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Gene Section
Review

NME1 (NME/NM23 nucleoside diphosphate kinase 1)

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Identity

Other names: AWD, GAAD, NB, NBS, NDKA, NDPK-A, NDPKA, NM23, NM23-H1

HGNC (Hugo): NME1

Location: 17q21.33

DNA/RNA

Note

The NME1 gene was identified in 1988 as the first metastasis suppressor, due to its reduced expression in metastatic murine melanoma cell line (Steeg et al., 1988; reviewed in: Lee et al., 2009). The human gene, also named NM23-H1, was shown to be homologous to the Drosophila AWD gene, involved in larvae development, and to exhibit an expression inversely correlated to the metastatic spread in primary breast tumors (Rosengard et al., 1989). It is the first described member of a family of ten genes in human (Desvignes et al., 2009; reviewed in: Boissan et al., 2009).

Description

The gene is composed of 6 exons of which five are coding exons. Exon 2 skipping results in a shorter transcript.

Transcription

Two transcripts have been reported for this gene (Rosengard et al., 1989; Ni et al., 2003). A read-through variant, resulting of the co-transcription of NME1 and the neighboring NME2, generates a transcript, which encodes a fusion protein sharing identity with each individual gene products (Valentijn et al., 2006).

Pseudogene

A pseudogene of NME1 (NME1P1) is located on chromosome 13q12.11 (NC_000013.10, Gene ID: 100874501).

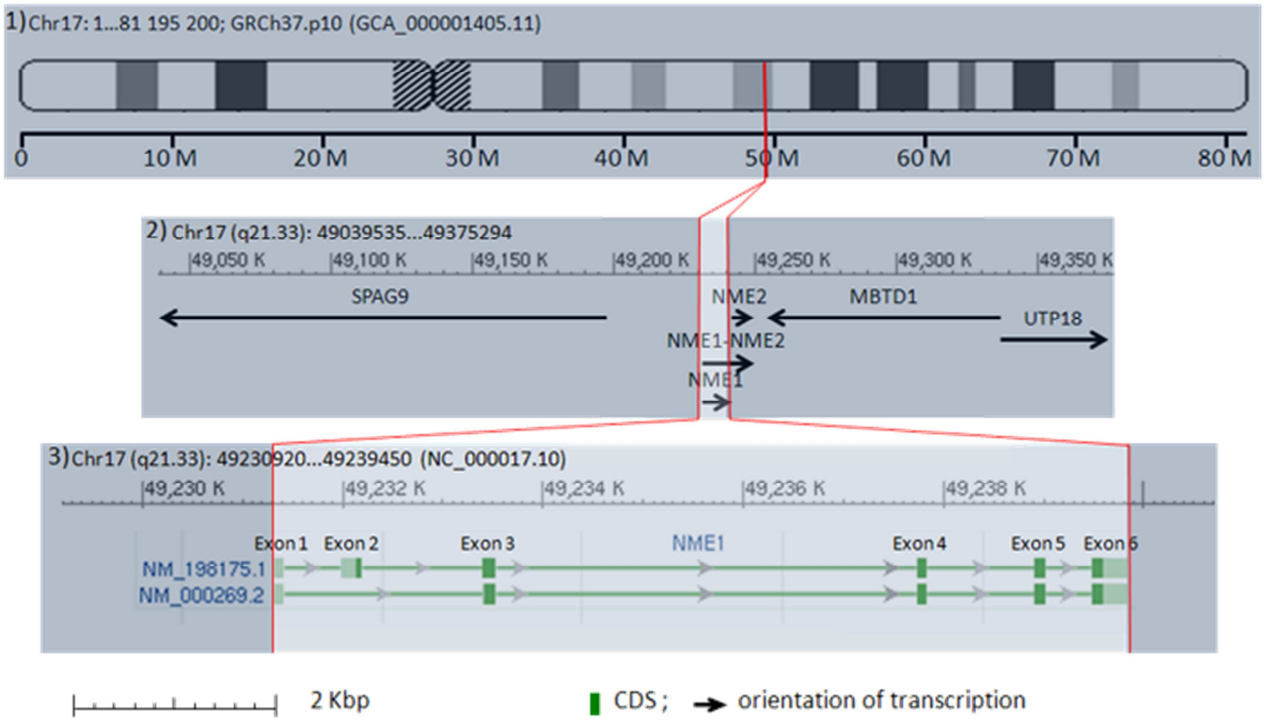
Protein

Note

The NME1 gene encodes a nucleoside diphosphate kinase (NDPK) (Wallet et al., 1990). The two most abundantly expressed and the most studied genes are NM23-H1 and NM23-H2 (NME1 and NME2). They encode, respectively the A and B subunits of NDPK, 88% identical in their amino acid sequences (Gilles et al., 1991).

NME1 was demonstrated to be a metastasis suppressor 1) by its enforced expression in various metastatic cell lines which decreased motility and invasion (Lee et al., 2009), 2) in double transgenic mice invalidated for Nme1 and prone to develop hepatocarcinoma in which lack of Nme1 increased incidence of pulmonary metastases (Boissan et al., 2005) and 3) by its invalidation (SiRNA) in non-aggressive hepatoma and colon cancer cell lines which induced a "metastatic phenotype" (Boissan et al., 2010). The role of the homologous gene, NME2, in cancer progression is less documented (reviewed in: Thakur et al., 2011).

The mechanisms involved in the metastatic potential control are largely unknown. They could involve several known NM23 enzymatic activities (nucleoside diphosphate kinase, histidine kinase, and 3'-5' exonuclease), protein-protein interactions and downstream gene regulation properties (for reviews: Marino et al., 2011; Marino et al., 2012).



The NME1 gene viewed at three different levels of detail (highlighted between two red vertical boundary lines). 1) Overview within chromosome 17. 2) Partial regional view within chromosome 17q21.3. 3) Detailed view within chromosome 17q21.33 showing two of the transcription variants of NME1, which include NME1 (NM_000269.2) and NME1B (NM_198175.1). Abbreviations: M and K: mega- and kilobase pairs from pter; Kbp: kilobase pairs; Chr: chromosome; CDS: coding sequence.

A Granzyme A activated DNAse (GAAD) activity involved in caspase-independent apoptosis was also reported for this gene (reviewed in: Lieberman, 2010).

Description

The main transcript encodes a protein of 152 amino acids with a Mr of 17 kDa (Rosengard et al., 1989). The longer transcript encodes a protein of 177 amino acids (Ni et al., 2003).

Expression

Ubiquitous.

Localisation

Mostly cytoplasmic but also reported in nucleus (Bosnar et al., 2009).

Function

Nucleoside diphosphate kinase activity responsible for the synthesis of most cellular (oxy- and deoxy-) nucleoside triphosphates (Parks and Agarwal, 1973).
Other functions have been proposed, histidine protein kinase activity, 3'-5' exonuclease activity (for review: Marino et al., 2011) and Granzyme A activated DNAse (GAAD) activity (Lieberman, 2010).

Homology

The human NME1 gene is conserved in amniota with a high percentage of identity in the pairwise alignment of protein vs. chimpanzee (99%), rat (95%), mouse (94%), dog (93%), cow (93%), opossum (89%) and chicken (84%). The paralogs of human NME1 include NME2, NME3, NME4, NME5, NME6, NME7, NME8 (TXNDC3), NME9 (TXL2) and RP2 (NME10) (Desvignes et al., 2009; Desvignes et al., 2010).
These genes encode one (NME1 to NME6 and NME9) or several (NME7 and NME8) conserved NDPK domains, either full length or truncated. The NDPK domain occurs individually or associated with extra-domains (NME5, NME7, NME8, NME9 and RP2) (Boissan et al., 2009).

Mutations

Note
A S120G mutation was reported in aggressive neuroblastoma (Chang et al., 1994).

Implicated in

Cancers

Note
NME1 plays a crucial role in cancer metastasis. Numerous clinical studies reported an inverse association between NME1 expression and the metastatic potential for human solid tumors of epithelial origin such as breast, liver, colorectal, ovarian and lung carcinomas and for melanoma (for review: Marino et al., 2012).
A dual expression was reported with an increased expression of NME1 and often NME2 in the primary

tumor as compared to the adjacent non-tumoral tissue (Lacombe et al., 1991; Flørenes et al., 1992; Martinez et al., 1995) and with a decreased expression of NME1 in the primary tumor correlated with metastatic spread. If, for liver, breast and lung carcinomas and for melanoma, the vast majority of studies reported an inverse correlation with metastasis and/or poor overall survival, this is less marked in colorectal, gastric and ovarian carcinomas for which more discrepant results are found. The conflicting data reported in the literature, might be due to the presence of the two closely related isoforms (NME1 and NME2) which are most often not discriminated by antibodies and probes, the heterogeneous expression in the primary tumors and the criteria to evaluate and grade NME1 expression in the clinical samples.
Remarkably, enforced NME1 expression induced by transfection in epithelial cancer derived cell lines and melanoma reverses their motility and invasive potential. This was observed with cell lines derived from melanoma (Leone et al., 1991) and from breast (Leone et al., 1993a), colon (Suzuki et al., 2004), lung (Nie et al., 2008), liver (She et al., 2010), ovarian (Li et al., 2006), prostate (Andolfo et al., 2011) and oral (Wang YF et al., 2008) carcinomas.
In other types of cancers such as neuroblastoma (Leone et al., 1993b; Garcia et al., 2012), hematopoietic malignancies (Yokoyama et al., 1998) and osteosarcoma (Liao et al., 2000), a high tumoral expression was noted which was most often correlated with poor clinical outcome.

Breast cancer

Note

Most clinical studies reported an inverse correlation between NME1 expression in breast primary tumors and the metastatic dissemination and/or shorter patient survival (Bevilacqua et al., 1989; Hennessy et al., 1991; Tokunaga et al., 1993; Toulas et al., 1996; Charpin et al., 1997; Caligo et al., 1997; Yamaguchi et al., 1998; Yoshida et al., 1998; Bertheau et al., 1998; Heimann et al., 2000; Mao et al., 2001; Terasaki-Fukuzawa et al., 2002; Niu et al., 2002; Ding and Wu, 2004; Bal et al., 2008; Dong et al., 2011). In some cases, an increased NME1 expression in tumoral cells as compared to normal tissue was observed (Lacombe et al., 1991; Goodall et al., 1994; Caligo et al., 1997). However, some studies showed that NME1 expression was of no prognostic relevance (Russo et al., 1996; Russell et al., 1997; Belev et al., 2002; Göhring et al., 2002) or even positively associated with a poor overall survival (Galani et al., 2002). Yoshida et al. (Yoshida et al., 1998) noted a low NME1 expression at the invasive front. Forced expression of NME1 by transfection in breast cancer cell lines reduced cell motility in vitro and metastasis formation in xenograft mouse models (Kantor et al., 1993; Leone et al., 1993a; Russell et al., 1998; Bhujwalla et al., 1999).

Hepatobiliary carcinoma

Note

Hepatobiliary carcinoma are among of the most aggressive cancers with a five year survival of less than 15%. Most studies reported an inverse association between NME1 expression and metastatic dissemination in hepatocellular carcinoma (reviews: Boissan and Lacombe, 2006 and An et al., 2010) and gallbladder carcinoma (Lee and Pirdas-Zivcic, 1994; Yang et al., 2008) but this is not always the case (Lin et al., 1998). Boissan et al. (2010) noted a low NME1 expression at the invasive front of hepatocellular carcinoma.
Also, highly aggressive hepatocarcinoma cell lines exhibited a decrease level of NME1 as compared to less aggressive counterpart (Lin et al., 1995; Qin et al., 2007).
NME1 overexpression, induced by transfection in the H7721 hepatocarcinoma cell line, inhibited cell migration and invasion (She et al., 2010). Conversely, NME1 silencing in non-aggressive hepatoma cell lines, HepG2 and PLC/PRF5 (Boissan et al., 2010), induced several parameters of a "metastatic phenotype" (loss of cell-cell contacts, increased motility and invasion).

Lung carcinoma

Note

In lung carcinoma, an inverse association with metastatic spread was reported by several studies (Huwer et al., 1994; Lai et al., 1996; Kawakubo et al., 1997; Ohta et al., 2001; Graham et al., 2002; Katakura et al., 2002; Goncharuk et al., 2004; Tang et al., 2005; Chen et al., 2005; Liu et al., 2011) but was not found by others (Higashiyama et al., 1992; Gazzeri et al., 1996; MacKinnon et al., 1997; Tomita et al., 1999; Wang et al., 2010).
Some studies noted an increased level of NME1 in tumors as compared to non-tumoral tissue (Huwer et al., 1994; Gazzeri et al., 1996). Few studies reported a positive association between NME1 expression and metastatic dissemination to lymph nodes (Tomita et al., 2001). NME1 determination in bronchial lavages was proposed as a diagnostic tool for lung cancer (Huwer et al., 1997).
In lung carcinoma cell lines, increased NME1 expression (Nie et al., 2008) and NME1 silencing (Zhao et al., 2013) decreased invasion and increased TGFβ-induced epithelial mesenchymal transition, respectively.

Melanoma

Note

An inverse correlation between NME1 mRNA levels and malignant potential of melanoma was reported in several studies (Flørenes et al., 1992; Xerri et al., 1994).

This correlation was also found by immunohistochemical studies (Lee et al., 1996; Sarris et al., 2004; Ferrari et al., 2007), one including more than 100 patients (McDermott et al., 2000). However, other studies, including fewer cases, concluded to a lack of correlation (Easty et al., 1996; Saitoh et al., 1996; van den Oord et al., 1997).
In uveal melanoma, high NME1 expression was related to better survival rate (Bakalian et al., 2007) and, inversely, with prognostic factors of metastasis (Greco et al., 1997). Moreover, the high expression of NME1 mRNA and NME1 protein in derived-cell lines from human uveal melanomas is closely correlated with a reduced metastatic behavior in experimental animals (Ma et al., 1996). In accordance with its role as a metastasis suppressor, NME1 deficiency promotes metastasis in a UV radiation-induced mouse model of human melanoma (Jarrett et al., 2013). Enforced expression of NME1 in aggressive melanoma cell lines inhibited their metastatic potential (Leone et al., 1991; Zabrenetzky et al., 1994).

Colorectal cancer

Note

Concerning colorectal cancers (CRC), data about NME1 expression are highly conflicting. An enhanced NME1 expression was noted in CRC as compared to normal adjacent tissues at the mRNA (Ayhan et al., 1993; Myeroff and Markowitz, 1993; Yamaguchi et al., 1993; Zeng et al., 1994; Martinez et al., 1995; Okuno et al., 2001) as well as at the protein (Haut et al., 1991; Ayhan et al., 1993; Martinez et al., 1995; Sarris and Lee, 2001; Kapitanovic et al., 2004; Lin et al., 2011; Alvarez-Chaver et al., 2011) levels. Several studies reported 1) an inverse correlation with tumor stages (Cheah et al., 1998) and metastatic spread (Ayhan et al., 1993; Yamaguchi et al., 1993; Royds et al., 1994a; Martinez et al., 1995; Tannapfel et al., 1997; Dursun et al., 2002; Bertucci et al., 2004; Su and Li, 2004; Kapitanovic et al., 2004; Liu et al., 2005; Elagoz et al., 2006; Chen et al., 2007; Lin et al., 2011) or 2) no correlation (Haut et al., 1991; Myeroff and Markowitz, 1993; Ichikawa, 1994; Heide et al., 1994; Lindmark, 1996; Heys et al., 1998; Tabuchi et al., 1999; Lee et al., 2001; Dusonchet et al., 2003; Soliani et al., 2004; Qian et al., 2012) and 3) even a positive correlation with aggressiveness (Berney et al., 1999; Brenner et al., 2003). Boissan et al. (2010) observed a low NME1 expression at the invasive front of CRC.
In contrast to other cancers, for which NME1 LOH was rarely reported, several studies reported LOH for NME1 gene in CRC (Lamb et al., 1996; Lenehan et al., 1997; Sugai et al., 2000; Tsai et al., 2002; Kapitanovic et al., 2004). Allelic loss was correlated with advanced stage of the disease (Kapitanovic et al., 2004), shorter disease free and overall patient survival (Campo et al., 1994) and with formation of distant metastases (Wang et al., 1993; Cohn et al., 1997; Yang et al., 2008) or not correlated (Lamb et al., 1996). In addition, few studies reported no allellic loss of NME1 in CRC (Heide et al., 1994; Cawkwell et al., 1994).
Enhanced expression of NME1 by transfection in colon cancer cell lines reduced their migratory ability and their metastatic potential to the liver (Suzuki et al., 2004).
Conversely, silencing NME1 expression by SiRNA in the colon cancer cell line, HCT8/S11, induced a "metastatic phenotype" (Boissan et al., 2010).

Gastric carcinoma

Note

In gastric carcinoma, many studies reported a negative correlation between NME1 expression in the primary tumors and advanced stage of the disease (Yang et al., 2008), metastatic dissemination (Kodera et al., 1994; Kim et al., 1995; Ura et al., 1996; Wang et al., 1999; Terada et al., 2002; Li et al., 2003; Chen et al., 2004; Liu et al., 2005; Guan-Zhen et al., 2007) and overall poor survival (Ura et al., 1996; Terada et al., 2002; Li et al., 2003).
However, in some studies, NME1 expression was not correlated (Müller et al., 1998; Yoo et al., 1999; Lee et al., 2003; Mönig et al., 2007; Wang LB et al., 2008) or positively correlated (Wang et al., 1998; Nesi et al., 2001) with metastasis and poor survival.

Ovarian carcinoma

Note

In ovarian cancers, conflicting results were obtained. If several studies observed an inverse correlation with metastatic dissemination and/or poor patient survival (Mandai et al., 1994; Kapitanovic et al., 1995; Okubo et al., 1995; Scambia et al., 1996; Ferrandina et al., 1996; Qian et al., 1997; Galani et al., 2002; Hua et al., 2008), other studies did not observed any correlation (Baekelandt et al., 1999) and others reported a positive correlation with advanced stages, lymph node invasion or shorter overall survival (Leary et al., 1995; Harlozinska et al., 1996; Schneider et al., 2000; Arik and Kulacoglu, 2011).
NME1 overexpression induced by transfection inhibited the metastatic potential of aggressive ovarian cancer cells (Li et al., 2006).

Neuroblastoma

Note

In neuroblastoma, all studies reported an enhanced expression of NME1 in tumors, correlated, when evaluated, with a poor prognosis (Hailat et al., 1991; Leone et al., 1993b; Chang et al., 1994; Hiyama et al., 2004; Garcia et al., 2012).
In accordance with a metastatic promoting role, enhanced NME1 expression in the NB69 neuroblastoma cell line increased cell migration and metastatic potential (Almgren et al., 2004). A S120G mutation of Nme1 was found in aggressive cases (Chang et al., 1994).

Leukemia

Note

In several hematopoietic malignancies, NME1 was overexpressed and this high expression was positively correlated with poor prognosis. This was the case for acute myeloid leukemia (Yokoyama et al., 1996; Okabe-Kado et al., 1998; Wakimoto et al., 1998; Yokoyama et al., 1998; Cui et al., 2004), for Hodgkin and non Hodgkin lymphoma (Aryee et al., 1996; Lee et al., 2006), for acute lymphoid leukemia (Koomägi et al., 1998; Ning et al., 2009), for peripheral T-cell lymphoma (Niitsu et al., 2003a; Huang et al., 2006; Niitsu et al., 2011) and for diffuse large B-cell lymphoma (Niitsu et al., 2004). Interestingly, the poor prognosis can be evaluated by ELISA test of the NME1 level in patient sera for Hodgkin lymphoma (Niitsu et al., 2008) and non-Hodgkin lymphoma (Niitsu et al., 2001a; Niitsu et al., 2001b), for acute myeloid leukemia (Niitsu et al., 2000), for myelodysplastic syndrome (Ito et al., 2002), for diffuse large B-cell lymphoma (Niitsu et al., 2004; Niitsu et al., 2006) and for extranodal NK/T-cell lymphoma (Niitsu et al., 2003b). However, Bircan et al. (Bircan et al., 2008) reported that the increased NME1 expression was of no prognostic value in a series of Hodgkin and non Hodgkin lymphoma. Pointing to a role of extracellular NME1, recombinant NME1 protein was shown to promote the survival of acute myeloid leukemia blast cells (Okabe-Kado et al., 2009a; Lilly et al., 2011) but to decrease the survival of primary cultured normal peripheral blood mononuclear cells (Okabe-Kado et al., 2009b).

Other cancers

Note

NME1 has been evaluated as a prognostic marker in other types of cancer with various results, against pointing to a potential interest of NME1 as a metastasis suppressor specifically in carcinoma.

Controversial data have been obtained for prostate cancers (reviewed in: Myers and Grizzle, 1997). Several studies reported NME1 overexpression in tumor cells as compared to normal prostatic tissue (Myers et al., 1996; Jensen et al., 1996; Igawa et al., 1996; Luo et al., 2001; Prowatke et al., 2007). NME1 expression was shown to be inversely correlated with metastatic dissemination (Konishi et al., 1993; Stravodimos et al., 2000; Ding et al., 2006), of no prognostic interest (Borchers et al., 1996; Prowatke et al., 2007) or even related to advanced stage of the disease (Igawa et al., 1994) and poor prognosis (Andolfo et al., 2011). Interestingly, NME1 overexpression induced by transfection in PC3 prostate cancer cells, inhibited cell motility (Andolfo et al., 2011).

Few reports exist for pancreas cancers. Friess et al. (Friess et al., 2001) reported that early pancreatic cancer stages exhibited higher NME1 immunostaining than advanced tumor stages, while Nakamori et al. (Nakamori et al., 1993) observed that NME1 expression was positively associated with lymph node metastasis.

In thyroid cancers, several studies reported an increased expression in primary tumors (Zou et al., 1993; Ferenc et al., 2004; Al-Maghrabi and Asa, 2005) and a reduced expression in the metastatic lymph nodes (Arai et al., 1995; Shirahige et al., 1997), most often not correlated with patient survival (Luo et al., 1993; Farley et al., 1993; Zou et al., 1993; Royds et al., 1994b; Okubo et al., 1995; Al-Maghrabi and Asa, 2005; Tabriz et al., 2009). Zafon et al. (2001) observed a decreased expression in metastatic lymph nodes from papillary and follicular carcinomas, correlated with a decreased overall patient survival in follicular carcinoma. Bertheau et al. (1994) noted a nuclear NME1 labeling associated with a longer disease free survival.

For head and neck squamous cell carcinoma, several studies pointed to the absence of prognostic interest for NME1 (Takes et al., 2001; Tsuzuki et al., 2005; Sheikh et al., 2006; Mhawech-Fauceglia et al., 2007).

However, some authors reported a lower NME1 level in lymph nodes as compared to the primary tumor (Takes et al., 2001; Wang YF et al., 2008) or an inverse correlation with lymph node invasion but not associated with better survival (Song et al., 2000). McDonald et al. (1996) have shown that a high NME1 expression in the primary tumor predicts a favorable outcome although the reverse was found by another study (Pavelic et al., 2000). Wang YF et al. (2008) have observed a low NME1 level in lymph node metastases of patient with oral squamous cell carcinoma and that induced NME1 overexpression in these cancer derived cell lines decreased their invasive potential.

In osteosarcoma, a positive immunoreactivity was detected in tumoral cells (Oda et al., 1995; Liao et al., 2000), but no correlation (Liao et al., 2000; Ozger et al., 2009) or a positive correlation (Oda et al., 2000) between NME1 expression and metastatic spread was observed.

In mesothelioma, high NME1 expression levels were observed by cDNA array (Røe et al., 2009) and by immunohistochemical analysis of tumors (Lumb and Suvarna, 2004) and of serous effusion samples (Zimmerman and Fogt, 2006) but were not related to prognosis.

Two studies reported that NME1 expression could possess a diagnostic interest by discriminating between benign and malignant pheochromocytoma (Ohta et al., 2005; Saffar et al., 2011) with a lower expression in malignant tumors. This observation should be confirmed by larger studies.

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