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Treating Cancer and many incurable diseases by resetting the cell programs

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Abstract

Cancer is a cell fleeing from death by blocking the intrinsic and extrinsic pathways of cell death programs. In this work, the experimental formula was designed to remove these blockers. It was applied on 120 Swiss albino mice inoculated intraperitoneally and subcutaneously with Ehrlich Ascites Carcinoma cells; $1 \times (10^6)$ cell/mouse. The activity of the cell death programs of the tumor was detected by measuring the volume of Ascites fluid, counting the number of dead cancer cells, measuring the size of the tumor, detecting the positive reaction of caspase enzyme in cancer cells and the presence of macrophages and apoptotic bodies in tumor tissue. Also, applied as the nutritional therapeutic program for a breast cancer patient, the activity of the cell death programs of cancer cells was detected by measuring the size of tumor mass. The experimental formula and the nutritional program succeeded in removing the blockers of the pathway of the cell death program in cancer cells and returning the cell death program to work again, the cancer mass disappeared within 34 days. Also, treating viruses by shutting down the activity of the transcription program of host cells, Psoriasis by controlling the activity of interleukin genes as well as, diabetes by preventing the activity of insulin resistance gene.

Keywords: Cancer; Detoxification Enzymes; Free Radicals; Glutathione; NF-kB

Introduction

ALLAH created the living cell and gave it a wide range of programs to perform its functions and remain healthy. So, any defect of one or more cell programs leads to loss of a certain cell function and a pathological condition occurs. The disease is a defect in one or more cell programs. So, we can treat any disease by resetting the cell programs. Resetting cell programming is an innovative and unique method to restore the body to health. The nuclear factor kappa –B(NF-Kb) is the master key for cell programs, it controls more than 500 genes so we can reset many cell programs we need and treat many incurable diseases. According to the World Health Organization Report in 2023, there were an estimated 20 million new cases of cancer people around the world, and there were 10 million of them died (1). There are many methods to treat cancer, such as surgery, radiation, monoclonal therapy, adoptive cell transfer, target therapy, an angiogenesis inhibitor, hormone therapy and stem cell transplant (2). But for all types of treatment out there, they all unable to save all cancer patients. Moreover, all of them have serious side effects. Therefore, it has become urgent to find a new method that is effective, safe and suitable for treating all cancer patients.

Cancer is defecting in one or more cell programs and appears as a pathological cell condition, to successfully treat cancer, it is necessary to identify the cell program responsible for the occurrence of cancer, as well as to identify what functions the cell lost that caused it to turn into a cancer cell, and then restore this program to work again normally (reset cell programs). To achieve this goal, a research plan has been drawn up as follows:

- Identify the cell program responsible for the occurrence of cancer
- Studying the cell death program.
- Why did ALLAH create two programs for cell death?

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- How does cancer occur? And what is the real cause of cancer?
- Studying the effect of carcinogen molecules and their relationship to cell death programs and cancer occurrence.
- Determining the stages of transformation of a normal cell into a cancer cell.
- Designing a therapeutic strategy to restore the cancer cell death program to work again.
- Applying the therapeutic strategy to experimental mice and then to humans.

Material and methods

Normal somatic cell divides into two similar cells; one lives and the other dies. A living cell to divide requires a division program and to die requires a death program. This means the normal somatic cells have two programs division program and death program. The death program controls the cell numbers which are produced by the division program of the cell.

Cancer cell which is somatic cell in origin divide mitotically as well into two similar cells but they both live. This means that the death program is impaired or blocked. Therefore, the cell death program is the program responsible for the occurrence of cancer.

1.1. Cell death program

All living cells have two death programs; one of them is located inside the cell called the intrinsic death program, and another program is located outside the cell called the extrinsic death program.

1.1.1. Extrinsic Death Program

It is an immune program that depends on immune cells which send molecules called ligands to cell death receptors which are located at the cell surface. When the ligands bind with death receptors activate the FADD protein, which activates the Caspase enzyme which decomposes the cell into fragments called apoptotic bodies leading to cell death (3&4) figure-1.

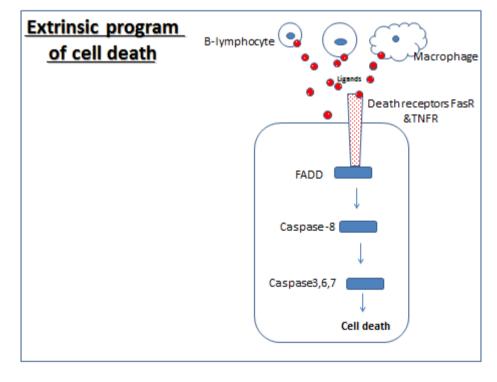


Figure 1 The pathway of extrinsic program of cell death

1.1.2. Intrinsic death program

It is a genetic program controlled by gene p53, the running pathway is done by a protein belonging to the bcl-2 protein family. All members of this family share a close homology in up to four characteristic regions termed the BH domains.

These BH domains are (BH1, BH2, BH3 and BH4). The proteins of this family are divided into; pro-apoptotic proteins which lead to the onset of cell death, and anti-apoptotic proteins which bind with pro-apoptotic proteins to prevent the onset of cell death.

Pro-apoptotic proteins divide into; multi-domain proteins and only domain proteins

Multi-domain proteins have two or more domains (Bax & Bak). The Bak protein is located on the outer surface of mitochondria while the Bax protein is located in the cell cytoplasm but in an inactive state. In addition, the only domain proteins that have only one domain (BH3-Only domain) as (Noxa & Poma).

The operation of the cell death program depends on the activity of gene P53. The activity of this gene is controlled by a factor called krupple-like factor which normally binds with the premotor region of gene P53 and keeps it in an inactive form. But, when the krupple-like factor is separated from the premotor region of gene P53, the Gene becomes active and stimulates the production of BH3-Only protein which activates the pro-apoptotic protein (Bax) whose presence in the cytoplasm and Trans locates it to the outer surface of mitochondria. Bax aggregates with each other as Bax-Bax or with Bak as Bax-Bak, forming a channel called the mitochondrial Apoptosis-Induced Channel (MAC). This channel open in between the outer and inner membranes of mitochondria, once this channel is formed, the Cytochrome –c enzyme which is present in between the inner and outer membrane of mitochondria releases into the cell cytoplasm (5&6)and engages the apoptotic protease activating factor-1(APAF1) and form apoptosome, which activates Caspase enzyme (Cysteiene aspartic proteases)(7). Once that happens, the death program runs and cannot stop.(8&9) (figure-2).It is worth noting that, the intrinsic and extrinsic death programs work in the same way by activating the same enzyme Caspase. So, one program of death is enough to get cell to die. Here the question arises: Why did ALLAH create two death programs?

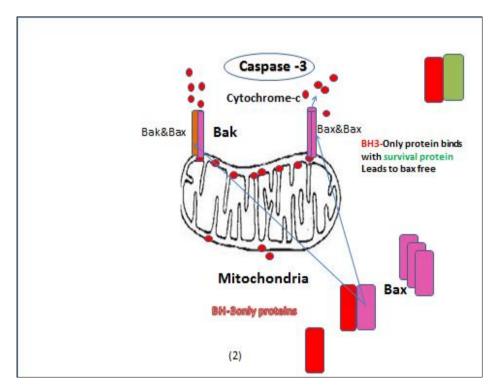


Figure 2 The activation of the Bax protein which is present in the cytosol by BH3-only protein and Trans locates it to the outer membrane of mitochondria forming a mitochondrial apoptosis-induced channel and releasing cytochrome-c to cytosol

1.2. The relationship between the death program and cancer

The active gene p53 is responsible for the death of the cell. Therefore, the mutation or destruction of this gene or prevention of its activity by muc-1 which binds the kurpple-like factor on the premotor region of the gene p53 leads to escape from death. On the other hand, the activity of the gene p21 which is responsible for mitotic cell division, is linked to the activity of the gene p53 is active, the gene p21 becomes active and binds with cyclin resulting in stop cell division. While the inactivity of gene p53 leads to the inactivity of gene p21. So, it is unable to bind to cyclin,

so the cell continues to divide (10). Therefore, the inactivity of gene p53 results in the cell escaping from death and continuing to divide, this means that the cell transfers to a cancerous cell (11) Figure 3.

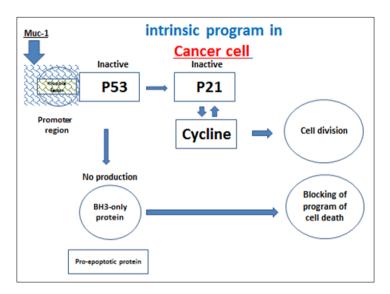


Figure 3 muc-1 interacts with gene P53 and makes the krupple-like factor tightly bound with the promoter region, leading to the inactivation of gene P53 ,by the extension suppression of gene P21 (cyclin- dependent kinase inhibitor)resulting in the prevention of cyclic arrest and also, blocks the BH3-only protein which leads to a block of the intrinsic apoptosis and cancer formation

1.3. Why did ALLAH create two programs for cell death?

To find the answer to this question, since there is a relationship between cancer and the cells escaping death. We had to return to the Holy Quran, which contains many scientific facts and research for verses that talk about escaping death. We found verse no.8 surat al-jumah is talking about escaping from death "Say to them, the death you try to flee from will meet you; then you will be conveyed back to him who knows the unseen and the witnessed, Then He will inform you of all that you have done". This translation is near to it is meaning.

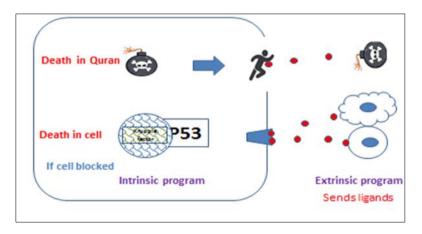


Figure 4 A comparison between trying to escape from death and not allowing this escape to happen in the cell and what was mentioned in the Quranic verse

This verse is talking about the phenomenon of fleeing from death and does not allow this fleeing to occur (12). This verse is talking about two deaths one becomes behind you when you flee and the second faces you and moves to meet you. This means that ALLAH Almighty created a system in his living creatures that does not allow escaping death. Accordingly, a comparison was made between trying to escape from death and not allowing this escape to happen in the cell and what was mentioned in the Quranic verse. It revealed that the manner of death in the cell is completely identical to the manner of death mentioned in the Quran verse, and therefore the attempt to escape death and not allow it to happen is completely identical, as follows; the death in the cell is represented by two opposite sides of death

(intrinsic death program & extrinsic death program). In addition; the movement of death, as mentioned in the verse, is represented by the immune cells sending ligands to meet the cell and bind with death receptors, see Figure 4.

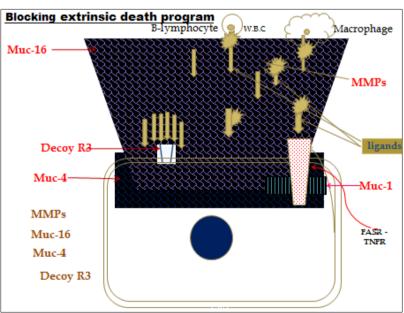
This comparison revealed that describing cancer as an escape from the intrinsic death program of cells is inaccurate because there is an extrinsic program that does not allow escape from death and sends ligands that bind with death receptors and destroy it. If the cell was destroyed via the extrinsic death program, this means there is no cell to transfer to cancer.

Only one program of cell death is enough to get the cell to die, but Allah created two programs of cell death. Thus, an important question arises in this context: "What will happen if Allah created only one program of cell death" i.e. only an intrinsic program?

Every day, the human body gets rid of 50 - 70 billion cells that are not needed by the intrinsic death program of cells (13). The work of the intrinsic program of cell death

Mainly depends on the activity of the p53 gene. When this gene is exposed to physical or chemical injurious agents, the p53 gene may be damaged or a genetic mutation may occur leading to block the intrinsic death program. If the intrinsic program of cell death is blocked in only one cell among 70 billion cells that die every day, it will result in cancer. Therefore, the probability of cancer occurring in people who have an intrinsic program of cell death only is 50 - 70 billion times daily. Thus, it is impossible for any human or living being to escape from cancer. But, Allah wisely created another program of cell death (an extrinsic death program).

If the cell succeeds in fleeing from death by blocking the intrinsic death program of cell, the extrinsic death program will meet the cell to get it to die by sending ligands to bind with its death receptor (FAS). This is identical to what is mentioned in Qur'an verse 8, surat aljomaa, "Say to them, The death you try to flee from will meet you; then you will be conveyed back to Him Who knows the Unseen and the Witnessed. Then He will inform you of all that you have done. "In addition, the accuracy of the Quranic pronunciation of fleeing means striving and making the necessary arrangements to ensure saving itself, which is more appropriate than the word escape.



1.4. How does cancer occur? And what is the real cause of cancer?

Figure 5 Muc-4 and Muc-16 prevent the binding of ligands with their death

Allah created the extrinsic death program to meet any cell that flees from death by blocking the intrinsic death program and destroy it. This leads to keeping the cell away from cancer. So, how does cancer form?

For the cancer to be formed, the cell must completely flee from death by shutting down both the death program of the cell (extrinsic and intrinsic). Here a logical question appears; how does the cell shut down the extrinsic death program?

For the cell to shut down its extrinsic death program, it produces five substances that it uses to prevent ligands from reaching the death receptors, which are located on the cell surface (see figure-5). The cell to be able to do that, it produces five substances as above.

Receptors and act like a mask on the death receptors. MMP3 (metalloproteinase enzymes) cleaves the ligands at the surface of the immune cells and the extracellular matrix so that no interactions occur with the death receptor. Decoy receptor-3 binds with ligands and keeps them away from the death receptor.

1.4.1. metalloproteinase enzyme (MMPs)

This enzyme breaks down the ligands; they found the ligands had been destroyed on the surface of white blood cells, as well as in the intercellular space (14&15).

1.4.2. DcR3 Receptors

These receptors are spread extensively on the surface of the cell, but they are ineffective as they don't have a domain that extends inside the cytoplasm to transmit the signal from outside the cell to inside it. These receptors are considered fake death receptors, and the ligands were shown clustering around them and sticking to them, thus the ligands are keeping away from the true death receptors (16&17).

1.4.3. Mucin-1(muc-1)

This muc-1 is a protein that binds with FADD protein and inhibits its activity (18).

1.4.4. Mucin-4 (muc-4)

It is a protein that extends inside the cytoplasm more than it has extended into the intracellular space. It acts as a cover for death receptors, preventing the ligands from binding the death receptors (18).

1.4.5. mucin-16 (muc-16)

This mucin is called tower mucin because it extends into the intracellular space almost reaches the neighboring cell and has a limited extension within the cytoplasm. This extension acts as a barrier that prevents the ligands from reaching the death receptors (18) See Fig -5.

Accordingly, a normal cell turns into a cancer cell when it completely flees from death, by shutting down both the intrinsic and extrinsic death programs. Based on the above, we can describe cancer correctly and accurately as follows; Cancer is a phenomenon of cell fleeing from death by shutting down the pathway of the intrinsic and extrinsic death programs.

1.5. What is the real cause of cancer?

When a cell is exposed to excessive free radicals and becomes close to death, the cell produces six substances that it uses to shut down both the intrinsic and extrinsic death programs. For a cell to shut down its intrinsic death program produces anti-apoptotic proteins (B-cl2) which bind with bax and bak inhibiting their activity, also it produces muc-1 that is directed to gene p53 and occupies its promoter region and binds the krupple-like factor strongly, keeping the gene p53 in an inactive form, this leads to blocking the intrinsic death program and continues cell division. This means the normal somatic cell transfers to the cancer cell. But it is not true cancer because there is another death program (the extrinsic death program) still active and can destroy it. So for the normal somatic cell to turn into a cancer cell, the extrinsic death program must also be blocked. For the cell to shut down the extrinsic death program produces five substances (muc-1, muc-4, muc-16, DcR3 and metalloproteinase enzyme), the cell uses those to prevent the ligands sent by immune cells from reaching the death receptors which located on the cell surface. blocking the intrinsic and extrinsic death programs lead to complete escape from death and continued cell division resulting in cancer formation. Thus, For the cell to produce the six substances that it uses to shut down both the intrinsic and extrinsic death programs, the nuclear factor kappa-B(NF-Kb) must be activated. Nuclear factor kappa-B presents in the cytoplasm and binds with the LC8 protein and inhibitor kappa-B. When hydrogen peroxide is formed inside the cell, it oxidizes the LC8 protein, this oxidation leads to separating the LC-8 protein from the structure leaving the nuclear factor kappa-B and inhibitor kappa-B bound together. This separation allows the kinase enzyme to phosphorylate the inhibitor kappa-B which separates from the nuclear factor kappa-B, then NF-kB becomes free and active and moves from cytoplasm into the nucleus and stimulates the genes (Bcl-2, muc-1, Decoy-R3, mmps and TNF- α) responsible for producing the six substances that the cell uses to shut down both the intrinsic and extrinsic death program (19&20&21) see Figure (6).

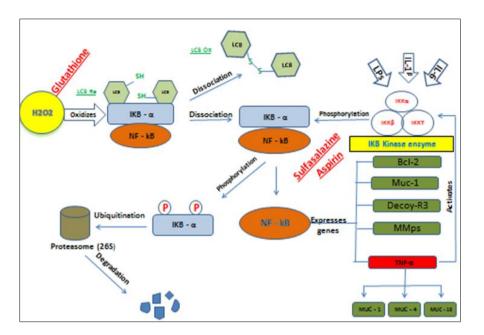


Figure 6 H2O2 oxidizes the LC8 leading to dissociation from IKB-α, then IKKs phosphorylate the IKB-α resulting in free NF-Kb, which Trans locates into the nucleus and stimulating the expression of genes responsible for the shutdown of the pathway of the intrinsic and extrinsic programs of cell death. TNF-α reactive the NF-Kb by stimulating LKKS

Based on the above, the real cause of cancer is the activity of the nuclear factor kappa-B (22). Also, to determine the stages of transformation of a normal cell into a cancer cell, we can refer to:

The direct cause of this transformation is the activation of nuclear factor kappa-B and hydrogen peroxide is the spark of this activity. While hydrogen peroxide is formed inside the cells as a result of the high level of phase-I detoxification enzymes .Since the accumulation of carcinogenic molecules leads to an increase in the levels of phase –I detoxification enzymes, we can determine the stages of transformation of normal cells into cancerous cells as the follows:

- First stage: Accumulation of carcinogenic molecules inside the normal cell.
- Second stage: increasing the level of phase-I detoxification enzymes.
- Third stage: formation of hydrogen peroxide.

Fourth stage: activation of the nuclear factor kappa-B, which expresses several genes, responsible for the production of the six substances that the cell uses to shut down the intrinsic and extrinsic death programs and becomes completely flee from death as cell division continues resulting in cancer formation. So, to treat cancer, all these stages must be blocked.

1.6. Treatment strategy of cancer

Treatment strategy is based on shutting down all stages of transformation of a normal cell into a cancer cell.

1.6.1. first stage

Removing the carcinogenic molecules from the cancer cell to reduce the levels of the phase -I detoxification enzymes, thus reducing free radicals and hydrogen peroxide formation. The carcinogenic molecules are hydrophobic. They move easily into the cell but they are difficult to get out of the cell, because the structure of the inner surface of the cell membrane which faces the cytoplasm is a phosphate group (hydrophilic), therefore, the carcinogenic molecules are kept away from the inner surface of the cell membrane and remain in the middle of the cell as a result of the force of expulsion from all direction.

To remove carcinogenic molecules from the cell to the outside, it needs two steps: the first step is moving the molecules from the middle of the cell to near to the inner surface of the cell membrane and the second step, is expelling it out of the cell. In the first step; the enzymes of phase –II detoxification must be activated to move the carcinogenic molecules from the middle of the cell to its pole, as it adds a hydrophilic group that binds to the carcinogenic molecule and

increases its polarity and moves it toward the phosphate group at the inner surface of the cell membrane in preparation for expulsion out the cell (23). Since each carcinogenic molecule is bound to the suitable group to be transported, it has become necessary to activate all enzymes of detoxification phase –II to ensure that all types of carcinogenic molecules are transported. Thus, to activate all phase –II detoxification enzymes, it must activate the nuclear factor erythroid-2 (Nrf-2(24).The Nrf-2 is activated by Sulphoraphane substance which present in the nature in cruciferous plants such as Broccoli and Cabbage (25).

The second step; is activating phase –III detoxification enzymes to expel carcinogenic molecules that have approached the inner surface of the cell membrane to out the cell).phase-III detoxification enzyme is activated by polyphenols (26)which present in the nature in apple and green coffee (27).

1.6.2. Shutting down the second stage of cancer formation

It can be done by reducing the level of phase-I detoxification enzymes to reduce the interaction with carcinogenic molecules hence, it leads to reduced hydrogen peroxide and superoxide free radicals formation. It can be achieved by reducing Cytochrome-p450 enzyme, xanthine dehydrogenase/oxidase enzyme and amino-oxidase enzyme.

Cytochrome p450 enzyme can be reduced by xanthohumol substance or naringenin substance which is present in the nature in grapefruits (28). Xanthine dehydrogenase/oxidase can be controlled by allopurinol substance or anthocyanidins substance which is present in the nature in blackberries or black grapes (29). Amino-oxidase enzyme reduced by curcumin which is present in the nature in turmeric(30).

1.6.3. Shutting down the third stage of cancer formation

It can be done by decomposing the hydrogen peroxide and preventing it from being formed again, it can be controlled by Glutathione which converts the hydrogen peroxide into water and oxygen (31). And preventing superoxide free radicals from converting to hydrogen peroxide by vitamin -E(32).

1.6.4. Shutting down the fourth stage of cancerous transformation

It can be done by preventing the activity of nuclear factor kappa- B which can be controlled through two steps;

- Decomposition of hydrogen peroxides and prevents it from being formed again.
- Inhibiting the activity of kinase enzyme by aspirin or dexamethasone.

Based on this strategy, we can create a hundred different drug combinations or therapeutic nutritional programs that can restore the death program of cancer cells. In any case, returning one death program of cancer cells to work again leads to a 100% cure rate. While returning the two death programs of cancer cells to work again achieving a 200% cure rate.

An experimental formula and a therapeutic nutritional program were created to return the death program of cancer cells to work again.

The experimental formula

(Broccoli extract 3.4 mg/mouse, Apple extract 14 mg/mouse, Grape fruit extract 6 mg/mouse, black berry 4 mg/mouse, Turmeric extract 2 mg/mouse, Vitamin A 3 mg/mouse, Vitamin C 3 mg/mouse, Vitamin-E 3 mg/mouse, Glutathione 0.4 mg/mouse, cysteine 0.4 mg/mouse, N-acetyl-L-cysteine 0.8 mg/mouse, selenium 0.07 mg/mouse and aspirin 0.6 mg/mouse. All plant extracts were moisture free. All ingredients of plants were extracted by ethyl alcohol 99% for one week then the alcohol was evaporated. Whole plant extracts were dissolved by Propylene Glycol then added the water, propylene did not exceed than 5%) and was applied to albino mice at the College of Veterinary Medicine at Suez Canal University, Egypt (33). Then it was applied to humans according to this strategy.

1.7. Appling the strategy to laboratory animals and then to humans

An experimental Formula was applied on120 female Swiss albino mice were maintained for a week acclimation period and were kept on a commercial standard diet and tap water until the end of the experiment. Mice were divided into six groups as follows:

• Group-1 (negative control): 20 mice were injected I.P with saline 0.9 NaCl.

- Group-2 (treated only): 20 mice were given the experimental formula in drinking water 3 ml/mouse daily.
- Group-3 (positive control): 20 mice were injected I.P with Ehrlich Ascites Carcinoma cells 1 × 10⁶ cell/mouse.
- Group-4 (positive treated): 20 mice were injected I.P with Ehrlich Ascites Carcinoma cells 1 × 10⁶ cell/mouse and were treated orally by the experimental Formula (3 ml/mouse daily) in drinking water.
- Group-5 (positive control): 20 mice were injected sub-cut with Ehrlich Ascites Carcinoma cells 1×10^6 cell/mouse.
- Group-6 (positive treated): 20 mice were injected sub-cut with Ehrlich Ascites Carcinoma cells 1 × 10⁶ cell/mouse and were treated by the experimental formula (3 ml/mouse daily) orally in drinking water. All procedures relating to the care and maintenance of the animals and fish were under the international guiding principles for the care and use of laboratory animals.

Results and Discussion

The method of treating diseases by resetting cell programs is innovative and can treat cancer, viral diseases, and incurable diseases at the same time

The Holy Quran led us to know a cellular phenomenon that was not known before. It is called the cell fleeing from death phenomenon, this phenomenon is the key to all cancer science, where it accurately re-describes cancer, determines the real cause of cancer, determines the stages of cancer formation and facilitates the creation of various drug combinations and many nutritional programs to treat cancer without any side effects.

- re-describing cancer as " Cancer is cell fleeing from death by blocking the pathway of the intrinsic and extrinsic programs of cell death (34).
- determining the real cause of cancer .when the cell is exposed to an excessive free radicals and becomes close to death, it activates the nuclear factor kappa-B which stimulates several genes, which responsible for producing the six substances that the cell uses to shut down both the intrinsic and extrinsic death programs to flee from death and keep itself alive (36).So, the activation of NF-Kb is the real cause of cancer.

4.1 Identification of the stages of cancer formation as follows

- First stage: Accumulation of carcinogenic substances inside the cell.
- Second stage: increasing the level of phase-I detoxification enzymes.
- Third stage: Formation of hydrogen peroxide.
- Fourth stage: activation of nuclear factor kappa-B.

One of the important features in the process of carcinogenesis is the ability of cancer cells to flee from death by blocking the program of cell death (12). The core of this paper is returning the death programs of cancer cells to work again.

The cancer treatment idea is to decompose cancer cells without any side effects, and this can be achieved by returning the death program of cancer cells to work again. The strategy of our study was designed to shut down all transformation stages, by preventing hydrogen peroxide formation and converting the present hydrogen peroxide into water and oxygen, and inhibiting kinase enzyme to completely inhibit the NF-kB activation (37).

The experimental formula was created and applied to albino mice. **It succeeded in** bringing the death program of cancer cells back to work again strongly and the cancer mass gradually decreased until it disappeared completely on the 34th day in mice(33). Then applied as a therapeutic nutritional program(figure 10) to humans also, it succeeded.

4.1.1 Applying to mice

An extract of plants was made and glutathione, vitamin C, vitamin E and aspirin were added to form an experimental formula and were applied to mice. The parameters of the death program activity of the cancer cells were detected by measuring the volume of Ascites fluid, counting the number of dead cancer cells, measuring the size of the tumor mass , detecting the positive reaction of caspase enzyme in cancer cells and the presence of macrophages and apoptotic bodies in tumor tissue. The experimental formula succeeded in removing the blockers of the cancer cell death program resulting in running the death program again (33).

The Volume of Ascites Fluid

On the 8th day of inoculation, the Ascites fluid volume of the controlled group of mice which were inoculation I.P with EAC cells was 1.63 ± 0.176 ml and continued increasing daily until reached 5.87 ± 0.384 ml then all mice of this group died on the 21^{st} day after inoculation. On the other side, the ascites fluid volume of the treated I.P inoculation group was 1.6 ± 0.153 ml and continued decreasing gradually until Reached zero ml on the 34th day of I.P inoculation of EAC, This means that the experimental formula succeeded in removing the blockers of death program pathways of cancer cells and returned the death programs to work again at the 8th day and disappeared completely on the 34th day(33).

Counting the Number of Cancer Cells

The result revealed increasing the EAC cells number in the I.P inoculation micegroup. The total number of cells in the positive control group was 217 ± 2.91 Million/mL, the number of live cells was 197.33 ± 2.91 million/mL and the number of death cells was 20.33 ± 0.67 million/mL, while the total number of cells in the treated group was 125.33 ± 4.67 million cells/mL, (the number of live cells were 83.67 ± 4.26 million cells/mL and the number of death cells was (41.63 ± 0.88)). The death cells in the treatment group represent 33.21% of all cells(33).

The Size of Solid Cancer

It was measured on the 18th day in the positive control sub-cut (EAC) cell inoculation group and the treated sub-cut inoculated group. The result revealed the size of the solid cancer was 0.251 ± 0.033 cm³ in the positive control group and gradually increased until reached 2.630 ± 0.221 cm³ on the 34th day of sub-cut EAC inoculation. While the size of solid cancer in the treated group was 0.127 ± 0.017 cm³ on the 18th day of inoculation and gradually decreased until reached zero on the 34th day of sub-cut EAC cells inoculation, this means that the death programs of cancer cells returned to work again(33).

Detecting the positive reaction of caspase enzyme in cancer cells

Immunohistochemical sections examined from tumor samples showed a positive reaction for caspase enzyme in tumor cells, starting from the base of tumor tissue, at the junction with the healthy tissue; it looks like an explosion of several water springs. This proves that the experimental formula succeeded in stimulating the death programs of cancer cells (Figure 7).

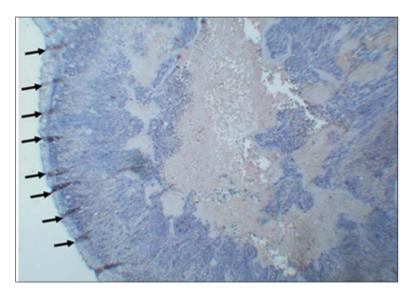


Figure 7 (IHC ×4) The Positive reaction (black arrows) in tumor cells beginning from base of the tumor representing abut 30% from the tissue. (sellbio, 2019)

The positive reaction to caspase enzyme in tumor tissue showed an extension of the response deep into cancerous tissue .Moreover, we observed the reduced density of the tumor cells near the areas of positive response to the caspase enzyme, indicating the success of activation of the apoptotic program in eliminating tumor cells (Figure 8).

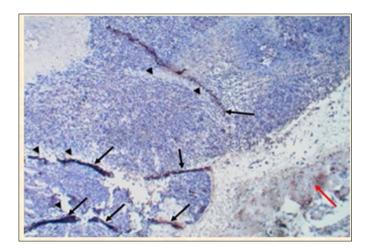
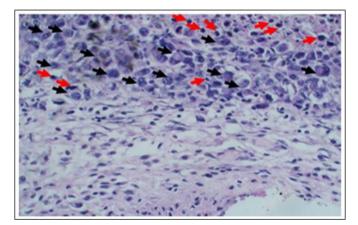
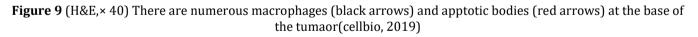


Figure 8 (IHC ×10) Multiple areas in tumor show such positive reaction to caspase enzyme, beginning from tumor base at the junction with muscle and extending deep into tumor cell. In addition, there is a reduced density of the tumor cells near the areas of positive reaction to caspase enzyme arrowheads (cellbio, 2019)

The presence of macrophages and apoptotic bodies in tumor tissue

In addition, histopathological examination revealed numerous macrophages and apoptotic bodies (red arrows) at the base of the tumor, indicating the successful elimination of tumor cells (Figure 9).





1.7.1. Applying to human

A therapeutic nutritional program (figure 10) was created to return the death program of cancer cells to work again in humans .It was applied to a 30 –year- old volunteer woman, who was suffering from a breast tumor, after histopathological examination of a sample of this tumor and ensure that she was suffering from malignant breast cancer.

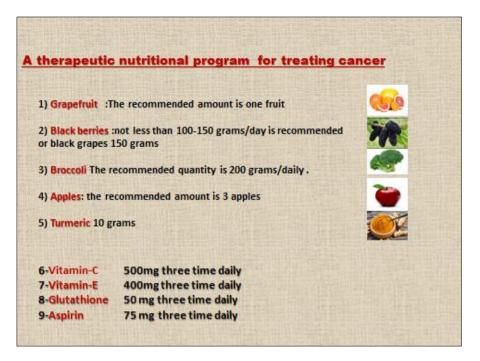


Figure 10 A therapeutic nutritional program for cancer treatment)

Some patients used glutathione 250mg one time daily instead of using 50 mg three time daily,the result was more responsive.

The results were recorded during successive time periods starting from taking the therapeutic nutritional program it was as follows:

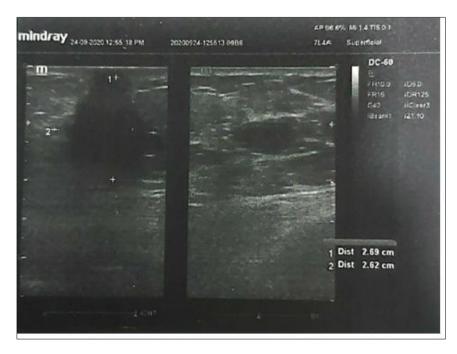


Figure 11 The first measurement of breast cancer after starting a diet program

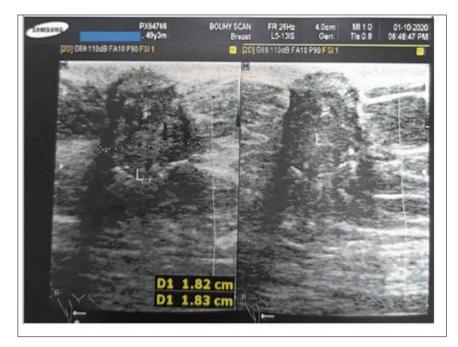


Figure 12 The measurement of breast cancer after one week of returning the death program to work again

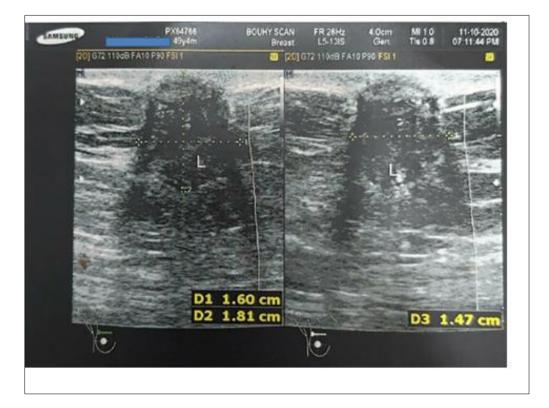


Figure 13 The measurement of breast cancer after 17 days of returning the death program to work again.



Figure 14 The measurement of breast cancer after 25 days of returning the death program to work again

total cancer decreased through 25 days after returning the death program of cancer cells to work again shown in table-1.

Table 1 The breast cancer decreased through 25 day

Date	size	Size /cm ³
24/9/2020	2.69x2.62x2.8	19.7cm ³
1/10/2020	1.82x1.83x1.8	5.99cm ³
11/10/2020	1.62x1.81x1.47	4.31cm ³
19/10/202	1.62x1.44x1.54	3.59cm ³
19.7 - 3.59 = 16.14cm ³		

According to the (table 1), 81% of the cancer mass disappeared within 25 days, and then doctors removed it through surgery.

1.8. The requirements for treatment with therapeutic nutritional program

- It should not be used except under medical supervision, as the medicinal nutritional program contains aspirin, and it should not be used with patients who suffer from stomach ulcers and hemophilia also, platelets should be measured before use of aspirin.
- In the case of using the therapeutic nutritional program with the chemical protocol; Caution must be taken when using grapefruit, as it reduces the cytochrome p450 enzyme any may be conflict with types of chemotherapy protocol medication.

- The therapeutic nutritional program regulates the blood pressure and diabetes so ,it is necessary to constantly measure blood pressure and diabetes before taking its medication.
- The threrapeutic nutritional program deals with all stages of cancer formation, therefore it must taken in full without any reducing, as each substance is complementry for what came before and paving the way for what comes after, also must take at accurat time.

Likewise, the nutritional program should be continous for 45 after the cancer disappears, as there is remnants of cancer cells that cannot be detected by x- rays.

Treating by resetting cell programs is a new effective, safe, and promising method that will solve many intractable pathological problems. The nuclear factor kappa -B, controls nearly 500 genes and therefore controls a very large number of cell programs. It is considered the master key to controlling cell programs, or the main controller of cell programming. So, keeping the nuclear factor kappa-B in a normal state leads to the return huge number of cell programs in a normal state and treats the most incurable diseases as cancer by returning the cancer cell death program to work again, treating viruses by shutting down the activity of the transcription program in the host cells. It has been used in Treating Covid 19(35). It can also be used to treat psoriasis by controlling the activity of interleukin 1 gene to interleukin 27gene, as well as diabetes by preventing the activity of the gene responsible for insulin resistance,

This treatment method deals with cancer cells only and doesnot affect neighboring cells. Therefore, it doesnot have any side effect like other treatment methods currently used.

This method deals with the cancer death cell program, every living cell has a death program. Therefore, it is suitable for the treatment of all types of cancer in all living organisms (human, animal, bird and marine creatures).

The Study revealed no toxic effect was observed during the use of the experimental formula. The experimental formula contains natural plants, vitamins, glutathione, and aspirin. So, it could be given orally in a diet program or a suspension form or in the form of capsules.

5. Conclusion

The method of treating diseases through resetting cell programming is an innovative and unique method to restore the body to health quickly. Nuclear factor kappa –B is the master key for cell programs, it controls more than 500 genes so can reset most cell programs we need. Treating cancer by returning the death program of cancer cells to work again, treating virus diseases by inhibiting the activation of the transcription program of host cells and keeping it in the normal state, also can treat psoriasis by controlling the activation of the interleukin genes.

Treating incurable diseases by resetting the cell programs as ALLAH created them is the best treatment method.

Compliance with ethical standards

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References

- [1] https://www.paho.org/en/campaigns/world-cancer-day-2023-close-care-gap (1) World Cancer Day 2023: Close the care gap.
- [2] National Cancer Institute. http://www.cancergov/about-cancer/treatmenttypes
- [3] Wajant,H.,(2002).Citation: The Fas Signaling Pathway: More than a paradigm.Science,296,1635-1636
- [4] Sessler, T., Healy, S., Samali, A., and Szegezdi, E(2013). Citation: Structural Determinants of DISC Function: New Insights into Death Receptors-Mediated Apoptosis Signaling. Pharmacology & Therapeutics, 140, 186-199.
- [5] Dejean, L.M., Martinez-Caballero, S. and Kennelly, K.W. (2006) Is MAC the Knife That Cuts Cytochrome C from Mitochondria during Apoptosis? Cell Death and Differentiation, 13, 1387-1395. https://doi.org/10.1038/sj.cdd.4401949

- [6] Dejean, L.M., Martinez-Caballero, S. and Kinnally, K.W. (2006) Regulation of the Mitochondrial Apoptosis-Induced Channel, MAC, by BCL-2 Family Proteins. Biochimica et Biophysica Acta , 1762, 191-201. https://doi.org/10.1016/j.bbadis.2005.07.002
- [7] Ow, Y.-L.P., Green, D.R., Hao, Z.Y. and Mak, T.W. (2008) Cytochrome-C: Function beyond Respiration. Nature Reviews Molecular Cell Biolog y, 9, 532-542. https://doi.org/10.1038/nrm2434
- [8] Albert, B., Johnson, A., Lewis, J., et al. (2002) Molecular Biology of the Cell. 4th Edition, Garland Science, New York.
- [9] Albert, B., Johnson, A., Lewis, J., Raff, M., Roberts, K. and Peter, W. (2008) Chapter18 Apoptosis: Programmed Cell Death Eliminates Unwanted Cells. In: Molecular Biology of the Cell, 5th Edition, Garland Science, New York, 1115.
- [10] Wei, X., Xu, H., and, Kufe, D. (2007). Citation: Human Mucin 1 Oncoprotein RepressesTranscription of the p53 Tumor Suppressor Gene.Cancer Research, 67, 1853-1858. Retrieved from URL, https://doi.org/10.1158/0008-5472.CAN-06-3063
- [11] Ahmed,R.,Alam,M.,Rajabi,H.,and Kufe,D.(2012).:Citation: The MUC1-C Oncoprotien Bind to the BH3 Domain of the Pro-Apoptotic Bax Protein and Blocks Bax Function. The Journal of Biological Chemistry,287,20866-20875.
- [12] Elkhodary ,m.s.m(2020)Cell fleeing from death phenomenon. Cellbio,9,1-13 https://doi.org/10.4236/cellbio.2020.91001
- [13] 10^A22 Atoms and 50-70 Billion Cells-Alpha Chiropractic Bethlehem, PA. https://www.alphachiropractic.net
- [14] Ebsen, H., Lettau, M., Kabelitz, D., and, Janssen, O. (2015), Citation: Subcellular Localization and Activation of ADAM Proteases in the Context of FasL Shedding in T Lymphocyte, Molecular Immunology, 65, 416-428.retrieved from URL, https://doi.org/10.1016/j.expneurol.2007.01.03.
- [15] Kiaei, M., Kipiani, K., Calingasan, N.Y., Wille, E., Chen, J., Heissig, B., et al. (2007), Citation: Matrix Metalloproteinase-9 Regulates TNF-α and FasL Expression in Neuronal Glial Cells and Its Absence Extends Life in a Transgenic Mouse Model of Amyo-trophic Lateral Sclerosis. Experimental Neurology, 205, 74-81.
- [16] Lau, W., Ramagopal, U., Cheng, H., Bonanno, J.B., Toro, R., Bhosle, R., Zhan, C., and, Almo, S.C. (2016), Citation: Crystal Structure of the Complex of Human FasL and Its Decoy Receptor DCR3. Structure, 24, 2016-2023.
- [17] Sheikh, M.S., and, Fornace, A.J. (2000), Citation: Death and Decoy Receptors and p53-Mediated Apoptosis. Leukemia, 14, 1509-1513. Retrieved from URL. https://doi.org/10.1038/sj.leu.2401865.
- [18] Hollingsworth, M.A., and, Swanson, B.J. (2004), Citation: Mucin in Cancer: Protection and Control of the Cell Surface. Nature Reviews Cancer, 4, 45-60. Retrieved from URL, https://doi.org/10.1038/nrc1251.
- [19] Hayden, M.S. and Ghosh, S. (2004), Citation: Signaling to NF-kappaB. Genes & Development, 18, 2195-2224.
- [20] Thomas D. Gilmore,(n.d), NF-kB Target Genes, Biology Department, Boston University 5 Cummington Mall, Boston, Massachusetts 02215-2406, USA ,Retrieved from URL, https://www.bu.edu/nf-kb/generesources/target-genes
- [21] Jung, Y., Kim, H., Min, S.H., Rhee, S.G. and Jeong, W. (2008), Citation: Dynein Light ChainLC8 Negatively Regulates NF-kb through the Redox-Dependent Interaction with IKB-αThe Journal of Biological Chemistry, 283, 23863-23871.
- [22] Elkhodary, M. (2018), Ciation: Quranic Verse No. 8 of Surat Al-Jumu'ah Leads Us to Describe Cancer and Determine Its True Cause. Part-II, CellBio, 2018, 7, 35-49 http://www.scirp.org/journal/cellbio,ISSN Online: 2325-7792, ISSN Print: 2325-7776 ISSN Print: 2325-7776.
- [23] J akoby, W.B. and Ziegler, D.M. (1990), Citation: The Enzymes of Detoxification. The Journal of Biological Chemistry, 256, 20715-20718.
- [24] Jung, K.A. and Kwak, M.K. (2010), Citation: The Nrf2 System as a Potential Target for theDevelopment of Indirect Antioxidants. Molecules, 15, 7266-7291. retrieved from , URL https://doi.org/10.3390/molecules15107266.
- [25] Dinkova-Kostova, A.T., Holtzclaw, W.D., Cole, R.N., et al. (2002), Ciation: Direct Evidence That Sulfhydryl Groups of Keap1 Are the Sensors Regulating Induction of Phase 2Enzymes That Protect against Carcinogens and Oxidants. Proceedings of the National Academy of Sciences, 99, 11908-11913.
- [26] Mizuno, N., Yotsumoto, T. and Sugiyama, Y. (2003), Citation: Impact of Drug Transporter Studies on Drug Discovery and Development. Pharmacological Reviews, 55, 425-461.

- [27] Veeriah, S., Miene, C., Habermann, N., et al. (2008), Citation; Apple Polyphenols Modulate Expression of Selected Genes Related to Toxicological Defense and Stress Response in Human Colon Adenoma Cells. International Journal of Cancer, 122, 2647-2655.
- [28] Fuhr, U. and Klittich Staib, A.H. (1993), Ciation: Inhibitory Effect of Grapefruit Juice and Its bitter Principle, Naringenin, on CYP1A2 Dependent Metabolism of Caffeine in Man. The Journal of Clinical Pharmacology, 35, 431-436.
- [29] Lin, S., Zhang, G., Liao, Y., Pan, J. and Gong, D. (2015), Citation: Dietary Flavonoids as Xanthine Inhibitors: Structure Affinity and Structure-Activity Relationships , Journal of Agricultural and Food Chemistry, 63, 7784-7794.
- [30] Wong, T.S., Chan, W.S., Li, C.H., Man, L.W., Tang, W.Y., Tsao, S.W., Tsang, K.Y., Ho, W.K. and Chan, Y.W. (2010), Citation: Curcumin Alters the Migratory Phenotype of Nasopharyngeal Carcinoma Cells through up-Regulation of E-Cadherin. International Institute of Anticancer Research, 30, 2851-2856.25.
- [31] Irshad, M. and Chaudhuri, P.S. (2002) Oxidant-Antioxidant System: Role and Significance in Human Body. Indian Journal of Experimental Biology, 40, 1233-1239.
- [32] Soonlae, R., Chul, J.Y. and Hwa, C.J. (2005), Citation: Effects of Vitamin E on PhospholipaseA2 Activity and Oxidative Damage to the Liver in Streptozotocin-Induced Diabetic Rats. Annals of Nutrition and Metabolism, 49, 392-396. https://doi.org/10.1159/000088930.
- [33] Mahmoud Saad Mohamed El-Khodary1,2, Sahar Ezeldien Hasan3, Wael A. Hassan4,5, Maather M. El-Lamie6, Ismail A. M. Eissa6, Waleed F. Khalil3, Salah M. Aly7(2019) How to Return the Death Programs of Cancer Cells to Work again and Cure Cancer within a short time. CellBio, 2019, 8, 17-39 https://www.scirp.org/journal/cellbio ISSN Online: 2325-7792,IssN print.2325-7776.
- [34] Elkhodary, M.S.M. (2018) Quranic Verse No. 8 of Surat Al-Jumu'ah Describes Cancer as a Complete and Accurate Description and Leads Us to Determine the True Cause of Cancer. "Part-1". CellBio, 7, 1-11. http://www.scirp.org/journal/cellbio https://doi.org/10.4236/cellbio.2018.7100
- [35] Elkhodary,M.S.M.(2020) Treatment of covid-19 by controlling the activity of the nuclear factor-kappa B"CellBio, 2020, 9, 109-121 https://doi.org/10-4236/cellbio-2020-92006. <u>https://www.scirp.org/journal/cellbio</u> ISSN Online: 2325-7792 ISSN Print: 2325-7776
- [36] El-Khodary, M.S.M. (2018) Quranic Verse No. 8 of Surat Al-Jumu'ah Leads us to Describe Cancer and Determine Its True Cause (Part-III).CellBio , 6, 35-49. https://doi.org/10.4236/cellbio.2018.73004.