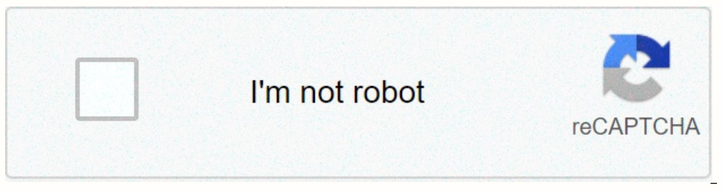

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Difference between chromosome and chromatid brainly

Sexual reproduction results in endless possibilities of genetic variation. In other words, sexual reproduction results in offspring that are genetically unique. They are different from both parents and also from each other. This is done for a number of reasons. All these mechanisms working together result in an amazing amount of potential variation. Every human couple, for example, has the potential to produce more than 64 trillion genetically unique children. No wonder we're all different! Cross-over occurs during prophase I, and it is the exchange of genetic material between non-sister chromatids of homologous chromosomes. Recall during prophase I, homologous chromosomes line up in pairs, gene-by-gene down their entire length, forming a configuration with four chromatids, known as a tetrad. At this point, the chromatids are very close to each other and some materials from two chromatids switch chromosomes, that is, the material is broken off and reattaches at the same position on the homologous chromosome (Figure \(\PageIndex{2} \)). This exchange of genetic material can happen many times within the same pair of homologous chromosomes, creating unique combinations of genes. This process is also called recombination. Image \(\PageIndex{2}\): Crossover. A mother's string of DNA is shown in red. A paternal piece of DNA is shown in blue. Crossing over produces two chromosomes that have not previously existed. The process of recombination involves breakage and reconnect of parent chromosomes (M, F). This results in the generation of new chromosomes (C1, C2) that share DNA from both parents. (CC NO LATER THAN 2.5; David Eccles (Gringer) via Wikimedia). Image \(\PageIndex{3}\): Crossover between homologous chromosomes Crossover occurs between non-sister chromatolls of homologous chromosomes. The result is an exchange of genetic material between homologous chromosomes. (CC THROUGH 4.0 via OpenStax College). During prophase I, chromosomes condense and become visible inside the nucleus. When the core envelope begins to break down, move homologous chromosomes closer together. The synaptonemal complex, a dithering of proteins between the homologous chromosomes, is formed in specific locations, which spread to cover the entire chromosomes. The narrow pairing of the homologous chromosomes is called synapsis. In synapsis, the genes of the chromatids in the homologous chromosomes are aligned. The synaptonemal complex also supports the exchange of chromosomal segments between non-sister homologous chromatids in a process called crossover. Crossover events are the first source of genetic variation produced by meiosis. A single crossover event between homologous non-sister chromatids leads to an exchange of DNA between chromosomes. After crossover, the synaptone mal complex is broken down and the cohesin connection homologous couples are also removed. At the end of prophase I, the pairs are held together only at chiasmata; They are called tetrads because the four systerkroomatimes of each pair of homologous chromosomes are now visible. We all know what a chromosome is. (if you don't: Chromosomes are wire-like structures found inside the nucleus of any living organism.) But most of us don't know what a chromatid is. This article aims to help you understand the meaning and difference between Chromosome and Chromatid. The following illustration clearly illustrates the difference between the two in terms of their structure. The two-stranded structures are the chromaperiod, and the structure as a whole forms the chromosome. Difference between Chromosome and Chromatid A chromosome is a genetic material that has all the functions and properties of an organism. Originates from Greek, Chroma and Soma, which are translated into color and body respectively. Now, a chromosome consists of two strands that are identical to each other and these are called Chromatids. The major difference between Chromosome and Chromatid is summarized below. Difference between Chromosome and Chromatid Chromosome Chromatid Their function is to carry the genetic material Their main function is to enable the cells to duplicate A chromosome occurs throughout the cell's life cycle A chromatid, on the other hand created only when the cell passes through mitosis or meiosis stages Chromosomes are not exact copies of each other. A copy of the gene comes from each parent Sister Chromatids are identical copies of each other Chromosomes have centromeres It is the sister Chromatids only that has centromeres DNA utilized during macromolecule synthesis (synthesis of complex proteins) DNA is not utilized during macromolecule conclusion A chromosome is made up two Identical Sister Chromatids. And each sister chromatids are united at the centromere. A copy of each chromosome is inherited from a male parent and a female parent and this explains the characteristic characteristics held by their offspring (such as facial features reminiscent of the parents). Related Links: Changes affecting the structure of chromosomes can cause problems with the growth, development and function of the body's system. These changes can affect many genes along the chromosome and interfere with the proteins made from these genes. Structural changes can occur during the formation of eggs or sperm, in early fetal development, or in any cell after birth. Pieces of DNA can be rearranged within a chromosome or transferred between two or more chromosomes. The effects of structural changes depend on their size and location, and whether any genetic material is won or lost. Some changes cause medical problems, while others may have no effect on a person's health. Changes in chromosome include: Migration rings A translocation occurs when one piece of a chromosome breaks off and attaches to another chromosome. This type of reorganization is described as balanced if no genetic material is gained or lost in the cell. If there is a gain or loss of genetic material, the migration is described as unbalanced. Deletions Deletions occur when a chromosome breaks and some genetic material is lost. Deletions can be large or small, and can occur anywhere along a chromosome. Duplication Duplication occurs when part of a chromosome is copied (duplicated) too many times. This type of chromosomal change results in additional copies of genetic material from the duplicate segment. Inversions An inversion involves the breakage of a chromosome in two places; the resulting piece of DNA is reversed and re-inserted into the chromosome. Genetic material may or may not be lost as a result of chromosome breaks. An inversion involving the narrowing point of the chromosome (centromer) is called a pericentric inversion. An inversion that occurs in the long (q) arm or short (p) arm and does not involve centromer is called a paracentric inversion. Isochromosomes An isochromowhich is a chromosome with two identical arms. Instead of a long (q) arm and a short (p) arm, an isochromosome has two long arms or two short arms. As a result, these abnormal chromosomes have an extra copy of some genes and are missing copies of other genes. Dicentric chromosomes Unlike normal chromosomes, which have a single narrowing point (centromer), a dicentric chromosome contains two centromers. Dicentric chromosomes are the result of the abnormal fusion of two pieces of chromosome, each of which includes a centromere. These structures are unstable and often involve a loss of certain genetic materials. Ring chromosomes Ring chromosomes usually occur when a chromosome breaks in two places and the ends of the chromosome arms merge to form a circular structure. The ring may or may not include the narrowing point of the chromosome (centromere). In many cases, genetic material near the ends of the chromosome is lost. Many cancer cells also have changes in their chromosome structure. These changes are not inherited; they occur in somatic cells (cells other than eggs or sperm) during the formation or progression of a cancerous tumor. The human body consists of trillions of somatic cells with the capacity to divide into identical daughter cells facilitating organism growth, repair, and response to the changing environment. This process is called mitosis. In gamete production, another form of cell division occurs called meiosis. The result of meiosis is the creation of four daughter cells, either sperm or egg cells, through the reduction division resulting in a haploid complement of chromosomes in each gamete. When fertilizing is is The sperm cell nucleus merges with the haploid egg cell nucleus, which restores the diploid chromosomal complement and confirms the formation of the zygote. During the anaphase of the cell cycle, chromosomes are separated to opposite ends of the cell to create two daughter cells. Nondisjunction is the failure of chromosomes to separate, which produce daughter cells with abnormal numbers of chromosomes. [1] [2] [3] The genome is encoded by the chemical sequence of DNA nucleotides in our cells. In periods of cell growth, histon proteins around DNA are acetylated causing less interaction between DNA and histone protein. This opened DNA is called euchromatin and gives transcription enzymes access to DNA. Before periods of cell division, deacetylated proteins allow the formation of a condensed form of DNA called heterochromatin are deacelated. Somatic human cells contain 23 paired chromosomes or 46 total chromosomes. Forty-six are considered diploid numbers (2n), while 23 are considered haploid numbers (1n) or half the diploid number. Aneuploidy refers to the presence of an abnormal number of chromosomes. Monosomy (n-1) is a form of aneuploidy characterized by missing a single chromosome resulting in 45 total chromosomes. Trisomy (n+1) is another form of aneuploidy that has an extra chromosome resulting in 47 total chromosomes. Each type of aneuploidy can be attributed to nondisjunction during mitosis or meiosis. [4] [5] [6] There are 2 parts to the cell cycle: interphase and mitosis/meiosis. Interphase can be further divided into growth 1 (G1), synthesis (S), and growth 2 (G2). During the G phases, the cell grows by producing different proteins, and during the S phase the DNA is replicated so that each chromosome includes 2 identical sister chromate times. Mitosis contains 4 phases: profas, metaphase, anaphase, and telophase. In prophase, the core envelope breaks down and chromatin condenses. In the metaphase line the chromosomes up along the metaphase plate, and microtubules attach to the kinetochors of each chromosome. In anaphase, chromatids separate and are drawn by microtubules to opposite ends of the cell. Finally, in telophase, the core envelopes recur, the chromosomes unwind in chromatin, and the cell undergoes cytokines, which divides the cell into 2 identical daughter cells. Meiosis goes through all 4 phases of mitosis twice, with modified mechanisms that ultimately create haploid cells instead of diploid. One modification is in meiosis I. Homologous chromosomes are separated instead of sister chromatids, creating haploid cells. It is during this process where we see cross over and independent assortment that leads to the increased genetic diversity of offspring. Meiosis II progresses in the same way as mitosis, but with haploid number of chromosomes, ultimately creating 4 cells are all genetically distinct from the original cell. Nondisjunction may occur during anafas of mitosis, meiosis I, or meiosis II. During anafas, sister chromatids (or homologous chromosomes for meiosis I), will separate and move to opposite poles of the cell, drawn by microtubules. In nondisjunction, the separation fails to occur causing both sister chromatids or homologous chromosomes to be drawn to a pole of the cell. Mitotic nondisjunction may occur due to inactivation of either topoisomeras II, condensation, or separase. This will result in 2 aneuploid daughter cells, one with 47 chromosomes (2n+1) and the other with 45 chromosomes (2n-1). Non-discrimination in meiosis I occur when tetrads fail to separate during anaphase I. At the end of meiosis I there will be 2 haploid daughter cells, one with n+1 and the other with n-1. Both of these daughter cells will then proceed to divide once more into meiosis II, producing 4 daughter cells, 2 with n+1 and 2 with n-1. Non-discrimination in meiosis II results from the failure of sister chromatids to separate during anafas II. Since meiosis I continued without error, 2 of the 4 daughter cells will have a normal complement of 23 chromosomes. The other 2 daughter cells become aneuploida, one with n+1 and the other with n-1. In-utero, the diagnosis of fetal chromosomal aneuploidy can be made by performing cytogenetic analysis of fetal cells, usually obtained by amniocentesis or chorionic villus sampling. The fetal chromosomal supplement is analyzed by performing a karyotype test, counting the chromosomes, and analyzing under light microscopy, all while looking for abnormalities in chromosome numbers or structure. Many prenatal screening tests are available to help provide an age-adjusted risk for fetal chromosomal aneuploidy by asalyzing various markers or cell-free fetal DNA in maternal serum. [7] [8] With in vitro fertilization (IVF), testing can also be performed prior to implantation through preimplantation genetic diagnosis (PGD), polar body diagnosis (PBD), or blastomere biopsy. PGD is a technique used to identify normal embryos that will be implanted in the mother, but technically demanding and with additional costs compared to prenatal diagnosis. PBD can detect maternally derived aneuploidies and is relatively quick to perform when compared to PGD. Finally, a blastomere biopsy can be obtained prior to implantation for genetic analysis. However, blastomere biopsy places the growing embryo at greater risk and therefore is not currently a recommended standard for practice. Mitotic nondisjunction can cause somatic mosaicism, with the chromosome imbalance only reflected in the direct offspring of the original cell where nondisjunction occurred. This can cause some forms of cancer, including retinoblastoma. Meiotic nondisjunction is of greater clinical because because aneuploidies are incompatible with life. However, some will result in viable offspring with a spectrum of developmental disorders. Autosomal TrisomiesPatau Syndrome: Trisomy of Chromosome 13Clinical Features: Rocker-bottom feet, microphthalmia (abnormally small eyes), microcephaly (abnormally small head), polydactyly, holoprosencephaly, cleft lip and palate, congenital heart disease, and severe intellectual impairment. Life expectancy is rarely longer than one year. Edwards Syndrome: Trisomy of chromosome 18Clinical Features: Rocker-bottom feet, low set of ears, micrognathia (abnormally small jaw), knotted hands with overlapping fingers, congenital heart disease, and severe intellectual impairment. Life expectancy is normally less than one year. Down syndrome: Trisomy of chromosome 21The most common viable aneuploidy. Clinical features: Single palmar folds, flat facies, prominent epicanthal folds, duodenal atresia, congenital heart disease, Hirschsprung disease, intellectual disability. Especially increased risk of developing Alzheimer's disease or leukemia. Life expectancy is about 60 years. Sex Chromosome TrisomiesKlinefelter Syndrome: An additional X chromosome in a male (47, XXY)Clinical features: Long, long extremities, gynecomastia, female hair distribution, testicular atrophy, developmental delay. Triple X syndrome: An extra X chromosome in a female (47, XXX)Clinical features: Phenotypically normal, some with unusually long stoning. X chromosomes are disabled as coniferous bodies. Therefore, 2 additional barr bodies are seen, but no clinical abnormalities results. XYY syndrome: An extra Y chromosome in a male (47, XYY)Clinical features: phenotypic normal, unusually high sensing. Most cases go undetected due to lack of clinical abnormalities. Sex Chromosome MonosomiesTurner Syndrome: Monosomy of the X chromosome in a female (45, X)The only chromosomal monosomy compatible with life. Clinical characteristics: Unusually short stature, shield breast, congenital heart disease, webbing, horseshoe kidney, ovarian dysgenesis. The most common cause of primary amenorrhea. No Barr bodies are visible. Further Training/Review Questions1.Kaser D. Status of Genetic Screening in Recurrent Pregnancy Loss. Obstetrics Gynecol Clin North Am. 2018 Mar;45(1):143-154. [PubMed: 29428282] 2.Skuse D, Printzlau F, Wolstencroft J. Sex chromosome aneuploidies. Handb Clin Neurol. 2018;147:355-376. [PubMed: 29325624] 3.Kurtas NE, Xumerle L, Leonardelli L, Delledonne M, Brusco A, Chrzanowska K, Schinzel A, Larizza D, Guerneri S, Natacci F, Bonaglia MC, Reho P, Manolakos E, Mattina T, Soli F, Provenzano A, Al-Rikabi AH, Errichiello E, Nazaryan-Petersen L, Giglio S, Tommerup N, Liehr T, Zuffardi O. Small superfluous marker chromosomes: A legacy of trisomi rescue? Hum Mutat. 2019 Feb;40(2):193-200. [PubMed: 30412329] 4.Ushijima K, Yatsuga S, Matsumoto T, Nakamura A, Fukami Kagami M. A A short-statured girl with 47.XX, + 14/46,XX,upd(14)mat, mosaicism. J Hum Genet. 2018 Mar;63(3):377-381. [PubMed: 29311684] 5.Saito TT, Mr Colaiácovo MEP. Regulation of Crossover Frequency and Distribution during meiotic recombination. Cold Spring Harb Symp Quant Biol. 2017;82:223-234. [PMC free article: PMC6542265] [PubMed: 29222342] 6.Li X, Liu Y, Yue S, Wang L, Zhang T, Guo C, Hu W, Kagan KO, Wu Q. Uniparental disomy and prenatal phenotype: Two case reports and review. Medicine (Baltimore). 2017 Nov;96(45):e8474. [PMC free article: PMC5690727] [PubMed: 29137034] 7.Coppedè F. Risk factors for Down syndrome. Arch Toxicol. 2016 Dec;90(12):2917-2929. [PubMed: 27600794] 8.Soellner L, Begemann M, Mackay DJ, Grønskov K, Tümer Z, Maher ER, Temple IK, Monk D, Riccio A, Linglart A, Netchine I, Eggermann T. Latest advances in Imprinting Disorders. Clin Genet. 2017 Jan;91(1):3-13. [PubMed: 27363536] 27363536]