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Diffusion Epidemic model for Analysis the effects of six-day SSRI administration on diurnal cortisol secretion in healthy volunteers using non-linear partial differential equations

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Abstract:
In this paper, we investigated the dynamics of a reaction diffusion epidemic model with specific nonlinear incidence rate. This specific nonlinear incidence rate includes the traditional bilinear incidence rate, the bedding ton-De Angelis functional response, and Crowley Martin functional response. The local stability of the disease-free equilibrium and endemic equilibrium is obtained via characteristic equations. Eventually, we come to the conclusion that medical solutions had obtained and evaluated the relevant mathematical findings. Ultimately, we conclude that the application part coincides with a mathematical model and the result is linked to the medical report. In the future, this paper will be beneficial in the medical field.

Keywords - *Cortisol, Epidemic model, Partial differential equation, HPA axis, Depression*
Mathematical subject classification: $62H_{xx}; 62NO_5; 90B25$.

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I. Introduction

In this article, we check out the following SIR disease model with a particular nonlinear occurrence rate as stated below

$$\frac{ds}{dt} = \Lambda - \mu S - \frac{\beta SI}{1 + \alpha_1 S + \alpha_2 I + \alpha_3 SI}$$
$$\frac{dI}{dt} = \frac{\beta SI}{1+\alpha_1 S+\alpha_2 I+\alpha_3 SI} - (\mu + d + r)I, \qquad (1)$$
$$\frac{dR}{dt} = rI - \mu R,$$

Where S, I, and R are susceptible, infectious, and recovered classes, respectively. $\Lambda$is the enrolment pace of the people, μ is the normal population mortality rate, d is the mortality levels due to sickness, r is the retrieval levels for Infectious Persons, β is the disease coefficient, and $\beta SI(1 + \alpha_1 S + \alpha_2 I + \alpha_3 SI)$ is the event rate, where $\alpha_1, \alpha_2, \alpha_3 \geq 0$ are constants. It is very important to note that this Beddington-DeAngelis functional response introduced in [1,2] and used in [3] when $\alpha_3 = 0$, and Crowley- Martin functional response presented in [4-6] if $\alpha_3 = \alpha_1\alpha_2$. Moreover, the function $\beta S/(1 + \alpha_1 S + \alpha_2 I + \alpha_3 SI)$ satisfies the hypothesis(H1), (H2), and (H3) of general incidence rate presented by Hattaf et al[7].

II. Mathematical Model And Assumptions

In traditional disease models, this rate was believed to be linear with the number of vulnerable and contaminated persons. This assumption is based on a rule of mass intervention that is more suitable for transmittable illnesses, but not for highly contagious diseases such as HIV/AIDS.

The following SIR disease model with a particular non-linear occurrence rate and spatial diffusion is considered:

$$\frac{\partial S}{\partial t} = d_s \Delta S + \Lambda - \mu S(x,t) - \frac{\beta S(x,t) I(x,t)}{1 + \alpha_1 S(x,t) + \alpha_2 I(x,t) + \alpha_3 S(x,t) I(x,t)}$$

$$\frac{\partial I}{\partial t} = d_1 \Delta I + \frac{\beta S(x,t) I(x,t)}{1+\alpha_1 S(x,t)+\alpha_2 I(x,t)+\alpha_3 S(x,t) I(x,t)} - (\mu + d + r) I(x,t)$$

$$\frac{\partial R}{\partial t} = d_R \Delta R + rI(x,t) - \mu R(x,t), \qquad (2)$$

Where S(x,t), I(x,t) and R(x,t) represent the numbers of susceptible, infected, and removal individuals at location x and time t, respectively. The optimistic constants $d_s, d_{I,} and\ d_R$ are the equivalent diffusion levels for all three groups of persons.

The goal of this work is to analyse the global dynamics of the diffusion-reaction mechanism (2). Note that R is not present in the first two equations; this helps one to research the process.

$$\frac{\partial S}{\partial t} = d_s \Delta S + \Lambda - \mu S(x,t) - \frac{\beta S(x,t) I(x,t)}{1 + \alpha_1 S(x,t) + \alpha_2 I(x,t) + \alpha_3 S(x,t) I(x,t)}$$

$$\frac{\partial I}{\partial t} = d_1 \Delta I + \frac{\beta S(x,t) I(x,t)}{1 + \alpha_1 S(x,t) + \alpha_2 I(x,t) + \alpha_3 S(x,t) I(x,t)} - (\mu + d + r) I(x,t), \qquad (3)$$

With the uniform equations of Neumann

$$\frac{\partial S}{\partial v} = \frac{\partial I}{\partial v} = 0, \text{ on } \partial\Omega \text{x } (0,+\infty), \qquad (4)$$

And primary conditions

$$S(x,0) = \phi_1(x) \geq 0,\ I(x,0) = \phi_2(x) \geq 0, x \in \bar{\Omega}. \qquad (5)$$

Here, Ω is a bounded domain in R^n with smooth boundary $\partial\Omega, \partial S/\partial v$ and $\partial I/\partial v$ are respectively, the normal derivatives of S and I on $\partial\Omega$.

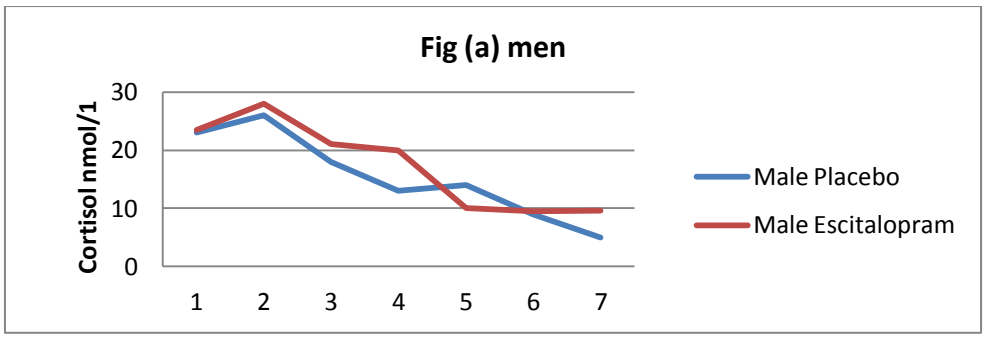
III. Applications

Depression is one of most common conditions associated with stress. The deficiency in cholinergic function is one of a neurobiology of anxiety. Hyperactivity of the hypothalamic-pituitary-adrenal (HPA) axis was often commonly documented in severe depression [12].There is data that the disturbances of the cholinergic stystem and the HPA axis are related, and this association may be a significant mechanism involved in the design of depression[].

Cortisol is the terminal point of HPA axis and is the main intention corticosteroid in people. Studies investigating the impact of selective serotonin reuptake inhibitors (SSRIs) on suicidal subjects have resulted in both short-and long-term SSRI increases and reductions in basal cortisol levels, and several investigations have indicated zero impact [1,3,4-5,7,17,10-11].

The secretion of Cortisol has a pronounced dium sequence. Cortisol is now at a high level on waking, followed by an increase that hits a plateau about 30 min after waking. This is referred to as the cortisol awaking reaction (CAR).

Cortisol curve was measured in nanomoles per litre per minute (nmol/L/min) by regressing cortisol at sampling period, except +30 min; higher values suggest a steeper decline in cortisol throughout the day.



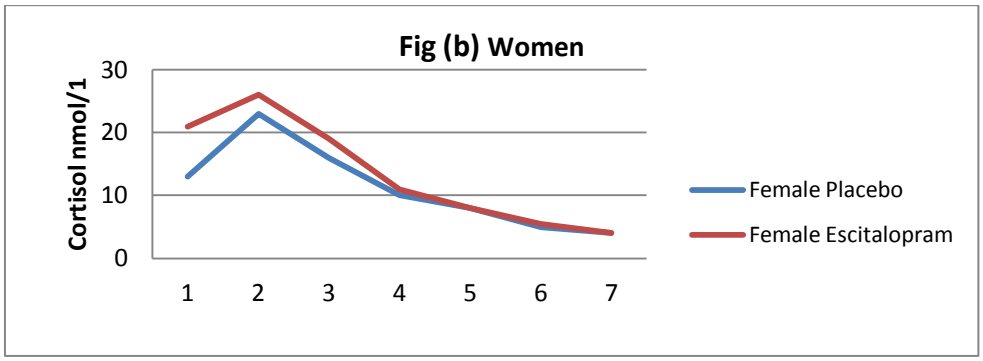
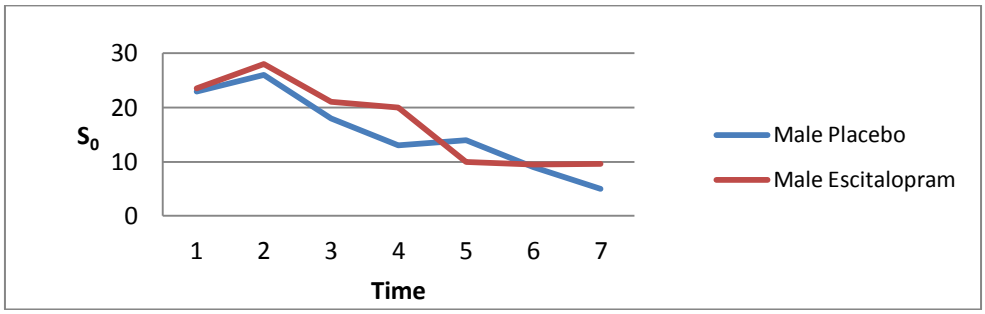
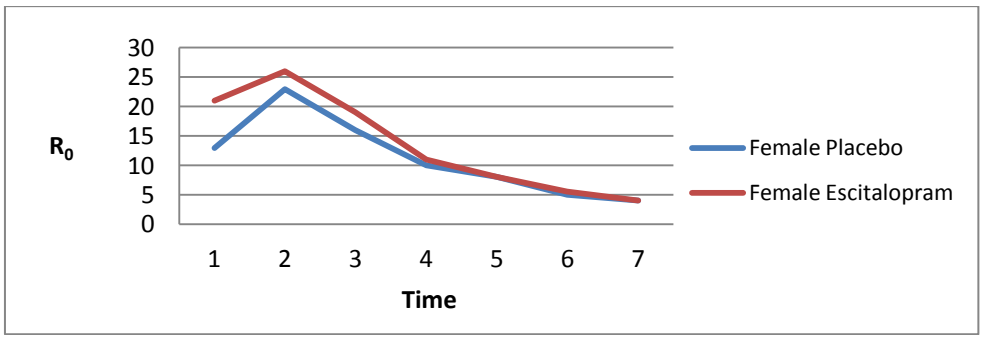


Fig 1 & 2 There was no significant impact of medications or sex on the slope of cortisol. However, ANOVA revealed an important drug category by having a sexual activity impact on hormone slope. Evaluating men and women subjects independently showed no impact of drugs on men's cortisol curve. There was however, an influence of medicine on women. People who take escitalopram saw heavier cortisol slopes relative to women getting placebo. Additions in the cortisol slope may be influenced by the amount of cortisol at awakening and/or at night. We then investigated the impact of escitalopram on waking and evening levels of cortisol in female participants. Medicine has a major key impact on cortisol awakening values with higher levels in female having taken escitalopram than placebo. There was no major effect of the drug on the values of cortisol in the evening.

The purpose of this research was to evaluate the impact of six-day administering of escitalopram on many different indicators of diurnal HPA activity in healthy volunteers. This was the first research to investigate the impact of SSRIs on cortisol release independently of symptom relief or mood shifts. We believed that escitalopram would lead to changes in the Vehicle, cortisol AUC, and cortisol slope, and more precisely, that escitalopram would lead to increased in levels of cortisol.

IV. MATHEMATICAL RESULTS





V. Conclusions

In this article, a diffusion epidemic model with complex nonlinear partial differential equations was used to analyse the impact of SSRI on diurnal cortical activity in healthy subjects. In the classical disease model, this rate was believed to be linear with the amount of vulnerable and contaminated persons. Finally, we reached the conclusion that the necessary statistical results have been collected and tested by medical solutions.

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