Sequence DNA(FASTA) isomorf 1:

>NM_002467.6 Homo sapiens MYC proto-oncogene, bHLH transcription factor (MYC), transcript variant 1, mRNA

AACTCGCTGTAGTAATTCCAGCGAGAGGCAGAGGGAGCGAGCGGGCGGCCGGCTAGGGTGGAAGAGCCGG
GCGAGCAGAGCTGCGCTGCGGGCGTCCTGGGAAGGGAGATCCGGAGCGAATAGGGGGCTTCGCCTCTGGC
CCAGCCCTCCCGCTGATCCCCCAGCCAGCGGTCCGCAACCCTTGCCGCATCCACGAAACTTTGCCCATAG
CAGCGGGCGGGCACTTTGCACTGGAACTTACAACACCCGAGCAAGGACGCGACTCTCCCGACGCGGGGAG
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Sequence protein (fasta) isomorf 1:

>NP_002458.2 myc proto-oncogene protein isoform 1 [Homo sapiens]
MDFFRVVENQQPPATMPLNVSFTNRNYDLDYDSVQPYFYCDEEENFYQQQQQSELQPPAPSEDIWKKFEL
LPTPPLSPSRRSGLCSPSYVAVTPFSLRGDNDGGGGSFSTADQLEMVTELLGGDMVNQSFICDPDDETFI
KNIIIQDCMWSGFSAAAKLVSEKLASYQAARKDSGSPNPARGHSVCSTSSLYLQDLSAAASECIDPSVVF
PYPLNDSSSPKSCASQDSSAFSPSSDSLLSSTESSPQGSPEPLVLHEETPPTTSSDSEEEQEDEEEIDVV
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ISNNRKCTSPRSSDTEENVKRRTHNVLERQRRNELKRSFFALRDQIPELENNEKAPKVVILKKATAYILS
VQAEEQKLISEEDLLRKRREQLKHKLEQLRNSCA

Sequence DNA (fasta) isomorf 2:

>NM_001354870.1 Homo sapiens MYC proto-oncogene, bHLH transcription factor (MYC), transcript variant 2, mRNA
GGAGTTTATTCATAACGCGCTCTCCAAGTATACGTGGCAATGCGTTGCTGGGTTATTTTAATCATTCTAG
GCATCGTTTTCCTCCTTATGCCTCTATCATTCCTCCCTATCTACACTAACATCCCACGCTCTGAACGCGC
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Sequence protein (fasta) isomorf 2:

>NP_001341799.1 myc proto-oncogene protein isoform 2 [Homo sapiens]
MDFFRVVENQPPATMPLNVSFTNRNYDLDYDSVQPYFYCDEEENFYQQQQQSELQPPAPSEDIWKKFELL
PTPPLSPSRRSGLCSPSYVAVTPFSLRGDNDGGGGSFSTADQLEMVTELLGGDMVNQSFICDPDDETFIK
NIIIQDCMWSGFSAAAKLVSEKLASYQAARKDSGSPNPARGHSVCSTSSLYLQDLSAAASECIDPSVVFP
YPLNDSSSPKSCASQDSSAFSPSSDSLLSSTESSPQGSPEPLVLHEETPPTTSSDSEEEQEDEEEIDVVS
VEKRQAPGKRSESGSPSAGGHSKPPHSPLVLKRCHVSTHQHNYAAPPSTRKDYPAAKRVKLDSVRVLRQI
SNNRKCTSPRSSDTEENVKRRTHNVLERQRRNELKRSFFALRDQIPELENNEKAPKVVILKKATAYILSV
QAEEQKLISEEDLLRKRREQLKHKLEQLRNSCA

Tamanho: DNA isomorf 1 - 3721 bp
DNA isomorf 2 - 4515 bp
Protein isomorf 1 - 454 aa
Protein isomorf 1 - 453 aa

Proteína que forma: NP_002458 - myc proto-oncogene protein isoform 1 [Homo sapiens]
NP_001341799 - myc proto-oncogene protein isoform 2 [Homo sapiens]

Quando foi descoberto: 1979. -> https://pmc.ncbi.nlm.nih.gov/articles/PMC3345192/? [The road to MYC's discovery was paved by early studies of fulminant chicken tumors caused by oncogenic retroviruses, leading to the identification of the v-myc oncogene that causes myelocytomatosis (leukemia and sarcoma) (Duesberg and Vogt, 1979; Hu et al., 1979; Sheiness and Bishop, 1979)]

Importância do gene e porque é estudado: A large body of evidence shows that Myc genes and proteins are highly relevant for treating tumors.Except for early response genes, Myc universally upregulates gene expression. Furthermore, the upregulation is nonlinear. Genes for which expression is already significantly upregulated in the absence of Myc are strongly boosted in the presence of Myc, whereas genes for which expression is low in the absence Myc get only a small boost when Myc is present.

Inactivation of SUMO-activating enzyme (SAE1 / SAE2) in the presence of Myc hyperactivation results in mitotic catastrophe and cell death in cancer cells. Hence inhibitors of SUMOylation may be a possible treatment for cancer.

Amplification of the MYC gene was found in a significant number of epithelial ovarian cases. In TCGA datasets, the amplification of Myc occurs in several cancer types, including breast, colorectal, pancreatic, gastric, and uterine cancers.

In the experimental transformation process of normal cells into cancer cells, the MYC gene can cooperate with the RAS gene.

Expression of Myc is highly dependent on BRD4 function in some cancers.BET inhibitors have been used to successfully block Myc function in pre-clinical cancer models and are currently being evaluated in clinical trials.

MYC expression is controlled by a wide variety of noncoding RNAs, including miRNA, lncRNA, and circRNA. Some of these RNAs have been shown to be specific for certain types of human tissues and tumors. Changes in the expression of such RNAs can potentially be used to develop targeted tumor therapy.

Bónus: Localização - 8q24.21
Nº de Exões - 3