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# COGNIZE HUMAN THROUGH IMPRINTING OR FINGER PRINTING AND DNA-FINGER PRINTING

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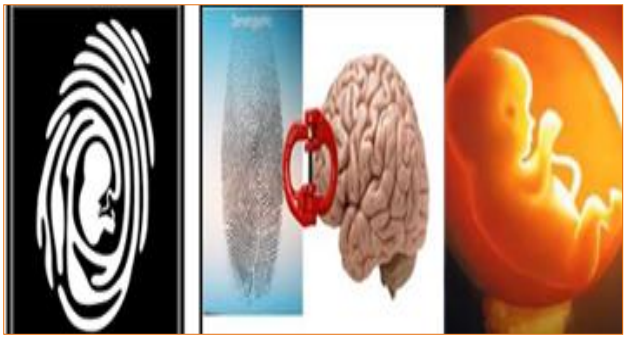
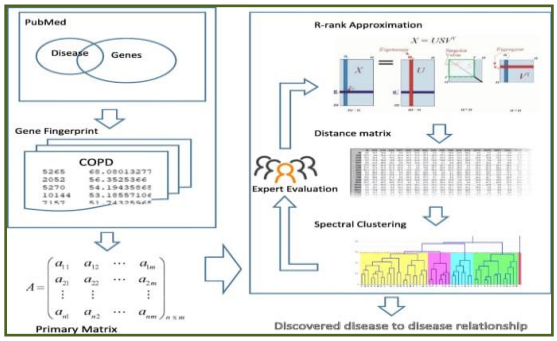
**Abstract:** Independent of the method of visualization through the physical pattern of finger is known as imprint or finger print where as derive the pattern through the DNA profiling is called DNA fingerprinting. Both are scientifically true tools. Imprinting or finger printing based on psychological and DNA finger printing based on gene; building blocks or coding of A-T/ C-G (adenine-thymine / cytosine-guanine). Human finger print forms when a baby is 13th to 21st week in her mother wombs (in pregnancy time). The fingerprints reveal to us what we need and how we learn, transforming our lives through a holistic education approaches. Discover our own aptitudes, interest, passion, good and bad qualities and improving or solution behavior are come to now through Dermatoglyphics Multiple Intelligent Test (DMIT). DNA profiling or Molecular Fingerprinting or DNA typing or Genetic Fingerprinting is the process of determining an individual's DNA characteristics. DNA analysis intended to identify a species, rather than an individual, is called DNA bar coding. The different sequence is the same as the word 'POST' has a different meaning from 'STOP 'or 'POTS' even though they use the same letters. Although 99.9% of human DNA sequences are the same in every person, enough of the DNA is different to distinguish one individual from another, unless they are monozygotic twins. In 1984 professor Alec Jeffreys, Leicester University first invented DNA finger printing. Finger print created by the friction ridge structure and identification by dactyloscopy. In 1926 Harold Cummins, the father of Dermatoglyphics coined the term. DMIT is the combined study of anthropology, genetics and embryology, psychology and neuro science.

**Key words:** Physical pattern, DNA profiling, building blocks, holistic education, Dermatoglyphics Multiple Intelligent Test, sequence, genetic, molecular finger print, dactyloscopy.

**Introduction:** Genome-wide association studies found single nucleotide polymorphisms within the gene ADAMTS9-AS2 on 3p14.1, which appeared to have an influence on the whorl pattern on all digits [1]. In February 2023 a study identified WNT, BMP and EDAR as signaling pathways regulating the formation of primary ridges on fingerprints, with the first two having an opposite relationship established by a Turing reaction-diffusion system [2,3,4]. The general characteristic of gene determine patterns is the slightly difference than the DNA fingerprinting. The composition of finger prints consists of water (95-99%), organic compounds (such as amino acids, proteins, glucose, lactase, urea, pyruvate, fatty acids and sterols) and inorganic compounds (such as chloride, sodium, potassium and iron) [5]. A very rare medical condition, adermatoglyphia, is characterized by the absence of fingerprints. Affected persons have completely smooth fingertips, palms, toes and soles, but no other medical signs or symptoms [6]. A 2011 study indicated that adermatoglyphia is caused by the improper expression of the protein SMARCAD1[7]. The condition has been called immigration delay disease by the researchers describing it, because the congenital lack of fingerprints causes delays when affected persons attempt to prove their identity while traveling [6]. Only five families with this condition had been described as of 2011 [8].



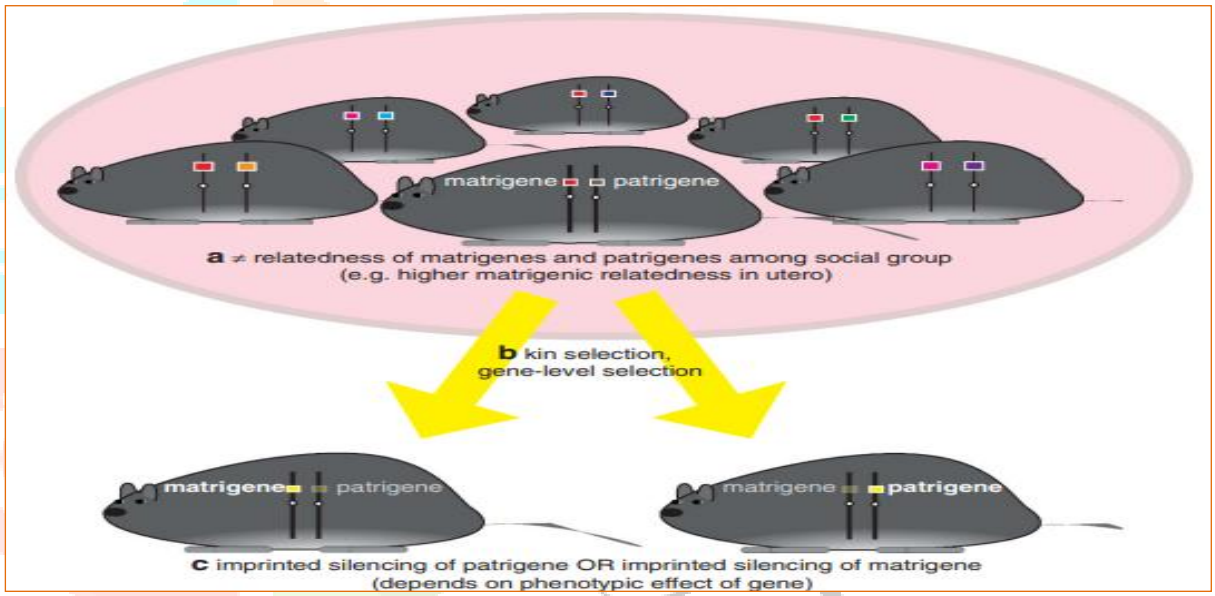
Figer:1: Finger print develop within 13th -21st week pregnancy.




Figer:2: Gene fingerprint model for literature based detection.

**Theories for the evolution of genomic imprinting:** It has motivated the development of numerous theories of the epigenetic phenomenon of genomic imprinting are described under the following

.**(a). Haig and colleagues' kinship theory:** The kinship theory is a kin selection model, where matrigenic and patrigenic alleles experience different patterns of relatedness in the social environment (for example, individuals tend to encounter more matrilineal than patrilineal relatives), and as a result, their expression has different consequences for their respective inclusive fitnesses [9]. The kinship theory focuses on genes whose expression level governs the extent of some physiological or behavioral interaction between individuals [10].




Figer-3: The kinship theory of genomic imprinting has two prerequisites: first, epigenetic marks that differentiate matrigenes from patrigenes; second, a difference in the relatedness of matrigenes and patrigenes to the social group.

Both strong and weak version are expressing by the sketch of logic. This version is depending in the family distinction of evolutionary biology between origin and maintenance of consequent of parental antagonism. This theory focused on multiple mating for matrigenes and patrigenes. The kinship theory can be applied to any behavior that influences the fitness of kin and is not limited to the solicitation behaviors of offspring [10].

**(b) The sexual antagonism theory:** The sexual antagonism theory for the evolution of genomic imprinting relies on sex-specific selection pressure [11]. Below the figure no-4, (a,b) The sexual antagonism theory of genomic imprinting starts with sexually antagonistic selection, which produces different allele frequencies, shown as pie charts, for genes of maternal and paternal origin. (c, d) Natural selection favors individuals that are able to express the fitter of the two alleles at a locus, which for males will be the patrigenic allele and for females will be the matrigenic allele. (In addition, the sexual antagonism theory may predict matrigenic or patrigenic expression in both sexes, such that the expressed allele derives from the parental sex that experiences stronger selection pressure. This scenario is not depicted) [10].

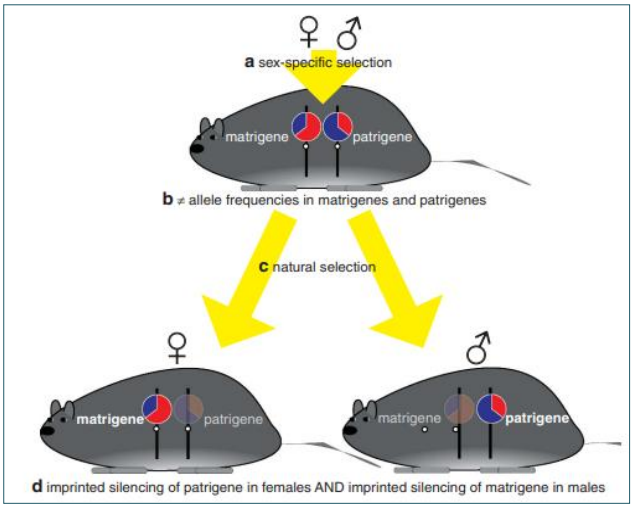



Figure-4: It express the sexual antagonism theory.

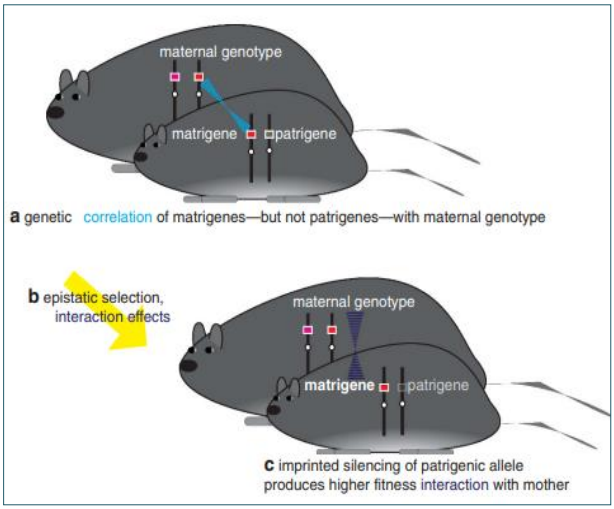



Figure-5: The maternal offspring co-adaptation theory.

In this theory the two alleles at a diploid locus carry, on average, different information or instructions, and one of these copies provides more adaptive information than the other. Actually this theory completely depends on the sex-specific selection pressure on a gene**.**

**(c) The maternal offspring co-adaptation theory:** The sexual antagonism and maternal–offspring co-adaptation theories view genomic imprinting as a mechanism to modify the resemblance of an individual to its two parents, with imprinting evolving to increase the probability of expressing the fitter of the two alleles at a locus [10]. This theory expressed to more successful social interactions by coordinating the traits expressed by interacting individuals. It is important for maintaining species persistence and keeping biodiversity.

**Theories of evolution of DNA-fingerprinting**: Most of our DNA is identical to each other. There are inherited regions of our DNA that can vary from person to person (such variations are termed as polymorphisms). The class of polymorphisms is known as tandem repeats, which vary within the individual of the species. This forms the basis of genetic fingerprinting. Tendon repeats occur in DNA when a pattern of two or more nucleotide is repeated and the repetitions are adjacent to each other and form different density band on density gradient centrifugation satellite. Example- A-T-T-C-G-A-T-T-C-G-A-T-T-C-G. Tandem repeats (i) satellite DNA, (ii) microsatellite, (iii) minisatellite. Determining the order of bases in a section of DNA is known as DNA sequencing. The main purpose of DNA sequencing are deciphering code of life, detecting mutations, typing microorganisms, identifying human halo types, designating polymorphisms. Methods of DNA sequencing techniques are under the following.

**(i) Maxam and Gilbert chemical degradation method:** On 1977 scientist A.M.Gilbert and W.Gilbert are invented the base of chemical sequencing treatment of DNA cuts into fragments and monitoring of sequences by high resolution gel electrophoresis and detection of the labeled fragments by autoradiography. Chemical treatment generates break at a small proportion of one or two of the four nucleotide bases in each of four reactions (G, A+G, C, C+T). After visualize the fragments, the gel is exposed to X-ray film for autoradiography, yielding a series of dark bands each corresponding to a radiolabelled DNA fragment, from which the sequence may be inferred.

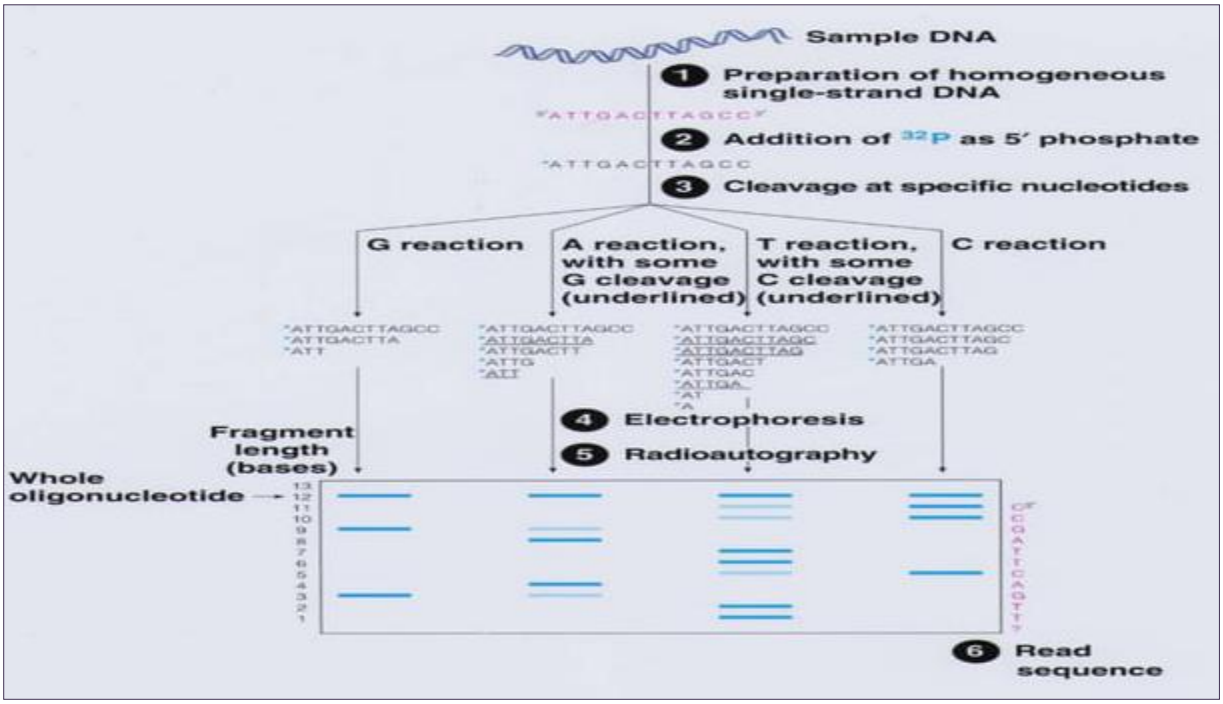



Figure:6- Maxam and Gilbert gene sequencing theory.

**(ii) Sanger methods or Chain Termination or Dideoxy method:** Fredrick Sanger adopted the primer extension strategy to develop more rapid DNA sequencing methods the MRC Centre, Cambridge, UK and published a method for "DNA sequencing with chain terminating inhibitors" in 1977[12]. After that he got Nobel Prize on 1980.The principle of DNA Sequencing is - the sequence of a single stranded DNA molecule is determined by enzymatic synthesis of complementary polynucleotide chains. This chains terminating at specific nucleotide position, separate by gel electrophoresis and read DNA sequence. Primer, DNA template, DNA polymerase, ddNTPs, dNPTs (A,T,C,G) are the components and there are 4 steps such as denaturation, primer attachment and extension of bases, termination and poly acrylamide gel electrophoresis of Sanger method.

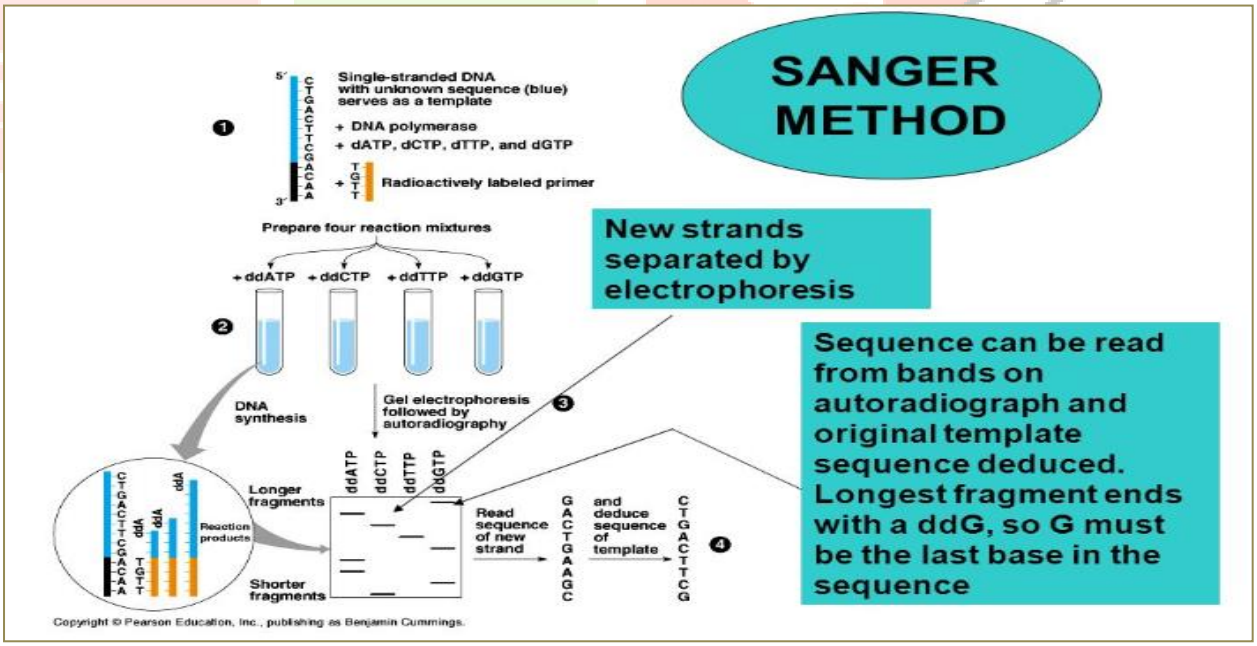
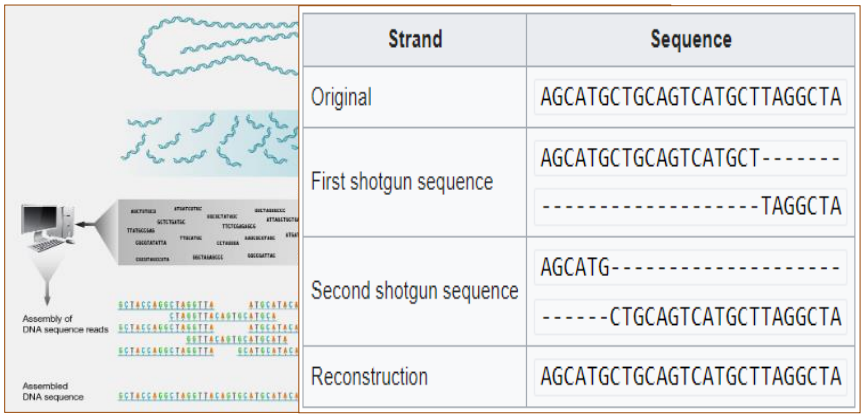



Figure:7- Sanger methods or Chain Termination or Dideoxy method.

**(iii)Shotgun cloning or sequencing:** It is a method used for sequencing long DNA strands or the whole genome. In this method, DNA is broken up randomly into numerous small segments and overlapping regions are identified between all the individual sequences that are generated. Multiple overlapping reads for the target DNA are obtained by performing several rounds of this fragmentation and sequencing. The computer programs then use the overlapping ends of different reads to assemble them into a continuous sequence. This process was first used successfully with the bacterium *Haemophilus influenzae*. Craig Venter used this method to map the Human genome project in 2001.




| Strand | Sequence |
|---|---|
| Original | AGCATGCTGCAGTCATGCTTAGGCTA |
| First shotgun sequence | AGCATGCTGCAGTCATGCT-------<br>------------------TAGGCTA |
| Second shotgun sequence | AGCATG--------------------<br>------CTGCAGTCATGCTTAGGCTA |
| Reconstruction | AGCATGCTGCAGTCATGCTTAGGCTA |

Figure:8 -: DNA sequencing from original to reconstructions (Shotgun sequencing method)

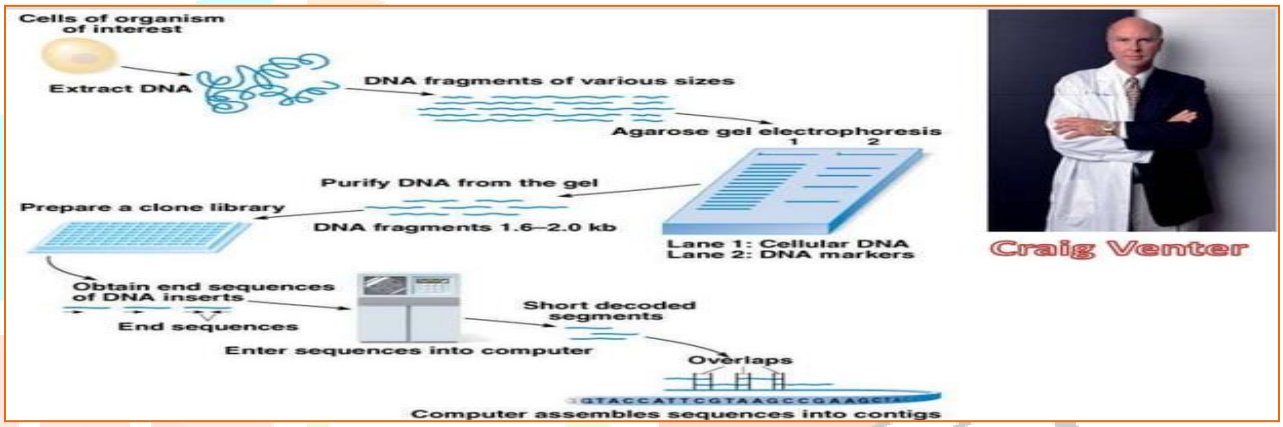



Figure-9:-Shotgun cloning or sequencing Procedure.

**(iv)2nd or Next Generation Sequencing:** Next Generation Sequencing (NGS) technology is similar to Capillary Electrophoresis (CE) sequencing, where DNA polymerase catalyzes the incorporation of fluorescently labeled deoxyribonucleotide triphosphates (dNTPs) into a DNA template strand during sequential cycles of DNA synthesis. During each process, the nucleotides are identified by fluorophore excitation at the addition of each nucleotide. The major difference is that instead of sequencing a single DNA fragment, NGS simultaneously extends this process across millions of fragments. 1st generation or Sanger sequencing involves the fragmentation and cloning of the target DNA into plasmid vectors. The DNA is then sequenced using a cyclic chain termination method with either radio isotopically labelled or fluorescently labelled dNTPs. The 2nd generation sequencing technologies are all based on sequencing by synthesis. Two common methods used are emulsion PCR and bridge PCR.3rd generation sequencing methods have been developed by many different companies and are based on different technologies. They all involve more direct examination of the target DNA.

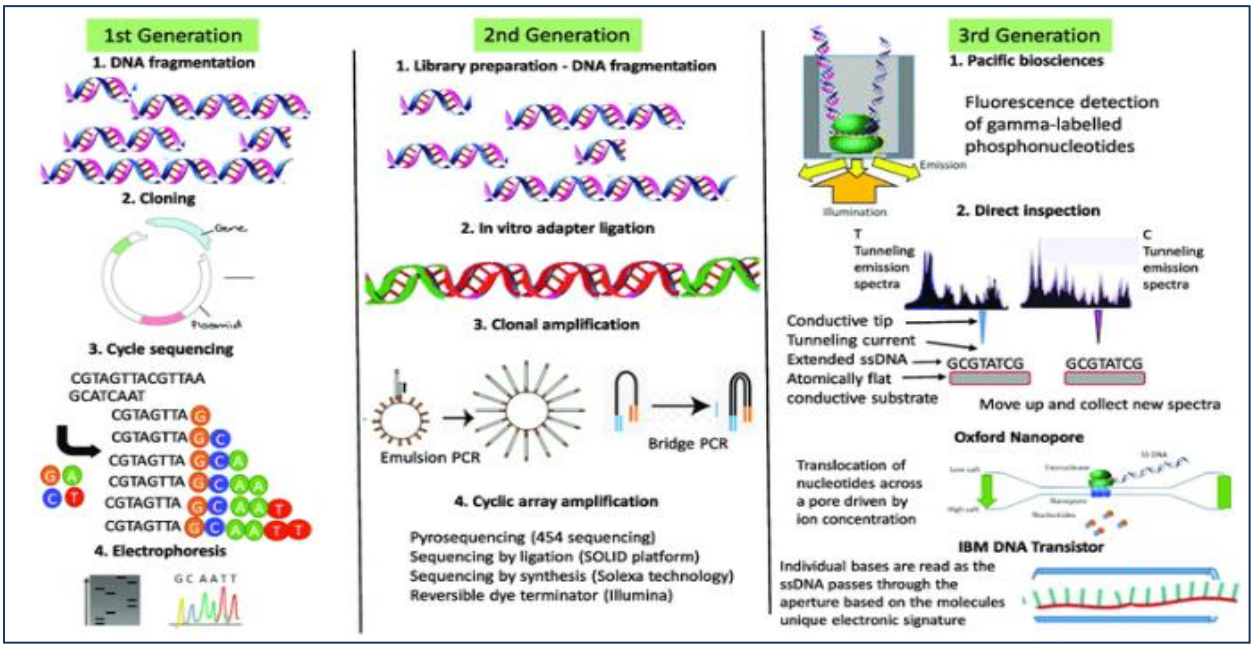



Figure 10: Compare 1st, 2nd,and 3rd generation of DNA sequencing.

**Methods or Styles of Fingerprinting**: DMIT=Dermatoglyphics +M.Intelligence. Dermatoglyphics is based on the most advanced scientific and medical research as a root. It refers to the growth of the human hands, finger print, protruding ridges on the soles of feet and tongue. The different types of styles are (such as – Learning style, Personality test, Intelligence quotients and Multiple intelligences). Dermatoglyphics is auto-assessment interface that it uniquely adaptive, intuitive and responsive to any child's unique needs and skill-set. The ideal education needs to be holistic and should facilitate the involvement of multiple intelligence based learning. The equal participation from both left and right brain are fostering creative and application of thinking process. For this reason parents should be focused to recognize the latent talent within the child.

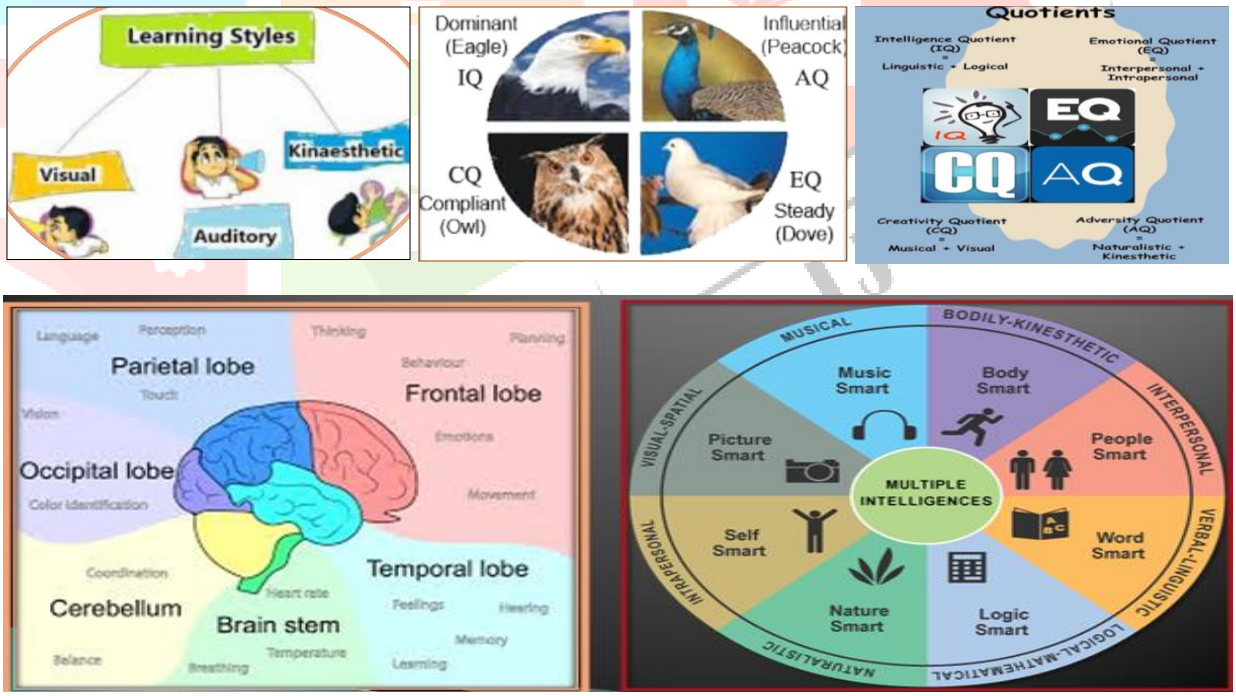



Figure:11 –Different types of knowledge are find out after getting DMIT.

There are four types of identification are found in our fingers. These are Arch(A), Whorl(W), Loop(Ulnar &Radial). So imprinting identification is an exceptionally flexible and versatile method of human identification. Radiation therapy system and company's access-control system are two main methods to

identify the unique fingerprint. The dryness of the skin, the surrounding temperature and the force are factors for influence hazards. Deltabit is one of the help to identify patients in healthcare and as well as replace the door key with fingerprint identification.

**Methods or types of DNA fingerprinting**: Simple Sequence Length Polymorphosis (SSLPS), Simple Sequence Repeats (SSRs), Inter Simple Sequence Repeats (ISSRs), Single Nucleotide Polymorphism (SNP) are the different types of DNA fingerprinting. SSLPs are displayed only length variations. Such as micro satellites or STRs have dinucleotide or tetranucleotide units. Where as minisatellites or VNTRs have the repeat unit which is up to25bp in length. SSRs is found in the genome of all eukaryotes. The nucleotide repeat sequence such as (dC-dA)n, (dG-dT)n is reported to occur in the human genome as many as 50,000 times with 'n' varying from 10-60. Primers can be extended outside or inside the ISSR in which case a unique region most likely will be amplified.

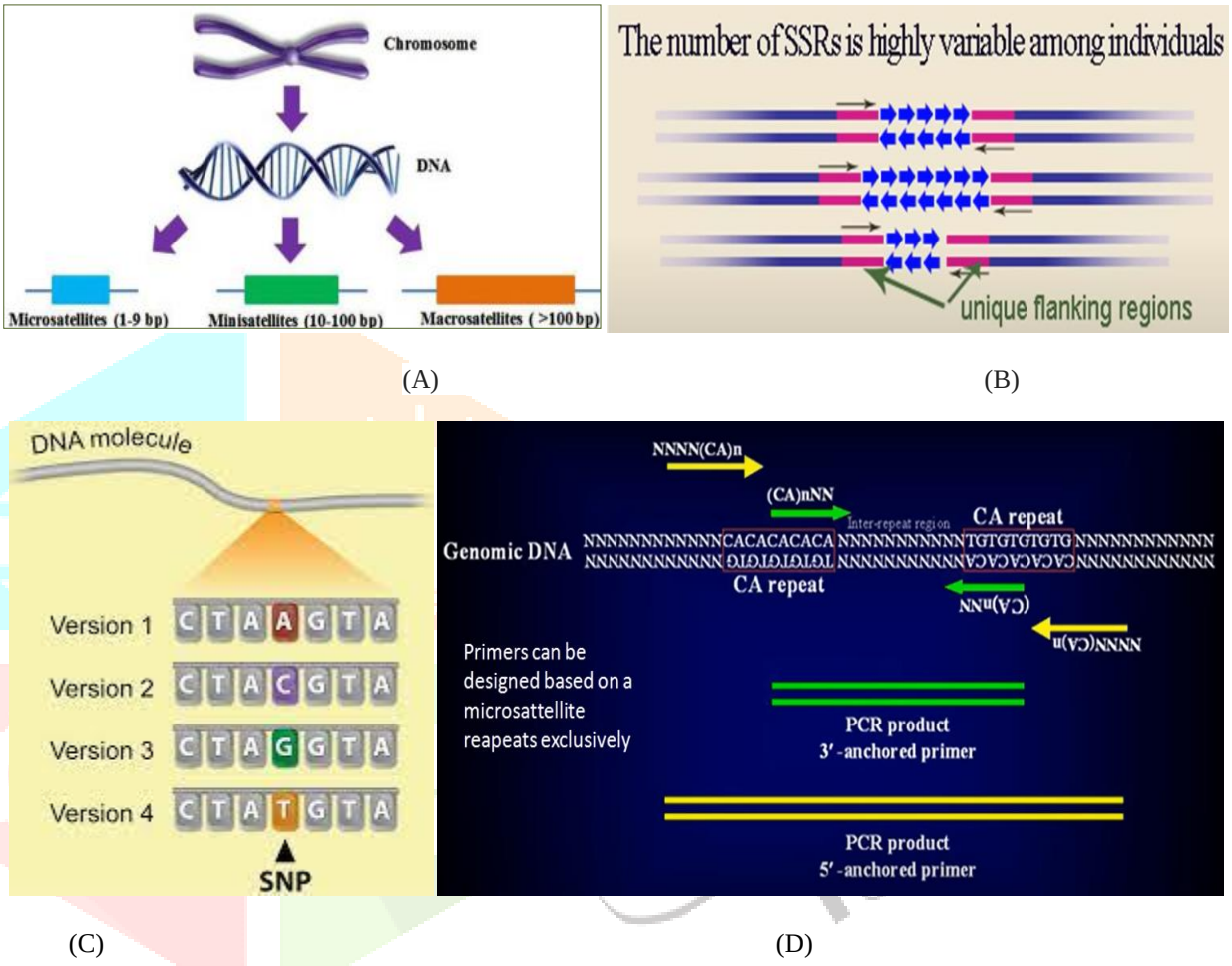
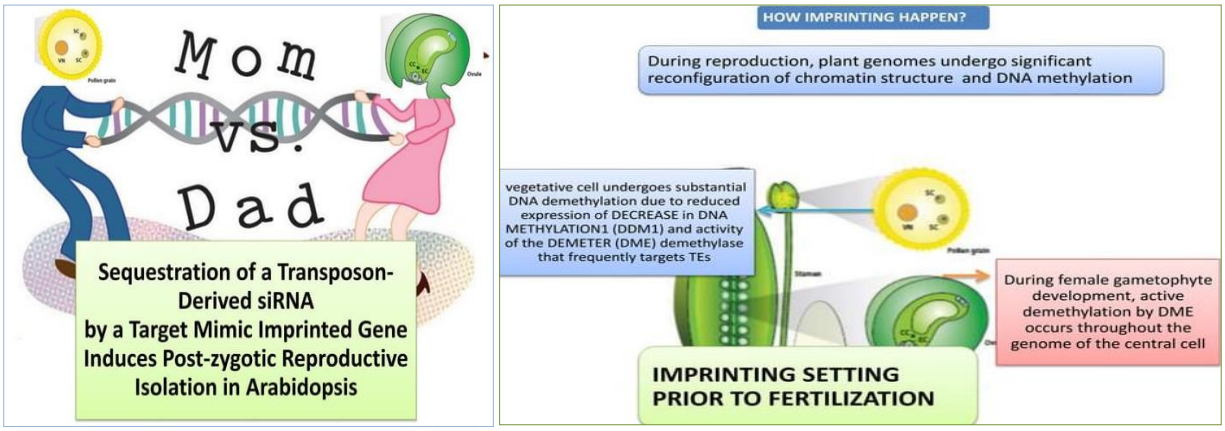



Figure:12- (A)SSLPs, (B)SSRs, (C)SNP, & (D)ISSR are types of DNA fingerprinting.

SNPs are found in the DNA between genes. They can act as biological markers, helping scientists locate genes that are associated with disease. When SNPs occur within a gene or in a regulatory region near a gene, they may play a more direct role in disease by affecting the gene's function.

**Mechanisms:** at present studies numerous mechanisms that regulate genomic imprinting in the mammalian genome and basic difference is regions of allele-specific differential methylation (DMRs) are present in all imprinted genes. Differential methylation is erased in germ cells at an early stage of their development, and germ-line-specific methylation imprints in DMRs are reestablished around the time of birth [13]. After fertilization, differential methylation is retained in core DMRs despite genome-wide demethylation and de novo methylation during preimplantation and early postimplantation stages [13]. Direct repeats near CG-rich DMRs may be involved in the establishment and maintenance of allele-specific methylation patterns. Imprinted genes tend to be clustered; one important component of clustering is enhancer competition, whereby promoters of linked imprinted genes compete for access to enhancers [13]. So the catalogue the

genome are-wide patterns of epigenetic marks (i) A Primer on Epigenetics, DNA Methylation and Histone Modifications (ii) Genomic Imprinting and Targeting DNA Methylation (iii) DNA Methylation and Gene Repression (iv) DNA Methylation Confer Effects on Gene Expression [14]. The alleles of imprinted genes are marked epigenetically at discrete elements termed imprinting control regions (ICRs) with their parental origin in gametes through the use of DNA methylation, at the very least [15]. Imprinted gene expression is subsequently maintained using noncoding RNAs, histone modifications, insulators, and higher-order chromatin structure [15].




(A) (B)

Figure:13- Both (A) & (B) are showing genomic imprinting is an epigenetic phenomenon.

DNA methylation can attract proteins that bring about gene repression through recruitment of chromatin modifiers. A group of proteins, collectively referred to as methyl binding proteins (MBPs) have been characterized and shown to specifically bind to methylated, but not unmethylated, DNA[16,17,18,19,20,21]. MBPs are known to interact with histone modifiers such as HDACs, e.g., in forming complexes, such as the nucleosome remodeling deacetylase (NuRD) complex, which through their histone deacetylase activity and subsequent chromatin condensation bring about gene repression[22,23,24,25,26,27]. Certain proteins may interact with DNA in a methylation dependent manner. Here, DNA methylation may be refractory to the binding of proteins, such as transcription factors or other regulatory proteins [28,29,30] that are necessary for gene expression. In the H19/Igf2 imprinted cluster, the protein coding gene Igf2 is expressed from the paternally inherited allele[31]. This expression pattern is dependent on the regional ICR[32], on its differential methylation[33,34,35] and on the insulator protein CTCF binding to the ICR. On the unmethylated maternal allele, CTCF can bind, while its binding is inhibited on the methylated paternally inherited chromosome [36, 37,38, 39 ]—thus CTCF binding to DNA is methylation-sensitive.

DNA extraction, DNA cutting, Gel electrophoresis, Southern hybridization and Autoradiography are steps of DNA fingerprinting methodology. Blood, hair, saliva,semen, body tissue cells are the biological materials to use for DNA profiling.

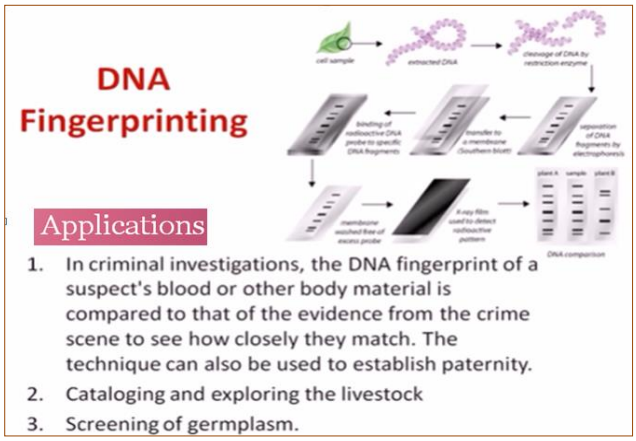



Figure:14-Application and process of DNA fingerprinting.

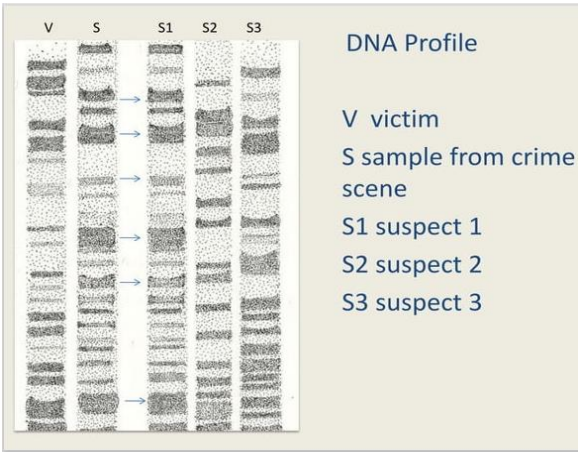



Figure:15-DNA profiling for identification of parent.

**Conclusion:** The robust genetic approaches applied to the regulation of imprinting have allowed it to be an excellent hypothesis-driven model to investigate and understand the epigenetic control of genome regulation [14]. DNA fingerprinting is using in forensic science, drug analysis, first extraction of DNA from plant cells. So imprinting and DNA fingerprinting both are vice versa to recognize inimitable of human generalize.

**Reference:**

1. Martin, N. G., & Medland, S. E. (2015). Yvonne YW Ho, David M Evans 3, 4, Grant W Montgomery, Anjali K Henders, John P Kemp 3, 4, Nicholas J Timpson 3, Beate St Pourcain 5, 6, Andrew C Heath 7, Pamela AF Madden 7, Danuta Z Loesch 8, Dennis McNevin 9, Runa Daniel 10, George Davey-Smith 3.
2. Glover, J. D., Sudderick, Z. R., Shih, B. B. J., Batho-Samblas, C., Charlton, L., Krause, A. L., & Headon, D. J. (2023). The developmental basis of fingerprint pattern formation and variation. *Cell*, *186*(5), 940-956.
3. "How fingerprints form was a mystery — until now". February 9, 2023. Retrieved February 15, 2023.
4. "Why don't identical twins have the same fingerprints? New study provides clues". *February 9, 2023.* doi:10.1126/science.adh0982.
5. Cadd, S., Islam, M., Manson, P., & Bleay, S. (2015). Fingerprint composition and aging: a literature review. *Science & Justice*, *55*(4), 219-238.
6. Burger, B., Fuchs, D., Sprecher, E., & Itin, P. (2011). The immigration delay disease: Adermatoglyphia–inherited absence of epidermal ridges. *Journal of the American Academy of Dermatology*, *64*(5), 974-980.
7. "The Mystery of the Missing Fingerprints". August 4, 2011. Archived from the original on February 16, 2016.
8. Al-Ahwal, M. S. (2012). Chemotherapy and fingerprint loss: beyond cosmetic. Oncologist 17 (2): 291-3. *Am J Hum Genet*, *89*(2), 302-307.
9. Haig D (2002). Genomic Imprinting and Kinship. Rutgers University Press: New Brunswick, NJ, USA.
10. Patten, M. M., Ross, L., Curley, J. P., Queller, D. C., Bonduriansky, R., & Wolf, J. B. (2014). The evolution of genomic imprinting: theories, predictions and empirical tests. *Heredity*, *113*(2), 119-128.
11. Day, T., & Bonduriansky, R. (2004). Intralocus sexual conflict can drive the evolution of genomic imprinting. *Genetics*, *167*(4), 1537-1546.
12. Ashish M,Parmar, Keyur D. Patel, Nilang S. Doshi, Girish M. Kapadiya, Bhavesh S. Patel and Dr. Dhrubo Jyoti Sen (2014), the research article " Correlation Approach Between Shotgun Sequencing With DNA Sequencing In Molecular Genomics, World Journal Of Pharmacy and Pharmaceutical Sciences, ISSN 2278-4357.
13. "Imprinting Mechanisms"- Miguel Constaˆncia, Benjamin Pickard, Gavin Kelsey, and Wolf Reik. Programme in Developmental Genetics, The Babraham Institute, Cambridge CB2 4AT, UK. Downloaded from genome.cshlp.org on July 7, 2015 - Published by Cold Spring Harbor Laboratory Press. ISSN 1054-9803/98.

14. Adalsteinsson, B. T., & Ferguson-Smith, A. C. (2014). Epigenetic control of the genome—lessons from genomic imprinting. *Genes*, *5*(3), 635-655.
15. Bartolomei, M. S. (2009). Genomic imprinting: employing and avoiding epigenetic processes. *Genes & development*, *23*(18), 2124-2133.
16. Klose, R.J.; Bird, A.P. Genomic DNA methylation: The mark and its mediators. Trends Biochem. Sci. 2006, 31, 89–97.
17. Meehan, R.R.; Lewis, J.D.; McKay, S.; Kleiner, E.L.; Bird, A.P. Identification of a mammalian protein that binds specifically to DNA containing methylated CpGs. Cell 1989, 58, 499–507
18. Lewis, J.D.; Meehan, R.R.; Henzel, W.J.; Maurer-Fogy, I.; Jeppesen, P.; Klein, F.; Bird, A. Purification, sequence, and cellular localization of a novel chromosomal protein that binds to methylated DNA. Cell 1992, 69, 905–914.
19. Hendrich, B.; Bird, A. Identification and characterization of a family of mammalian methyl-CpG binding proteins. Mol. Cell. Biol. 1998, 18, 6538–6547.
20. Hendrich, B.; Tweedie, S. The methyl-CpG binding domain and the evolving role of DNA methylation in animals. Trends Genet. 2003, 19, 269–277.
21. Bird, A.P.; Wolffe, A.P. Methylation-induced repression—Belts, braces, and chromatin. Cell 1999, 99, 451–454.
22. Jones, P.L.; Veenstra, G.J.C.; Wade, P.A.; Vermaak, D.; Kass, S.U.; Landsberger, N.; Strouboulis, J.; Wolffe, A.P. Methylated DNA and MeCP2 recruit histone deacetylase to repress transcription. Nat. Genet. 1998, 19, 187–191.
23. Nan, X.; Ng, H.-H.; Johnson, C.A.; Laherty, C.D.; Turner, B.M.; Eisenman, R.N.; Bird, A. Transcriptional repression by the methyl-CpG-binding protein MeCP2 involves a histone deacetylase complex. Nature 1998, 393, 386–389.
24. Ng, H.-H.; Zhang, Y.; Hendrich, B.; Johnson, C.A.; Turner, B.M.; Erdjument-Bromage, H.; Tempst, P.; Reinberg, D.; Bird, A. MBD2 is a transcriptional repressor belonging to the MeCP1 histone deacetylase complex. Nat. Genet. 1999, 23, 58–61.
25. Wade, P.A.; Gegonne, A.; Jones, P.L.; Ballestar, E.; Aubry, F.; Wolffe, A.P. Mi-2 complex couples DNA methylation to chromatin remodelling and histone deacetylation. Nat. Genet. 1999, 23, 62–66.
26. Zhang, Y.; Ng, H.-H.; Erdjument-Bromage, H.; Tempst, P.; Bird, A.; Reinberg, D. Analysis of the NuRD subunits reveals a histone deacetylase core complex and a connection with DNA methylation. Genes Dev. 1999, 13, 1924–1935.
27. Sarraf, S.A.; Stancheva, I. Methyl-CpG binding protein MBD1 couples histone H3 methylation at lysine 9 by SETDB1 to DNA replication and chromatin assembly. Mol. Cell 2004, 15, 595–605.
28. Prendergast, G.C.; Lawe, D.; Ziff, E.B. Association of Myn, the murine homolog of Max, with c-Myc stimulates methylation-sensitive DNA binding and Ras cotransformation. Cell 1991, 65, 395–407.
29. Watt, F.; Molloy, P.L. Cytosine methylation prevents binding to DNA of a HeLa cell transcription factor required for optimal expression of the adenovirus major late promoter. Genes Dev. 1988, 2, 1136–1143.
30. Comb, M.; Goodman, H.M. CpG methylation inhibits proenkephalin gene expression and binding of the transcription factor AP-2. Nucleic Acids Res. 1990, 18, 3975–3982.
31. DeChiara, T.M.; Robertson, E.J.; Efstratiadis, A. Parental imprinting of the mouse insulin-like growth factor II gene. Cell 1991, 64, 849–859.
32. Thorvaldsen, J.L.; Duran, K.L.; Bartolomei, M.S. Deletion of the H19 differentially methylated domain results in loss of imprinted expression of H19 and Igf2. Genes Dev. 1998, 12, 3693–3702.
33. Tremblay, K.D.; Duran, K.L.; Bartolomei, M.S. A 5' 2-kilobase-pair region of the imprinted mouse H19 gene exhibits exclusive paternal methylation throughout development. Mol. Cell. Biol. 1997, 17, 4322–4329.
34. Ferguson-Smith, A.C.; Sasaki, H.; Cattanach, B.M.; Surani, M.A. Parental-origin-specific epigenetic modification of the mouse H19 gene. Nature 1993, 362, 751–755.
35. Li, E.; Beard, C.; Jaenisch, R. Role for DNA methylation in genomic imprinting. Nature 1993, 366, 362–365.
36. Szabó, P.E.; Tang, S.-H.E.; Rentsendorj, A.; Pfeifer, G.P.; Mann, J.R. Maternal-specific footprints at putative CTCF sites in the H19 imprinting control region give evidence for insulator function. Curr. Biol. 2000, 10, 607–610.

37. Kanduri, C.; Pant, V.; Loukinov, D.; Pugacheva, E.; Qi, C.-F.; Wolffe, A.; Ohlsson, R.; Lobanenkov, V.V. Functional association of CTCF with the insulator upstream of the H19 gene is parent of origin-specific and methylation-sensitive. Curr. Biol. 2000, 10, 853–856.
38. Hark, A.T.; Schoenherr, C.J.; Katz, D.J.; Ingram, R.S.; Levorse, J.M.; Tilghman, S.M. CTCF mediates methylation-sensitive enhancer-blocking activity at the H19/Igf2 locus. Nature 2000, 405, 486–489.
39. Bell, A.C.; Felsenfeld, G. Methylation of a CTCF-dependent boundary controls imprinted expression of the Igf2 gene. Nature 2000, 405, 482–485.