

Non-coding RNA

A non-coding [RNA](#) (ncRNA) is an [RNA molecule](#) that is not translated into a [protein](#). The [DNA](#) sequence from which a functional non-coding RNA is transcribed is often called an RNA gene.

Less-frequently used synonyms are non-protein-coding RNA (npcRNA), non-messenger RNA (nmRNA) and functional RNA (fRNA). The DNA sequence from which a non-coding RNA is transcribed is often called an RNA gene.

Many of the newly identified ncRNAs have not been validated for their function.

It is also likely that many ncRNAs are non-functional (sometimes referred to as Junk RNA), and are the product of spurious transcription.

Types

Abundant and functionally important types of non-coding RNAs include [microRNAs](#), [Long non-coding RNAs](#), and [circular RNAs](#), transfer RNAs (tRNAs) and ribosomal RNAs (rRNAs), as well as small RNAs such as microRNAs, siRNAs, piRNAs, snoRNAs, snRNAs, exRNAs, scaRNAs and the long ncRNAs such as Xist and HOTAIR.

The number of ncRNAs encoded within the human genome is unknown, however recent transcriptomic and bioinformatic studies suggest the existence of thousands of ncRNAs.

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At present, although drugs such as [temozolomide](#), [cisplatin](#), and [bevacizumab](#), are effective in improving the overall survival of patients with Glioblastoma, most patients eventually develop [drug resistance](#), leading to poor clinical prognosis. The development of multidrug resistance has therefore become a major obstacle to improving the effectiveness of chemotherapy for Glioblastoma. The ability to fully understand the underlying mechanisms of [chemotherapy resistance](#) and to develop novel therapeutic targets to overcome resistance is critical to improving the prognosis of patients with Glioblastoma. Of note, growing evidence indicates that a large number of abnormally expressed [noncoding RNAs](#) (ncRNAs) have a central role in glioma [chemoresistance](#) and may target various mechanisms to modulate [chemosensitivity](#). [noncoding RNAs](#) is a research direction for tumor drug resistance mechanisms and targeted therapies, which not only provides novel perspectives for reversing glioma drug resistance but may also promote the development of precision medicine for clinical diagnosis and treatment ¹⁾.

Small non-coding RNA

[Small non-coding RNA](#).

Long non-coding RNA

Long non-coding RNA.

Non-coding RNAs contribute to diseases including cancers, autism, and Alzheimer's.

While accumulating studies have investigated coding gene-associated biomarkers in malignant glioma, research on comprehensive coding and [non-coding RNA](#)-associated biomarkers is lacking. Furthermore, few studies have illustrated the [crosstalk](#) signalling pathways among these biomarkers and mechanisms in detail.

Huang et al. identified differentially expressed genes and [Competing endogenous RNA](#) (ceRNA) networks in malignant glioma and then constructed [Cox/Lasso regression models](#) to further identify the most valuable genes through stepwise refinement. Top-down comprehensive integrated analysis, including functional enrichment, SNV, immune infiltration, transcription factor binding site, and molecular docking analyses, further revealed the regulatory maps among these genes. The results revealed a novel and accurate model (AUC of 0.91 and C-index of 0.84 in the whole malignant gliomas, AUC of 0.90 and C-index of 0.86 in LGG, and AUC of 0.75 and C-index of 0.69 in Glioblastoma) that includes twelve ncRNAs, 1 miRNA and 6 coding genes. Stepwise logical reasoning based on top-down comprehensive integrated analysis and references revealed cross-talk signalling pathways among these genes that were correlated with the circadian rhythm, tumour immune microenvironment and cellular senescence pathways. In conclusion, our work reveals a novel model where the newly identified biomarkers may contribute to a precise diagnosis/prognosis and subclassification of malignant glioma, and the identified cross-talk signalling pathways would help to illustrate the noncoding RNA-associated epigenetic regulatory mechanisms of [glioma tumorigenesis](#) and aid in targeted therapy ²⁾.

1)

Zeng Z, Chen Y, Geng X, Zhang Y, Wen X, Yan Q, Wang T, Ling C, Xu Y, Duan J, Zheng K, Sun Z. NcRNAs: Multi-angle participation in the regulation of glioma chemotherapy resistance (Review). Int J Oncol. 2022 Jun;60(6):76. doi: 10.3892/ijo.2022.5366. Epub 2022 May 4. PMID: 35506469.

2)

Huang Y, Gao X, Yang E, Yue K, Cao Y, Zhao B, Zhang H, Dai S, Zhang L, Luo P, Jiang X. Top-down [stepwise refinement](#) identifies coding and noncoding RNA-associated [epigenetic](#) regulatory maps in malignant glioma. J Cell Mol Med. 2022 Feb 22. doi: 10.1111/jcmm.17244. Epub ahead of print. PMID: 35194922.

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