THE INTERACTIVE STAGES OF DISEASE TRANSITIONAND THEIRNEUTROSOPHICPROJECTION MATRIX WITH GENERATING FUNCTION$A_n = \frac{P_n-1}{2} P_n = 1,3,5,7$

by

A. Zubairu, A.A Ibrahim, A. Mustafa

Shehu Shagari College of Education, Sokoto, Nigeria. Usmanu Dan Fodiyo University, Sokoto, Nigeria. Email: Zubairuabdul44@yahoo.com

Abstract

The classical SIR disease model was extended to include the exposed stage where the host population is composed of susceptible (S), Exposed (E) Infected $(\check{I})$ and recovered individuals (R). Such that the total population size at any given time $N(t)$ will be
$N(t) = S(t) + E(t) + \check{I}(t) + R(t)$*and the generating function is given by*
$A_n = \frac{P_n-1}{2} P_n = 1,3,5,7$. *So we attached an indeterminate factor "I" to A_n and obtained $N(G) = A_n \cup I$ a neutrosophic group. Here also we show the interaction between two successive stages and their corresponding effects on the cyclic nature of disease transition. Finally the results were portrayed on the neutrosophic projection matrix so that important epidemiological quantities such as (R_0) and (λ) could easily be estimated.*

Keywords: *Generating function, Neutrosophic algebra, cyclic transition, projection matrix.*

1. Introduction

Neutrosophic theory is now being studied and applied to many fields in order to solve real life situations are vague, imprecise and contradictory therefore, it is absolutely necessary to investigate this spectrum of neutrality in other to obtained a realistic result. A lot of study on infectious disease reveals that the transition moved in stages such as S,I,R *or* S,E,I,R *or* S,I,S *or* S,I,R,I among others and the total population size at any given time $N(t)$ is given by:

$$N(t) = S(t) + E(t)\check{I}(t) + R(t) \quad (1)$$

We introduced a cyclic disease transmission with permutation patterns generated from

$$A_n = \frac{P_n - 1}{2}, \qquad P_n = 1,3,5,7 \quad (2)$$

When attached an indeterminate factor "I"to A_n, then

$$N(G) = A_n \cup I \quad (3)$$

is a neutrosophic group [2]. Therefore the total population size at any given time (t) with an indeterminate factor "I" attached to the transmission states is a function of$N(G)$, hence

$$N(G) = \{S(t) \oplus I\} + \{E(t) \oplus I\} + \{\check{I}(t) \oplus I\} + \{R(t) \oplus I\} \quad (4)$$

Taken the transmission rates from the subclass of the neutrosophic group and varying the permutation scheme to obtained cyclic transmission graph from which a neutrosophic projection matrix is derived. In this paper, we extend the S,I,R type model to include the exposed state that is, the period the disease is starting to showup. So once the transmission is portrayed into neutrosophic matrix epidemiological concepts such as threshold value, basic reproduction ratio(R_o), population growth rate(λ)associated with disease transmission could easily be estimated.

2. Literature review

Neutrosophy is a branch of philosophy introduce by [3] that studies the origin nature and scope of neutralities as well as their interaction with different ideational spectra. This theory considered every entity $< A >$ with its opposite or negation $< antiA >$ together with its spectrum of neutralities$< neutA >$in between them entity supportingneither $< A >$nor$< antiA >$ hence is a generalization of the Hegel dialectics which is based on $< A >$ and $< antiA >$ only. The theory assert that every entity $< A >$ tends to be neutralized and balanced by $< neut\ A >$ and $< anti\ A >$ as a state of equilibrium, so that in classicalway$< A >$,$< neutA >$ and $< antiA >$ are disjoint two by two. And $< neut\ A >$which of course depends on $< A >$ can be regarded as indeterminacy and is labelled as $'I'$. So neutrosophic theory means neutrosophy applied in many field to solve problems related to indeterminacy.

T*he neutrosophic group N(G) = {⟨G ∪I⟩, *} is generated by I and G, under* * [4]. Where 'I' is the indeterminate and

$$I^2 = I, \quad I + I + I, \ldots n\ times = nII - I = 0,0I = 0.$$

It is however to note that:

I. N(G) in general is not a group by itself.

II. N(G) always contains a group.

David N. Koons et al (2005) presented a transient population dynamics: Relations to life history and initial population state using matrix population model. They calculated the short-term (i.e., transient) population growth rate and its sensitivity to changes in the life-cycle parameters for three bird and three mammal species with widely varying life histories [5].

Madan k. Oii et al (2006) provide a unified framework for modeling disease dynamics in discrete time within the framework of matrix population models [6].First, they outline methods for determining model structure for infectious diseases with any number of disease states, and present methods for asymptotic analyses of the model, they then describe methods for estimating model parameters such as $R_o$using rigorous statistical techniques [7].

Zubairu. A (2017), provided a cyclic transition graphs and their corresponding neutrosophic projection matrix with generating function $A_n = \frac{P_n - 1}{2}$ $with$ $P_n = 1, 2, 3$. By transforming A_n into neutrosophic group and varying the permutations of:

$A_n = \left(0, \frac{1}{2}, 1\right)$, in the S, I, R model, it was found that disease transmission is cyclic and that neutrophic projection matrices so derived are strong instrument for further statistical analysis [8].

The motivation for the present work stem from the prime generating function of Aunu sequence $A_n = \frac{P_n - 1}{2} P_n \geq 5$ [9], that generates positive integers upon insertion of primes (≥ 5). The distinction between Aunu generating function and Prime number generating polynomial $3n^2 + 3n + 23$[10]or Mersene primes[11], is that Aunu inputs primes greater than or equal to 5 and output sequence not necessarily prime. These numbers are permuted as a class of (123) and (132) avoiding permutation patterns and is being applied in cellular algebra, circuit design, automata theory, lattices and association scheme.

The Purpose is to explore the same polynomial $A_n = \frac{P_n - 1}{2}$,$P_n = 1, 3, 5, 7$. Also by attaching a neutrosophic spectra of neutrality to A_n, i.e. $\frac{P_n - 1}{2} \cup I$ the generated function is then assigned to the different states in the $S,\ E,\ I,\ R$ type model.

3. Methodology

3.1 Observation

Observed that $A_n = (0, 1, 2, 3)$, this formed a group modulo 4 with respect to addition operator. The set A_n could be permuted in twenty four different permutations and denote the set of permutations of A_n by:

$a_1 = (0, 1, 2, 3).$ $a_2 = (0, 1, 3, 2).$
$a_3 = (0, 2, 3, 1).$ $a_4 = (0, 2,1, 3).$
$a_5 = (0, 3, 1, 2).$ $a_6 = (0, 3, 2, 1).$
$a_7 = (1, 0, 2, 3).$ $a_8 = (1, 0, 3, 2).$
$a_9 = (1, 2, 3, 0).$ $a_{10} = (1, 2, 0, 3).$
$a_{11} = (1, 3,0, 2).$ $a_{12} = (1,3,2, 0).$
$a_{13} = (2,1,0,3).$ $a_{14} = (2,1,3,0).$
$a_{15} = (2,0,3,1).$ $a_{16} = (2,0,1,3).$
$a_{17} = (2,3,1,0).$ $a_{18} = (2,3, 0,1).$
$a_{19} = (3,1,2, 0).$ $a_{20} = (3,1,0,2).$
$a_{21} = (3,2,0,1).$ $a_{22} = (3, 2,1,0).$
$a_{23} = (3,0,1,2).$ $a_{24} = (3,0,2,1)$

The cardinality of A_n is four, discrete, $0 \leq A_n \leq 3$ for each permutation.

3.2 Deduction

Clearly the generated set A_n formed a group modulo 4 $\oplus$ and when we attached and indeterminate factor I to A_n the structure so formed is neutrosophic group.

So $N(G) = A_n \cup I = \begin{pmatrix} 0+0I, 0+I, 0+2I, 0+3I, \\ 1+0I, 1+I, 1+2I, 1+3I, \\ 2+0I, 2+I, 2+2I, 2+3I, \\ 3+0I, 3+I, 3+2I, 3+3I \end{pmatrix}$

This neutrosophic group is also a group modulo 4.

by partitioning $N(G)$ into different subclasses $N_1(G)$, $N_2(G)$, $N_3(G)$,$N_4(G)$ we will again arrived at neutrosophic subclasses of $N(G)$.

$N_1(G) = (0 + 0I, \quad 1 + 0I, \quad 2 + 0I, \quad 3 + 0I)$
$N_2(G) = (0 + I, \quad 1 + I, \quad 2 + I, \quad 3 + I)$
$N_3(G) = (0 + 2I, \quad 1 + 2I, \quad 2 + 2I, \quad 3 + 2I)$
$N_4(G) = (0 + 3I, \quad 1 + 3I, \quad 2 + 3I, \quad 3 + 3I)$

.3.3 Assumptions

We assumed that different subclasses of $N(G)$ belongs to the interval $[0, 3]$ and the probability of transition also belongs to the same interval. The population is sample at discrete time and in $SEIR$type model each individual in the population is assigned to one of the disease states and the transition is serial.

3.4 Model formulation

$$A_n = \begin{Bmatrix} (0,1,2,3),(0,1,3,2),(0,2,3,1),(0,2,1,3),(0,3,1,2),(0,3,2,1), \\ (1,0,2,3),(1,0,3,2),(1,2,3,0),(1,2,0,3),(1,3,0,2),(1,3,2,0), \\ (2,1,0,3),(2,1,3,0),(2,0,3,1),(2,0,1,3),(2,3,1,0),(2,3,0,1), \\ (3,1,2,0),(3,1,0,2),(3,2,0,1),(3,2,1,0),(3,0,1,2),(3,0,2,1). \end{Bmatrix}$$

Lets denote by $(a_i, b_j, c_k, d_l)$the set of all first, second, third, and fourth coordinates of the different permutations in A_n of interval$[0, 3]$and assigned to the different states of the disease transmissionobserved that:

$$\sum_{i=1}^{n} a_i > 3$$
$$\sum_{j=1}^{n} b_j > 3$$
$$\sum_{k=1}^{n} c_k > 3$$
$$\sum_{l=1}^{n} d_l > 3$$

In general by composing two neutrosophic functions, the indeterminacy increases.

Example:

$f_1(x) = x^3$,

$f_2(x) = [2.1, 2.5]^x$, then

$$(f_1 o f_2)(x) = f_1(f_2(x)) = [2.1,2.5]^{3x}$$

Theorem: During a cyclic disease transition the number of susceptible (S), Exposed (E) Infected $(\check{I})$ and recovered (R) changes at any given time (t).

Proof: From equation1 above:

$N(t) = S(t) + E(t)\check{I}(t) + R(t)$, where $N(t)$ denotes the total population.

Assign $N(t)$ to the neutrosophicgroup $mod\ n$. Hence:

$N(t) \cup I = S(t) \cup I + E(t) \cup I\check{I}(t) \cup I + R(t) \cup I$. Where

$$S(t) \cup I = (s_1 + I_1) \oplus (s_2 + I_2) \oplus \ldots (s_n + I_n)$$
$$E(t) \cup I = (e_1 + I_1) \oplus (e_2 + I_2) \oplus \ldots (e_n + I_n)$$
$$\check{I}(t) \cup I = (\check{I}_1 + I_1) \oplus (\check{I}_2 + I_2) \oplus \ldots (\check{I}_n + I_n)$$
$$R(t) \cup I = (r_1 + I_1) \oplus (r_2 + I_2) \oplus \ldots (r_n + I_n)$$

Suppose an individual is introduced into the population, say $n_1(t)$, obviously the R.H.S of equation1 will increase. More precisely $n_1(t)$ could only belong to one of the transition stage at time (t) and recall that the terms on the R.H.S of equation1 are fixed at time (t). So the insertion of $n_1(t)$ to either $S(t)\ or\ E(t)\ or\ \check{I}(t)\ or\ R(t)$is being controlled by the mapping definition below.

Now let $n_1(t)$ be inserted at $S(t)$, then we would at least have:

$$(s_1 + I_1) \oplus (\check{s}_1 + \check{I}_1) = (s_1 + \check{s}_1, \quad I_1 + \check{I}_1) mod n$$

So that the value of $S(t)$ is altered. And since $N(t)$ is neutrosophic group $mod\ n$, and $S(t)$ is fixed the resultant effects of this interaction is being transferred to the next stage at time (t). Continuously and obtained the cyclic disease transition.

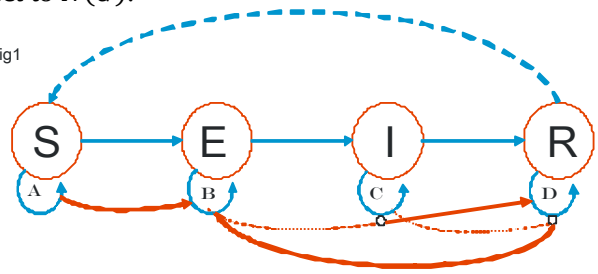
Definition: Let$N(G)$ be a neutrosophic group $mod\ n$ and let $A = a + bI$, $B = c + dI$ be two sets of neutrosophicsubsets of $N(G)$assigned to two successive stages of viral transmission, then the mapping from $A \longrightarrow B$defined by$f: A \longrightarrow B = (ac + (bd)I) mod n$defines the interaction between two successive stages of the viral transmission.The effects of the interactions can be display on the cyclic transitiongraphs.

3.5 Disease interactive transitiongraphs

Let $N_1(G) = A,\ \ N_2(G) = B,\ \ N_3(G) = C,\ \ N_4(G) = D$. Then assign the permutation of A, B, C, D to the transition state of the viral transmission, hence we obtained 24, such permutations.

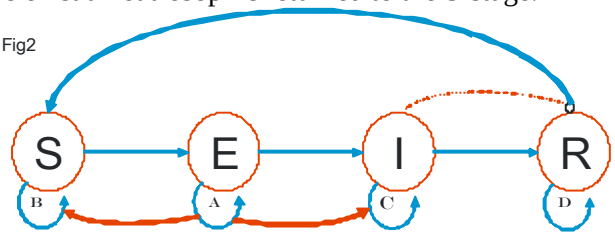
Note that with the exception of $N_1(G)$ which is a group, the neutrosophic subclasses are closed only with respect to $N(G)$.

Fig1



The interaction between S-stage and E-stage(SE) results in the E-satge, while E-stage and I-stage (EI) results in R-stage. Furthermore interaction between I-stage and R-stage results in E-stage of which there exist a neutrosophic returned to the S-stage.

Fig2



In Fig2 the interaction between the S-stage and the E-stage (SE) remained S-stage, while between E-stage and I-stage results at I-stage and finally the interaction between I-stageand R-stage returned S-stage.

Fig3

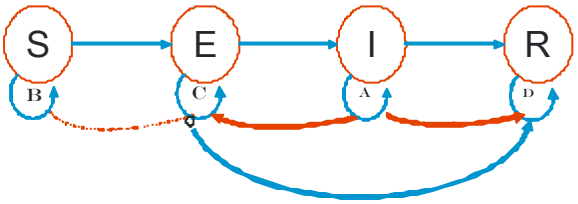


Fig3: The interaction between S-stage and E-stage result on R-stage, while the interaction between E-stage and I-stage remained at E-stage. Furthermore the interaction between I-stage and R-stage result at R-stage

Fig4

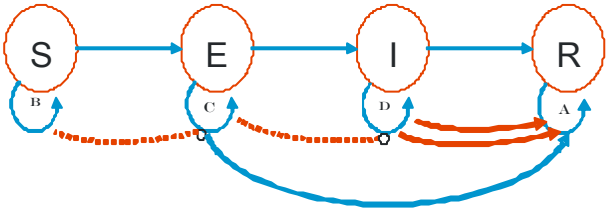


Fig4:The interaction between S-stage and E-stage resulted on R-stage, likewise E-stage and I-stage, also I-stage and R-stage. This is an indication that there are a lot of recoveries in this transition.

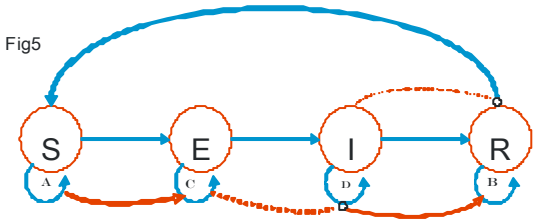


Fig5: The interaction between S-stage and E-stage resulted and E-stage, while the interaction between E-stage and I-stage resulted at R-srage. Finally the interaction between I-stage and R-stage resulted at S-stage.

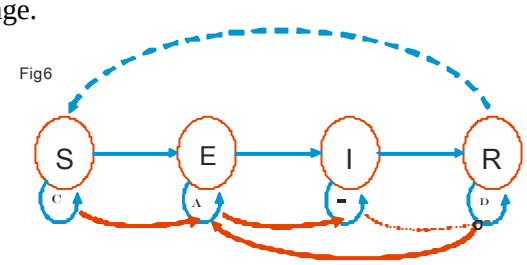


Fig6: The interaction between S-stage and E-stage resulted at E-stage, the interaction between E-stage and I-stage resulted at B. But the interaction between I-stage and R-stage resulted on E-stage and there is neutrosophic retuned to the S-stage.

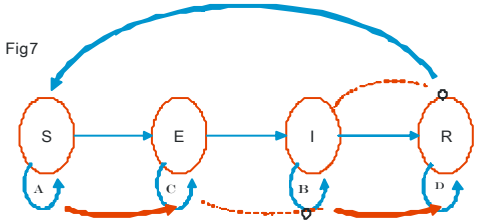


Fig7: The interaction between S-stage and E-stage results at E-stage, while E-stage and Istage result at R-stage. Finally the interaction between I-stage and R-stage returned S-stage.

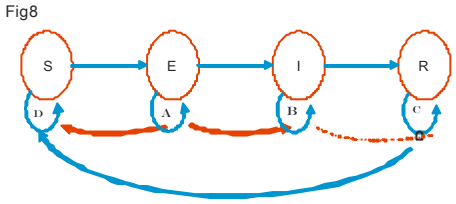


Fig8: The interaction between S-stage and E-stage remains S-stage, while the interaction between E-stage and I-stage result at I-stage. Finally the interaction between I-satge and R-stage returned S-stage.

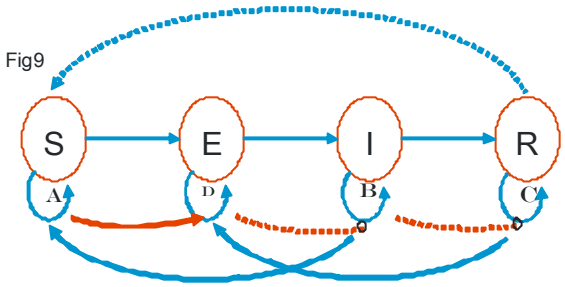


Fig 9: The interaction between S-stage and E-stage result at E-stage, while E-stage and I-stage returned S-stage. Finally the interaction between I-stage and R-stage resulted at E-stage indicating that there is a neutrosophic return to S-stage.

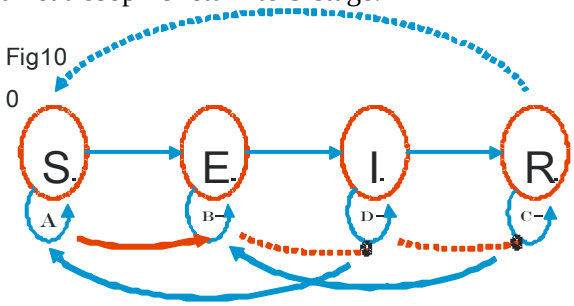


Fig10: The interaction between S-stage and E-stage resulted at E-stage, while the interaction between E-stage and I-stage returned S-stage. Finally the interaction between I-stage and R-stage returned E-stage indicating a neutrosophic returned to the S-stage.

4. Results/Discussion

The corresponding neutrosophic projection matrices related to these disease transition interactive cyclic graphs were obtained as follows:

0 =no connection between nodes.

1 =directly proportional connection.

-1 =inversely proportional connection.

I =directly proportional indeterminate connection.

$-$I =inversely proportional indeterminate connection.

4.1 Neutrosophicprojection matrix

$$\text{Fig1}\begin{bmatrix} 1 & 1 & 0 & -I \\ -1 & 0 & I & -1 \\ 0 & -I & 0 & 1+I \\ I & 1 & -(1+I) & 0 \end{bmatrix} \qquad \text{Fig2}\begin{bmatrix} 0 & -1 & 0 & -I \\ 1 & 1 & 1 & 0 \\ 0 & -1 & 0 & I \\ 1 & 0 & -I & 0 \end{bmatrix}$$

$$\text{Fig3}\begin{bmatrix} 0 & I & 0 & 1+I \\ -I & 0 & -1 & 1 \\ 0 & 1 & 1 & 1 \\ -(1+I) & -1 & -1 & 0 \end{bmatrix} \qquad \text{Fig4}\begin{bmatrix} 0 & I & 0 & 1+I \\ -I & 0 & I & 1 \\ 0 & -I & 0 & 1 \\ -(1+I) & -(1+I) & -1 & 1 \end{bmatrix}$$

$$\text{Fig5}\begin{bmatrix} 1 & 1 & 0 & -1 \\ -1 & 0 & I & 1+I \\ 0 & -I & 0 & 1+I \\ 1 & 0 & -(1+I) & 0 \end{bmatrix} \quad \text{Fig6}\begin{bmatrix} 0 & 1 & 0 & -I \\ -1 & 1 & 1 & -1 \\ 0 & -1 & 0 & I \\ I & 1 & -I & 0 \end{bmatrix}$$

$$\text{Fig7}\begin{bmatrix} 1 & 1 & 0 & -1 \\ -1 & 0 & I & 1+I \\ 0 & -I & 0 & 1+I \\ 1 & 0 & -(1+I) & 0 \end{bmatrix} \quad \text{Fig8}\begin{bmatrix} 0 & -1 & 0 & -1 \\ 1 & 1 & 1 & 0 \\ 0 & -1 & 0 & I \\ 1 & 0 & -I & 0 \end{bmatrix}$$

$$\text{Fig9}\begin{bmatrix} 1 & 1 & -1 & -I \\ -1 & 0 & I & -1 \\ 1 & -I & 0 & I \\ I & 1 & -I & 0 \end{bmatrix} \quad \text{Fig10}\begin{bmatrix} 1 & 1 & -1 & -I \\ -1 & 0 & I & -1 \\ 1 & -I & 0 & I \\ I & 1 & -I & 0 \end{bmatrix}$$

4.2 Discussion

We viewed the SEIR disease transmission as an interactions between two successive stages of the transition and consequently obtained the resultant effects of such interactions. These resultant effects are indicated as shown to be within the limit on the transition graphs, since the stages were allocated with subclasses of the neutrosophic group.

The projection matrices are quantities assigned to the directed neutrosophic graphs, so that important epidemiological statistical concepts such as eigenvalue (λ) (average number of new infected persons generated by one primary case), could easily be estimated.

4.3 Conclusion

The paper provided the interactive stages of disease transition and their corresponding effects on the cyclic nature of disease transition. Here the S, E, I, R model is adopted and with the permutations generated from $A_n = \frac{P_n - 1}{2}$, $P_n = 1, 3, 5, 7$ and assigned to the SEIR disease transmission we arrived at 24transition graphs and consequently 24 neutrosophic projection matrices. But we presented only 10 graphs here which are neutrosophically isomorphic with the remaining. Finally the results could be a sufficient tools for further epidemiological statistical analysis.

References

Vasantha, K. and Florentin, S. (2014), Algebraic structures on real and neutrosophic semi open squares: Educational publishers. Inc, Ohio, USA. http://www.gallup.unm.edu/eBooks.

Vasantha, K. and Florentin, S. (2006), neutrosophic rings: Hexis phoenix, Arizona, USA. ISBN 1-931233-20-9, http://www.gallup.unm.edu.

[Florentin, S. (1995), A unifying field in logic: Third edition. American Research Press RehobothISBN 1.879585-766.

[Florentin,S.(2014), neutrosophic theory and its applications:Collected papers.vol.1 Europa Nova, Brussels, Belgium, http://www.europanova.be

Koons, D.N. et al (2005),Transient population dynamics: Relations to life history and initial population state: Journal, ecological modelling 185(2005) 283-297www.sciencedirect.com

Caswell, H. (2001), Matrix Population Models: Construction, Analysis, and Interpretation. 2nd edition. Sinauer Associates, Sunderland MA.

Madan, K.O et al (2006), Population dynamics of infectious diseases:a discrete time model: Journal ecological modelling 198(2006) 183-194. doi:10.1016/j.ecolmodel.2006,04.007.

Zubairu. A et al (2017), Cyclic disease transition state and its corresponding neutrosophic projection matrix with generating function$A_n = \frac{P_n-1}{2} P_n = 1, 2, 3$: Journal, mathematical association of Nigeria, abacus, mathematical science series.volume 44(1), pp 362-371

Mustafa, A et al (2013), Implementation of Aunu permutation patterns using computer algebra system: A computational approach. Net journals physical sciences research international Vol 1(3), pp 54-61.

Jeevan,M.(2016), Prime number generating polynomial$3n^2 + 3n + 23$: International journal of mathematics and its applications. Vol 4, Issue 1-C, pp.149-150.

Gilbert, I.A. and Joseph R.K. (2015), Invited by the Mersene primes: The perfect numbers and the Mersene composite numbers. ARPN Journal of science and technology, Vol.5 (1), pp. 50-52.