Bio-Science Research Bulletin Vol. 33 No.2, July-December 2017: P.100-103 Print version ISSN 0970 0889 Online version ISSN 2320 3161 DOI 10.5958/2320-3161.2017.00012.8

# THE DOWNFALL OF THE GENETICS IN CELLULAR STUDIES

# Rishan Singh

## Author's Affiliation:

Discipline of Biological Sciences, University of KwaZulu-Natal, Durban, 4001, South Africa

# **Corresponding Author:**

**Rishan Singh**, Discipline of Biological Sciences, University of KwaZulu-Natal, Durban, 4001, South Africa

E-mail: rshnsingh1@yahoo.com or rshnsingh1@webmail.co.za

**Received on** 12.10.2017, **Accepted on** 19.12.2017

#### **Abstract**

Cellular studies are a prominent area of research, due to the variety of celled organisms in biology. These organisms have an organised structure, organelles (or organs), and are required to perform specialised functions in their habitat. In all of them, the metabolic efficiency, regulated by nucleic acid substances, must be kept at an optimum. However, genetics in cellular studies could be misguided, because of the interplay of nucleic acid substances with administered substances. This paper addresses the downfall of using genetics as a prominent method in analysing cellular data.

**Keywords:** compounds, membrane, proteins, signal transduction, ligands, pH, calcium release, mitotic catastrophy.

#### 1. INTRODUCTION

Genetics in cellular studies usually refers to analysing gene or protein functions in particular pathways of cells, since these molecules control the optimum functioning of cells [Woese and Fox, 1977; Singh, R, 2017]. Some of the major genes are found in the sugar, protein and nucleic acid processing pathways [Nogucho et al., 2013]. Often it is found that mutations in those genetic pathways lead to the interruption of these pathways, but recent studies have provided evidence that this is not always the case [Singh, R, 2017]. A cellular study, in research, is a broad field involving not only plant, animal and human organisms, but also bacteria and viruses, since they have a defined structure in the natural state [Singh, October 2011]. However, it is the genetics of these organisms that pose as a major hindrance to researchers, because seldom are studies involving the molecular basis of cellular structure easy, and reproducible without any difficulty [Singh, October 2011; Singh, R, 2017]. This is further complicated by the fact that often it is the compounds that are used that have dire consequences on the genetic basis of cellular studies [read Lee et al., 2004]. Now and then,

researchers may find a solution to this impending factor by attempting to confirm cellular studies by isolating active compounds of plants, for example [Singh, R, 2017]. However, the downfall of the genetics in cellular studies is one that's not easily sorted, due to interlinked pathways and signal transduction events, which are contingent of proper sugar and nucleic acid processing.

## 2. THE UNSUCCESSES OF MOLECULAR GENETICS

Studies on molecular genetics in cells are varied, and are also widely published. However, although this may be the case, there are many failures that are not very well understood [Singh, R, 2017]. One unsuccess is that often ceramide, a compound of the cell membrane in mammalian cells, is ignored when results involving compounds are obtained [Singh, R, read Rothman and Lenard, 1977]. This is a major downfall in cellular studies, because signal transduction events are reliant on optimum ceramide function. Ceramide, a fatty acid composed of sphingomyelin, insulates cells, and therefore, it's probably a requirement to consider this in cellular studies [Singh, R, 2017]. Another unsuccess of molecular genetics is that often when a compound, or a mixture of them are tested, laboratory analysis of the cells involved can't consider all the genes / proteins participating in metabolic pathways [Wolf and Green, 1999; Noguchi et al., 2013; Aiston et al., 2003; Singh, R, 2017]. This indicates that perhaps phenotypic and microscopy testing will be a benefit to researchers, so as to obtain a starting point for analysing genes in cells, rather than merely performing molecular biology tests on compound-stimulated cells [Singh, R, 2017]. In addition, in many reports there is no evidence that genetic testing had accounted for whether compounds have entered a cell, or not [Singh, R, 2017; Singh, October 2011]. This indicates that many studies are inaccurate, since optimum genetic testing involving compounds, like in a previous publication in this journal on cytotoxicity and drug susceptibility tests, rely on them disrupting the function of the nucleus, marked by morphology and topological cellular changes [Van Cruchten and Van Den Broeck, 2002; Rogers, 2005]. In the plants however, toxins are used to induce signal transduction events, but since plants cells are rigid in nature, heating of them definitely disrupts the genetic machinery [Roger, 2005; Reape et al., 2003; Salisbury and Ross, 2010]. This is another downfall of molecular studies, since in this case, the effect of the toxin (e.g. Fusarium spp.) in genetic testing isn't completely accurate [Singh, R, 2017]. Mitotic catastrophy, a process that terminates cell division in cells, is often misguided by tested compounds, since researchers don't report whether characterised compounds of optimum concentration have entered the cell [Singh, R, 2017]. This indicates that studies involving apoptosis, oncosis and necrosis, have some degree of inaccuracy that is very difficult to eliminate [read Majno and Joris, 1995 and Golstein and Kroemer, 2006]. The role of the p53, the main cell regulation proteins, is thus, affected by both natural, synthesised, isolated and extracted compounds [Chance and Williams, 1956; read Wolf and Green, 1999, Kondo and Kondo, 2006], since p 53 may still continue to regulate the cellular functions optimally, once they are administered with these substances or chemicals [Singh, R, 2017].

#### 3. SIGNAL TRANSDUCTION EVENTS AND MOLECULES

In order for signal transduction to take place optimally, compounds must enter the cell, and induce a signal [Singh, R, 2017]. Often signal transduction is misguided by compounds, causing cell death that's classified as apoptosis, or simply a form of induced mitotic catastrophy by a substance [read Rogers, 2005; read Wolf and Green, 1999, Lockshin and Zakeri, 2004; Singh, R, 2017]. Thus, it is in this case, that classified compounds become important to be studied in cells, but still a problem persists [Singh, R]. One major hindrance is that in standardised cellular studies, often the susceptibility of ligands to compounds are ignored [Rothman and Lenard, 1977]. This indicates that sometimes signals may be induced in cellular organisms by solvents alone [Singh, October 2011, Singh, R, 2017], rather than the prepared formulations (as in the case of extracts or prepared drug compounds), causing morphology changes, like cellular shrinkage, chromatin breakdown and cytoskeletal protein inactivation [Van Cruchten and Van Den Broeck, 2002; Majno and Joris, 1995; Golstein and

#### Rishan Singh / The downfall of the genetics in cellular studies

Kroemer, 2006, Kondo and Kondo, 2006]. It's evident from this that the pH of compounds are vital for inducing a signal for mitotic divisions, since their polarity determines their degree of interaction with cellular ligands, and ceramide, as mentioned. It's, thus an essential requirement that successful signal transduction events be confirmed using phenotypic tests like cell staining, and histochemical methods [Singh, R, 2017].

## 4. CONCLUSION: THE CYTOLOGY, PROTEIN AND CELLULAR ACTIVITIES

The downfall of the genetics in cellular studies includes events that occur in the cytosol [Singh, R, 2017]. Since the cytosol is made up of water mostly, the proteins and ribosomes are in consistent contact with an environment that's hydrated [Salisbury and Ross, 2010]. However, interruption occurs when chemicals enter the cells, since the electrolyte consistency of the cytoplasm becomes altered. If the pH is too low, cellular activities become inactive. But there is an exception, in this case. The exception is that if the pH of the administered compound is sufficiently high, then, the cellular ionic concentration may become normal, inducing its activity. However, the setback is that the result may not be reflective of that in metabolically active cells [Singh, R, 2017]. In bacteria and viruses, on the other hand, the scenario is the same, but over there the simplicity of the genome is important [Singh, October 2011]. In both microorganisms, if the pH is to denature or inactivate replication proteins, chemicals of whatever concentration, may not induce their effects. If bacteria are resistant, or are developing resistance, then the converse situation occurs [Singh, October 2011]. In this case, it has been found that plant extracts have the potential to cause proliferative effects [Lee et al., 2004]. Papers have emphasised that, in cancer cells for example, that extracts entering the cytoplasm promotes cell growth by stimulating calcium channels. In the human body, the same occurs but body temperature is of vital consideration there [Singh, R, 2017].

#### REFERENCES

- 1. C. R. Woese, G.E. Fox. (1977). The concept of cellular evolution. *Journal of Molecular Evolution* v (10): 1-6.
- 2. S. Van Cruchten, W. Van Den Broeck. (2002). Morphological and biochemical aspects of apoptosis, oncosis and necrosis. *Anatomy, Histology and Embryology* v (31): 214-223.
- 3. G. Majno, I. Joris. (1995). Apoptosis, oncosis and necrosis. An overview of cell death. American Journal of Pathology v (146): 3-15.
- 4. P. Golstein, G. Kroemer. (2006). Cell death by necrosis: towards a molecular definition. *Trends in Biochemical Sciences* v (32): 37-43.
- 5. J.Y. Lee, W.I. Hwang, S.T. Lima. (2004). Antioxidant and anticancer activities of organic extracts from *Platycodon grandiflorum* A. De Candolle roots. *Journal of Ethnopharmacology* v (93): 409-415.
- 6. R. Singh. (October 2011). Mycobacterium tuberculosis: Telling a Story of Truths. Journal of Pure and Applied Microbiology v (5) No (2): 541-544.
- 7. T.E. Rothman, J. Lenard. (1977). Membrane asymmetry. *Science* v (195): 743-753.
- 8. R.A. Lockshin, Z. Zakeri. (2004). Apoptosis, autophagy, and more. *The International Journal of Biochemistry and Cellular Biology* v (36): 2405-2419.
- 9. H.J. Rogers. (2005). Cell death and organ development in plants. *Current Topics in Developmental Biology* v (71): 225-261.
- 10. B. Chance, G.R. Williams. (1956). The respiratory chain and oxidative phosphorylation. *Advances in Enzymology* v (17): 65-134.
- 11. T. Kondo, S. Kondo. (2006). Autophagy and cancer therapy. Autophagy v (2): 85-90.
- 12. R. Noguchi, H. Kubota, K. Yugi, T. Toyoshima, Y. Komori, T. Soga, S. Kuroda. (2013). The selective control of glycolysis, gluconeogenesis and glycogenesis by temporal insulin patterns. *Molecular Systematics and Biology* v(9): 664.
- 13. T.J. Reape, E.M. Molony, P.F. McCabe. (2008). programmed cell death in plants: distinguishing between different modes. *Journal of Experimental Botany* v (59) No (3): 435-444.

## Bio-Science Research Bulletin / Volume 33 Number 2 / July-December 2017

- 14. B.F. Salisbury, W.C. (2000) Plant Physiology (Fifth Edition). *Wadsworth Publishing Company*, Belmont, California, United States of America.
- 15. B.B. Wolf, D.R. Green. (1999). Suicidal tendencies: apoptotic cell death by caspase family proteinases. *Journal of Biological Chemistry* v (274): 200049-20052.
- 16. S. Aiston, B. Andersen, L. Agius. (2003). Glucose-6-phosphate regulates hepatic glycogenolysis through inactivation of phosphorylase. *Diabetes* v (52): 1333-1339.
- 17. Singh, R. (2017). Personal writing and statement, representing the Republic of South Africa.