

A Review of Epigenetic Imprints in Aquatic Animals

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Abstract

Epigenetics is one of the most rapidly expanding fields in biology. On a molecular level, covalent modifications of cytosine bases and histones, and changes in the positioning of nucleosomes are commonly regarded as the driving epigenetic mechanisms. They are fundamental to the regulation of many cellular processes, including gene and microRNA expression, DNA protein interactions, suppression of transposable element mobility, cellular differentiation, embryogenesis, X-chromosome inactivation and genomic imprinting. Genomic imprinting is an epigenetic gene-marking phenomenon that occurs in the germ line, leads to parental-origin-specific expression of a small subset of genes in mammals and oviparus. The epigenetic imprints regarding the parental origin are established during male and female gametogenesis, passed to the zygote through fertilization, maintained throughout development and adult life, and erased in primordial germ cells before the new imprints are set.

Keywords: Epigenetics; Imprinting; CpG Islands; Histone modification; Nucleosome positioning; Fish crustacean

Epigenetic and its Mechanism's

Before discovering DNA as inheritance component, scientists were concluded that all the gens in all time are not active in all organisms, although carry the same data. Then Epigenetic was introduced as a mechanism that controls the gene expression in an inheritable pathway. In a molecular level the modification of cytosine and histone alkaline modification and changes in nucleosome location are known as the typical mechanisms of epigenetic. These mechanisms are essential for regulating cellular process such as: difference, transposon movements, genomic imprinting, × deactivation and Embryogenesis. In a multi cellular organism the ability of preservation and retention of Epigenetic during genesis and transition to next generation is essential to make a lot of phenotypes that originates from the same genotype. In human, although monozygotic twins are alike in DNA sequence they are different in DNA methylation and the profiles related to histone modification. This matters even in one single cell. How stem cell can become any cell will be answered by epigenetic. The importance of epigenetic came out when their inappropriate outbreak leads to a number of diseases such as cancer [1].

Epigenetic modification and related complexes:

Epigenetic modifications divided into three branches:

- 1) DNA methylation
- 2) Histone DNA
- 3) Nucleosome positioning

DNA methylation

DNA methylation generally occurs in CpG dinucleotide. These nucleotides are gathered in CpG islands- which about 60% of human gene promoters are in contact with them and usually they are not methylated and have the permission to be copied and make a suitable chromatin formation for gene expression but their malapropos hypo methylation leads to deactivation of copying, but some of them will be methylated during the tissue differentiation. Generally CpG islands methylation is correlated with gene silence. DNA methylation doesn't occur just in CpG Island. The idiom of CpG Island Shores refers to

the areas with low concentration of CpG which are in vicinity of CpG Islands, and methylation in these areas are completely in contact with copying suppress. It seems that most of tissue methylation occurs in CpG Island Shores instead of CpG Island. DNA methylation has a key role in genomic imprinting; it means hyper methylation in one or two maternal alleles is related to mono allele gene expression [2].

DNA methylation has several mechanisms to suppress gene expression:

- 1) Methylated DNA increases use of MBD protein. The family member of MBD protein calls the histone modifier complexes and chromatin remodeling's to methylated sites [3].
- 2) DNA methylation suppresses transcription directly by blocking the use of joining protein (such as E2F, NF-KB, AP2) to DNA in Cis binding element areas [3].

It is so rare that DNA methylation be along with transcription activation but in plants and vertebrates, gene bodies' methylation (the exon of some genes which is under transcription and translation) is positively related to gene expression. It is suggested that this matter be along with elongation step blockage of spurious initiation of transcription. Gene bodies tend to DE methylation in disease that leads to transcription initiation in several wrong sites.

Histone modification

Histones are key elements in epigenetic. H2B, H2A (two dimer form) and H3, H4 (one tetra dimer form) form nucleosome.

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H1 histone is copulative and will join the DNA which enters to nucleosome. All histones will be revised after translation and most of this correction is done on histone tail including: stilation, methylation, phosphorylation, obi coientination and ADP rebozilation [1,4].

These corrections have a prominent role in transcription regulation and chromosome compaction. Refer to the transcription form, genomes are divided into two form: a) Eu-chromatin active form b) de active hetero chromatin form. In Eu-chromatin form high levels of H3K4, H3K36, H3K79 estilasion and methylation is detected but in hetero chromatin form low estilasion and high methylation levels will be detected. Totally transcriptionally active gene be spotted in promoters by high level of H4K20 me1, H2BK5ac, H3K27ac, H3K4me3 and in gene bodies by H4K20 me1, H3K79me1. Several Non coding RNAs become syntheses from some hetero chromatin areas. For instance a SiRNA expressed from hetero chromatin pombe saccharomyces centromere area that will jointranscription silence inductor RNA complex and affect it [1].

Nucleosome positioning

Nucleosome is as an obstacle for transcription, and block the transcription activator factors availability to DNA sites, also they rein transcription elongation by capturing polymerases. Exact Nucleosome position around Transcription Start Site (TSS) has an important role in transcription. About 30bp Nucleosome relocating in TSS leads to change in RNAPol2 activity. 3 and 5 terminals are vacant of Nucleosome where is an appropriate space for aggregation and deployment of transcription machine. Lack of Nucleosome on the TSS is extremely in contact with transcription activation, and blocking the TSS by Nucleosome will rein transcription. Accurate Nucleosome action will be affected by histone different variant. These histones are synthesis from the mRNA that has polyA tail and has different key amino acid and histone tail. These histones affect on Nucleosome positioning and gene expression, and everyone could be assumed as an index of a specific chromatin [1].

Genome imprinting

Genome imprinting is an epigenetic phenomenon which occurs in alive layers and leads to some specific maternal gene subset expression. This phenomenon has a prominent effect on genesis, differentiation, germinal growth and metabolism. The gene imprinting occurs in gametogenesis and after ovum and spermzygosis will transmute to zygote and also will be kept during genesis and in primordial germ cells will be deleted before new imprint organization [5,6].

Vertebrate spaternal allele hasn't same performance always, in most of diploid genes if just one gene copy that inherits from one of parent have had defection, other allele can somehow make it up. But in imprinted gene although there are two copies of one gene it means that the person is haploid, and this is the reason of high sensitivity of imprinted genes to different changes and mutation. In 1991 Igf2r gene was identified as the first imprinted gene in mouse, which becomes expressed from maternal 4 allele merely [6].

Imprinted genes are not spread out in all genome, but they are as cluster on genome. Aimprinting cluster can include imprinted maternal and paternal gene. In addition to this they include non-coding RNA and genes without imprinting. There is some area enriched with dinucleotide which is methylated just on one of two paternal chromosomes, which is called Differently Methylated Regions (DMR). Different DNA methylation between sperm and ovum is detected in some DMRs which are called Germline DMR or Gametic DMR.

Some of Germline DMRs act as Imprinting Control Region (ICR). These elements are methylated on one of paternal allele, and they are mono allele expression controller of imprinted gene and other DMRs methylation in the cluster and they are able to affect Bi directional on far distances. ICRs perform as Cis. ICR include an Insulator which blocks enhancer to join promoter in some domains and in other domains Non coding RNAs have hand in, and they makes gene silenced by absorbing chromatin modifiers complexes (just like chromosome deactivation), epigenetic correction has key role in ICR formation since ICR is just for alleles [6,7].

Elements have hand in imprinting: DNA methylation, histone correction, insulator protein such as CTCF, Non coding RNAs.

Epigenetic in aquatic animals

During embryonic development, epigenetic modifications of DNA occur through various processes (e.g. DNA methylation and histone acetylation) and are assumed to facilitate differentiation into specific cell types. An epigenetic alteration can be defined as a mitotically and/or meiotically heritable change in the function of a gene without alterations in the gene sequence [8]. Genes that are to be silenced from one of the parental alleles (i.e. expressed by only one allele) become methylated during the embryonic development in a process called imprinting [9]. There is only limited evidence of genomic imprinting in oviparous species, such as fish [10]. However, DNA methylation reprogramming was observed during the early embryonic development of zebrafish (*Danio rerio*) in a recent study using an anti-5-methylcytosine antibody in immunohistochemistry and southwestern immunoblotting [11]. This reprogramming of DNA methylation found in zebrafish is similar to the reprogramming during mammalian development [12].

Vitellogenesis is the production of yolky eggs in oviparous species, and involves transport of gene products from the liver to the ovary where proteins are deposited in the maturing oocytes. Strömqvist [13] shown that exposure to EE2 results in an alteration of DNA methylation levels in zebrafish which warrants studies of epigenetic changes in fish toxicological studies. They have for the first time shown sex and tissue differences in the level of DNA methylation in adult zebrafish in a franking region of the vitellogenin I gene. Further, following exposure to 100 ng EE2/L during 14 days the DNA methylation levels they decreased in liver of both females and males. The results of them demonstrate the usefulness of the zebrafish as a model organism for studying epigenetic processes and possible epigenetic alterations following exposure to chemicals.

Pyrosequencing technology represents a tool to determine methylation levels of multiple CpG sites specific genes of interest and this study shows its applicability to investigate pollutant-induced alterations of methylation levels in fish [13].

Future work should include investigations into the epigenetic nature of such changes.

Genetic and epigenetic interactions between redundant genes in polyploid fish have probably influenced their evolutionary fate, leading to their current impressive biological diversity [14]. Spontaneous polyploids have been observed in several phylogenetically distant orders, including both wild and farmed fish species [15,16]. In the vertebrates, polyploid species are not exclusive to fish, since they have been reported in different groups, from amphibians [17] to occasionally even in mammals [18]. Polyploids can originate either from alterations of meiotic or mitotic processes in specimens within

a species (autopolyploidy) or by reproductive contact among species (allopolyploidy) [19].

Regulatory changes in gene expression following tetraploidisation may result in epigenetic instability, because they are more likely to be deleterious than advantageous [19].

The genetic sources of variation can be associated with the presence of the extra maternal set of chromosomes and can involve simple gene dosage (additivity) between chromosome sets or positive or negative dosage compensation effects (heterosis), epigenetic mechanisms, and transcriptional co-suppression (negative gene dosage compensation) [19].

Studies on gene dosage compensation in the allotriploid endemic Iberian minnow showed that the allelic expression patterns differ between genes and between different tissues [20]. Thus, it appears that in triploids rather than a whole haploid chromosome set (haplome) being silenced, regulatory mechanisms involve selective individual gene-copy silencing [21]. Feng et al., expressed that the live cell clusters are located at the base of the antennules and antenna, as well as the cephalic lobe, implying an epigenetic mechanism of germ cell specification in *Fenneropenaeus chinensis*. These cells migrate to a dorsolateral position in naupliar and zoeal stages, and gradually enter the genital ridge at the mysis 1 stage. Their findings show that the developmental expression pattern of Fc-vasa-like is different from that of other Crustaceans, and suggest an epigenetic mechanism of germ cell development in Chinese shrimp. The epigenetic mode, in which germ cell was conditionally specified and depended on inductive signals amongst embryonic cells at later embryonic stages, has been reported in the *M. musculus* [22,23] Strong *locentrotus purpuratus* [24,25] *Blatta germanica* (reviewed by Extavour and Akam) [26] *Platynereis dumerilii* [27] and *Mnemiopsis leidyi* (reviewed by Extavour and Akam) [26]. The expression pattern of Fc-vasa-like, and the origin and migratory characteristics of germ cell suggest that the specification of germ cell in *F. chinensis* may begin at the limb bud stage and display an epigenetic mode [21]. Characteristics of the origin and migration of the germ cell suggest that the specification of the germ cell of *F. chinensis* is epigenetically induced, which is different from those reported for other Crustaceans [21].

In zebrafish modulation of parental abiotic environment and nutrition confers increased resistance to the environmental stressor and alterations in cardiovascular parameters (stroke volume, heart rate, cardiac output and red blood cell concentration) to the subsequent generation [28,29].

Conclusion

One of the extremely promoting science in world is epigenetic. These science usages in different branches are spreading and the raise of studies in this field show that. Epigenetic is a process in which without affecting on main DNA succession gene expression pattern is changed. Epigenetic changes leads to malapropogense silence in tumors, better epigenetic recognition which blocks transcription, enables researchers to identify new elements for molecular treatment.

References

1. Portela A, Esteller M (2010) Epigenetic modifications and human disease.
2. Blackledge NP, Klose RJ (2011) CpG Islands chromatin (A platform for gene regulation).
3. Ho DH, Burggren WW (2010) Epigenetics and transgenerational transfer: a physiological perspective. The Journal of Experimental Biology 213: 3-16.
4. Rodenhiser D, Mann M (2006) Epigenetics and human disease: translating basic biology into clinical applications.
5. Feil R, Goto Y, Umlauf D (2007) Centre National de la Recherche Scientifique, Montpellier, France. Molecular Genetics of Genomic Imprinting.
6. Yufeng Li, Hiroyuki Sasaki (2011) Genomic imprinting in mammals: its Life cycle, molecular mechanism and reprogramming.
7. Soejima H, Wagstaff J (2005) Imprinting Centers, Chromatin Structure and disease.
8. Youngson NA, Whitelaw E (2008) Transgenerational epigenetic effects. Annu. Rev. Genom. Hum Genet 9: 233-257.
9. Bird A (2002) DNA methylation patterns and epigenetic memory. Genes Dev. 16: 6-21.
10. Shimoda N (2007) DNA methylation in zebrafish. In: Neumann HA (ed) Progress in DNA Methylation Research. Nova Science Publishers, Inc: 133-152.
11. MacKay AB, Mhanni AA, McGowan RA, Krone PH (2007) Immunological detection of changes in genomic DNAmethylation during early zebrafish development. Genome 50: 778-785.
12. Santos F, Dean W (2004) Epigenetic reprogramming during early development in mammals. Reproduction 127: 643-651.
13. Strömquist M, Tooke N, Brunström B (2010) DNA methylation levels in the 5'flanking region of the vitellogenin I gene in liver and brain of adult zebrafish (*Danio rerio*)—Sex and tissue differences and effects of 17 α -ethinylestradiol exposure. Aquatic Toxicology 98: 275-281
14. Le Comber SC, Smith C (2004) Polyploidy in fishes: patterns and processes. Biol J Linn Soc 82: 431-442.
15. Schulz RJ (1967) Gynogenesis and triploidy in the viviparous fish *Poeciliopsis*. Science 157: 1564-1567.
16. Thorgaard GH, Gall GAE (1979) Adult triploids in a rainbow trout family. Genetics 93: 961-973.
17. Stöck M, Lamatsch DK, Steinlein C, Eppel JT, Grosse WR (2002) A bisexually reproducing all-triploid vertebrate Nat Genet 30: 325-328.
18. Gallardo MH, Bickham JW, Honeycutt RL, Ojeda RA, Köhler N (1999) Discovery of tetraploidy in a mammal. Nature 401: 341
19. Piferrer F, Beaumont A, Falguière J, Flajšhans M, Haffray P (2009) Polyploid fish and shellfish: Production, biology and applications to aquaculture for performance improvement and genetic containment. Aquaculture 293: 125-156.
20. Pala I, Coelho MM, Scharl M (2008) Dosage compensation by gene-copy silencing in a triploid hybrid fish. Curr. Biol. 18: 1344-1348.
21. Feng Z, Zhang Z, Shao M, Zhu W (2011) Developmental expression pattern of the Fc-vasa-like gene, gonadogenesis and development of germ cell in Chinese shrimp, *Fenneropenaeus chinensis*. Aquaculture 314: 202-209.
22. McLaren A (2003) Primordial germ cell in the mouse. Dev. Biol. 262: 1-15.
23. Saga Y (2008) Mouse germ cell development during embryogenesis. Curr Opin Genet Dev 18: 337-341.
24. Juliano CE, Voronina E, Stack C, Aldrich M, Cameron AR (2006) Germ line determinants are not localized early in sea urchin development, but do accumulate in the small micromere lineage. Dev Biol 300: 406-415.
25. Voronina E, Lopez M, Juliano CE, Gustafson E, Song JL (2008) Vasa protein expression is restricted to the small micromeres of the sea urchin, but is inducible in other lineages early in development. Dev Biol 314: 276-286
26. Extavour CG, Akam M (2003) Mechanisms of germ cell specification across the metazoans: epigenesis and preformation. Development 130: 5869-5884
27. Rebscher N, Zelada-González F, Banisch TU, Raible F (2007) Vasa unveils a common origin of germ cell and of somatic stem cells from the posterior growth zone in the polychaete *Platynereis dumerilii*. Dev Biol 306: 599-611.
28. Schwerte T, Voigt S, Pelster B (2005) Epigenetic variations in early cardiovascular performance and hematopoiesis can be explained by maternal and clutch effects in developing zebrafish (*Danio rerio*). Comp Biochem Physiol. A Mol Integr Physiol 141: 200-209.
29. Ho DH (2008) Morphological and physiological developmental consequences of parental effects in the chicken embryo (*Gallus gallus domesticus*) and the zebrafish larva (*Danio rerio*). Diss: University of North Texas.