Original Article

GROUP THEORY CONCEPTS IN MOLECULAR SYSTEMS

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Abstract

In this work, we explore group theory ideas implemented to biology and in selective systems of biology. Symmetry performs a notable role in biology of bilaterians over radially symmetric bodies. The distinction between living and non-living organisms is best known. We concentrate on the group theory and abstract algebra covering the biology of molecular systems. We provide the applications of group theory to cancer cells. We review applications to the cells cycle and explore how cells growing and forming cycles. We applied group theory to Cancer Cells and present example to support the validity of the article.

Keywords: Group theory, Cells, Molecular system, Cancer cell.

INTRODUCTION

Cancer causes by the division of cells without spreading and stopping into surrounding tissues is called cancer. The cancer can develop in any part of the body that it is composed of tons in cells. Generally, the cells must expand and split into a shape that the body produces to have new cells. Whenever the cells become new or old cells become damaged replace it, as the needed for a body and this extras will grow without stopping and spreading. Currently in certain instances, cancer is induced by hard tissue tumors while blood cancer is not produced by solid tumors, Cancer is malignant, meaning that it may spread to or invade neighboring tissues, causing severe damage. In addition, as these tumors expand and other cancer cells disperse they move through the bloodstream or immune system to different locations in the body to form new tumors far from the initial tumor. Unlike other tumors, malignant tumors may not grow into or form healthy tumorsor invade the nearby tissues. However, benign tumors may often be very big. When removed, tumors may not usually regrow, although malignant tumors often do. Malignant brain tumors, unlike other benign tumors from elsewhere in the body, can be life-threatening and harmful. Cancerous cells differentiate from normal cells, allowing them to grow out of control and become violent in a variety of ways. Another big difference is that there are less aggressive cancer cells than normal cells. It is, while it's a normal cell with related functions evolve into somewhat specific cell types, but cancerous cells do not. Unlike normal cells and cancer cells, this is one example of why would keep separating and not preventing. In fact, cancer cells can disregard signals that usually alert cells not to divide or begin the process such as apoptosis or the cellular senescence which the body produces to be get out of defective cells.[9] Cells may affect the normal cells, proteins, and tissues surrounding but feeding the cancer in a region called as the cell membrane. For example, cells may induce surrounding cancer cells to shape the blood flow they need to build that supply nutrients to cancers. Tumors can also be used to stay stable and to work in the immune response. For example, Cancer cells can effectively prevent cancer cells from suppressing the immune response with that assistance of certain nervous system cells which normally resist escape from the immune response.

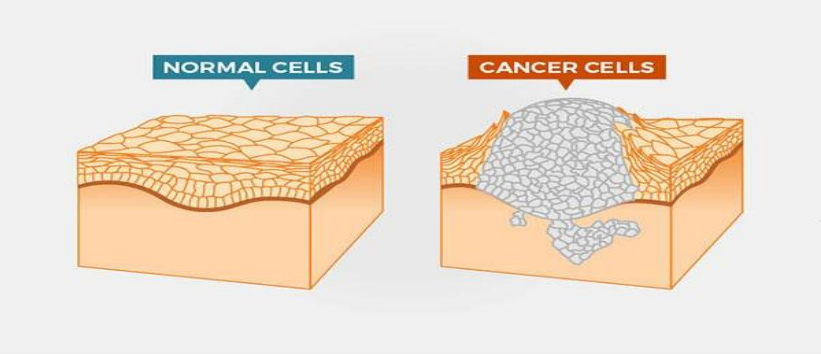

Figure 1

There have been a number of variations among normal cells and cancer cells (see Figure 1). The worst significant is really the amount of checkpoints that have to be prevented to order with the cell becoming cancerous.

- The cell needs to be having many growth factors that forces it's to grow, even if it is not necessary.
- We start to reduce due to the influence of evading proteins (undesirable enzymes) that force cells to avoid their development and death because they are unhealthy.
- The cell tries to stop impulses originating from several other cells.
- Such cells just have to sacrifice the usual thickness or adhesion which regular cancers create.

It is quite rare for human cell of becoming cancerous. [6&7]This might sound impossible, including how once in three people are developing cancer early throughout their lifetime. Certain mutations in our cells can contribute to the creation of a cancer cell. There are three billion cells that generate any single cell in our human body. These errors in the replication of certain genes by heredity nor substances throughout the atmosphere throughout the separation of certain cells that contribute to cancer cells. We subsection this paper into the following. Section 2, we introduce a few important facts and discussions associated with our study that is employed, which utilizes throughout the discussion of this article. Section 3 is reserved for discussion about group theory. Section 4, we applied group theory to Cancer Cell and present example to support the validity of the article.
Lastly, in Section 5 conclusion of the work.

How Cancer Develops

Cells have several specific ways to limit cell differences. We restore DNA damage to keep cancer from growing. Due to this new method, assumed it might evolve into a dual-step cancer cycle. To which such various pathways have collapsed until the tipping point is achieved and the cell has been specifically Split a bit more slowly. It is doubtful that they might be cancerous, however the tumor is benign. A collection of cells that differentiate into far more than average cells which do not have the ability to enter or destroy other substances. Through years, changes have arisen inside one of the declining cells, which have contributed to enhanced control of the disease. [10] The mutation may not induce cancer that has its own, although the descendants of such cells may therefore grow quickly and build a wide pool of cells whereby the additional mutant may still take effect. Finally, each cell could have enough mutants to bring over the features of a cancer cell and to give birth to a malignant cell.

Characteristics of Normal and Cancer Cells

Such genes produce enzymes which detect and restore DNA injury, receive DNA binding substances, preserve telomere caps at the edges of genomes, but perform certain important maintenance functions.
When one of these genes is altered and yet another mutant is also not activated. Thus, when a cell does have a component in the survival of a semi-functional gene, its descendants will be influenced by the small core of replication which creates a cancer cell even slower than normal cells (see Figure 2).

Cell Cycle and Regulators

Specific forms of cancer require various kinds of genes, so each tumor will have a specific range of genetic changes. In fact, defects of both forms of cell cycle receptors can facilitate the growth of cancer: positive controllers could be over-activated (become oncogenic), whereas neutral controllers, often called tumor suppressors, might be denatured.

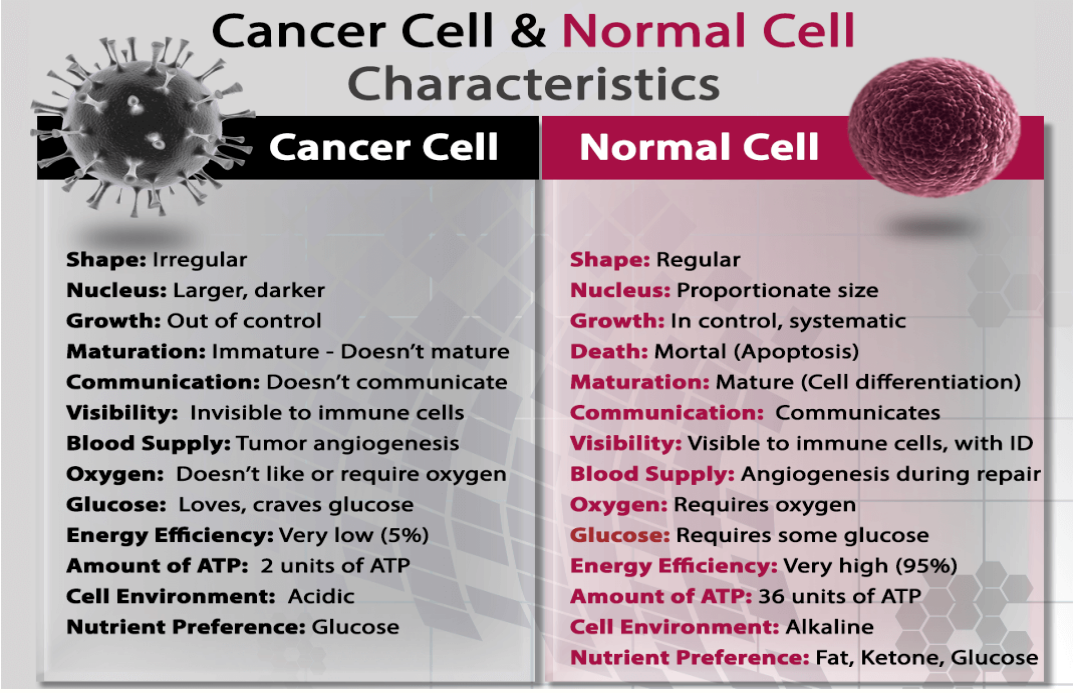

Figure 2

Group Theory

Group theory is a subset of mathematical algebra that has been established to research and exploit abstract principles concerning symmetry. Until explaining group theory to more precise words, it would help to start with an illustration from such an abstract definition, the circle group. A flat square board in a true 3-dimensional space (see Figure 3). The radians, i.e. 180 degrees, can be rotated along the X, Y and Z axes. Let us show these rotations by (r1, r2, r3) [7].

They would also believe that there can be no do-nothing project defined by e. If we circle our card of r1 accompanied by a rotation of r2 and then we have the inverse of a combination of r3. Therefore, we will fill the Cayley table with the "multiplication" row, since the process is not really a standard multiplication (see Table 1).

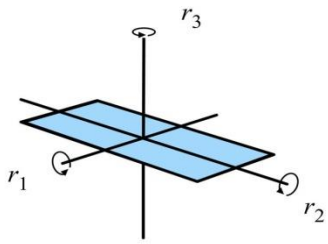

Figure 3

The similarity of the diagonal in the Cayley table informs us that the group is abelian, they are commutative while the rotations are done in pairs, so rm*rn = rn*rm.

Such four operations for groups can also be written in matrix form:

$$E = \begin{bmatrix} 1 & 0 & 0 \\ 0 & 1 & 0 \\ 0 & 0 & 1 \end{bmatrix} \quad R_1 = \begin{bmatrix} 1 & 0 & 0 \\ 0 & -1 & 0 \\ 0 & 0 & 1 \end{bmatrix} \quad R_2 = \begin{bmatrix} -1 & 0 & 0 \\ 0 & 1 & 0 \\ 0 & 0 & -1 \end{bmatrix} \quad R_3 = \begin{bmatrix} -1 & 0 & 0 \\ 0 & -1 & 0 \\ 0 & 0 & 1 \end{bmatrix}$$

We have become able to define the thorough explanation of group G: it is a non-empty set with a binary operation (referred to hereby *) that satisfy characteristics:

i. Closure: for all m, n ∈ G, m*n ∈ G.

ii. Associativity: for all m, r, f ∈ G, (m*r) * f = m* (r*f).

iii. Identity: There is an identity element e ∈ G, such that v*e = e* v = v, for all v ∈ G.

iv. Inverse: For any v ∈ G there is an element u ∈ G, such that v*u = u*v = e.

*	e	r_1	r_2	r_3
e	e	r_1	r_2	r_3
r_1	r_1	e	r_3	r_2
r_2	r_2	r_3	e	r_1
r_3		r_2	r_1	e

Table 1 shows Cayley rotations:

Cancer Cell Applied In Group Theory

Now, we applied the cell cycle in application of graph theory. The steps of cell cycle include G1 → S → G2 →M, and back to G1. G0 are also present in this cycle. G0 is basically as brief in some cases as to be non-existent so we will disregard that condition. The only logical approach to put that cell cycle into group theory is to use that square symmetries to remember the definition of the group we gave earlier. Table 2 shows a group graph of the Cell Cycle. It is abelian and isomorphic to the cyclic group Z_4.

*	G1	S	G2	M
G1	G1	S	G2	M
S	S	G2	M	G1
G2	G2	M	G1	S
M	M	G1	S	G2

Table 2

Reporting the cycle of the cell cycle as the combinations we have,

$$R_0 = \begin{pmatrix} G1 & S & G2 & M \\ G1 & S & G2 & M \end{pmatrix}$$

$$R_{90} = \begin{pmatrix} G1 & S & G2 & M \\ S & G2 & M & G1 \end{pmatrix}$$

$$R_{180} = \begin{pmatrix} G1 & S & G2 & M \\ G2 & M & G1 & S \end{pmatrix}$$

$$R_{270} = \begin{pmatrix} G1 & S & G2 & M \\ M & G1 & S & G2 \end{pmatrix}$$

Where for example R90 can be expressed as the mapping.

$$G1 \Longrightarrow S$$
$$S \Longrightarrow G2$$
$$G2 \Longrightarrow M$$
$$M \Longrightarrow G1$$

Designed for the Population Cell Cycle table, it proposes to analyze the collective behaviors of some possible genetic cell control. Rao and Johnson and Johnson and Rao also conducted experiments to transfer nucleus through each cell to the next

to produce multiple nucleus cells (see Figure 4 & 5). Such studies discussed wider questions regarding genome moisture and the control of DNA production. Here, by way of a group map, we examine the integration of such binocular cells. In certain cases, less than one nucleus has already been connected to a cell in another area. [1] For example, two G1 nuclei have been applied to both the S-phase cell, and if the G2 nucleus has also been introduced to the G1 cell, there has essentially been little change. Those were just tentative assumptions given extra space. All the enzymes converge to condition M, which is the greatest identifier in cell cycle nuances. In order to show that such ties exist with real community ideas, we will have to demonstrate associativity, to find an identity and an inverse part or associativity, to show an isomorphism with a known group.[2]

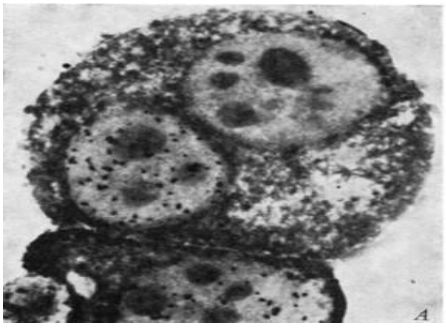

Figure 4

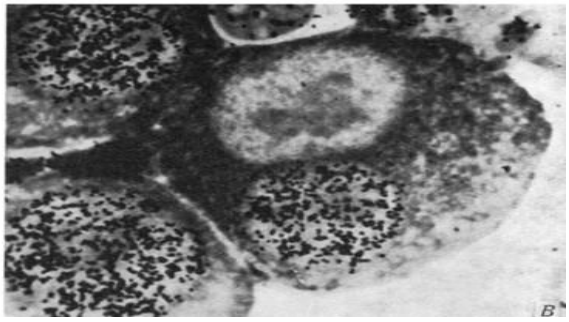

Figure 5

Figure reproduced after Rao and Johnson. Rietman et al, Theoretical Biology and Medical Modelling 2011, 8:21.

Cell Cycle Control

The cell cycle requires controlled reproduction, division and cell growth. The cell cycle forms of four different stages: [5]G1 (gap step 1), S (DNA synthesis), G2 (gap step 2), and M (mitosis). Gene expression control (all other relaxation and secretion) is based on various cell growth receptors that deter and extend irregular cell growth stimulation.

For example: The G2/M checkpoint means which damaged DNA cell would not enter mitosis. Such cell growth checkpoints were controlled through CDK+cyclin linking sets. CDK4/6+cyclin D, RB1/E2F, CDK2+cyclin E, CDK2+cyclin A, CDK1+cyclin A, CDK1+cyclin B.

That phase campaign is a sequence of phases wherein the organism is replaced by two separate new cells. The process loop consists of four steps, namely G1 (gap step 1), S (DNA synthesis), G2 (gap step 2) and M (mitosis).
The cell cycle is regulated by a strong mix between storm-dependent cytokines (CDK-1,-2,-4,-6,-8,-12) and storms (Cyclin-A,-B,-D,-E). Commuting homodimers and CDKs happen at lower stages of cycle development. There are a few term limits in operation, called checkpoints, in the cell division in order to ensure the health of new cells. The checkpoints shall consist of the G1 / S checklist and the G2 / M checklist. Such checkpoints can also become essential throughout the assessment of DNA injury. DNA loss triggers these checkpoints to maintain genetic stability by restoring destroyed DNA or by pressuring the cell to follow the conditioned cell division process if DNA should not be restored.

Checkpoints and DNA injury effects were changed in such a variety of cancers. Relevant points in the system which are clinically prosecutable are reported (see Figure 6 & 7).

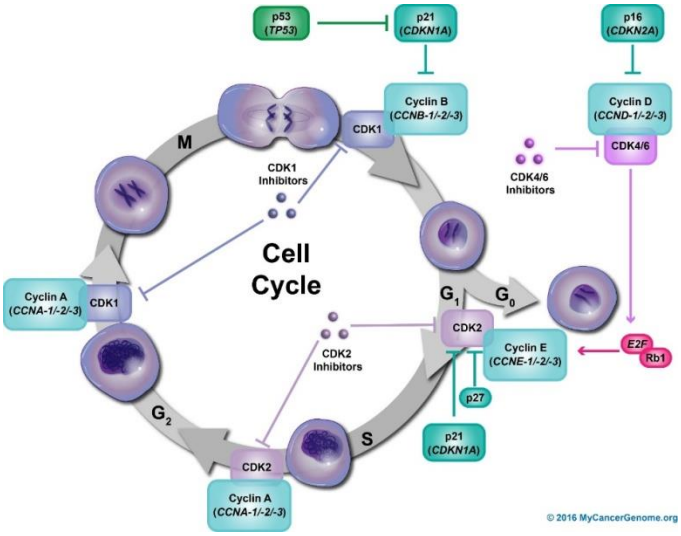

Figure 6

CANCER CELL AND NORMAL CELL RESULT

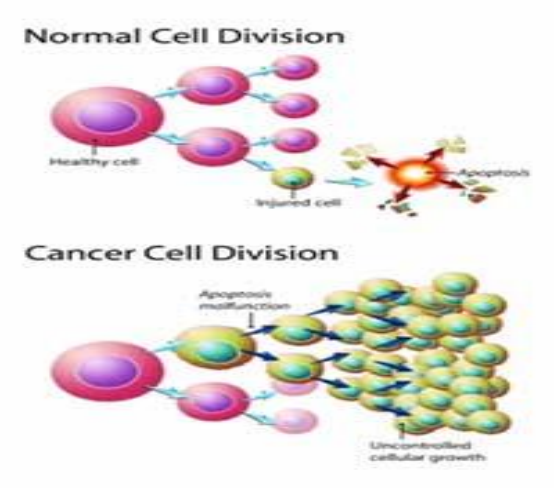

Figure 7

GROWTH

Human cells are not grow until there are adequate cells. For examples, when cells are produced to repair a wound in the body, fresh cells are also no more developed unless are there enough cells that pick up the slack, until the fixing cell is complete. [5] On the other hand, cancer cells cannot cease to develop until there are more cells necessary. This ongoing growth also contributes in the creation of a tumor. Every of these enzymes is a transcription factor, a material that causes cells to expand and differentiate on the basis of cell injury. Unless the genes that scripts for any of these factors is trapped in a mutation location, the enzyme proteins may begin to be generated. The cells begin to expand in reaction.

Development rate- Normal cells replicate themselves and avoid when enough cells are available. Cancer cells multiply quickly until they have matured.

Ripening- The ripeness in human cells. Cancerous cells, because they expand quickly and separate until the cells are totally mature, remain immature. Neuroscientists have used the word 'unformed' to define immature cells. A further way to understand this would be to consider cancer cells as cells which do not "develop" and concentrate in mature cells. The stage of cell development correlates to the rate of cancer.

*	G1	S	G2	M
G1	G1	S	G1/G2	M
S	S	S	S	M
G2	G1/G2	S	G2	M
M	M	M	M	M

Table 3: Group table for the converged stated of binucleated cells

Proof: Now, how cell forming a cycle and producing daughter cells.

The table shows that the group is abelian-which commutative always holds: $x \circ y = y \circ x$ for all $x, y \in G$, where G is the group. We can also show associativity, $x \circ (y \circ z) = (x \circ y) \circ z$ for all $x, y, z \in G$.

For example: $G1 \circ (S \circ G2) = (G1 \circ S) \circ G2$

$G1 \circ S = S \circ G1$

$S = S$

And,

$G1 \circ (M \circ G2) = (G1 \circ M) \circ G2$

$G1 \circ M = M \circ G2$

$M = M$

That are on the other end, it is indeed observed from the Table 3 of multiplications but on set {G1, G2, S, M} we could not have a structure. Notably, every row or column of the multiplication table in classification G includes exactly the G constituents once, and this will be a possible combination of components. The asset in row S and M fails. This same factor G1 and G2 are also unspecified. Even so, the Set is {G1, G2, S, M} brings that groupoid form-to be mentioned following.

Specific conditions occur as the mix cells with various types or the state with separation. Such kinds of tests, as examined in Hanna, were conducted for various stem cells. He's seen how and how the cells merge and expand. The fusion-type approach includes the transfer of nuclear power in another diploid cells form to the next, and the detection of the result. That is the migration of RNA species within cells and the detection of shifts in the morphology of the cells.

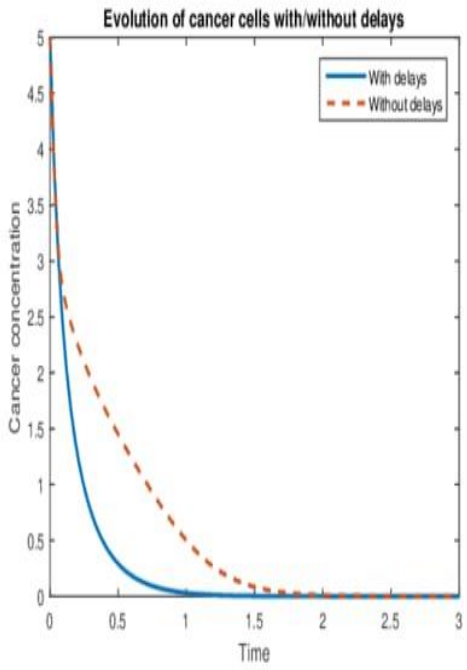

Figure 8

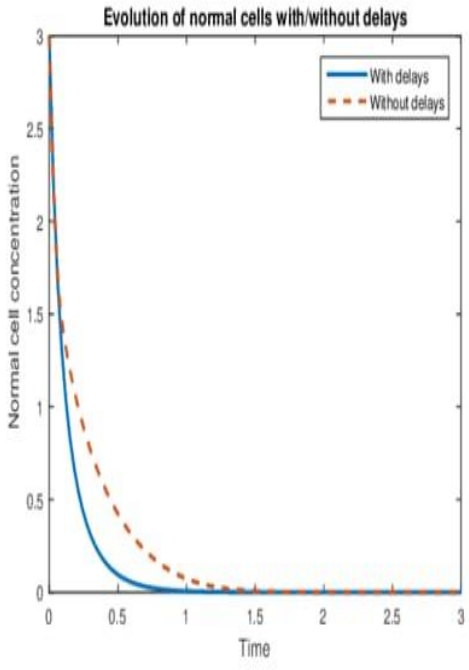

Figure 9

In this above graph (see Figure 8 & 9), we shown how normal and cancer cell growth. whatever cancer cell or normal cell both are having a same cycle path. Only differ in growth and spread.

CONCLUSION

Throughout this paper, we have briefly described how cancer cell and normal cell forming a cycle. Both cells are producing same G1 cell. Normal cell producing two daughter cells and cancer cells producing multiple cells. The distinction between living and non-living organisms is best known. We concentrate on the group theory and abstract algebra covering the biology of molecular systems. Finally, we proved the cycle in group theory application. Not only some specific cells. Every cells forming a cycle based upon their growths.

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