumors expressed GATA-6 compared to

those whose tumors had no GATA-6 expression, and the relationship was highly statistically significant.

Adrenocortical cancer

Disease

GATA6 has been implicated in development of the normal adrenal gland. GATA6 mRNA, although expressed in the normal adrenal cortex was found to be absent from experimental mouse adrenocortical tumors, whereas GATA-4 showed the opposite pattern (Kiiveri, 1999; Rahman et al., 2001). GATA-6 expression was also decreased in human adrenocortical carcinomas compared to normal adrenal tissue and adenomas (Kiiveri et al., 2004). The physiologic relevance of altered GATA6 expression in adrenocortical tumorigenesis has not yet been elucidated. However, based on expression of the CDK inhibitor p21 and proliferation marker Ki67, GATA-6 expression in adrenocortical tumors does not appear to be linked to regulation of cell proliferation.

Prognosis

The prognostic significance of GATA-6 in adrenocortical tumors has not been determined.

Germ cell tumors

Disease

Germ cell tumors comprise a heterogeneous group of lesions, including teratomas, yolk sac tumors and embryonal carcinoma. Using in situ hybridization and immunohistochemical staining, GATA6 was evaluated in pediatric germ cell tumors and was found to be expressed in a majority of yolk sac tumors. GATA6 expression was also evident in distinct cell types comprising teratomas, including gut and airway epithelia (Siltanen et al., 2003), but was variable in carcinoma in situ of the testis and absent from embryonal carcinomas and choriocarcinomas (Salonen et al., 2010).

Prognosis

The prognostic role of GATA6 in germ cell tumors is unknown.

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