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## Heterochromatin formation model

The compact and highly viscous form of Heterochromatin chromatin is a tightly packaged form of thick DNA or DNA, which comes in several varieties. This variety lies on a continuum between two constitutive heterochromatin extremes and constitutive heterochromatin. Both play a role in gene expression. Because it is tightly packaged, it is considered inaccessible to polymerases and therefore not transcribed, but according to Volpe et al. (2002), [1] and many other papers since, [2] many of these DNAs are in fact transcribed, but are constantly submitted through RNA-induced transcriptional silencing (RITS). Recent studies with electron microscopy and OsO<sub>4</sub> staining revealed that solid packing is not due to chromatin. [3] Constitutive heterochromatin can affect genes nearby (e.g. positional effect varied). It usually repeats and forms structural functions such as flashlights or telomeres, in addition to acting as a redeemer for signaling gene expression or other repression. Heterochromatin is the result of genes silenced through mechanisms such as histone deacetylation or RNA (piRNA) that interact with Piwi through RNAi. It does not recur and shares a compact structure of constitutive heterochromatin. However, under certain developmental or environmental signal cues, it can lose its viscous structure and become transcriptionally active. [4] Heterochromatin has been associated with di- and tri-methylation of H3K9 in certain parts of the genome. [5] H3K9me3-related Methyltransferase appears to have played an important role in modifying heterochromatin during lineage commitments at the beginning of organogenesis and in maintaining lineage fidelity. [6] The nucleus of the human cell indicates the location of heterochromatin. Note that the informal diagram shown here may be incorrect about the location of heterochromatin. The inactive X chromosome (aka Barr's body) migrates to the nuclear membrane only, leaving active X and other chromosomes in the nucleoplasm (far from the membrane in general). Other heterochromatin appears as particles separated from the membrane. Heterochromatin appears as small, dark-stained, irregular particles scattered throughout the nucleus.... [7] The structure of Heterochromatin vs. euchromatin Chromatin is found in two varieties: euchromatin and heterochromatin. [8] Initially, both forms were cytologically distinguished by how intensely they were stained - euchromatin was less intense, while intense heterochromatin stains, indicating tighter packaging. Heterochromatin is usually realized to the periphery of the core. Despite this early dichotomy, recent evidence in both animals [9] and plants [10] has shown that there are more than two different heterochromatin states, and that it may actually exist in four or five 'states', respectively with different combinations of Sign. Heterochromatin mainly consists of genetically inactive satellite sequences, [11] and many genes are suppressed to varying degrees, although some cannot be expressed in euchromatin at all. [12] Both the flashlight and telomeres are heterochromatic, as is Barr's body from the second X-chromosome that is inactive in women. A common model of function for heterochromatin duplication during the heterochromatin cell division has been associated with several functions, from gene regulation to protection of chromosomal integrity; [13] some of these roles can be attributed to dense DNA packing, which makes them less accessible to protein factors that typically bind to DNA or related factors. For example, the double-stranded tip of naked DNA will usually be interpreted by the cell as damaged or viral DNA, triggering the capture of cell cycles, DNA repair or destruction of fragments, such as by endonuclease in bacteria. Some areas of chromatin are very dense with fibers that display conditions comparable to chromosomes in mitosis. Heterochromatin is generally inherited clonally: when cells divide, two female cells typically contain heterochromatin within the same region of DNA, resulting in epigenetic inheritance. Variations cause heterochromatin to penetrate adjacent genes or repress genes in extreme domains. Transmissible materials can be pressed by positioning (in cis) in this boundary domain. This gives rise to a level of expression that varies from cell to cell, [14] which may be indicated by the degree of position effect. [15] Insulator sequences can act as a barrier in rare cases where constitutive heterochromatin and highly active genes juxtapose (e.g. insulator 5HS4 upstream of β-globin loci). [16] and looms in two *Saccharomyces* spp. [17] [18]. Main article constitutive heterochromatin: Constitutive heterochromatin All cells of a particular species, pack the same area of DNA in constitutive heterochromatin, and thus in all cells, any genes contained in constitutive heterochromatin will be poorly expressed. For example, all human chromosomes 1, 9, 16, and Y chromosomes contain large areas of constitutive heterochromatin. In most organisms, constitutive heterochromatin occurs around the centromere chromosome and near the telomeres. The heterochromatin Region of DNA packed in radioactive heterochromatin will not be consistent between cell types in the species, and thus the sequence in one cell packed in radioactive heterochromatin (and the genes in it is poorly expressed) can be packaged in euchromatin in other cells (and the genes in it are no longer silenced). However, the formation of heterochromatin is regulated, and is often associated with morphogenesis or differentiation. An example of heterochromatin is the X chromosome in female mammals: one X chromosome is packaged as heterochromatin and silenced heterochromatin, while the other X chromosome is packaged as euchromatin and expressed. Among the molecular components that seem to regulate the spread of heterochromatin are polycomb group proteins and non-coding genes such as Xist. The mechanism of the spread is still the subject of controversy. [19] The repressive complexes of POLYCOMBE PRC1 and PRC2 regulate chromatin compaction and gene expression and have a fundamental role in the developmental process. PRC-mediated epigenetic deviations are associated with genome instability and malignancy and play a role in DNA damage response, DNA repair and in replication fidelity. [20] *Saccharomyces cerevisiae* heterochromatin yeast, or bud yeast, is a model of eukaryote and its heterochromatin has been thoroughly defined. Although most of its genome can be characterized as euchromatin, *S. cerevisiae* has a region of DNA that is very poorly transcribed. These loci are the so-called silent mating type loci (HML and HMR), rDNA (RNA ribosomal coding), and sub-telomeric regions. Fission yeast (*Schizosaccharomyces pombe*) uses another mechanism for the formation of heterochromatin on its flashlight. Gene decay at this location depends on the components of the RNAi pathway. The double-stranded RNA is believed to result in the dissolution of the region through a series of measures. In the *Schizosaccharomyces pombe* fission yeast, two RNAi complexes, the RITS complex and the RNA-directed RNA polymerase complex (RDRC), are part of the RNAi machine involved in the initiation, propagation and maintenance of heterochromatin assembly. Both of these complexes mediate localization in the chromosome, at the location of heterochromatin assembly. RNA polymerase II synthesizes transcripts that serve as a platform for recruiting RITS, RDRC and possibly other complexes necessary for heterochromatin assembly. [22] Both the RNAi and the exosome-dependent RNA degradation process contribute to the decay of heterochromatic genes. This mechanism of *pombe* *Schizosaccharomyces* can occur in other eukaryotes. [23] A large RNA structure called RevCen has also been involved in the production of siRNAs to mediate the formation of heterochromatin in some fission yeast. [24] See also Heterochromatin Centric Reference ^ Volpe TA, Kidner C, Hall JM, Teng G, Grewal SI, Martienssen RA (September 2002). Regulation of heterochromatin and histone H3 lysine-9 methylation regulation by RNAi. *Science*. 297 (5588): 1833–7. doi:10.1126/science.1074973. PMID 12193640. S2CID 2613813. ^ What is the current evidence that indicates active transcription in it ... www.researchgate.net. Retrieved 2016-04-30. 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PMID 30606806. ^ Shown here: An image of a nucleus electron microscope with annotated heterochromatin particle [1] ^ Elgin, S.C. (1996). Heterochromatin and gene regulation in *Drosophila*. *Current Opinions in Genetics & Development*. 6 (2): 193–202. doi:10.1016/S0959-437X(96)80050-5. ISSN 0959-437X. PMID 8722176. van Steensel B (May 2011). Chromatin: build the big picture. *EMBO Journal*. 30 (10): 1885–95. doi:10.1038/emboj.2011.135. PMC 3098493. PMID 21527910. Roudier F, Ahmed I, Bérard C, Sarazin A, Mary-Huard T, Cortijo S, et al (May 2011). Integrative epigenomic mapping defines the four main chromatin states in *Arabidopsis*. *EMBO Journal*. 30 (10): 1928–38. doi:10.1038/emboj.2011.103. PMC 3098477. PMID 21487388. ^ Lohe AR, Hilliker AJ, Roberts PA (August 1993). Mapping of repetitive DNA sequences in simple heterochromatin *Drosophila melanogaster*. *Genetics*. 134 (4): 1149–74. PMC 1205583. PMID 8375654. ^ Lu BY, Emtage PC, Duyf BJ, Hilliker AJ, Eissenberg JC (June 2000). 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