

Modellings of Infectious Diseases and Cancers under Wars and Pollution Impacts in Iraq with Reference to a Novel Mathematical Model and Literature Review

Mohemid Maddallah Al-Jebouri 

Department of Medical Laboratory Technology, Al-Qalam University College, Kirkuk, Iraq
Email: profaljebouri@yahoo.com

How to cite this paper: Al-Jebouri, M.M. (2023) Modellings of Infectious Diseases and Cancers under Wars and Pollution Impacts in Iraq with Reference to a Novel Mathematical Model and Literature Review. *Open Journal of Pathology*, 13, 126-139.
<https://doi.org/10.4236/ojpathology.2023.133013>

Received: May 6, 2023

Accepted: July 4, 2023

Published: July 7, 2023

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Abstract

Microbial pathogens include bacteria, viruses, fungi, and parasites and together account for a significant percentage of acute and chronic human diseases. In addition to understanding the mechanisms by which various pathogens cause human disease, research in microbial pathogenesis also addresses mechanisms of antimicrobial resistance and the development of new antimicrobial agents and vaccines. Answering fundamental questions regarding host-microbe interactions requires an interdisciplinary approach, including microbiology, genomics, informatics, molecular and cellular biology, biochemistry, immunology, epidemiology, environment and interaction between host and microbe. Studies investigating the direct effects of pollutants on respiratory tract infections are very vast, but those interested in the role of a pre-existing disease and effects of the exposure on the response to secondary stresses are few. In an experimental study at concentrations of air pollutants found in urban environments, frank toxicological responses are rarely observed, however, exposure to secondary stress like the respiratory challenge with infectious bacteria can exacerbate the response of the experimental host. The models like experimental, mechanical, and mathematical are the most abstract, but they allow analysis and logical proofs in a way that other approaches do not permit. The present review is mostly concerned with these model representations particularly with a novel mathematical model explaining the interaction between pathogen and immunity including the equivalence point.

Keywords

Infectious Diseases, New Mathematical Model, Immunity, Environment,

1. Life and Environment

Environmental pollution is defined as the undesirable change in physical, chemical and biological characteristics of our air, land and water. According to the essential concepts of ecology, any form of life could be called as “an ecosystem” provided that the two domains of factors are present: the first part is named as the “biotic factors”, this includes: producers, consumers and decomposers. The second part of an ecosystem is the “abiotic factors” representing chemical and physical factors [1].

Organisms interact among themselves. Hence, all organisms, such as plants, animals and human beings as well as the physical surroundings with whom we interact form a part of our environment. All these constituents of the environment are depended upon each other, thus, they maintain the balance in nature [2]. As the environment is the supplier of all forms of resources and the wastes are cleaned up by the environment itself, it maintains the genetic diversity and stabilizes the ecosystem [3].

The environmental imbalance gives rise to various environmental problems. Some of the environmental problems are pollution, soil erosion leading to floods, salt deserts and sea recedes, desertification, landslides, change of river directions, extinction of species, depletion of natural resources, waste accumulation, deforestation, thinning of ozone layer and global warming [4]. The environmental problems are visualized in terms of pollution, growth in population, development, industrialization, unplanned urbanization etc. As a result of over-populations, rapid industrialization, heavy industries, and other human activities like agriculture and deforestation etc. as shown in the diagrammatical model (**Figure 1**), the earth became loaded with diverse pollutants that were released as by-products [5].

2. Self Purification of Environment and Balance of Nature

In spite of receiving large quantities of pollutants, which sometimes strongly affected the stability of ecosystems, most of times, nature have the ability to get back its previous stable state via multiple mechanisms, this recovery often depends on kind and rate of pollutants, generally, pollutants are grouped under two classes [6]:

1) Non-Biodegradable Pollutants: Nonbiodegradable pollutants are stronger chemical bondage, do not break down into simpler and harmless products. These include various insecticides and other pesticides, mercury, lead, arsenic, aluminum, plastics, radioactive waste etc. [7].

2) Biodegradable Pollutants: Biodegradable pollutants are broken down by the activity of micro-organisms and enter into the biogeochemical cycles. Examples

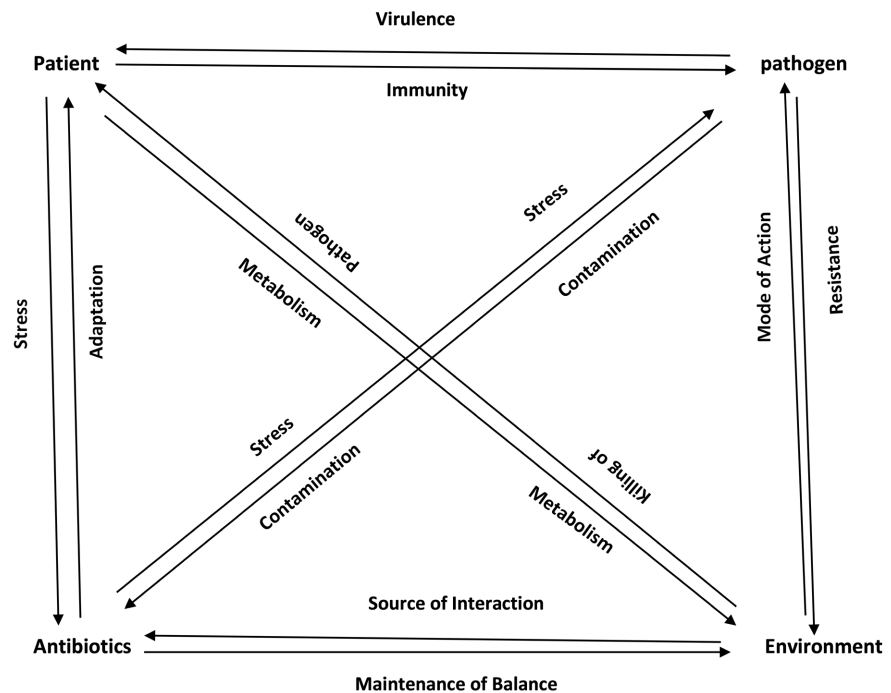


Figure 1. Modelling of pathogenicity parameters.

of such pollutants are domestic waste products, urine and fecal matter, sewage, agricultural residue, paper, wood and cloth and even oil [8]. The process by which organisms act to breakdown oil spillage is called “bioremediation”. It is the process done by many kinds of living organisms like plants, fungi, and bacteria. In the recent years, new techniques applied to get rid of hazardous pollutants, for example: spots of oil leaked through oil transporting or production processes can be treated by distinct genera of bacteria which have been collected, bred in vast quantities and stored in powder form to be used to clean up oil spills [9]. On the other hand, presence of heavy metals in air, water or in soil at ratios more than allowable levels could be considered as one of the most dangerous kinds of pollution due to their carcinogenic properties beside accumulation in living organisms. As microorganisms represent the heaviest ring in the chain of living organisms [10], it become very logical to focus on the effect of such pollutants on this kind of organisms, especially, large number of bacteria causing serious diseases to the man and his domestic animals and plants. As the high levels of heavy metals known to be toxic to microorganisms, they develop different mechanisms to resist higher concentrations of these metals, thus they defend their existence in the nature, eventually, they kept their role in the environment [11] [12].

3. Interrelationship between Respiratory Tract Infections and Oil Refinery Pollutants

Studies investigating the direct effects of pollutants on respiratory tract infections are very vast, but those interested in the role of a pre-existing disease and

effects of the exposure on the response to secondary stresses are few. In an experimental study at concentrations of air pollutants found in urban environments, frank toxicological responses are rarely observed, however, exposure to secondary stress like the respiratory challenge with infectious bacteria can exacerbate the response of the experimental host [13].

The relationship between exposure to air pollutants and the resistance to respiratory infections has been investigated on mice. The gaseous pollutants included in that study are ozone and nitrogen dioxide, and the experimental group is artificially infected with *Streptococcus pyogenes* and *Klebsiella pneumoniae* and previously they are exposed to different concentration of those gases, while the control group also infected with bacteria but let to breathe filtered air. The results showed higher rates of death and shorter time of survival in the experimental group [14].

Chronic inhalation of metal particles leads to a lower respiratory tract response characterized by an accumulation of mononuclear and polymorphonuclear phagocytes, epithelial cell damage, and interstitial fibrosis [15]. Other study shows that sheep obligated to inhale inorganic dust for 2 weeks/month for 18 months results in priming lung inflammatory cells and markedly enhanced their capacity to release toxic oxygen radicals [16]. Mononuclear phagocytes (MPs) recovered from healthy subjects are strongly inhibitory for cytotoxic lymphocytes, thus, natural killer (NK) cells and several phenotypes of T cells become dysfunctional after contact with autologous MPs and eventually enter a suicidal program of apoptosis [17]. NK cells are more sensitive to this suppressive signal than other subsets of cytotoxic lymphocytes [18]. Studies on the mechanisms responsible for the triggering of lymphocyte dysfunction by MPs have demonstrated a pivotal role for reactive oxygen species [19]. Although phagocyte derived oxidants play an essential role in defending the lung against inhaled microorganisms, they can also inactivate extracellular proteins such as alpha-1-antitrypsin induce lipid peroxidation in cell membranes cause cytogenetic injury and mediate cell death [20].

4. Bacterial Resistance to Antimicrobial Agents

Antimicrobial agent: A general term for drugs, chemicals, or other substances that either kill or slow the growth of microbes. Among the antimicrobial agents are antibacterial drugs, antiviral agents, antifungal agents, and antiparasitic drugs [21]. The two domains of most frequent in studies are the antibiotics and heavy metals. Many of them have shown a correlation between metal tolerance and antibiotic resistance in bacteria because of the likelihood that resistance genes to both (antibiotics and heavy metals) may be located closely together on the same plasmid in bacteria, thus more likely to be transferred together in the environment [22].

The intensive use and misuse of antibiotics have resulted in antibiotic resistance among several human pathogens. They reduce the possibilities for infec-

tions treatment and jeopardizing medical procedures such as organ transplantations or implants of prostheses where infective complications are common and antibiotic therapy is needed to prevent or treat those infections [23]. There are two main mechanisms involved in the development of antibiotic resistance, namely mutation [24] and acquisition of resistance genes [25] by horizontal gene transfer (HGT). Human pathogens are susceptible to antibiotics before the use of these drugs for the treatment of infections. The origin of antibiotic resistance determinants acquired by HGT must necessarily lie in the non-pathogenic micro biosphere. In some instances, human commensals can provide antibiotic resistance to pathogens [26]. However, in most cases, the antibiotic resistance genes have originated in the environmental microbiota [27]. The term antibiotic is originally coined to the name of those compounds produced by micro-organisms and capable of inhibiting bacterial growth [28]. Although any type of drug (natural or synthetic) used for treating bacterial infections is frequently termed as an antibiotic nowadays (Figure 2). Since several antibiotics are produced by environmental bacteria, it is conceivable that antibiotic-producing organisms could be the origin of HGT-acquired antibiotic resistance genes because these micro-organisms must have systems to avoid the activity of the antimicrobials they produce [29]. It is clear that antibiotic resistance genes originated in environmental bacteria so that changes in natural ecosystems may impact upon antibiotic resistance and consequently human health. Among those changes, the release of antibiotics together with human-linked microbiota eventually containing antibiotic resistance genes can be particularly important for the future evolution of antibiotic resistance in pathogenic bacteria [30]. Because of the prevalence of antibiotic resistant pathogenic bacteria, infectious diseases are becoming more difficult and more expensive to be treated [31].

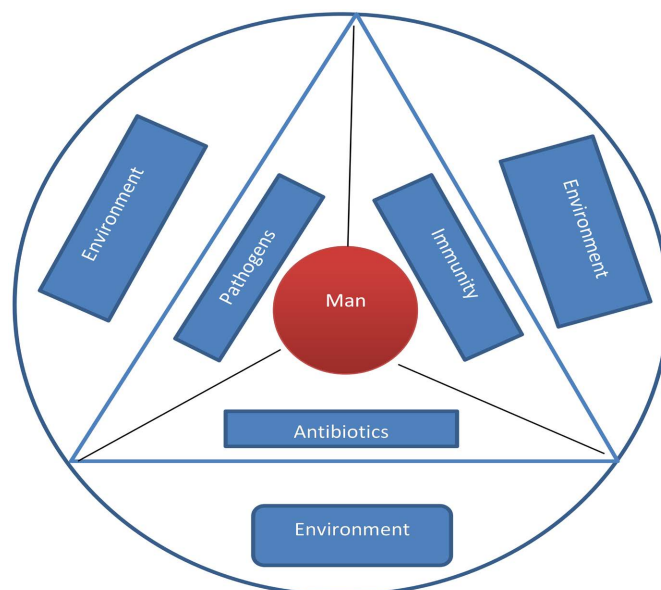


Figure 2. A model of interrelationship between different factors affecting infectious diseases.

Environmental monitoring of microbial contaminants is important for crew health and assessing functionality of engineering systems. Routine monitoring of air and surfaces on the International Space Station found *Staphylococcus spp.* to be the most common bacterial species whereas *Aspergillus spp.* were the most common fungi. The levels of microbial contaminants in the air and surfaces were typically low and within the acceptability limits. Bacterial levels in the potable water from the hot water port were uniformly low. The space environment, stress, and other factors may also diminish the host immune system. The status of antimicrobial functions of neutrophils and monocytes was determined by flow cytometry. Shuttle astronauts had decreased functionality of NK cells, neutrophils, monocytes, and changes in other elements of immunity. Latent virus reactivation is an important new approach to assessing immunity and can be monitored in body fluids collected during space flight. Reactivation of Epstein-Barr virus (EBV), cytomegalovirus (CMV), and varicella-zoster virus (VZV) was determined by a polymerase chain reaction assay to detect viral DNA. Space flight resulted in increased incidence of EBV and CMV shedding and an increased number of copies of EBV DNA, and the incidence of VZV reactivation increased in astronauts during and after flight. Increased plasma levels of virus-specific antibodies substantiated reactivation of EBV, CMV, and VZV. Increases in cortisol and catecholamines were consistent with elevated stress levels. The indicative Cytokines of viral reactivation were elevated. These data indicate that space flight is a unique stress environment that may produce stress-induced changes in immunity that jeopardize the host-microbe relationship [32] [33] [34] [35] [36].

5. Models

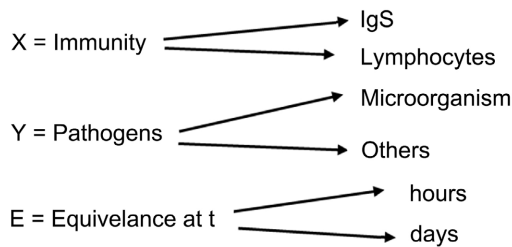
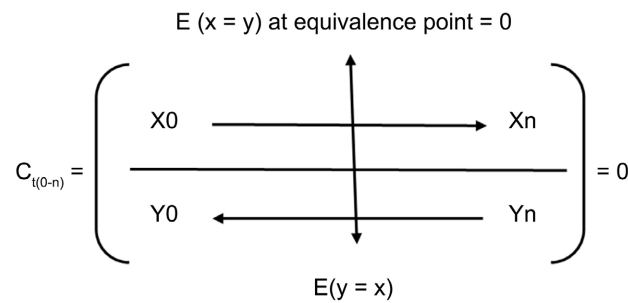
A model is just a simplified representation of a complex phenomenon. We are all familiar with the use of models in various context, by architects, economists and many branches of biomedicine, for example, the use of laboratory animals as models when carrying out research on drugs or toxic materials. The more complex a phenomenon is, or the more difficult and expensive it is to study, or the greater the ethical implications of carrying out by research, then the greater is the motive to explore models. By such logic, infectious diseases in human are obvious subjects for modelling which is complex. However, research in human populations is difficult and expensive, and such research raises many ethical issues. Thus, it is no surprise that there is a long history of modelling relevant to human infectious diseases. There are three sorts of models have been used which are animal, mechanical and mathematical models,

However, with the mathematical models, the population parameters are described by symbols and linked by algebraic formulae, and nowadays, analyzed using computers. These models are the most abstract, but they allow analysis and logical proofs in a way that other approaches do not permit. There is a long tradition of such models [37] [38] [39]. The present work emphasizes another

possible mathematical model we can concluded from manner of infectious diseases dissemination in our exceptional locality due to series of wars and laden of pollution we passing through in Iraq.

6. Mathematical Hypothesis and/or Model Representation of Immunity vis Pathogen

Single celled microorganisms were the first forms of life to develop on earth, approximately 3.5 billion years ago [40] [41]. On the other hand, it was shown that humans first evolved in Africa and much of human evolution occurred in that continent [42]. The fossils of early human who lived between 6 and 2 million years ago come entirely from Africa. However, it easy to expect that the interaction between humans and microorganisms was evolved millions years ago, and in spite of the presence of pathogenic microorganisms which attack human body since that time ,the human species is still existed . At the same time, the antimicrobials are recently discovered and manufactured not more than a century .In this case the human survival before discovery of antibiotics is entirely depended on immunity of human body and selection of nature for millions of years .This antagonistic relationship between man and pathogens which living together can be illustrated by the following hypothesis formula (Figure 3). This formula shows that the number of microorganism enters human body (Y) will be defeated by an equivalent components of human immunity (X). This equivalence or so called equilibrium will occur at a specific time of reaction (t). The sum of reactants will be constant Cat t time [Al-Jebouri, unpublished work].



C = Constant of Survival
 T = Time
 IgS = Immunoglobulins

Figure 3. Propositional formula shows the infection vs immunity of human body.

It is suppose that the number of pathogen (Y) at the moment of entry at (t_0) is 10^6 when the number of specific immunity (X) for the same pathogen is zero; soon the pathogen enters the human body it will initiate the immunity to proliferate and/or produce the equivalent specific number (n) of immunological orchestra components which starts to steadily increase to reach the equivalence point which supposes to be 10^5 (**Table 1, Figure 4**). The equivalence point will be reached after certain time (t_n). Furthermore, the sum of pathogen (Y) and immunity components (X) is constant (C) at any time of reaction (t_n) as shown in the above formula (**Figure 3**). However, the correlation coefficient (R) is equal -1 . Furthermore, the R-squared (R^2) or the coefficient determination is 1.

Table 1. Hypothesized equivalence point of pathogen enters human body *vis* immunological orchestra.

Pathogen	Immune components
1,000,000	0
900,000	100,000
800,000	200,000
700,000	300,000
600,000	400,000
500,000	500,000
400,000	600,000
300,000	700,000
200,000	800,000
100,000	900,000
0	1,000,000

Al-Jebouri (unpublished data).

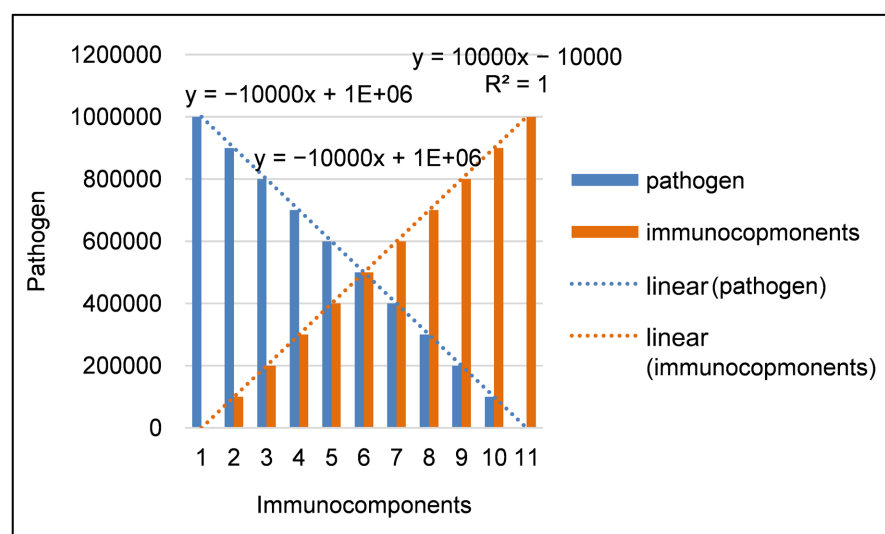


Figure 4. Point of equivalence between pathogen and immunocomponents interaction. $R = -1$.

Iraq is one of few developing countries faced a horrible long series of wars for more than three decades and still involving in this destructive types of wars. This series of wars result in elevation of so many diseases as a result of war impact. Male-factor infertility is a well-known health issue all over the world including Middle East countries, Africa and other developing countries; it presents a particularly vexing clinical problem [2]. The effect of war impact was very effective on male infertility of Iraq. A mixed pathology of semen sperms *i.e.* oligotera-toastherozoospermia was the most common feature of semen analyzed [43]. Farther investigation particularly genetical and various war impact parameters assessment is needed (Table 2, Figure 5).

Moreover, the series of wars occurred in this country might increase the risk of water contamination that leads to public health hazards particularly the infectious diseases, cancers and genetic drifts. Finally, further investigations are desperately needed to explore the various changes that might occur in this locality. At1998 Baghdad Conference, Al-Jebouri and his co-workers reported his findings after analyzing cancer data for two separate time periods in Iraq: August 1989 to March 1990 and August 1997 to March 1998. The data from this study showed almost 5-fold increase in the incidence of cancer in 1998 when compared to 1990, and this might be due to using depleted uranium bombs attacking tanks of Iraqi special forces [44] (Table 3, Figure 6).

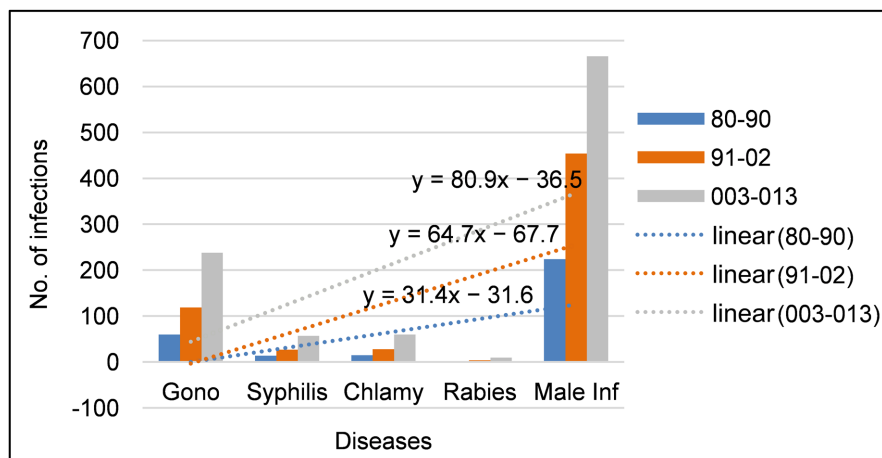


Figure 5. Linear distribution of infectious diseases among a period of study.

Table 2. Distribution of various infectious diseases among three periods of war.

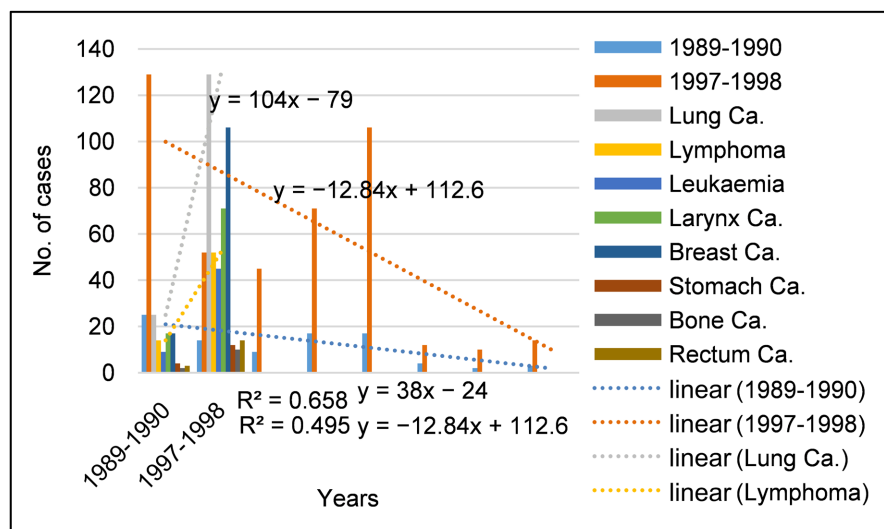
Disease	1980-1990	1991-2002	2003-2013
Gonorrhoeae	60	119	238
Syphilis	14	27	57
Chlamydia	15	28	60
Rabies	0	4	10
Male Infertility	224	454	666

From Al-Jebouri, M. M. (2013) A regional study on the infertility of Iraqi males under war impact from 1980 to 2013. World Journal of Pharmaceutical Research, 4 (5), 497-503 [43].

Table 3. Incidence of cancers among of war impact.

Cancers	1989-1990	1997-1998
Lung Ca.	25	129
Lymphoma	14	52
Leukemia	9	45
Larynx Ca.	17	71
Breast Ca.	17	106
Stomach Ca.	4	12
Bone Ca.	2	10
Rectum Ca.	3	14

From Al-Jebouri, M. M., Al-Ani, I. A., Al-Jumaily, S. A. (1998). The effect of the war of the American and the affiliated forces against Iraq on the distribution and elevation of cancer diseases in Mosul. In: Conference on Health and Environmental Consequences of Depleted Uranium Used by U.S. and British Forces in the 1991 War. Hotel Al-Rahid, Baghdad, Iraq, December 2-3, 1998 [44]. <https://www.researchgate.net/publication/259802474>.

**Figure 6.** Incidence of cancers among two periods of wars.

Fresh water bodies particularly rivers always considered as a main source of drinking and relevant human uses for maintenance of life. These natural water bodies usually under stress of environmental pollution include chemical, physical and biological aspects that might turn the nature of these waters to be risky for human beings uses. Environmental stress as well as human population growth associated with an increased industrial and civilian activities can all cause a damage for the natural water quality that might be necessary for human needs which lead to continuous environmental survey and assessment to reveal the degree of pollution impact particularly on raw water sources like rivers, lakes, springs and deep wells waters *i.e.* ground waters. Raw water quality can be assessed using various parameters particularly the bacterial indicators of biolog-

ical pollution. Chemical and physical factors affecting water quality have been utilized for evaluation of degree of pollution and these included oxygen biological demand, turbidity, electrical conductivity, temperature, and pH of waters under investigations [45].

7. Conclusion

It was quite clear from the proposed modelling that microbial pathogenicity was a complex process including pathogen, immunity, environment and antimicrobial agents. There were secondary factors related and interacting with pathogenicity primary factors such as pollution, disturbance of environmental balance, stress of both man and pathogens and any defect in immunity or inactivation of antibiotics. The present new mathematical formula revealed that the number of pathogens reacted with the number of immune components is always constant reaching the equivalence point when the number of both reactants is equal at t time of exposure.

Conflicts of Interest

The author declares no conflicts of interest regarding the publication of this paper.

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