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Current Review on Genetic Glitch

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Review Article

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ABSTRACT

Genetic Glitches are those which once occur cannot be restored. Glitches can be actually seen in computer programs, Video Games etc. But, here the term 'Genetic Glitch 'is used to sort out the things which underwent wrong path at the time of formation of the first Embryo and then its divisions. This is a bit difficult to understand in general words. I will take you in depths of this in order to understand this term Gene Glitch. Because every change that occurs in any part of body of an animal is from the genetic levels of the organism or the gene expressions and mutations. Mutations occur in the genome or the genetic sequence of the person.

INTRODUCTION

Everything we can see has got one thing in common. That is life. It can't be described but the basic thing essential for an organism to survive is DNA (Deoxy–Ribo Nucleic Acid). A single DNA has over 30000-35000 genes which perform more than 1000000+ functions. But it is said that the functions of 94% of the genes we have is yet a mystery. And it is also proved that DNA stores a second layer of information in it. So, from where did life originate? Different people have different theories and explanations for this.

I personally feel that evolution is the best suitable as stated by Sir. Charles Darwin [Father of evolution]. No one actually knows the differences between the facts and myths of Biogenesis. Life was given to us approximately 6.4 Billion years ago. The Beginning of everything is big bang. The first sign of life on the Earth is a Carbohydrate. It doesn't possess life but it facilitated the origin of life, a kick start to what is visible to us today. This was proved by scientists of UK a couple of decades ago.

Experiments on artificial life (Abiogenesis)

Experiment 1: A scientist took some common sugar in a jar filled with half water in it and oxygen as well. He started to heat the jar assuming the normal temperatures. The sugars started to form protein like structures in the jar. When observed deeply they were actually amino acids formed artificially.

Experiment 2: Students of California University conducted an experiment to know the formation of life. They performed the experiments in such a place where it was life the earth after big bang. More content of sulphur and oxygen in the atmosphere and they exposed it to excess lightning and phosphorus rains. Result after an year was formation of simplest bacterium with only 500 genes in the total genome of the bacterium. Hence, it's proved that life can be created and can be transformed as we like.

POSSIBILITIES OF ARTIFICIAL LIFE (OR) CREATION OF NEW SPECIES

Mutations, The only thing that kept us alive or made us evolve from the day the first living creature has appeared on the Earth. The easiest way one can understand what mutations are is through observations. People say practice makes you perfect. Ever thought why? It's because of mutations and adaptions. Mutagenesis is the term that describes the origins of mutations in an individual.

Example: No one knows or can understand music by birth; they can neither sing perfectly nor compose music in the infant stages. But as they keep practicing music daily the genetics take over. And it is also proved that gene AVPR1A on the chromosome 12q is only for music perception and gene SLC6A4 on gene 17q is for memorising the music and creating it. In the same way mutations keep us help in and getting adapting to everything around us.
Possibilities include:

Gene editing

This technique is now used to treat some inborn diseases like diabetes and asthma. But these can also be used in creating and designing the body the way we like. For example, all he wrestlers and body builders show utmost 40% more Myosin II fibres than normal people. If the gene is edited and the production of these Myosin fibres is alternated then people can get the body they desire of.

Gene silencing

This is a new technique that is still in human trials using which the unwanted genes expression can be silenced and the effect can be supressed. Thanks to the new technologies and new tools of bioinformatics developed. CRIPSR/CAS 9 is one that tool that channels the facility to silence the genes.

Genome rearrangement

The Genome Rearrangement has to be done in the embryonic stages of the foetus of animal. Though this is opposed by many ethics and Religious issues but there is a possibility to design a new foetus the way we desire. For example, we can insert the OCA2 gene and substitute it with HERC2 genes. OCA2 gene is responsible for blue eyes and whereas HERC2 is for black ones.

Natural mutations

We see these in many daily lives of animals. They help most of us to survive the situations and sort out the situations. Examples:

One who leaps a lot grows taller compared to those who don't. This happens because the Gene has codons and sensors which sense the leaping daily. They sense that the animal must grow a bit taller to reach out for something. GSH is released to facilitate the growth and help the animal.

Epigenetics: This happens accordingly to atmospherically occurring changes to body of the animal. The simplest example is Tan. We tan if we go out in the sun. It's because the genes sense UV radiation contact with the skin of the animal. They are aware of the damage caused by UV. So they form melanocytes only at the areas exposed to the son and tan the area. Melanocytes are dense and are hard for UV to penetrate. This mechanism saves the endodermis of the skin and other internal parts.

No matter what the type of mutation is, everything has got its own glitch. Or a Drawback we say. The techniques discussed above could be a boon or a curse, none knows yet.

CONCLUSION

No matter what we do in our day to day life. Genetics and Glitches are always a part of our life and animal life. The mutations and adaptations facilitate the survival and Instincts we and animals encounter with the environment and nature daily. Genetics, DNA and Mutations. Three main this that help everyone and most of us are unaware of it. They play a major role in the life of every being that survives today. Right from the first day of the appearance of life on earth.

REFERENCES

1. Dogan S et al. Comparison of MLL Fusion Genes Expression among the Cytogenetics Abnormalities of Acute Myeloid Leukemia and Their Clinical Effects. J Biom Biostat. 2016;7:312.
2. Nikhil A et al. High Protein/Fish Oil Diet Prevents Hepatic Steatosis in NONcNZO10 Mice; Association with Diet/Genetics-regulated Micro-RNAs. J Diabetes Metab. 2016;7:676.
3. Shankar A et al. Anti-VEGFR2 Driven Nuclear Translocation of VEGFR2 and Acquired Malignant Hallmarks are Mutation Dependent in Glioblastoma. J Cancer Sci Ther. 2016;8:172-178.

4. Eskenasi M and Mehrandezh M. The Permutation Flow-Shop Scheduling Using a Genetic Algorithm-based Iterative Method. Ind Eng Manage. 2016;5:191.
5. Talaat H. Genetic Redundancy Eliminates the Dream of Beneficial Mutations. J Data Mining Genomics & Proteomics. 2016;7:201.
6. Kamath MP et al. A Case Report of a Metastatic Adenocarcinoma of Lung with Dual Positivity for EGFR Mutation and ALK Fusion. J Nucl Med Radiat Ther. 2015;6:262.
7. Romaniello R and Borgatti R. Epilepsy in Multigene Tubulin Family Mutations. J Neurol Neurophysiol. 2015;6:325.
8. Polzer H et al. Individualized Treatment Strategy with Small-Molecular Inhibitors in Acute Myeloid Leukemia with Concurrent FLT3-ITD and FLT3-TKD Mutation. 2015;8
9. Etienne H et al. Are Genetic and Epigenetic Instabilities of Plant Embryogenic Cells a Fatality? The Experience of Coffee Somatic Embryogenesis. Human Genet Embryol. 2016;6:136.
10. Lee HH. Modifying Other's Originality without Quote is an Act of Piracy. Human Genet Embryol. 2015;5:128.
11. Yovich JL et al. Cumulative Live Birth Rate: An Outmoded Term. JFIV Reprod Med Genet. 2016;4:165.
12. Baykal B. Time Lapse Embryo Imaging: We Don't Wanna Miss A Thing. JFIV Reprod Med Genet. 2016;4:121.
13. Vazifehmand R. Two Novel Somatic Mutations in Exon 15 of the Adenomatous Polyposis Coli Gene in Iranian Familial Adenomatousis Polyposis Coli Patients. Hereditary Genet. 2016;5:157.
14. Hossain A. Henrik Ibsen's Ghosts: A Critical Study of Hereditary Genetics. Hereditary Genet. 2016;5:162.
15. Liu X et al. Evaluation of AAVMediated Gene Therapy with Reduced Vector Volume in Cngb3 Knockout Mice, a Model of Achromatopsia. Hereditary Genet. 2016;5:163.
16. Jaffer ATF et al. α-Actinin-4 Gene Mutations, Steroid Responsiveness and FSGS in Adult Onset-Nephrotic Syndrome. Hereditary Genet. 2016;5:167.
17. Coscia MR. Antarctic Fish IgT, a Weird Option of Immunoglobulin Genes. Immunogenet open access. 2016;1:107.
18. Baratchi S. Importance of Mechanotransduction in Immune Responses. Immunogenet open access. 2015;1:102.
19. Saggini R et al. Viscosupplementation with Hyaluronic Acid or Polynucleotides: Results and Hypothesis for Condro-synchronization. J Clin Trials. 2014;4:198.
20. Marycz K et al. The Autologous Gelsolin Combined with Exogenous Nucleotides enhance Chondrogenic Differentiation in Equine Adipose Derived Mesenchymal Stromal Cells - An In Vitro Research. J Cell Sci Ther. 2014;5:174.
21. Ringo E et al. Use of immunostimulants and nucleotides in aquaculture: a review. J Marine Sci Res Development. 2012;1:104.
22. Mandrich L. The "Evolution" of Mutagenesis. Clon Transgen. 2015;4:e121.
23. Allam AR et al. Computational Analysis of Mutations in Colon Cancer Genes Reveals a Possible Role of Micro Satellite in Mutagenesis. J Proteomics Bioinform. 2008;S1: S041-S045.
24. Melzer M. Initial Phase of Epigenetic Pathways in Carcinogenesis and Mutagenesis. J Carcinog Mutagen. 2014;5:180.
25. Giorgio M et al. Mitochondrial Apoptosis Reduces Mutagenesis Regardless Oxidative Stress. J Carcinog Mutagen. 2014;S3:005.
26. Karn N and Karn SK. Evaluation and Characterization of Protease Production by Bacillus Sp. Induced By UV-Mutagenesis. Enz Eng. 2014;3:119.
27. Shafie NH et al. The CRISPR-Cas9 System: A New Dawn in Gene Editing. J Bioanal Biomed. 2014;6:45-48.
28. Yogindran S et al. Artificial miRNAs for Specific Gene Silencing and Engineering Virus Resistance in Plants. Cell Dev Biol. 2015;4:e137
29. Sontakke S et al. Different Types of Transgene Silencing in Animals: A Natural Foundation for RNAi Technology. Mol Biol. 2015;4:137.
30. Yin C and Hulbert S. Host Induced Gene Silencing (HIGS), a Promising Strategy for Developing Disease Resistant Crops. Gene Technology. 2015;4:130.
31. Fateme M et al. Gene Silencing with Herbal Compounds against Bacteria. J Med Microb Diagn. 2015;4:196.

32. Husso T et al. Regulation of VEGF-A Expression in Endothelial Cells by Transcriptional Gene Activation or Transcriptional Gene Silencing: Analysis of Genome Wide Transcriptional Response. Gene Technol. 2015;4:122.
33. Gupta B et al. Recent Advances on Virus Induced Gene Silencing (VIGS): Plant Functional Genomics. J Plant Biochem Physiol. 2013;1:e116.
34. Govindan R et al. Gene Silencing – Targeting at Gene Methylation and Demethylation Patterns for the Arrest of Cancer Progression. J Cancer Sci Ther. 2014;6:174-176.
35. Shimanovich U et al. Gene Silencing by siRNA Nanoparticles Synthesized via Sonochemical Method. J Nanomed Nanotechnol. 2014;5:204.
36. Niculescu VF. Pathogenicity of Entamoeba Species Depends on Cell Line Conversion, Genome Reprogramming and Epigenetic Gene Regulation. J Cell Sci Ther. 2016;7:245.
37. Kheiavi EK et al. Genome Mining of Rice (Oryza sativa ssp. indica) for Detection and Characterization of Long Palindromic Sequences. J Data Mining Genomics & Proteomics. 2016;7:199.
38. Liu Y and Zheng J. Variant Maps to Identify Coding and Non-coding DNA Sequences of Genomes Selected from Multiple Species. Biol Syst Open Access. 2016;5:153.
39. Refolo MG et al. Modulation of Doxorubicin Actions in Hepatocellular Carcinoma Cells by Insulin-Like Growth Factor-I. Biochem Anal Biochem. 2016;5:256.
40. Popa-Wagner A and Buga AM. Identification of New Therapeutic Targets for Cerebral Ischemia by Genome-Wide Analysis. Biochem Anal Biochem. 2016;5:e162.
41. Loyola DE et al. Genealogy of the Genome Components in the Highly Homogeneous Pandemic Vibrio parahaemolyticus Population. J Phylogen Evolution Biol. 2016;4:165.
42. Frischmeyer-Guerrerio PA et al. TGF Receptor Mutations Impose a Strong Predisposition for Human Allergic Disease. Science Translational Medicine. 2013;5:195.
43. Horev G et al. Proc. Natl. Acad. Sci. USA. 2011;108:17076-17081.
44. Pucilowska J et al. J. Neurosci. 2015;35:3190-3200.
45. Keeling K et al. "Gentamicin-mediated suppression of Hurler syndrome stop mutations leads to a partial restoration of a-L-iduronidase activity and a reduction of lysosomal glycosaminoglycan accumulation". Human Molecular Genetics. 2001;10:291-299.
46. Davies J et al. "Misreading of RNA codewords induced by aminoglycoside antibiotics". Molecular Pharmacology. 1965;1:93-106.
47. Lederberg EM et al. "Interaction of streptomycin and a suppressor for galactose fermentation in E. coli K-12". Proceedings of the National Academy of Sciences. 1964;51:678-682.
48. Wilschanski M et al. "A pilot study of the effect of gentamicin on nasal potential difference measurements in cystic fibrosis patients carrying stop mutations". American Journal of Respiratory Critical Care Medicine. 2000;161:860-865.
49. Barton-Davis ER et al. "Aminoglycoside antibiotics restore dystrophin function to skeletal muscles of mdx mice". Journal of Clinical Investigation. 1999;104:375-381.
50. Steiner CC et al. The genetic basis of phenotypic convergence in beach mice: similar pigment patterns but different genes. Mol. Biol. Evol. 2009;26:35-45.
51. Rosenblum EB et al. Molecular and functional basis of phenotypic convergence in white lizards at White Sands. Proc. Natl Acad. Sci. 2010;107:2113-2117.
52. Chan YF et al. Adaptive evolution of pelvic reduction in sticklebacks by recurrent deletion of a Pitx1 enhancer. Science. 2010;327:302-305.
53. Conte GL et al. The probability of genetic parallelism and convergence in natural populations. Proc. Biol. Sci. 2012;279:5039-5047.
54. Hubbard JK et al. Vertebrate pigmentation: from underlying genes to adaptive function. Trends Genet. 2010;26:231-239.
55. Manceau M et al. Convergence in pigmentation at multiple levels: mutations, genes and function. Philos. Trans. R. Soc. Lond. B Biol. Sci. 2010;365:2439-2450.
56. Skotko BG et al. Am. J. Med. Genet. 2009;149:2361-2367.
57. Korenromp MJ et al. Am. J. Obstet. Gynecol. 2007;196:149.e1-149.e11.
58. Albrecht GL and Devlieger P. J. Soc. Sci. Med. 1999;48:977-988.
59. Brickman P et al. J. Pers. Soc. Psychol. 1978;36:917-927.

60. Anders S et al. Detecting differential usage of exons from RNA-seq data. Genome Res. 2012;22:20082017.
61. Gilliland FD et al. Effects of glutathione S-transferase M1, maternal smoking during pregnancy, and environmental tobacco smoke on asthma and wheezing in children. Am J Respir Crit Care Med. 2002;166:457-463.
62. Gilliland FD et al. Effect of glutathione-S-transferase M1 and P1 genotypes on xenobiotic enhancement of allergic responses: randomised, placebo-controlled crossover study. Lancet. 2004;363:119-125.
63. Kabesch M et al. Glutathione S transferase deficiency and passive smoking increase childhood asthma. Thorax. 2004;59:569-573.
64. Romieu I et al. Genetic polymorphism of GSTM1 and antioxidant supplementation influence lung function in relation to ozone exposure in asthmatic children in Mexico City. Thorax. 2004;59:8-10.
65. Simpson A et al. Endotoxin exposure, CD14, and allergic disease: an interaction between genes and environment. Am J Respir Crit Care Med. 2006;174:386-392.
66. LeVan TD et al. A common single nucleotide polymorphism in the CD14 promoter decreases the affinity of Sp protein binding and enhances transcriptional activity. J Immunol. 2001;167:5838-5844.
67. Baldini M et al. A polymorphism in the 5′ flanking region of the CD14 gene is associated with circulating soluble CD14 levels and with total serum immunoglobulin E. Am J Respir Cell Mol Biol. 1999;20:976-983.
68. Koppelman GH et al. Association of a promoter polymorphism of the CD14 gene and atopy. Am J Respir Crit Care Med. 2001;163:965-969.
69. Ober C et al. A second-generation genomewide screen for asthma-susceptibility alleles in a founder population. Am J Hum Genet. 2000;67:1154-1162.
70. Shows TB et al. International system for human gene nomenclature. Cytogenet Cell Genet. 1979;25:96-116.
71. Shows TB et al. Guidelines for human gene nomenclature. An international system for human gene nomenclature. Cytogenet Cell Genet. 1987;46:11-28.
72. McAlpine PJ. International system for human gene nomenclature. Trends Genet. 1995;11:39-42.
73. White JA et al. Guidelines for human gene nomenclature. HUGO Nomenclature Committee. Genomics. 1997;45:468-471.
74. Wain HM et al. Guidelines for human gene nomenclature. Genomics. 2002;79:464-470.
75. Nebert DW. Proposal for an allele nomenclature system based on the evolutionary divergence of haplotypes. Hum Mutat. 2002;20:463-472.
76. Duplus E and Forest C. Is there a single mechanism for fatty acid regulation of gene transcription. Biochem Pharmacol. 2002;64:893-901.
77. Nelson DR et al. Cytochrome P450 superfamily: update on new sequences, gene mapping, accession numbers, and nomenclature. Pharmacogenetics. 1996;6:1-42.