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On The Chemoprevention of Petroleum Induced Carcinogenesis and The Role of Nanocatalysis

A.Badawi[1], A.Al-Sabagh[1], Nadia Kandile[2], Dina Abdelkader[1], Amal Abdelhafiz[1], Sahar Ahmed[1], M. Reyad[1], D. Harding[3], P. Collery[4], Eman Noaman[5], Nahla Mansour[1], Nadia Zakhary [6], A.Summan[7], M. Al-Ghobashy[8], Salwa M. Morsy[1,9] and Seham A. Shaban[1]

[1] Egyptian Petroleum Research Institute, Nasr City, [2] Chemistry Department, Faculty of Women, Ain Shams University, No. 11757 Cairo, Egypt,[3] Chemistry, Institute of Fundamental Sciences, Massey University, Palmerston, New Zealand,[4]Service de Cancerologie Medicale, Polyclinique Maymard, Rue Marcel Paul, 20407 Bastia, France, [5] National Radiation Technology Research Center, Nasr City, [6] National Cancer Institute, Cairo University, Cairo , Egypt. [7]. Faculty of Science, Umm-Al-Qura University, Holy Makkah, Saudi Arabia, [8] Analytical Chemistry Department, Faculty of Pharmacy, Cairo University, Egypt. and [9]Chemistry Department, Faculty of Science, Taif University, Saudi Arabia.

NANOTECHNOLOGY is a field which has been at the forefront of research over the past decades and its full potential has yet to be fully realized. One subset of nanotechnology that has emerged is nanomedicine, which has been able to exploit the unique properties of nano-sized particles for therapeutics. This review aims to discuss the current state of nanomedicine in the field of early detection of cancer and the subsequent targeted treatment. Current cancer detection methods rely on the patient to contact their provider when they feel ill or rely on non-specific screening methods, which unfortunately results in cancers being detected only after they have become too expensive for effective treatment. Furthermore, current treatment paradigms of cancer include mainly whole body treatment with chemotherapy agents exposing the patient to medications which non-specifically kill rapidly dividing cells, leading to debilitating whole body side effects. Nanomedicine has the potential to increase the specific treatment of cancer cells while leaving healthy cells intact through the use of novel nanocatalysis (nanoparticles) to seek and treat cancer in the human body. Co-polymer poly (lactic-co-glycolic acid) (PLGA) nanotechnology has been approved by the FDA for the use of drug delivery, diagnostics and other applications

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Dedicated to the late Prof. Amal Abdelhafiz and to the M.Sc. Chemist, Shahera Elshishini who were victims of occupational cancer.

including cardiovascular disease, cancer, vaccine and tissue engineering. However, there are undoubtedly toxicities of nanomedicines which have not yet been fully elucidated. This review discusses nanoparticles for the early detection and treatment of cancer such as: nanoshells, nanocantilevers, nanoprobes, nanocrystals, nanopolymers, nanocells, quantum dots, viruses and dendrimers. Known toxicities and possible mechanisms for toxicities of nanomedicines are also discussed.

Keywords: Cancer, Nanocatalysis, Chemoprevention and Petroleum.

It has been known for many years that some chemicals can cause cancer in man. More recently, there has been a growing awareness of the possibility that chemicals may also produce mutations in human germ cells thus influencing the frequency of genetic or heritable diseases. Many thousands of chemicals, including petroleum products, pharmaceutical products, domestic and food chemicals, and pesticides products are present in the environment and new chemicals are being introduced each year. In addition, there are many compounds that occur naturally, which are known to be mutagenic and/or carcinogenic (*e.g.,* mycotoxins in foods). It is important, therefore, that chemicals to which people are exposed, either intentionally (*e.g.,* therapeutically), in the course of their daily life (*e.g.,* in domestic products, cosmetics…, etc.), or inadvertently (*e.g.,* in pesticides) are tested for their potential to produce cancer and genetic damage (mutations). It is well established that benzene causes leukemia and other blood-related cancers and diseases. Benzene and benzene-based molecules (phenyls) are part of many popular medications used to relieve pain, alleviate cold and flu symptoms, and as a weight-loss aid. We know that benzene is a human carcinogen with a unique electromagnetic potential that enables it to insert itself into human DNA.

Most powdered laundry detergents contain benzene as part of the surfactant linear alkylbenzene sulfonate (LAS), which is added to help water penetrate fabric [(1)] . LAS concentrations of 7 to 13 percent in powdered detergents are noted in Material Safety Data Sheets provided by detergent manufacturers. You can easily verify this by examining the Household Products Database1 published by the National Institutes of Health.

Detergent residue remains in laundered items, as you can easily verify by wringing a clean, dry cloth in hot water. The white film left floating in the water likely contains benzene as part of linear alkylbenzene sulfonate LAS. Could benzene left on laundered clothing be causing cancer, especially in the pelvic and chest regions of the body where undergarments are in close contact with skin for prolonged periods of time? These areas become moist and salty with perspiration, which could facilitate the absorption of benzene through the skin. Skin is very porous; drugs such as nicotine, nitroglycerine, and birth control hormones are popularly administered through skin via adhesive patches. It was

believed that the body's macrophage cells release the benzene from the linear alkylbenzene sulfonate LAS molecules so the benzene with its unique electrostatic potential is free to wreak havoc on the cells it encounters in the body [2].

In 1965, new U.S. regulations required laundry detergent manufacturers to replace foaming phosphates with non-foaming linear alkylbenzene sulfonate LAS in detergents. According to the Centers for Disease Control and Prevention [3], more than 1,500 people a day die of cancer and about 1.4 million new cases are diagnosed each year. Urgent attention is needed to determine the role of benzene and polycyclic aromatic hydrocarbons in our current cancer epidemic. Petrochemicals and combustion products, including motor vehicle exhaust and polycyclic aromatic hydrocarbons (PAHs), linked to cancers of the bladder, lung, and skin.

This review aims to present the state-of-the-art knowledge of the role of surfactant, amino acids (or peptides), selenium, and magnesium in prevention of petroleum carcinogenesis. Also , the nutrition against petroleum induced free radical toxicity, bioactive food components, steps and mechanism of carcinogenesis, nutraceuticals and functional foods for chemoprevention of cancer, and finally nanotechnology in chemoprevention of carcinogenesis.

Surfactant prevention of complication from cancer

Polyethylene glycol (PEG) is a clinically widely used agent with profound chemopreventive properties in experimental colon carcinogenesis. Over the past several years, Corpert *et al.* have indicated that polyethylene glycol (PEG) has remarkable efficacy as a chemopreventive agent [4,5] . Indeed, the ability of this novel agent to suppress tumors or aberrant crypt foci in the azoxymethane (AOM) treated rat model was > 90%, generally outperforming reported efficacies of nonsteroidal anti-inflammatory drugs or those of other known chemopreventive agents [6]. Previous studies have suggested that PEG is a remarkably potent chemopreventive agent with effects seen throughout the spectrum of carcinogenesis. Specifically, PEG has been shown to cause regression of established lesions such as aberrant crypt foci [7] and also to inhibit the earliest stages of colon carcinogenesis including at the predysplastic mucosa [8]. Recently, it was found that epidermal growth factor receptor (EGFR) is the proximate membrane signaling molecule through which PEG initiates antiproliferative activity with snail/β-catenin pathway playing the central intermediary function [9]. Tween 80 is described as a nonionic, surface-active detergent, polyoxyethylene sorbitan monooleate. Tween 80 and some other nonionic and ionic surfactants appear to increase permeability of the cell membrane and to enhance uptake of dyes and proteins [10-15]. Tween 80 enhances uptake of the antibiotics AD and DM, especially in drug-resistant cells, as demonstrated by radioautography and as suggested by growth response in combination experiments [16] . Tween-80 has been shown to potentiate the cytotoxicity of etoposide (VP16) against several human lung adenocarcinoma

cells by increasing the accumulation of Vp16 in vitro. Tween-80-mediated sensitization of lung adenocarcinoma cell to Vp16 is considered to be related to both the characteristics of the cell membrane in adenocarcinoma cells and the lipotropic properties of Vp16. These results suggest this combination might have the potential to improve the therapeutic index of Vp16 in human lung adenocarcinoma [17]. The nonionic detergent Tween 80, which is used as a solvent for lipophilic drugs such as VP-16 and Taxotere, was found to reverse VP-16 resistance of the P-glycoprotein-associated multidrug resistance phenotype via increasing VP-16 influx. In adriamycin-resistant human chronic myelogenous leukemia K562 cells (K562/ADM), which overexpress mdr1 mRNA, the accumulation of VP-16 was only about 10% that in wild-type K562 cells. Tween 80 enhanced VP-16 accumulation in K562/ADM cells but did not influence VP-16 accumulation in parental K562 cells. VP-16 efflux was rapid and similar in both sensitive and resistant cell lines and was not blocked by Tween 80 or verapamil. Under glucose-free conditions, VP-16 accumulation in K562/ADM cells was only half of that in K562 cells. Tween 80 increased VP-16 accumulation in K562/ADM cells in glucose-free medium. In growth inhibition assay, Tween 80 reversed K562/ADM sensitivity to VP-16 without cell damage. Taken together, Tween 80 reverses VP-16 sensitivity in multidrug-resistant K562 cells by increasing influx, which is considered to be the primary mechanism of VP-16 resistance in K562/ADM cells [18].

The role of amino acids (or peptides) in the etiology and treatment of cancer

As outlined in other sections of this review, there are many factors involved in the etiology and treatment of cancer. Amongst these factors, one must consider the important role played by amino acids and their polymeric forms – peptides and proteins. The following outlines a very small number of examples of the parts that these moieties can play in the world of cancer. The action of amino acids and their derivatives can be helpful or harmful. For example, the amino acids phenylalanine and tyrosine as well as glutamine and methionine are reported to support tumor growth [19]. Human bradykinin (BK), a nonapeptide, is reported to be a vasodilator that dilates all blood vessels including those in tumors and hence also contributes to tumour growth [20]. On the other hand, the amino acid derivative N-acetylcysteine [21] and the peptide lunasin (43 amino acid peptide) [22,23] show much promise for anti-tumor therapy. Amino acids, peptides and proteins can complex with metals and aid in our fight against cancers. For example the dairy protein lactoferrin (80kDa) shows potential for the prevention of breast cancer [24]. Of course the best cure is prevention, which in the case of this presentation is to take all possible precautions to not breathe or come in direct contact with petroleum PAHs. In the case of the amino acids cited above by Fu *et al.* [19], the results of their study point toward tumor control based on the differing requirements of tumors versus normal cells for certain food intake requirements - in short they recommend dietary intake control. The BK nonapeptide amino acid sequence can be altered to produce peptide analogs that reverse BK's activity and show promise for human lung and prostate cancers [25]. In one study, BK analogs were shown to be less toxic and more

effective in suppressing human small cell lung cancer cell growth in mice than the well known ant-cancer agent, cis-platin [26].

N-acetylcysteine (NAC), as an example of an antioxidant, has been shown in vivo experiments with mice to reduce the incidence of lung cancer from birth due to the inhalation for volatile carcinogens [21] as may occur with petroleum industry workers. The NAC was administered during pregnancy and the babies studied for 210 days. Cysteine also plays a role in tumour therapy as a metal delivery agent. For example, L-methylcysteine is combined with selenium to give L-Se-methylselenocysteine for use as an anti-cancer agent [27] . Other bioorganic metallic agents involving amino acids include platinum [28], palladium[29], ruthenium [30] and germanium [31].

Peptides and proteins also play a major role in the world of cancer. An example of is lunasin a soya bean peptide of 43 amino acids [22,23] To date, it has shown strong promise *in vitro* against colon, breast and prostate cancers. In vivo studies are currently being carried out [23]. In its 43 amino acid sequence, this peptide to date exhibits a segment that delivers lunasin into the cells, a segment that targets histones (very basic proteins) in the cells and a highly acidic segment that binds to chromatin (a DNA/protein chromasomal complex in cell nuclei). Another recent example of peptide-anticancer activity is found in soricidin, a 27 amino acid peptide which is claimed to be useful in preventing/treating cancers resulting from over expression of calcium channels in the intestine [32]. Bovine lactoferrin has been studied for its potential as an anti-carcinogenic and anti-metastastic agent with colon studies proving the most promising to date.

Selenium inhibits petroleum carcinogenesis

Selenium has been frequently used as inhibitor of chemical carcinogenesis induced dimethylbenz(a)anthracene (DMBA) [33-35] . Sodium selenite inhibited aryl hydrocarbon hydroxylase (AHH) activity to a maximum of ≈ 70 % and suppressed the overall metabolism of benzo[a]pyrene [36]. Compared to the sulfur structured analogs, selenium compounds are much more active in cancer prevention and may have a multimodal mechanism in preventing cellular transformation as well as in delaying or inhibiting the expression of malignancy after DMBA exposure [37]. Intake of vegetables, selenium and particularly citrus fruit protects the renal VHL gene from mutation insults [38].

Selenium in cancer chemoprevention

Studies examining the relationship between the intake of dietary selenium and the risk of various cancers have shown that low selenium intake is associated with higher cancer rates, including liver cancer. These studies show that dietary organoselenium compounds induce enzymes that hydroxylate or oxidize the carcinogens and decrease DNA alkylation [39]. Previous studies in animals and humans have shown that selenium compounds can prevent cancer development. The association between production of TNT and associated

disorders and chemoprevention or anticarcenogenesis is insufficiently recognized in developing countries. Data from a sequential animal model of experimental hepatocarcinogenesis showed that sodium selenite is able to reduce the risk for cancer development in liver [(40)]. The mammalian genome encodes 25 selenoprotein genes, each containing one or more molecules of selenium in the form of selenocysteine. There is evidence that several selenoprotein genes may be involved with the mechanism by which selenium provides its anticancer effect [(41)]. Data on the differential expression patterns reported for selenoprotein genes in tumors versus normal tissue support their role in chemoprevention by selenium. Converging data from epidemiological, ecological, and clinical studies have shown that selenium can decrease the risk of some types of human cancers. Inducing apoptosis is considered an important cellular event that can account for the cancer preventive effect of selenium [(42)].

Selenium (Se) compounds are well known to inhibit cell proliferation and induce cell death in human cancer cells. Respective chemical forms of Se are intracellularly metabolized via complicated pathways, which target distinct molecules and exhibit varying degrees of anti-carcinogenicity in different cancer types; however, the precise mechanisms by which Se activates apoptosis remain poorly understood. The effects of Se compounds, Se-methylselenocysteine (MSC), selenomethionine (SeMet), and selenite on cell proliferation, apoptosis and its pathway in established human carcinoma cell lines (HSC-3, -4, A549, and MCF-7) were investigated. Cancer cells were treated with each Se compound during different periods. Cell apoptosis, caspase activity and ER stress markers were analyzed by flow cytometric or immunoblotting analysis, respectively [(43)]. Epidemiologic evidence in humans suggests a role for selenium in reducing cancer incidence and mortality. The ability of selenium dioxide (SeO_2) to enhance the lymphocyte progression through the cell cycle in patients with advanced (stage IV) cancer. Ten patients (mean age 51.9 years, range: 32-74; M/F ratio: 3/7) with tumors at different sites were included in the study. The addition into culture of SeO_2 1.5 microM enhanced significantly the progression into S phase of PBMCs isolated from cancer patients, whilst no significant effect was observed on PBMCs isolated from controls. ROS levels were significantly higher, whereas GPx activity was significantly lower in cancer patients than controls. Serum levels of IL-6 and TNF alpha were significantly higher in cancer patients than controls[(44)] . In 2010 there are many researches on Selenium as a chemoprotective anti-cancer agent. [(45-53)] .

The role of magnesium on prevention of petroleum carcinogenesis

Magnesium deficiency may favour the development of cancer

A low dietary Mg intake has been shown as associated with poorer DNA repair capacity and an increased risk of lung cancer [(54)]. The effects were more pronounced among older subjects (>60 years), current or heavier smokers, drinkers, those with a family history of cancer in first-degree relatives, small cell lung cancer and late-stage disease. Petroleum derivatives should also be

more carcinogenic in these conditions. Statistically significant inverse trends in risk were also observed in overweight subjects for colon and proximal colon cancer across increasing quintiles of magnesium uptake [55].

Food groups have been identified in the European Prospective Investigation into Cancer and Nutrition (EPIC) showed variations in absolute intakes of 23 key nutrients and food components as possible among the 10 countries participating in this EPIC study [56]. There was a clear geographical variability in intakes, with differences ranging to 35% for magnesium in men [57]. Differences can also be expected in the Mg deficiency that could be observed in the different countries in the world.

Nutrition against petroleum induced free radical toxicity

Cancer cells are the result of multiple genetic defects resulting from exposure to environmental, dietary and infectious agents. The dietary carcinogens such as N-nitroso compounds polycyclic aromatic hydrocarbons and heterocyclic amines are present in cured or spoiled foods and crude oil contaminated diet. The level of exposure of the cell DNA to those and other carcinogens depends largely on the general quality of diet, the presence of bioactivated dietary constituents, including antioxidant vitamins found in abundance in fruits and vegetables. In addition, normal cellular metabolizing enzymes which convert particular chemicals to more water- soluble compounds that can be excreted in the urine [58].

Oxidative stress induction by crude oil was indicated by increased lipid peroxidation and decrease in superoxide dismutase and catalase activities. However, pre-treatment of the diet with vitamins C and E exhibited a protective role on the toxic effect of crude oil. The order of protection was vitamin E + C > vitamin E > vitamin C [59].

As early as 1550 B.C., Egyptians realized the benefits of garlic against certain human cancers. Natural garlic cultivated with selenium fertilization have protective roles in cancer prevention [60]. Garlic and onion, broccoli, wild leek, have the ability to accumulate the selenium (Se) from soil. These Se-enriched plants present a higher protection against carcinogenesis [61]. Earlier studies on curcumin contribute to its antioxidant properties. Curcumin affects the production of interleukin 6 (IL-6) and 8 (IL-8) in head and neck squamous cell carcinoma, and exhibits therapeutic promise for prostate cancer [62-64].

Research on eating behavior and changing dietary patterns must be included in any cancer prevention strategy. A new paradigm for diet, nutrition and cancer prevention can be developed using multidisciplinary approaches that include lifestyle and environmental changes, dietary modifications and physical activity consciousness to reduce the burden of cancer not only for high risk individuals but for the general population as well [65].

Bioactive food components in cancer prevention

There is a new paradigm in nutrition research focusing on the preventing and controlling cancer which based on a strong scientific basis, and understanding the specific role of nutrients is critical. Scientists are focusing their research on how essential and nonessential nutrients influence genetic pathways and how dietary constituents interact with individual genetic profiles (polymorphisms). The current approach to nutrition research may not be adequate clinical, basic, and translational research must work together and inter-institutional and interdisciplinary collaborations will be essential for achieving these new research goals.

Reviewing the potential nutrient modifiers of cancer illustrates the complexity, especially the difficulties in using blood levels to measure their response, their intake, and their actions. Nutrient modifiers being studied for some cancer as prostate cancer include: allylsulfides, considered as the most important potential nutrient modifier; calcium and Vitamin D (the latter causes differentiation and regulates calcium metabolism), epigallocatechin-3-gallate (EGCG), obtained from tea and related compounds (this modifier has been related to prostate cancer prevalence), fatty acids found in fish, which appear to relate to the decrease in cancer incidence with fish intake, genistein from soy, which has estrogenic activities and appears to be an important agent in cancer prevention.

There are some water soluble derivatives of allicin, principally s-allyl methylcysteine, which is derived from γ-glutamylcysteine. It is highly reactive, very unstable, and difficult to measure but very important in cancer prevention.

Vitamin A and its precursor ß-carotene are also considered critical nutrient modifiers, even though the conversion of ß-carotene to vitamin A is slow and inefficient and the binding and action of vitamin A is a complex process. The use of vitamin A supplements has also not been proven to be effective in reducing cancer risk in humans. It appears that the combination of micronutrients in fruits, vegetables, legumes, and grains is more likely to be helpful than individual vitamins. Synthetic retinoids that are more potent than natural vitamin A or beta carotene have shown some ability to reverse pre-malignancies in the cervix, mouth, throat, and skin. They also may help prevent new tumors in people who have already been treated for these forms of cancer.

Vitamin E also has been studied as a nutrient modifier of prostate and other cancers. Vitamin E is absorbed into the intestinal tract, enters the mucosal cells, gains access to the bloodstream through the thoracic duct, is transported to the liver, joins the lipoproteins (particularly low-density lipoprotein [LDL]), and is transported into the cell. The bioavailability of vitamin E depends upon how efficiently it is absorbed, how efficiently it is transported in the blood, and how well it binds to serum lipoproteins, which will transport it into the cell. Vitamin

E is a powerful antioxidant protects cells from the damaging effects of free radicals, especially reactive oxygen species (ROS) [66-68].

Vitamin C is an antioxidant that is able to neutralize free radicals, molecules that can harm a cell's membrane and genetic material. Vitamin C may also help regenerate Vitamin E, which not only protects cells from damage that leads to cancer, but enhances the immune system as well. In addition to neutralizing free radicals, vitamin C helps neutralize cancer-causing nitrosamines, which are produced during the digestion of nitrites and nitrates, preservatives found in meats and vegetables

A large number of scientific studies of many types have provided evidence suggesting that vitamin D may have a role in cancer prevention.

A high calcium intake may decrease the risk of one or more types of cancer, whereas studies suggest that a high calcium intake may actually increase the risk of prostate cancer.

Nutraceuticals and functional foods for chemoprevention of cancer

The term "Nutraceuticals" was first defined in 1989 by Stephen DeFelico as food ingredients or dietary supplements that demonstrate specific or medicinal health benefits [69,70]. Nutraceutical products fall into two categories, ingredients isolated from food to be consumed in a medicinal form and those added to food products, often referred to as "functional food" or "fortified food" [71] . A large volume of literature is available on nutraceuticals inhibitory effects on cancer cell growth. These findings are based on observations of cultured cancer cell proliferation, enhanced apoptosis and the antioxidant action [72]. Several examples of Nutraceuticals have been reported in the literature such as Lycopene (tomato products), Insoluble fibre (wheat bran), soluble fibres (Psyllium Protects), Betaglucan (oats, barley Protects) and recently, Milk Fat Globule Membrane (MFGM) [73,74].

MFGM was shown to prevent the development of cancer, especially breast cancer in human [75] . It originates from the plasma membrane of the mammary gland secretary cells, surrounds fat globules in milk and is formed of a large number of polypeptides, at least 50 (10 to 300 kDa). The main polypeptide components of the MFGM are; the glycoprotein butyrophilin (about 40%), and xanthine oxidase (12 to 13%) of the total protein of the MFGM. The rest of the other polypeptides are present in MFGM at low abundance, 5% or less [76]. However the physiological role of the MFGM proteins is not completely clear, there are some reasonable suggestions about a cancer prevention function of these proteins [75].

In addition to the role of MFGM-derived proteins and polypeptides in cancer prevention, the phospholipids contained in the MFGM were found also to affect the development of cancer [77]. Moreover, milk fat globules can act as a drug

delivery system for other cancer chemopreventive agents such as selenium, fat soluble vitamins and even anti-cancer drugs [74,78].

Nanotechnology in chemoprevention of carcinogenesis

Advances in nanotechnology are the impetus for the next industrial revolution for the detection of diseases. This might obviate patient care with a shift towards early detection and prevention. Nanotechnology will help define cancers by molecular signatures denoting processes that reflect fundamental changes in cells and tissues that lead to cancer [79]. To support the measurement, analysis, and manipulation of molecular processes at scale and in context, new technologies will be needed [80] . These ultimately will become the first wave of clinical tools to examine tissues and samples, pushing towards an entirely new method of addressing cancer that integrates detection, diagnosis, and intervention on a common technology platform. Nanotechnology opportunities emerging for cancer diagnostics, therapeutics, and prevention will involve single-molecule analysis, single-cell analysis small cell populations, and multi-parameter analysis [81] . Microfluidics and microelectronics will provide key and striking opportunities, and these technologies will move down in scale to the nano-realm. Nanotechnology provides new ways to decipher biological information. One possibility is the application of nanopores to sequence DNA and characterize proteins and other molecules. The ability to genotype at very high throughput will be exceptionally important. It was reported that nanotechnology offers possibilities for detection of Ribonucleic acid (RNA) splicing, examining proteins, and as saying cellular behaviors at the level of single cells. Researchers, when developing technology at the nanometer scale, should bear in mind dynamic range, techniques for RNA expression and RNA splicing. Particularly in cancer, detection methods capitalize on amplification technologies, while proteomics initiatives are disadvantaged by the lack of appropriate technology for protein amplification, although phage display technologies do offer potential in that the detection molecule can be amplified. In this and other approaches, sensitivity of detection is preeminent.

The current approach begins with microarrays to interrogate multiple tissues to determine specific genetically determined characteristics, or cellular phenotypes. Nanotechnology holds promise for providing new tools for understanding the cell, the differences between normal and abnormal cells, and the mechanisms of communication between them. This information is fundamental to design of cancer detection and prevention strategies. Nanotechnology may also provide nondestructive “windows” into cells, with the ability to manufacture particles or probes that are small enough to be inserted into cells and monitor in real time without damaging the cell. Current research is shifting the focus from chemotherapy to chemoprevention [82-85].

The field of nanotechnology is currently undergoing explosive development on many fronts. The technology is expected to generate innovations and play a critical role in cancer therapeutics. Among other NP systems, there has been

tremendous progress made in the use of gold nanoparticles, nanorods, nanoshells and nanocages in cancer therapeutics. These nanostructures play a bigger role in effective drug delivery due to their ability to carry anticancer drugs and targeting molecules. In addition, these nanostructures are being used as a therapeutic agent in photothermal therapy, as well as an anticancer drug enhancer. These new opportunities allow innovations leading to effective combinational therapy in the fight against cancer. In this review article, the recent progress in the development of gold-based NPs towards improved therapeutics will be discussed. A multifunctional platform based on gold nanostructures with targeting ligands and therapeutic molecules will hold the possibility of promising directions in cancer research [(86)].

Conclusion

The overall goal was to develop a low dose, novel combination of chemopreventive agents delivered using a unique nanotechnology-based colon-targeting delivery system for synergistic chemoprevention.

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الوقاية الكيماوية من التسرطن الناتج من البترول ودور الحفازات النانوية

عبد الفتاح بدوى[1]، احمد الصباغ[1] ، نادية قنديل[2] ، دينا عبد القادر[1] ، امال عبد الحافظ[1] ، سحر أحمد[1] ، محمود رياض[1] ، ديفيدهارديخ[3] ، فليب كوليرى[4] ، ايمان نعمان[5] ، نهلة منصور[1] ، نادية زخارى[6] ، عبد الهادى شومان[7] ، مدحت الغباشى[8] ، سلوى مرسى[1،9] وسهام شعبان[1]

[1] معهد بحوث البترول – مدينة نصر ، [2]قسم الكيمياء – كلية البنات – جامعة عين شمس – القاهرة – مصر ، [3]معهد العلوم الاساسية – جامعة ماسى – بالمرستون – نيوزيلاندا ، [4] المركز الطبى – بانستيا – فرنسا ، [5]مركز بحوث تكنولوجيا الاشعاع – مدينة نصر ، [6] المعهد القومى للسرطان ، [7] كلية العلوم – جامعة أم القرى– مكة المكرمة – السعودية ، [8] قسم الكيمياء التحليلية – جامعة القاهرة – مصر و [9] قسم الكيمياء – كلية العلوم – جامعة الطائف – المملكة العربية السعودية .

تكنولوجيا النانو هو مجال الذي كان في طليعة الأبحاث على مدار العقود الماضية وإمكاناتها الكاملة لم تتحقق بالكامل .مجموعة فرعية واحدة من تكنولوجيا النانو التي ظهرت هوالطب النانوي، والتي تمكنت من استغلال الخصائص الفريدة لحجم جسيمات النانو للمداواه .هذه المشاركة تهدف إلى مناقشة الوضع الحالي للطب النانوي في مجال الكشف المبكر عن السرطان وتستهدف معالجة لاحقة .طرق الكشف عن سرطان الحالية تعتمد على المريض في الاتصال بالطبيب عندما يشعر بالمرض أو الاعتماد على أساليب الفحص غير محددة، مما يؤدي للأسف إلى أكتشاف السرطان فقط بعد حدوثه والتكلفة لتلقي العلاج الفعال أصبحت مكلفة للغاية .وعلاوة على ذلك، نماذج المعالجة الحالية لسرطان تشمل تعريض كامل الجسم للعلاج الكيميائي و تعريض المريض للأدوية يؤدي إلى آثار جانبية منهكة للجسم كله. الطب النانوي لديه القدرة على زيادة علاج محدد للخلايا السرطانية بينما يترك باقى الخلايا السليمة على حالها من خلال استخدام حفازات نانوية جديدة (nanoparticles) للحصول على علاج السرطان في جسم الإنسان بإستخدام تكنولوجيا النانو للبوليمر ((poly(lactic-co-glycolic acid) PLGA))) تمت الموافقة من قبل ادارة الاغذية والعقاقير لاستخدام تكنولوجيا النانو لتقديم الأدوية ووسائل التشخيص وغيرها من التطبيقات بما في ذلك مرض القلب والأوعية الدموية والسرطان واللقاحات وهندسة الأنسجة .ومع ذلك، هناك بلا شك من السميات من nanomedicines لم يتم توضيح بشكل كامل. يناقش هذا التعليق النانوية للكشف المبكر وعلاج سرطان مثل nanoshells :، وnanocantilevers، nanoprobes، البلورات النانوية، nanopolymers، nanocells، نقاط الكم والفيروسات و .dendrimers وتناقش أيضا السميات المعروفة والآليات الممكنة لإزالة السميات من nanomedicines.