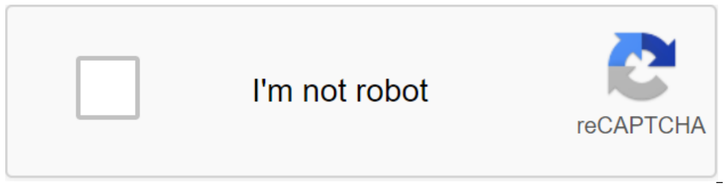

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Home Genes of Leukemia Solid Tumors Cancer-Prone Deep Insight Case Reports Magazines Portal Training X Y 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 16 17 18 19 20 21 22 NA - Long version of French English - I Concept heterohroromatin Definition of heterrochromatin in eukaryotic organisms, as opposed to prokaryotes, DNA is packed in the form of nucleoprotein complex called chromatin that contains a hereditary message. Chromatin is located in the nucleus and is organized into several separate entities, chromosomes. The concept of heterochromatin In 1928, Emil HEIC, based on histological observations, defined heterohroromatin (HC) as highly condensed and dark chromosomal segments in the interphase nucleus. In fact, chromatin is formed from a tangle of fibers, the diameter of which not only changes during the cell cycle, but also depends on the area of the observed chromosome. Active euchromatin is formed by a diameter fiber corresponding to nucleosome, which is a segment of bicameral DNA wrapped around the homodimers of histones H2A, H2B, H3 and H4. In inactive euchromatin, this fiber winds on itself thanks to H1 histones to form a solenoid. Interaction with other non-sourced proteins (topoisomerase II, scaffolding proteins, laminins,) causes a higher degree of organization. As for heterhoromatin, the fiber that makes it more condensed and often consists of aggregates. Its formation requires numerous additional proteins, including HP1 proteins (heterochromatin Protein 1 or protein heterochromatin 1). II Two types of heterhoromatin there are two types of heterhoromatin, composite HC and optional HC that differ little, depending on and they contain. The richness of satellite DNA determines both the constant and reversible nature of heterochromatin, as well as its polymorphism and coloring properties. Table I: Properties that distinguish composite heterchromatin from optional constituent heterochromatin contain a specific type of DNA called satellite DNA, formed by a large number of short sequences repeated in tandem. The main types of this DNA are alpha-satellite DNA and satellite DNA I, II and III. These satellite DNA sequences are capable of forming over themselves and can play an important role in the formation of a highly compact composite heterochromatin structure. Composite heterocheromatine is stable and retains its heterocheromatic properties at all stages of development and in all tissues. The composite heterchromatin is very polymorphic, probably due to satellite DNA. This polymorphism can affect not only its size, but also the location of heterohromatine, and apparently has no phenotypic effect. The composite heterchromatin is strongly colored in the C-band method, which is the result of very rapid renaturization of satellite DNA after denature. II.2 Additional heterroromatin optional heterhroromatin is characterized by the presence of repetitive LINE sequences. These sequences, scattered throughout the genome, can contribute to the spread of the condensed chromatin structure. Optional heterohroromatin reversible, its heterochromatic state depends on the stage of development and cell type. Two examples of this type of heterohromatine are the inactive X chromosome (Barra body) of female somatic cells and inactive sexual vesicula at the stage of the groin of male meiosis. Optional heterochromatin is not particularly rich in satellite DNA and therefore not polymorphic. Optional heterochromatin is never stained in the technique of bands C. III Properties heterchromatin Despite the differences described above, composite heterochromatin and optional heterromomatin have very similar properties. This is, in fact, what defines heterochromatin, and therefore applies to both composite and optional heterhromatine. This high condensation makes it highly chromophilic and inaccessible to DNAsa I and, in general, to other enzyme restriction. The inclusion of several nucleotide analogues shows that the DNA of both types of heterochromatin is replicated late. This is the result, on the one hand, of a high degree of condensation, which does not allow replication technology to easily access DNA, and on the other hand, its location in the peripheral nuclear field is poor in active elements. The DNA of the compound heterhroromatin is strongly methylated in cytosin. Thus, the anti-5-methyl cytosine antibody strongly marks all regions of this type of heterochromatin. As for optional heterochromatin, methylation of its DNA is lower, although analysis of methylation-sensitive enzymes shows significant methylation of CpG islets specifically located in regions that control gene expression. Histons may be subject to a number of post-translational changes in their N-terminal ends, which can affect their own genetic activity of chromatin. Hypoacetylation of N-terminal tails of histones, mainly in lysions, is associated with inactive chromatin. In contrast, hyperacetyl histones active chromatin. Acetylation/deacetylization of histones is an absolutely necessary mechanism for controlling gene expression. There are many transcription factors that have histon acetyltransferase (HAT) or giston deacetylase activity (HDAc or histon de-Acetylase). Methylation of giston H3 lysine 9 (H3-K9) appears to be closely related to the process of heterocheromatization of the genome, both in the formation of composite and optional heterocheromaticin. Unlike drosophila, human composite heteroromatin does not contain genes and the inclusion of tritiatized uridine in cellular cultures does not produce any type of marking at this level. Additional heterochromatin is relatively poor in genes, and they are generally not transcribed in heterichromatin. It is generally accepted that composite heterroromatine is not involved in genetic recombination. The insignificance of pre-pairing of homologous heterhoromatic regions may be associated with the polymorphism characteristic of these regions, which makes it difficult, though not impossible. The constituent heterochromatin also works by suppressing recombination in adjacent euchromatic areas. As for optional heterochromatin, it also does not participate in retotic recombination when it is inactive. A study of several organisms has shown that composite heterhroromatin tends to be added during the interface. In the larvae of drosophila it is possible to add centurmers of polythene chromosomes, rich in heterochromatine, to form chromocenters during the interface. In mice, the number of heterichromatic blocks that can be observed in the interphase nucleus is always lower than the number of heterochromatin regions observed on metaphasic chromosomes. In humans, short arms of aerocentric chromosomes, mostly formed from heterochromatin, are often associated in the nuclei in the interface with other chromosomes that have a large block of heterhoromatin (1, 9 and 16). This tendency to add heterichromatin appears to be closely related to the presence of satellite DNA sequences, although additional sequences may also be involved. IV Factors involved in heterchromatization Some observations have led to the identification of several elements that play an important role in the formation of heterocheromatine, whether composite or optional. Satellite DNA observed by FISH accurately places it with composite heterhroromatic. In addition, satellite DNA has the characteristic ability to bend and fold and this can be an important determinant in the formation of an extremely compact structure characteristic of this type of heterochromatin. However, this does not apply only to satellite DNA. In plants, Drosophila, and in mice, some multicopies transgenes express themselves little, or not, even if they are not repressed centrom'rica. These various observations suggest that the tandem repetition of DNA sequences in large numbers of copies is sufficient on its own to promote the direct formation of heterohromatin. Such repetitive sequences can allow the majority of chromatin to be compacted by forming characteristic structures. These structures can be recognized as specific proteins, such as HP1 proteins, which can guide the formation of higher chromatin structures. Long-term transgene repetitions do not always lead to transcription inactivation of transgene. Tandem repetitions of induced muffled silence appear to be associated with the presence of CPG-rich DNA sequences, probably

methylated. Therefore, a very basic composition of tandem repetitions can play an important role in the formation of heterochromatin. It has recently been described that the meCP2 methyl binding protein, which is usually attached to methylated cytosins DNA, also has the ability to recruit histon deacetyls (HDAc) (Figure 1). Thus, DNA methylation can cause deacetylization of histones and thus contribute to heterchromatization. However, DNA methylation is not necessary for the formation of heterohromatin, although this may be an element involved in stabilization. In fact, the marsupial, inactive X chromosome is not methylated, and this condition is less stable than in mammals. Figure 1: DNA methylation causes gisethoton deacetylation, a modification that characterizes histones present in both heterochromatin and suppressed euchromatin expression. MeCP2 specifically attaches to methylated DNA and recruits HDAc, which deacetylyates histones (ac-acetyl group; Me-methyl group; MeCP2 protein 2 binding groups (Methyl-CpG protein binding 2); Histon de Acetylase (HDAc)). We have already seen that hypoacetylation of histon is a feature of silenced chromatin, whether in the form of heterhoromatin or not. Thus, blocking the deacetylation of histones by adding triostatin A causes hyperacetylation of these, causing the discovery of the chromatin structure. In fact, lysine acetylation eliminates the positive load of histones, reducing strength attraction with a negative charge of DNA phosphates. This contributes to the discovery of chromatin. In contrast, deacetylizing lysins restores their positive load, so promotes their attractiveness to DNA, leading to more condensed chromatin. Methylation of lysine 9 in hisstone H3 is an epigenetic modification that has recently been described, participating in the process of heterhoromatization, not only the composite heterchromatin, but also in the formation of an inactive X chromosome. The enzyme responsible for this methylation is histon methyltransferase SUV39H1. Acetylation and methylation H3-K9 appear to be mutually exclusive. In Drosophila, Suv39h methyltransferase is associated with gistone dicetylase, offering a unique molecular mechanism for the direct conversion of acetylated lysine 9 into methylated lysine 9. In addition, H3-K9 methylation creates a place of high binding proximity to the HP1 heterchromatin protein. Suvar39h co-immunoprecipitation with HP1 involves a heterocheromatization mechanism based on the interaction between two proteins and lysine 9. Finally, Neurospora crassa recently described that H3-K9 methylation can cause DNA methylation (Figure 2). Figure 2: Chiton 3 methylation in lysine 9 (H3-K9) causes DNA methylation, a modification that characterizes DNA in heterhoromatin or suppressed euchromatin expression. SUVAR39H is a methyltransferase that specifically methylates 9 giston h3 gistones. This methylation creates a binder for the protein HEterochromatin HP1 (Heterochromatin Protein 1), which collects methyltransferase DNA capable of methylming CpG in DNA (Meo methyl group; methyl methyl group H3-K9TM in lysine 9 histonE H3; HP1o protein 1 hetererhoromatin; methyltransfers. HP1 proteins appear to play an important role in the organization of heterochromatin. Position effect variegated (PEV effect) studies in Drosophila and transgene studies in the same body and in mice have made it possible to better understand the role of these proteins. In Drosophila, the HP1 protein is encoded by the Su(var)205 gene, which is a variation suppressor that can alter the PEV effect. The change in position can be described as follows: genes that are usually located in active euchromatin, so that, after chromosomal reordering, located near the heterchromatic central region. The freshly-hardened chromatin then becomes more compact and begins to be associated with HP1 proteins, which are usually limited to centr based. Also genes present in this translucent chromatin begin to be suppressed. The mouse insertion of the transgene near the centerer can have similar effects. Interestingly, even when the transgene is suppressed, not as a result of the centur center effect, but as a result of its presence in several copies, HP1 proteins are also associated with repressed chromatin. HP1 proteins appear to be an important link in the formation of heterochromatin, and may play the role of domain chromatin organizers. These proteins appear to be able to recognize specific structures created by pairing and/or association DNA sequences. They are also able to establish a secondary interaction with a large number of other proteins thanks to chromodomen (CD) and the cromoshadow domain (CSD). It appears that some nuclear RREs may also contribute to the formation of optional heterhoromatin. In this sense, for example, the transcribed XIST gene plays an important role in initiating the inactivation of one of the X chromosomes of female mammalian somatic cells. Some recent mouse studies have shown that nuclear transcribed may also be involved in the formation of constituent heterhoromatin. In mouse cells, the center heterochromatin is characterized by a high concentration of gistonE H3 methylated in lysin 9 (H3-K9) and HP1 proteins that move quickly after incubation with RNSA A. This suggests that there is nuclear RNA, which may be an important structural component of the composite heterohrophatine. For a long time, the specific role of heterohromatine was a mystery, as its polymorphism did not seem to have any functional or phenotypic effects. Heterochromatin and euchromatin occupy various nuclear areas. Heterochromatin is usually located on the periphery of the nucleus attached to the nuclear membrane. In contrast, active chromatin is in a more central position. The preferential arrangement of heterochromatin against the nuclear membrane may be due to the interaction of the HP1 protein with the leaf B receptor, a component of the inner membrane of the nucleus. The peripheral location of heterohromatine concentrates active elements in the central part of the nucleus, allowing active euchromatine to multiply and transcribed with maximum efficiency. In most eukaryotes, centurion is surrounded by a significant mass of heterhoromatin. It has been suggested that heterhromatin can be used to cohesion the chromatide sister and that this will allow the normal separation of the mitotic chromosomes. Schizosaccharomyces pombe yeast, Swi6 analogue protein HP1 is absolutely essential for effective cohesion of sister chromatids during cell division. Experiments in which satellite DNA was removed show that a large area of repetition of this type of DNA is necessary for the proper functioning of the centr. It is assumed that the center heterhoromatin can de facto create a compartment by increasing the local concentration of the center version of histones, CENP-A, and by promoting the inclusion of CENP-A instead of histon H3 during replication. The expression of the gene can be controlled on two levels: first, local or transcription control, thanks to the formation of local transcription complexes. This level includes relatively small DNA sequences attached to genes. On a more global level, in this case he said that there is control over transcription. This control includes longer sequences representing a large chromatin domain that may be active or inactive. In this case, it appears to be heterochromatin. Genes are usually found in euchromatin so can be drowned out when they are near the heteroromatin domain. Cis inactivation mechanism : Chromosome reordering can lead to juxtapse of the euchromatic region to the heterchromatic region. At a time when reordering removes certain barriers that protect the euchromatin heterhoromatic structure is able to spread in cis to neighboring euchromatin, inactivating the genes found in it. Mechanism of trans-inactivation: During cell differentiation, some active genes can be transferred to the heterchromatic nuclear area, causing their inactivity. This mechanism is proposed as an explanation for the joint location in the nuclei of the lymphocytes of the IKAROS protein with the center of heterochromatin and the genes it controls. VI Heterochromatin-related diseases These diseases are usually the result of changes in cell differentiation. Some are constitutional, such as ICF syndrome or Roberts syndrome. ICF syndrome links immunodeficiency, instability of centriet and facial abnormalities. It is a rare recessive disease associated with mutations in the DNMT3B gene, methyltranspherous DNA. Satellite II and III DNA, rich in G-C, are particularly demethylated in this disease, which may lead to abnormal segregation of sister chromatids, multiradial structures, removals, micronucleids, etc. Some of them are acquired: in many types of cancer, changes in the compound heterohromatine have been found to affect both DNA and heterhoromatin proteins. In particular, it was described that non-Hodgkin's lymphoma and multiple myeloma are associated with changes in the secondary narrowing of chromosome 1. These changes are similar to those observed in ICF syndrome. In fact, global genome hypomethylation has been demonstrated, associated in particular with the hypomethylation of satellite DNA II. it is a protein that is commonly found in heterichromatic areas of chromosomes. These diseases may be the result of an X chromosome inactivation defect in female somatic cells (by mutations in the XIST gene) and may lead to expression of recessive diseases associated with the X chromosome in women. They may also be the result of defects in the sexual gallbladder condensation in male germ cells, which can lead to infertility due to the stopping of meiosis in the groin phase. In conclusion, although heterichromatic is a seemingly aephetic and isolated structure on the nuclear periphery, it seems to play an absolutely important role in the organization and functioning of the genome. In this review, we have shown the main characteristics of heterhroromatin, both composite and optional. We have also shown that the properties of composite heterchromatin do not differ in their basics from the properties of optional heterohromatin. It is therefore quite clear that the mechanisms responsible for optional etherhomaticization are mostly epigenetic mechanisms, the same as those associated with repression of euchromatin in general. Translation : Jose Luis Ismanos. Department of Genetics, Faculty of Sciences. University of Navarre. Pamplona, Spain. Written2003-01Mari-Genevivi Mattei, Judith Luciani INSERM U 491, Faculty deDecin, Bd Jean Moulin, 13385 Marseille, France © Atlas of Genetics and Cytogenetics in Oncology and Hematology Indexed on : Sun May 10:08:01 CEST 2020 Home Genes leukemia Solid Tumors Cancer-Prone Deep Insight Case Reports Magazines Portal Teaching X Y 2 2 3 4 5 6 7 8 9 10 11 12 13 15 15 16 17 18 19 20 21 22 NA For comments and suggestions or contributions, please contact us jlhuret@AtlasGeneticsOncology.org. jlhuret@AtlasGeneticsOncology.org. solenoide adn definicion. modelo solenoide adn. empaquetamiento del adn solenoide