OPEN ACCESS Freely available online

Journal of Down Syndrome & Chromosome Abnormalities

Short Communication

Model Review Selection for Aneuploid States

Jennifer Grove*

University of Nebraska Medical Center, Omaha, NE, 68198-5440, USA

ABSTRACT

Aneuploid segments of chromosomes, or abnormal numbers of chromosomes, are linked to a variety of genetic abnormalities and cancers. Many chromosomal defects are believed to be caused by a disruption of the genic equilibrium of protein complexes or pathways. The karyotype and gene expression profiles of 19 Drosophila modENCODE cell lines show how favourable gene copy numbers evolved when genic equilibrium was maintained. The copy number changes within genes encoding components of multiprotein complexes are consistent in these highly aneuploid cells, which may indicate good selection for genic balance.

KEYWORDS: Down Syndrome, Trisomy 21, Chromosome Abnormality.

INTRODUCTION

While this is far from uniform, gene dosage effects may be compensated or buffered [1]. There are chromosome-wide compensating pathways in the case of sex chromosomes. In mammalian females, for example, X inactivation results in a genetically monosomic state, which is countered by increased X chromosome expression in both females and males. Understanding dose compensation mechanisms may help researchers determine the effects of chromosomal abnormalities and perhaps improve therapeutic strategies. The cells in this study had increased cell growth and neurogenesis, showing how dose compensation can be used to deal with chromosomal anomalies [2]. Dosage compensation is another way for cells to deal with harmful gene dose changes. Genes inside duplicated or deleted regions of Drosophila cell lines display differing degrees of compensated gene expression, as determined by RNA sequencing. Alternatively, the differing levels of compensation may be the result of different optimization routes in response to copy number variations in different cell lines. This can be accomplished by focusing on the copy number and/or dose payout. If the above is accurate, additional general systems for obtaining dose compensation can be discovered. Watching the generation of aneuploidy across passages when forming cell lines may be a future path. We believe that such efforts will result in a better understanding of how aneuploidy develops [3].

CONCLUSION

In conclusion, we explain numerical anomalies found in Drosophila cell line genomes and how they can support cell lines during the immortalization process. Our results have implications for other cell-level selection processes, such as tumorigenesis, which is characterised by substantial genomic aberration. Most notably, the Drosophila cell lines' tiny but strongly rearranged genome provides an effective model system for studying numerical changes in genome, their effect, and dose compensation against the effect. Owing to widespread failures in early childhood, studying numerical modifications of chromosomes in animal models is often challenging. It's particularly true in humans, where the massive genome size and genetic pathway redundancies make interpreting copy number effects difficult. We assume that using Drosophila cell line systems, which have smaller genomes and simplified pathways, would be helpful. Watching the generation of aneuploidy across passages when forming cell lines may be a future path. Such efforts, we hope, will contribute to a deeper understanding of how aneuploidy develops. Investigating how cell lines respond to in vitro growth will help not only to clarify cell line establishment, but will also contribute to a better understanding of gene dose effects, gene balance, and dosage compensation.

REFERENCES

1. Dierssen M, Herault Y, Estivill X. Aneuploidy: from a physiological mechanism of variance to Down syndrome. Physiol Rev. 2009;89(2): 887-920.
2. Cook EH Jr, Scherer SW. Copy-number variations associated with neuropsychiatric conditions. Nature. 2008;4(5): 919-923.
3. Veitia RA, Potier MC2. Gene dosage imbalances: action, reaction, and models. Trends Biochem Sci. 2015;40(2): 309-317.

***Corresponding author:** Jennifer Grove, University of Nebraska Medical Center, Omaha, NE, 68198-5440, USA, E-mail: Jennifeergrove2016@gmail.com

Received date: Feb 25, 2021; **Accepted date:** Mar 10, 2021; **Published date:** Mar 15, 2021

Citation: Grove J (2021) Model Review Selection for Aneuploid States. Journal of Down Syndrome and Chromosomal Abnormalities 7:163. doi: 10.4172/2472-1115.21.7.163