



## MIR17HG gene

miR-17-92a-1 cluster host gene

### Normal Function

The *MIR17HG* gene provides instructions for making the miR-17~92 microRNA (miRNA) cluster. MiRNAs are short pieces of RNA, a chemical cousin of DNA. These molecules control gene expression by blocking protein production. The miR-17~92 cluster includes six miRNAs: miR-17, miR-18a, miR-19a, miR-19b-1, miR-20a, and miR-92a-1. MiRNAs in this cluster control the expression of hundreds of genes. These miRNAs help regulate signaling pathways that direct several cellular processes involved in growth and development, including cell growth and division (proliferation), cell maturation (differentiation), and the self-destruction of cells (apoptosis). Studies suggest that the miR-17~92 cluster is necessary for normal development of the skeleton, heart, kidneys, lungs, and nervous system.

The *MIR17HG* gene belongs to a class of genes known as oncogenes. When mutated, oncogenes have the potential to cause normal cells to become cancerous.

### Health Conditions Related to Genetic Changes

#### Feingold syndrome

Genetic changes that reduce the amount of the *MIR17HG* gene cause Feingold syndrome type 2. This developmental disorder is characterized by abnormalities of the fingers and toes, particularly shortening of the second and fifth fingers (brachymesophalangy). Other common features include an unusually small head size (microcephaly) and learning disabilities. The mutations involved in this condition, known as 13q31.3 microdeletions, remove (delete) a small region of chromosome 13 that includes the *MIR17HG* gene and sometimes part or all of other nearby genes. Loss of the *MIR17HG* gene is thought to underlie the characteristic features of the disorder, although loss of other genes may play a role in some cases.

Deletion of one copy of the *MIR17HG* gene reduces the amount of miR-17~92 cluster miRNAs available to control the activity of specific genes during development before birth. While it is likely that the resulting disruption of signaling pathways leads to the problems with growth and development characteristic of Feingold syndrome type 2, it remains unclear exactly how a shortage of miR-17~92 cluster miRNAs causes the specific features of the condition.

#### Cancers

Genetic changes that result in extra copies of the *MIR17HG* gene have been found in cancer. These genetic changes likely occur when DNA makes a copy of itself

(replicates) in preparation for cell division. Errors in the replication process can result in one or more extra copies of a gene within a cell, which is known as gene amplification. The amplifications are somatic, which means they are not inherited but instead occur in cells that give rise to the tumor. Gene amplifications involving *MIR17HG* have been found in a cancer of immune system cells called diffuse large B-cell lymphoma. Such amplifications are thought to increase the amount of miR-17~92 miRNAs. The resulting alterations to cell signaling pathways may lead to too much cell proliferation or too little apoptosis. As a result, cells can grow and divide uncontrollably, leading to the development of cancer.

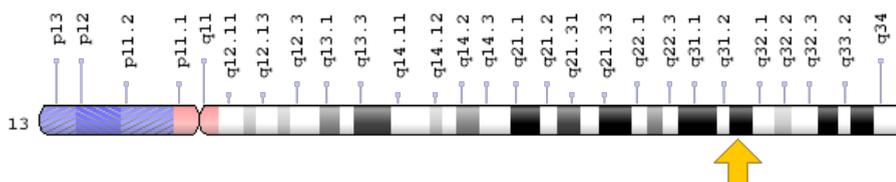
### Other disorders

Genetic changes, called microduplications, that result in an extra copy of the *MIR17HG* gene have been associated with developmental problems in a small number of people. Some individuals with these microduplications have overgrowth of the skeleton, resulting in extra fingers (polydactyly) and an unusually large head size (macrocephaly). Others with these microduplications have impaired growth, leading to short fingers (brachydactyly) and short stature. Affected individuals may also have features of autism spectrum disorder, which is characterized by impaired communication and social interaction. Researchers suggest that an extra copy of the *MIR17HG* gene disrupts normal skeletal growth, although they are unsure why the microduplications can either increase or reduce growth.

### **Chromosomal Location**

Cytogenetic Location: 13q31.3, which is the long (q) arm of chromosome 13 at position 31.3

Molecular Location: base pairs 91,347,820 to 91,354,575 on chromosome 13 (Homo sapiens Updated Annotation Release 109.20190607, GRCh38.p13) (NCBI)



Credit: Genome Decoration Page/NCBI

### **Other Names for This Gene**

- C13orf25
- FGLDS2
- FLJ14178

- LINC00048
- MIHG1
- miR-17-92
- MIRH1
- MIRHG1
- NCRNA00048

## **Additional Information & Resources**

### Educational Resources

- Stembook (2008): MicroRNA Biogenesis and Function  
<https://www.ncbi.nlm.nih.gov/books/NBK27061/#theroleofmicrornasingermline.sec1-3>

### Scientific Articles on PubMed

- PubMed  
<https://www.ncbi.nlm.nih.gov/pubmed?term=%28%28MIR17HG%5BTIAB%5D%29+OR+%28miR-17-92a-1+cluster+host+gene%5BTIAB%5D%29%29+OR+%28%28C13orf25%5BTIAB%5D%29+OR+%28FGLDS2%5BTIAB%5D%29+OR+%28MIRHG1%5BTIAB%5D%29+OR+%28miR-17-92%5BTIAB%5D%29%29+AND+%28%28Genes%5BMH%5D%29+OR+%28Genetic+Phenomena%5BMH%5D%29%29+AND+english%5Bla%5D+AND+human%5Bmh%5D+AND+%22last+720+days%22%5Bdp%5D>

### Catalog of Genes and Diseases from OMIM

- MICRO RNA 17 HOST GENE  
<http://omim.org/entry/609415>

### Research Resources

- Atlas of Genetics and Cytogenetics in Oncology and Haematology  
[http://atlasgeneticsoncology.org/Genes/GC\\_MIR17HG.html](http://atlasgeneticsoncology.org/Genes/GC_MIR17HG.html)
- ClinVar  
<https://www.ncbi.nlm.nih.gov/clinvar?term=MIR17HG%5Bgene%5D>
- HGNC Gene Symbol Report  
[https://www.genenames.org/data/gene-symbol-report/#!/hgnc\\_id/HGNC:23564](https://www.genenames.org/data/gene-symbol-report/#!/hgnc_id/HGNC:23564)
- Monarch Initiative  
<https://monarchinitiative.org/gene/NCBIGene:407975>
- NCBI Gene  
<https://www.ncbi.nlm.nih.gov/gene/407975>

## Sources for This Summary

- Fang LL, Wang XH, Sun BF, Zhang XD, Zhu XH, Yu ZJ, Luo H. Expression, regulation and mechanism of action of the miR-17-92 cluster in tumor cells (Review). *Int J Mol Med*. 2017 Dec; 40(6):1624-1630. doi: 10.3892/ijmm.2017.3164. Epub 2017 Sep 29. Review.  
*Citation on PubMed:* <https://www.ncbi.nlm.nih.gov/pubmed/29039606>  
*Free article on PubMed Central:* <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5716450/>
- Grote LE, Repnikova EA, Amudhavalli SM. Expanding the phenotype of feingold syndrome-2. *Am J Med Genet A*. 2015 Dec;167A(12):3219-25. doi: 10.1002/ajmg.a.37368. Epub 2015 Sep 11.  
*Citation on PubMed:* <https://www.ncbi.nlm.nih.gov/pubmed/26360630>
- Hemmat M, Rumpel MJ, Mahon LW, Strom CM, Anguiano A, Talai M, Nguyen B, Boyar FZ. Short stature, digit anomalies and dysmorphic facial features are associated with the duplication of miR-17 ~ 92 cluster. *Mol Cytogenet*. 2014 Apr 16;7:27. doi: 10.1186/1755-8166-7-27. eCollection 2014.  
*Citation on PubMed:* <https://www.ncbi.nlm.nih.gov/pubmed/24739087>  
*Free article on PubMed Central:* <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4005632/>
- Kannu P, Campos-Xavier AB, Hull D, Martinet D, Ballhausen D, Bonafé L. Post-axial polydactyly type A2, overgrowth and autistic traits associated with a chromosome 13q31.3 microduplication encompassing miR-17-92 and GPC5. *Eur J Med Genet*. 2013 Aug;56(8):452-7. doi: 10.1016/j.ejmg.2013.06.001. Epub 2013 Jun 20. Review. Erratum in: *Eur J Med Genet*. 2014 Feb;57(2-3):123-4.  
*Citation on PubMed:* <https://www.ncbi.nlm.nih.gov/pubmed/23792790>
- OMIM: MICRO RNA 17 HOST GENE  
<http://omim.org/entry/609415>
- Mirzamohammadi F, Kozlova A, Papaioannou G, Paltrinieri E, Ayturk UM, Kobayashi T. Distinct molecular pathways mediate Mycn and Myc-regulated miR-17-92 microRNA action in Feingold syndrome mouse models. *Nat Commun*. 2018 Apr 10;9(1):1352. doi: 10.1038/s41467-018-03788-7.  
*Citation on PubMed:* <https://www.ncbi.nlm.nih.gov/pubmed/29636449>  
*Free article on PubMed Central:* <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5893605/>
- Mogilyansky E, Rigoutsos I. The miR-17/92 cluster: a comprehensive update on its genomics, genetics, functions and increasingly important and numerous roles in health and disease. *Cell Death Differ*. 2013 Dec;20(12):1603-14. doi: 10.1038/cdd.2013.125. Review.  
*Citation on PubMed:* <https://www.ncbi.nlm.nih.gov/pubmed/24212931>  
*Free article on PubMed Central:* <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3824591/>
- de Pontual L, Yao E, Callier P, Faivre L, Drouin V, Cariou S, Van Haeringen A, Geneviève D, Goldenberg A, Oufadem M, Manouvrier S, Munnich A, Vidigal JA, Vekemans M, Lyonnet S, Henrion-Caude A, Ventura A, Amiel J. Germline deletion of the miR-17#92 cluster causes skeletal and growth defects in humans. *Nat Genet*. 2011 Sep 4;43(10):1026-30. doi: 10.1038/ng.915.  
*Citation on PubMed:* <https://www.ncbi.nlm.nih.gov/pubmed/21892160>  
*Free article on PubMed Central:* <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3184212/>

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