

Research Article

Mathematical Considerations for the Infectious Infertility of Male in Iraq

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Abstract

Aims: Male infertility is a multifactorial condition influenced by anatomical, hormonal, genetic and infectious causes. While advancements in diagnostics and treatments have improved outcomes, infertility remains a challenge, particularly in regions where access to specialized care is limited. Understanding both the success rates of various treatments and the etiological role of pathogens is essential for developing effective strategies. **Methods and Results:** This retrospective analysis examines the prevalence of urogenital pathogens isolated from male patients diagnosed with infertility across three decades: 1980-1990, 1991-2002, and 2003-2012. Bacterial and atypical pathogens were identified using standard microbiological and molecular techniques available during each respective period. Mathematical modeling, particularly through regression analysis, is a powerful tool for uncovering relationships between variables in clinical research. Patterns and quantify of different factors influence outcomes were identified, such as treatment effectiveness or disease prevalence. Regression equation was created for better predictive model that not only describes the current dataset but can also be used to estimate outcomes under different conditions. A total of 3,600 patients were e treated across various infertility types, yielding an overall cure rate of 11.5%. Azoospermia and Oligospermia showed the highest recovery rates, while Oligoteratoasthenozoospermia had the lowest. Pathogen prevalence data from 1980 to 2012 was analyzed to understand shifts in microbial contributors to infertility. The presented data revealed a decline in classic sexually transmitted infections like *Neisseria gonorrhoeae* and *Treponema pallidum*, with increasing presence of opportunistic pathogens such as *Escherichia coli* and *Streptococcus faecalis*. Azoospermia showed the highest treatment success rate, while Oligoteratoasthenozoospermia showed the lowest. The regression model captured the general trend of patient cure rates. **Conclusion, significance and impact of study:** The present study highlights evolving trends in pathogen prevalence among infertile male patients over 32 years. While classic sexual transmitted infectants like *Neisseria gonorrhoeae* have declined and opportunistic and uropathogenic bacteria like *E. coli* and *S. faecalis* have become more prominent. Outliers showed larger deviations suggesting a possible non-linearity in the real relationship using linear regression equation $Y = a + bX + \varepsilon_i$.

Keywords

Bacteria, Infections, Infertility, Male Infection, Treatment, Semen Analysis, Mathematical Modelling

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1. Introduction

One of the most serious social problems facing developed countries today is the declining birth rate, although it is generally not well recognized that the number of infertile couples is on the rise in these countries. While both social (i.e., social progress for women and the resulting increase in the age at which women marry) and environmental (i.e. pollution and global warming) factors are behind part of the increase in the number of patients with infertility, infertility in the male partner contributes to approximately half of all cases [1-3]. 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 civil wars. This series of wars resulting 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 [1]. It has been estimated that infertility of couples affects 10-15% of the general population. The prevalent rate varies between and within countries. For instance, in the United Kingdom and the United States of America it is estimated to be 6% and 10% respectively [4]. In Denmark, it is estimated to be in the region of 15.7% [5]. In Nigeria and some parts of sub-Saharan Africa including the Republic of Sudan and Cameroon, infertility rate could exceed 30% [2, 3, 6]. Some studies reported in South-eastern Nigeria, have demonstrated a 65% and 35% prevalent rate for primary and secondary infertility respectively [7]. Similarly, some countries, most notably Kenya, Gabon, Botswana, Zimbabwe and many other African countries, have shown a trend toward lower fertility [8-10]. However, we have not come across a national study in Iraq concerning infertility of couples, so the present study is an attempt covering part of Iraq to assess the changing picture of male infertility in a peculiar condition which is the series of wars. Male infertility is a multifactorial condition influenced by anatomical [11], hormonal, genetic and infectious causes [12]. While advancements in diagnostics and treatments have improved outcomes, infertility remains a challenge, particularly in regions where access to specialized care is limited. Understanding both the success rates of various treatments and the etiological role of pathogens is essential for developing effective strategies [13].

This paper presents a dual analysis: one part evaluates the efficacy of treatments among 3,600 infertile male patients across six categories of infertility [14] and the other explores the temporal distribution of urogenital pathogens [15] from 1980 to 2012. Mathematical modeling, particularly through regression analysis, is a powerful tool for uncovering relationships between variables in clinical research [16-19]. Developing a regression equation allows us to create a predictive model that not only describes the current dataset but can also be used to estimate outcomes under different conditions which is the task of the present study.

2. Materials and Methods

2.1. Patients

3600 male patients attended clinics complaining from infertility were studied. These patients were from different localities, Baghdad, Mosul, Tikrit, Al-Hawija and Al-Zab i.e from middle to North of Iraq. The seminal fluid analysis was carried out following WHO Laboratory Manual [20].

2.2. Isolation and Identification of Bacteria

All bacterial isolates were identified following Cowan and Steel's Manual for Identification of Medical Bacteria [21].

2.3. Mathematical Models

Mathematical modeling, particularly through regression analysis, is a powerful tool for uncovering relationships between variables in clinical research. Analyzing data collected would identify patterns and quantify and how different factors influence outcomes in relation to treatment effectiveness or disease prevalence. Developing a regression equation allows us to create a predictive model that not only describes the current dataset but can also be used to estimate outcomes under different conditions. The present study focuses on calculating and investigating a regression equation regarding the data collected.

2.3.1. A Simple Linear Regression Model

To construct a regression model from data collected, numerically encode time was needed which fit a model that describes the trend in pathogen prevalence over the three time periods.

Step 1: Time Encoding

The time periods were represented as numerical values (Table 1) as follow:

1. 1980 – 1990 → $x = 1$
2. 1991 – 2002 → $x = 2$
3. 2003 – 2012 → $x = 3$

Step 2: extracting pathogen data

Let's take one example: *Escherichia coli*

1. Time points (x): [1, 2, 3]
2. Prevalence (y): [22, 29.6, 24.7]

It can be now build a simple linear regression model:

$$y = a + bx$$

Formula for b (slope):

$$b = \frac{n \sum xy - \sum x \sum y}{n \sum x^2 - (\sum x)^2}$$

Given:

1. $x = [1, 2, 3]$

$$2. y = [22, 29.6, 24.7]$$

$$3. n = 3$$

Step-by-step:

$$1. \sum x = 1 + 2 + 3 = 6$$

$$2. \sum y = 22 + 29.6 + 24.7 = 76.3$$

$$3. \sum xy = (1 \times 22) + (2 \times 29.6) + (3 \times 24.7) = 22 + 59.2 + 74.1 = 155.3$$

$$4. \sum x^2 = 1^2 + 2^2 + 3^2 = 1 + 4 + 9 = 14$$

Now plug into the formula:

$$b = \frac{3(155.3) - 6(76.3)}{3(14) - 36} = \frac{8.1}{6} = 1.35$$

So, $b = 1.35$ — indicating that the prevalence increases by about 1.35% per time period.

Formula for a (intercept):

$$a = \frac{\sum y - b \sum x}{n}$$

Given:

$$\sum y = 76.3, \sum x = 6, b = 1.35, n = 3$$

Calculate a:

$$a = \frac{76.3 - (1.35 \times 6)}{3} = \frac{76.3 - 8.1}{3} = \frac{68.2}{3} = 22.73$$

So, the intercept $a = 22.73$

Final Regression Equation:

$$\text{Prevalence (\%)} = 22.73 + 1.35 \times \text{Time Period (Figure 1)}$$

2.3.2. Approximate Linear Regression Model

To model the relationship between X (e.g., number of patients) and Y (percentage of patients cured), we construct a simple linear regression equation:

$$Y = a + bX + \varepsilon_i$$

Using two points to estimate:

First, From the following two points:

$$1. (410, 27.3)$$

$$2. (1500, 4.8)$$

We calculate slope b_1 :

$$b_1 = \frac{27.3 - 4.8}{410 - 1500} = \frac{22.5}{-1090} \approx -0.02064$$

Then solve for a_1 using (410, 27.3):

$$27.3 = a_1 - 0.02064 \cdot 410 \Rightarrow$$

$$a_1 \approx 27.3 + 8.4704 = 35.7704$$

Second, From:

$$1. (80, 22.5)$$

$$2. (1500, 4.8)$$

We calculate slope b_2 :

$$b_2 = \frac{22.5 - 4.8}{80 - 1500} = \frac{17.7}{-1420} \approx -0.01246$$

Then solve for a_2 using (80, 22.5):

$$22.5 = a_2 - 0.01246 \cdot 80 \Rightarrow a_2 \approx 22.5 + 0.01246 \cdot 80 = 22.5 + 0.9968 = 23.4968$$

Third, From:

$$1. (630, 19.8)$$

$$2. (1500, 4.8)$$

We calculate slope b_3 :

$$b_3 = \frac{19.8 - 4.8}{630 - 1500} = \frac{15.0}{-870} \approx -0.01724$$

Then solve for a_3 using (630, 19.8):

$$19.8 = a_3 - 0.01724 \cdot 630 \Rightarrow a_3 \approx 19.8 + 0.01724 \cdot 630 = 19.8 + 10.8612 = 30.6612$$

Fourth, From:

$$1. (360, 15.6)$$

$$2. (1500, 4.8)$$

We calculate slope b_4 :

$$b_4 = \frac{15.6 - 4.8}{360 - 1500} = \frac{10.8}{-1140} \approx -0.00947$$

Then solve for a_4 using (360, 15.6):

$$15.6 = a_4 - 0.00947 \cdot 360 \Rightarrow$$

$$a_4 \approx 15.6 + 0.00947 \cdot 360 = 15.6 + 3.4092 = 19.0092$$

Fifth, From the points (430, 7.4) and (1500, 4.8), we calculate slope b_5 :

$$b_5 = \frac{7.4 - 4.8}{430 - 1500} = \frac{2.6}{-1070} \approx -0.00243$$

Then solve for a_5 using (430, 7.4):

$$7.4 = a_5 - 0.00243 \cdot 430 \Rightarrow$$

$$a_5 \approx 7.4 + 0.00243 \cdot 430 = 7.4 + 1.0449 = 8.4449$$

$$a = \frac{\sum_{i=0}^5 a_i}{5} = \frac{35.7704 + 23.4968 + 27.6498 + 19.0092 + 8.4449}{5} = \frac{114.3611}{5} = 22.8722$$

$$b = \frac{\sum_{i=0}^5 b_i}{5} = \frac{-0.02064 - 0.01246 - 0.01724 - 0.00947 - 0.00243}{5} = \frac{-0.06224}{5} = -0.012448$$

Final regression Model:

$$Y = a + bX + \varepsilon_i$$

This equation serves as an approximate rule for estimating the cure percentage Y based on the number of patients X , with ε_i representing the prediction error.

Where

$$Y = \text{Patients Cured (\%)}$$

$$X = \text{Patients treated}$$

$$a = 22.87422.$$

$$b = -0.012448$$

$$Y = 22.87422 - 0.012448 \times X$$

The regression equation describes the relationship between the number of patients treated and the percentage of patients cured. Components of the Equation:

1. Dependent Variable (Y):

Patients Cured (%) - this is the predicted percentage of patients who are cured.

2. Independent Variable (X):

Patients Treated - the number of patients who received treatment.

3. Intercept ($a = 22.87422$):

This is the estimated cure rate (%) when the number of patients treated is zero. It represents the theoretical starting point of the model.

4. Slope ($b = -0.012448$):

This tells us that for each additional patient treated, the cure rate decreases by about 0.0124%. The negative sign indicates an inverse relationship between the number of patients treated and the cure rate percentage.

3. Results

A total of 310 pathogen isolates were identified across the three periods, and *Escherichia coli* was consistently among the most prevalent pathogens (Table 1, Figure 1), peaking in 1991-2002 (29.6%) and remaining high in 2003-2012 (24.7%). *Neisseria gonorrhoeae* demonstrated a significant decline, from 27.8% in 1980-1990 to only 4.1% by 2003-2012. *Treponema pallidum* cases were rare and decreased further over time but *Chlamydia trachomatis* and *Ureaplasma urealyticum* initially rare or absent, showed a small increase in 2003-2012.

Table 1. Prevalence of pathogens in male infertile patients.

Pathogens	No. (%)		
	1980-1990	1991-2002	-2012
<i>Escherichia coli</i>	20(22)	21(29.6)	43(24.7)
<i>Streptococcus Faecalis</i>	10(11)	12(16.9)	43(24.7)
<i>Staphylococcus aureus</i>	18(20)	12(16.9)	32(22.1)
<i>Proteus vulgaris</i>	13(14.3)	16(22.5)	20(13.8)
<i>Neisseria gonorrhoeae</i>	25(27.8)	8(11.3)	6(4.1)
<i>Treponema pallidum</i>	3(3.3)	2(2.8)	1(0.7)
<i>Chlamydia trachomatis</i>	1(1.1)	0	3(2.1)
<i>Ureaplasma urealyticum</i>	1(1.1)	0	4(2.8)
Total	91	71	152

The clinical and microbiological findings indicate a measurable association between the prevalence of certain pathogens—such as *Escherichia coli*, *Streptococcus faecalis*, *Chlamydia trachomatis*, and *Ureaplasma* species—and the

incidence of male infertility over the span of several decades (1980-2012). To statistically investigate this connection, it was employed a multiple linear regression analysis as demonstrated below (Figures 1, 2).

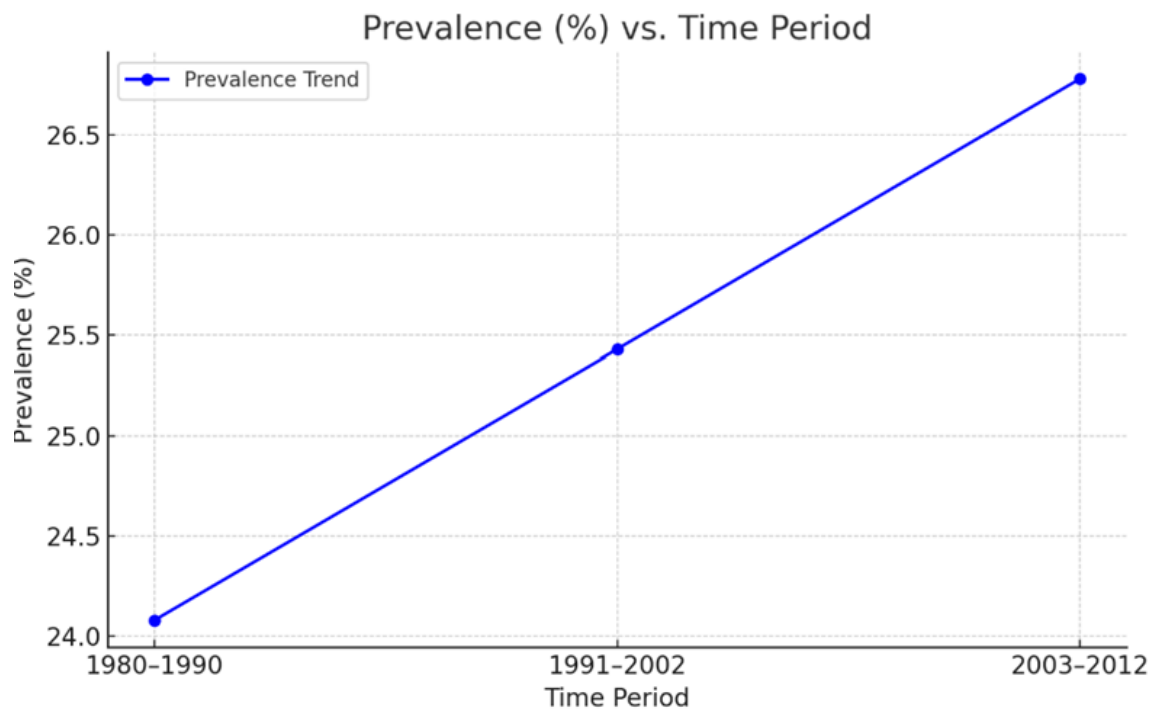


Figure 1. Prevalence (%) vs. Time Period.

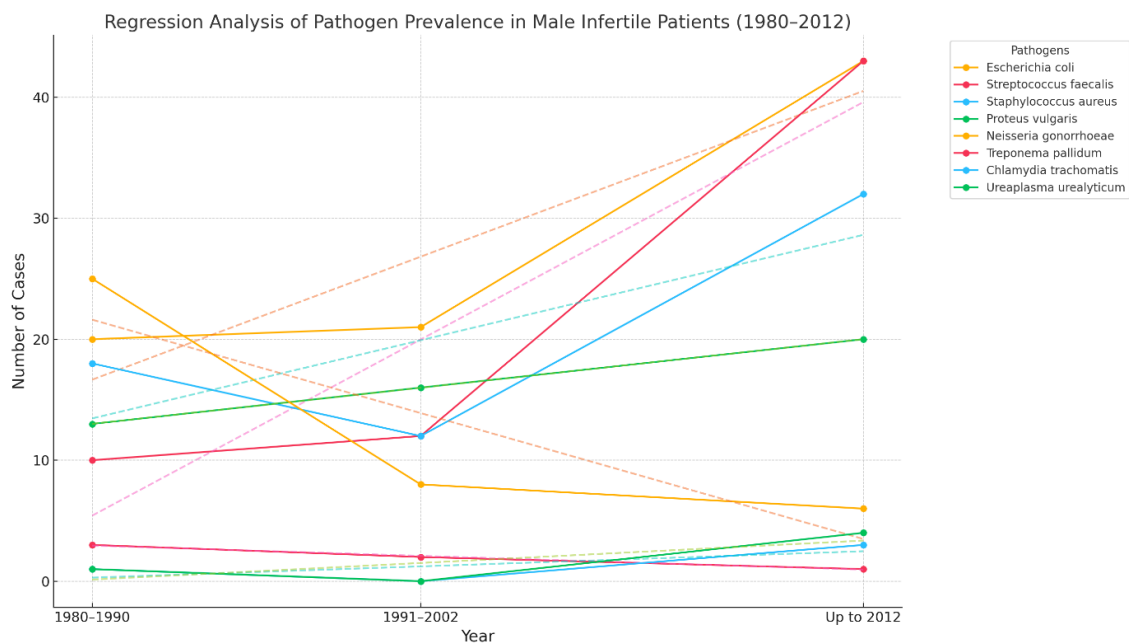


Figure 2. Regression analysis of pathogen incidence causing male infertility (1980-2012).

Regression Equation:

$$Prevalence\ (\%) = 22.73 + 1.35 \times Time\ Period$$

4. Analysis of Treatment Outcomes for Male Infertility Types

Understanding the Error Column (ϵ_i):

The error is defined as the absolute difference between the observed and predicted patient cure percentages. It reflects how well the model fits each data point (Table 2; Figures 3, 4).

The error (ϵ_i) from The Table 2, is calculated as:

$$\epsilon_i = |Observed\ Cure\ \% - Predicted\ Cure\ \%|$$

It represents how far off the regression model's prediction is

from the actual observed data for each case. Figure 2 shows an inflection point in the infectious agents dissemination among

Iraqi peoples which occurred at 1991 to 2002 period.

Table 2. Prevalence of Patients treated.

Infertility Type	Patients treated	Patients cured (%)	Patients cured (%) from regression equation	$\varepsilon_i(\text{error})$
Azoospermia	410	112 (27.3%)	17.7%	9.6%
Oligospermia	80	18 (22.5%)	21.9%	0.6%
Oligozoospermia	630	125 (19.8%)	15.03%	4.77%
Astherozoospermia	360	56 (15.6%)	18.4%	2.8%
8	430	32 (7.4%)	17.5%	10.1%
Oligoteratoasthero-zoospermia	1500	72 (4.8%)	4.2%	0.6%
Total	3600	415 (11.5%)	15.7%	4.2%

Observations About the Errors:

Largest Error:

Case: 112 patients (27.3%) vs predicted 17.7% → Error: 9.6%

Interpretation: The model significantly underestimates the cure rate here. This may indicate an outlier or unaccounted factor for higher cure success.

Smallest Errors:

Case: 18 patients (22.5%) vs 21.9% → Error: 0.6%

Case: 72 patients (4.8%) vs 4.2% → Error: 0.6%

Interpretation: These predictions are very close to the actual values, showing good local fit.

Moderate Errors:

Most other cases (e.g., 125, 56, 415 patients) show errors between 2.8% and 4.77%, which is acceptable in many epidemiological or clinical modeling contexts.

Regression model:

$$Y = 22.87422 - 0.012448 \times X + \varepsilon_i$$

X-axis: Number of patients; Y-axis: Predicted cure rate (%).

The regression equation describes the relationship between the number of patients treated and the percentage of patients cured. The downward slope shows that as the number of patients increases, the predicted percentage of patients cured decreases. An threshold was seen among the prevalence of of different forms of infertility (Figure 4).

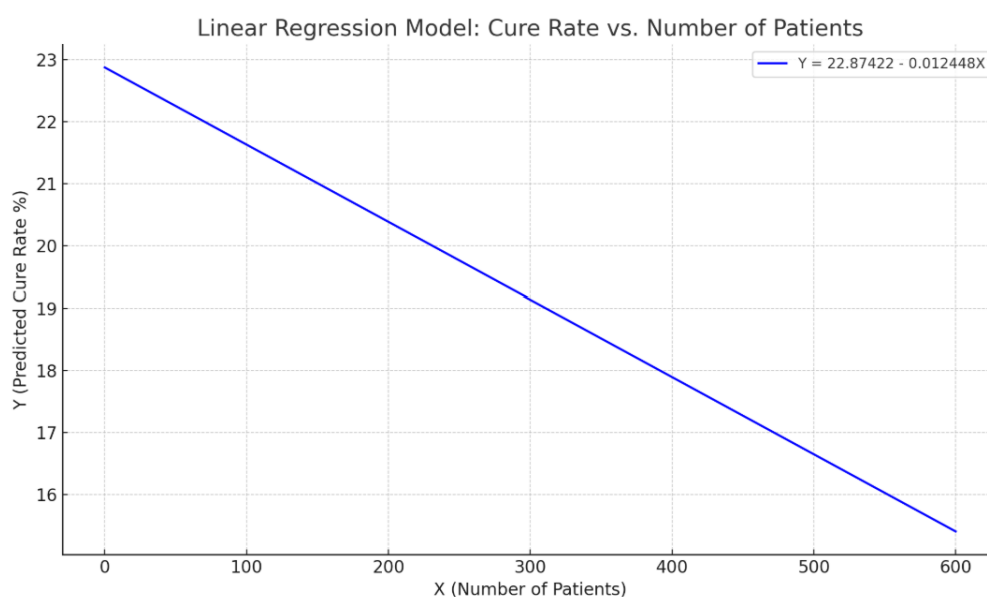


Figure 3. The plot of the regression model.

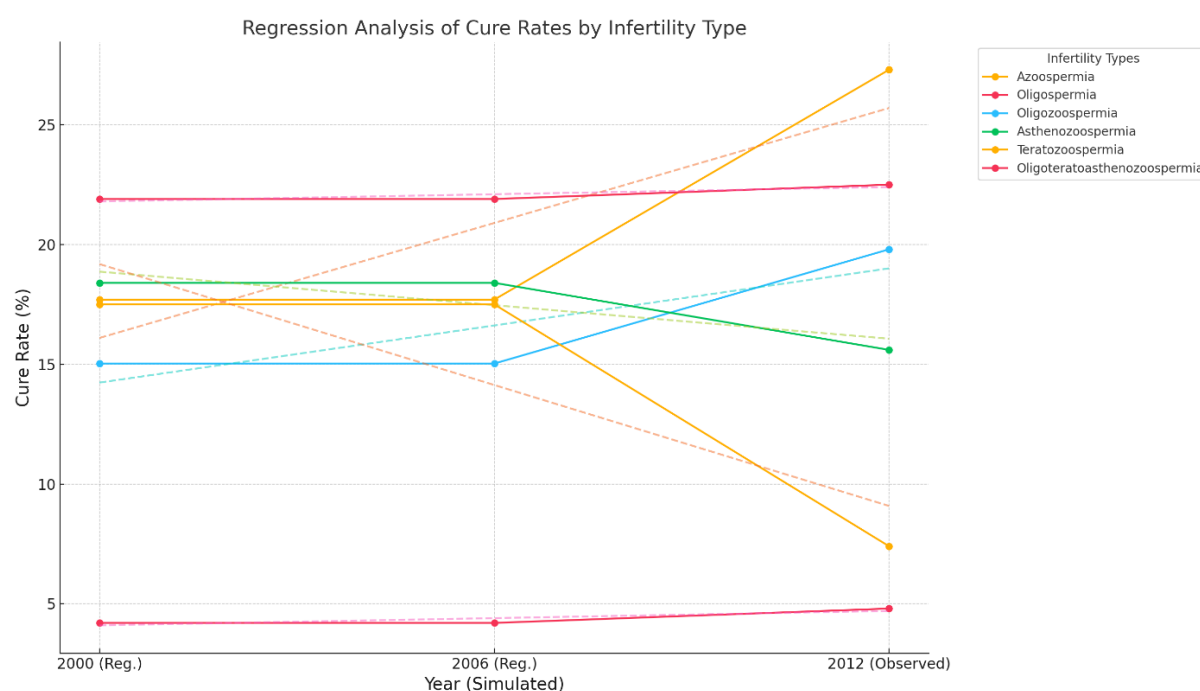


Figure 4. Shows the regression analysis concluded among three periods of study for Iraq males.

5. Discussion

The present study showed that the incidence of male infertility was elevated as the war and its impact went on in the country since 1980 to 2013. The impact of war and its parameters highly influenced the fertility of males. Male infertility has many causes from hormonal imbalances, to physical problems, to psychological and/or behavioral problems. Moreover, fertility reflects a man's —overall health. Men who live a healthy lifestyle are more likely to produce healthy sperm. The following list highlights some lifestyle choices that negatively impact male fertility, it is not all-inclusive: Smoking significantly decreases both sperm count and sperm cell motility, prolonged use of marijuana and other recreational drugs, chronic alcohol abuse, anabolic steroid use causes testicular shrinkage and infertility, overly intense exercise produces high levels of adrenal steroid hormones which cause a testosterone deficiency resulting in infertility, inadequate vitamin C and zinc in the diet, tight underwear increases scrotal temperature which results in decreased sperm production, Exposure to environmental hazards and toxins such as pesticides, lead, paint, radiation, radioactive, substances, mercury, benzene, boron, and heavy metals, malnutrition and anemia, excessive stress and modifying these behaviors can improve a man's fertility and should be considered when a couple is trying to achieve pregnancy [1, 22]. However, it was concluded that 90% of the attended clinic suspected primary infertility were young males of under 30 years old. Most of the infertile males were suffered from oligoteratoasthenozoospermia. This mixed pathology type of semen abnormality

was recorded by Abarikwa [23]. Furthermore, the present study revealed that *Escherichia coli* was the most prevalent pathogen isolated from infectious semens tested. This finding was also recorded by Okonofua et al. [24]. The infectious semens found in the present study were confirmed by the association of high number of leucocytes which elevated sometimes to reach 107. Genetic polymorphisms may also increase susceptibility to some forms of male infertility. Workers have identified polymorphisms of several genes that are associated with the human azoospermic population—*MEII*, *PRDM9* (*MEISETZ*), *SPATA17*, *PARP-2*, and *UBR2* genes are genetic risk factors for the patients with azoospermia by meiotic arrest [25-30] and polymorphisms of the *SEPTIN12* gene are associated with patients with Sertoli cell-only syndrome [30]. Genetic polymorphisms and male infertility have been under much investigation recently.

The data reveal a clear shift in the microbial landscape of male infertility over three decades. The decline in *Neisseria gonorrhoeae* and *Treponema pallidum* reflects improvements in public health, STI awareness, and antibiotic therapy. In contrast, the increased detection of *Streptococcus faecalis* and *Escherichia coli* [9, 10] may be attributed to changes in hygiene, diagnostic accuracy, or antibiotic resistance patterns.

Emerging detection of *Chlamydia trachomatis* [11] and *Ureaplasma* unrealistic [31, 22] in the 2003-2012 period may be due to advancements in molecular diagnostics and recognition of their role in subclinical infections. These pathogens, while less common, may have disproportionate impacts on sperm quality and fertility outcomes.

This study highlights evolving trends in pathogen prevalence among infertile male patients over 32 years. While

classic STIs like *Neisseria gonorrhoeae* have declined, opportunistic and uropathogenic bacteria like *E. coli* and *S. faecalis* have become more prominent. These findings underscore the importance of routine microbiological screening in male infertility workups and adapting treatment strategies to current microbial profiles. Ongoing surveillance and incorporation of molecular diagnostics are recommended for early and accurate detection of emerging pathogens [6, 32-34].

The data illustrates notable disparities in treatment outcomes across different types of male infertility. Higher success rates in Azoospermia and Oligospermia suggest these conditions may be more amenable to existing therapeutic interventions. In contrast, the complexity of Oligoteratoasthenozoospermia, which involves a combination of low count, poor motility, and abnormal morphology, is reflected in its poor treatment outcome.

The predominance of Oligoteratoasthenozoospermia cases (41.7% of the total cohort) also emphasizes the clinical significance of this multifactorial condition. The findings highlight a need for more personalized and comprehensive treatment approaches for patients with complex infertility profiles [31, 35, 36].

These findings highlight the importance of tailoring treatment protocols to specific infertility types and refining predictive models to improve clinical decision-making [37].

Furthermore, the overall fertility restoration rate among treated male patients was 11.5%. Cure rates varied significantly by infertility type, with Azoospermia and Oligospermia having the most favorable outcomes. Conversely, patients with Oligoteratoasthenozoospermia demonstrated the greatest resistance to current treatment modalities. These results underscore the importance of advancing targeted therapies, especially for multifactorial infertility cases [38, 39]. Future studies should explore novel interventions and long-term follow-up strategies to enhance treatment efficacy.

6. Conclusions

Mathematical modeling, particularly through regression analysis, is a powerful tool for uncovering relationships between variables in clinical research. By analyzing data collected within a table, we can identify patterns and quantify how different factors influence outcomes, such as treatment effectiveness or disease prevalence. Developing a regression equation allows us to create a predictive model that not only describes the current dataset but can also be used to estimate outcomes under different conditions. In this study, we focus on calculating and investigating a regression equation based on the data presented in the table, aiming to better understand the underlying trends and support evidence-based decision-making. The regression model appears to capture the general trend of patient cure rates fairly well. A few outliers (especially the first and fifth cases) show larger deviations, suggesting a possible non-linearity in the real relationship.

Abbreviations

STIs Sexual transmitted infections

Author Contributions

Mohemid Maddallah Al-Jebouri: Conceptualization, Supervision, Writing - review & editing

Mohammed Nokhas Murad Kaki: Data curation, Formal analysis, Writing - original draft

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Conflicts of Interest

The authors declare that they have no conflict of interests.

References

- [1] Miyamoto, T., Tsujimura, A., Miyagawa, Y., Koh, E., Namiki, M. & Sengoku, K. (2011). Male Infertility and Its Causes in Human. *Advances in Urology* 2012, 1-7.
- [2] 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 (3): 126-139.
- [3] Al-Jebouri, M. M. (2024). Impact of sublethal disinfectant exposure on antibiotic resistance patterns of *Pseudomonas aeruginosa*. *Medical Principles and Practice* 13: 1-9. <https://doi.org/10.1159/000542322>
- [4] Ugwuja, E., Ugwu, N. C., & Ejikeme, B. N. (2008). Prevalence of Low Sperm Count and Abnormal Semen Parameters in Male Partners of Women Consulting at Infertility Clinic in Abakaliki, Nigeria. *African Reproductive Health* 12, 67-73.
- [5] Schmidt, L., Minister, K., & Helm, P. (1995) Infertility and the seeking of infertility treatment in a representative population. *Britain Journal Obstetrics and Gynaecology* 102, 978-984.
- [6] Apari, P., de Sousa, J. D., & Müller, V. (2014). Why sexually transmitted infections tend to cause infertility: An evolutionary hypothesis. *PLoS Pathogens* 10: e1004111.
- [7] Ikechebelu, J. I., Adinma, J. I., Orie, E. F., & Ikegwonu, S. O. (2003). High prevalence of male infertility in South-eastern Nigeria. *Journal of Obstetrics and Gynaecology* 23: 657-659.
- [8] Larsen, U. (2000). Primary and secondary infertility in sub-Saharan Africa. *International Journal of Epidemiology* 29, 285-291.
- [9] Okonofua, F. E. (2003). Infertility in Sub-Saharan Africa. In: Okonofua F, and L Odunsi, eds. *Contemporary Obstetrics and Gynaecology for Developing Countries*. Women's Health and Action Research Centre, 128-156.

- [10] Agyei-Mensah, S. (1996). New perspective on the fertility situation in Sub-Saharan Africa *NorskGeografiskTidsskrift - Norwegian Journal of Geography* 50: 101 - 112.
- [11] Tahmasbpour, E., Balasubramanian, D., & Agarwal, A. (2014). A multi-faceted approach to understanding male infertility: gene mutations, molecular defects and assisted reproductive techniques (ART). *Journal of Assisted Reproduction and Genetics* 31(9), 1115-1137.
- [12] Stormont, G. D., & Deibert, C. M. (2021). Genetic causes and management of male infertility. *Translational Andrology and Urology* 10(3), 1365-1372.
- [13] Salonia, A., Bettocchi, C., Boeri, L., Capogrosso, P., Carvalho, J., Cilesiz, N. C., & Fijak, M. (2023). Infections and inflammation as etiological factors in male infertility. *Human Reproduction Update* 24(5), 416-440.
- [14] Guiton, R., & Drevet, J. R. (2023). Viruses, bacteria and parasites: infection of the male genital tract and fertility. *Basic and Clinical Andrology*, 33, Article 19. Springer.
- [15] Henkel, R. (2021). The role of infections and leukocytes in male infertility. *Andrology* 9(4), 101-116.
- [16] Vynnycky, E. & White, R. (2010). *An Introduction to Infectious Disease Modelling*. Oxford University Press, Oxford. 282-315.
- [17] Garnett, G. P., Swinton, J., Brunham, R. C. & Anderson, R. M. (1992). Gonococcal infection, infertility, and population growth: II. The influence of heterogeneity in sexual behaviour. *IMA Journal of Mathematics and Applied Medical Biology* 9(2), 127-144.
- [18] White, R. G., Orroth, K. K., Glynn, J. R., Freeman, E. E., Bakker, R., Habbema, J. D. F., et al. (2008). Treating curable sexually transmitted infections to prevent HIV in Africa: still an effective control strategy? *Journal of Acquired Immune Deficiency Syndrome* 47, 940-946.
- [19] Hethcote, H. W. (1976). Qualitative analysis for communicable disease models. *Mathematical Biosciences* 28: 335-356.
- [20] WHO Laboratory Manual for the Examination of Human Semen and Sperm-Cervical Mucus Interaction. 4th edition, Geneva, World Health Organization., 1999.
- [21] Cowan and Steel's Manual for Identification of Medical Bacteria. Edited by G. I. Barrow, R. K. A. Feltham, Cambridge University Press, 2009.
- [22] Samli H, Samli MM, Solak M., & Imirzalioglu, N. (2006). Genetic anomalies detected in patients with non-obstructive azoospermia and oligozoospermia. *Archives of Andrology* 52.263-267.
- [23] Abarikwu, S. O. (2013). Causes and Risk Factors for Male-Factor Infertility in Nigeria: A Review of *African Journal of Reproductive Health* 17(4), 150-166.
- [24] Okonofua, E. F., Harris, D., Odebiyi, A., Kane, T. & Snow, R. (1997). The social meaning of infertility in Southwest Nigeria. *Health Transformation Review* 7, 205-220.
- [25] Nwabuisi, C. & Onile, B. A. (2001). Male infertility among sexually transmitted diseases clinic attendees in Ilorin, Nigeria. *Nigerian Journal of Medicine* 10: 68-71.
- [26] Sule-Odu, A. O., Oladapo, O. T., Jagun, O. E., & Awosile, J. (2005). Microbial isolates and HIV infection in couples attending fertility clinics in Sagamu, *Nigerian Journal of Obstetrics and Gynaecology* 25, 685-688.
- [27] Esimai, O. A., Orji, E. O., & Lasisi, A. R. (2002). Male contribution to infertility in Ile-Ife, Nigeria. *Nigerian Journal of Medicine* 11, 70-72.
- [28] Obiechina, N. J., Okoye, R. N., & Emelife, E. C. (2002). Seminal fluid indices of men attending infertility clinic at St. Charles Borromeo Hospital, Onitsha, Nigeria (1994-1998). *Nigerian Journal of Medicine* 11, 20-22.
- [29] Ekwere, P. D. (2004). Profile of sub-fertile Nigerian men in an infertility Clinic in Calabar, Nigeria. *Nigerian Journal of Surgery* 10, 37-42.
- [30] Agbakoba NR, Adetosoye AI, & Ikechebelu JI. (2007). Genital mycoplasmas in semen samples of males attending a tertiary care hospital in Nigeria: any role in sperm count reduction? *Nigerian Journal of Clinical Practice* 10: 169-173.
- [31] Huang, J., Tian, Y., Xu, X., & Wang, Y. (2015). Mycoplasma and Ureaplasma infection and male infertility: A systematic review and meta-analysis. *Andrology* 3(6), 1121-1130.
- [32] Maissey, K., Nardocci, G., Imarai, M., Cardenas, H., Rios, M., Croxatto, H. B., et al. (2003). Expression of S150-161. Proinflammatory cytokines and receptors by human fallopian tubes in organ culture following challenge with *Neisseria gonorrhoeae*. *Infection and Immunity* 71: 527-32.
- [33] Malhotra, M., Sood, S., Mukherjee, A., Muralidhar, S., & Bala, M. (2013). Genital *Chlamydia trachomatis*: An update. *Indian Journal of Medical Research* 138: 303-16.
- [34] Turner, K. M., Garnett, G. P., Ghani, A. C., Sterne, J. A., & Low N. (2004). Investigating ethnic inequalities in the incidence of sexually transmitted infections: mathematical modelling study. *Sex Transmission Infection* 80(5): 379-385.
- [35] Zeighami, H., Peerayeh, S. N., Yazdi, R. S., & Sorouri, R. (2009). Prevalence of Ureaplasma urealyticum and Ureaplasma parvum in semen of infertile and healthy men. *International Journal of Sexual Transmitted Diseases & AIDS*, 20(6), 387-390.
- [36] Pai, M. O., Venkatesh, S., & Gupta, P. (2020). The role of infections in infertility: A review. *International Journal of Academic Medicine* 6(3), 189-196.
- [37] Ruggeri, M., Cannas, S., Cubeddu, M., Molicotti, P., Piras, G. L., Dessole, S., et al. (2016). Bacterial agents as a cause of infertility in humans. *New Microbiology* 39: 206-209.
- [38] Mascarenhas, M. N., Flaxman, S. R., Boerma, T., Vanderpoel, S., & Stevens, G. A. (2012). National, regional, and global trends in infertility prevalence since 1990: A systematic analysis of 277 health surveys. *PLoS of Medicine* 9, e1001356.

- [39] Kamath, M. S., & Deepti MK. (2016). Unexplained infertility: An approach to diagnosis and management. *Current Medical Issues* 14, 94-100.