International Research Journal of Mathematics, Engineering and IT
Vol. 3, Issue 9, September 2016 IF- 3.563 ISSN: (2349-0322)
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THE ARA_{∞} MODEL FOR THE ADRENOCORTICAL RESPONSIVENESS TO INFUSIONS OF PHYSIOLOGICAL DOSES

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ABSTRACT

In this paper we discussed theinitial intensity, minimal wear intensity of failure process without repair is supposed to be a Non Homogeneous Poisson Process (NHPP). The repair effect is characterized by the change induced on the failure intensity before and after failure. The magnitude and time course of changes in concentrations of plasma cortisol and DHEA in response to bolus infusions ofphysiological doses of ACTH in PTSD patients and control subjects and no evidence for PTSD-related alterations in cortisol or DHEA secretion in response to stimulation by low doses of ACTH and conclude that adrenocortical responsiveness is normal in PTSD.The occurrence of defects in HPA function in PTSD may be specific responses to particular combinations of trauma type, genetic susceptibility,and individual history.

Keywords: ACTH,NHPP,DHEA,PTSD

Introduction

Attribution of relationships between posttraumatic stress disorder (PTSD) and hypothalamic–pituitary–adrenocortical (HPA) dysfunctionis intuitively apposite because of the importance of

theHPA axis in the mediation of stress responses, and several earlystudies of PTSD reported HPA dysregulation .

This study was approved by the institutional review board atthe University of Washington, and all subjects provided writteninformed consent. All subjects underwent a medical history, screeningphysical examination, 12-lead electrocardiogram, and screening laboratory tests of blood samples for electrolytes, glucose, blood urea nitrogen, creatinine, serum glutamic oxaloacetic transaminase, serum glutamic pyruvic transaminase, thyroid function tests, andcomplete blood count, and of urine samples for routine urinalysis, urine toxicology screen, and for female subjects of child bearing potential, urine pregnancy test. Use of any psychoactive medication or any medication knownto affect the HPA axis within 2 weeks of the study was exclusionary for all subjects. These medications included antidepressants, antipsychotics, anticonvulsants, tranquilizers, sedative/hypnotics, antihypertensives, and oral or inhaled glucocorticoids. The only medications taken by participants in the study were ibuprofen, the antihistamines cetirizine and loratadine, and glucosamine andchondroitin for treatment of osteoarthritis.Participants taking medications to treat PTSD were withdrawn from those medications, under medical supervision, for up to 30days. The period required for withdrawal was dependent on thebiological half-life of each medication and management of potentialwithdrawal symptoms. Female subjects of childbearing potential underwent urinepregnancy tests on each study day to ensure that no pregnant participantswere exposed to ACTH. Female subjects of child bearing potential agreed to use a non-hormonal barrier form of birth control for the period of the study. Hormonal contraceptives were not allowed for at least 3 months prior to the study because of their stimulation of corticosteroid-binding globulin (CBG) that results in elevated levels of total plasma cortisol.

All participants were free of current (past 6 months) acuteor chronic Axis I psychiatric disorders (with the exception of PTSD in the PTSD subject group) based on SCID assessment. Two control and one PTSD subject had past histories of depression. Absence of substance abuse within 3 months of the study by subject report and urine toxicology screen was required. One control and seven PTSD subjects had a past history of alcohol abuse, and one individual in each group had a history of alcohol dependence. Two control and one PTSD participant had past histories of substance abuse (cannabis). One subject in each group was a current smoker; no information about past smoking history was requested. Participants included nine PTSD subjects:

eight males with combat trauma PTSD and one female with civilian trauma PTSD,50.3 ± 2.5 years old [mean ± standard error of the mean (SEM)].There were nine healthy non-trauma exposed aged-matched controls: six males and three females, 41.2 ± 3.5 years old. The ages of the two groups did not differ significantly. The PTSD participants had moderately severe symptoms according to the CAPS.

Blood samples were collected every 5 min for 60 min following ACTH administration and then every 15 min for the next 90 min. Blood was collected into chilled tubes containing ethylenediaminetetraacetic acid; the tubes were placed on ice until processed. The samples were cold-centrifuged within 1 h of collection. The plasma was aliquoted and stored at −80°C until assayed. For each hormone, all samples from a given subject were included in a single assay, and each assay contained samples from an equal number of subjects from each group.

The peak ACTH concentrations achieved after the higher of our two doses were approximately which are comparable to those reached after administration ofthe standard insulin tolerance test to control subjects.

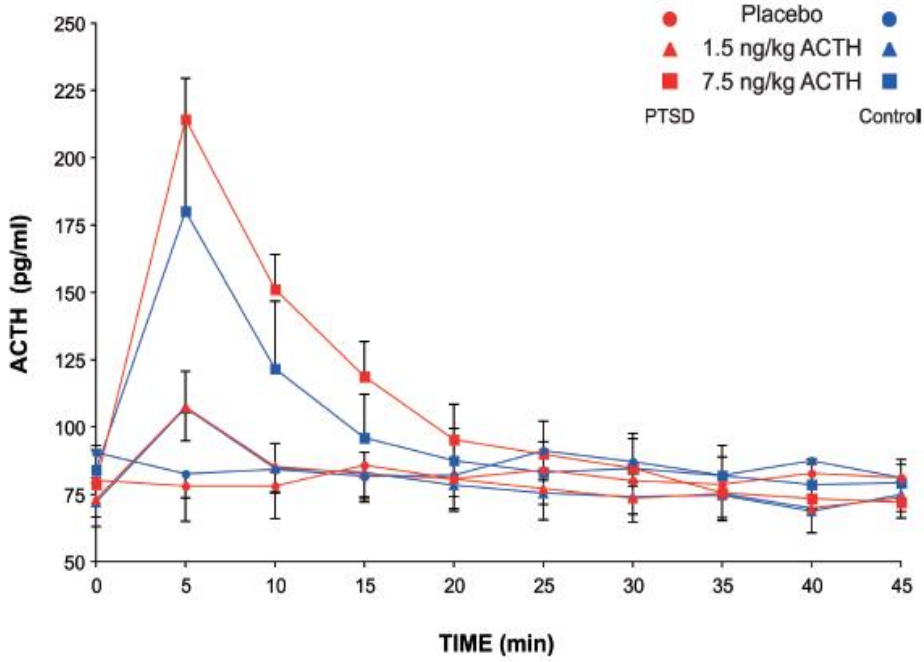

FIGURE 1 | Plasma ACTH concentration in PTSD and control subjects after ACTH infusion. Sampling occurred at 5-min intervals for 60 min and at 15-min intervals for the following 90 min. Mean and SEM values are plotted for each subject group and each dose of ACTH for the first 45 min after infusion during the dynamic portion of the ACTH concentration profile.

Mathematical model

Some important industrial systems (like nuclear power plants, planes, trains) come to the end of their planned life, but they seem to be still in normal working conditions. To extend their functioning life, one must justify some reliability requirements. One way to do it is to take into account the effect of repair actions or corrective maintenance. Repair is carried out after a failure and intends to put the system into a state in which it can perform its function again. Modelling the effect of these repair actions is of great practical interest and is the first step in order to be able to assess maintenance efficiency.

The basic assumptions on repair efficiency are known as minimal repair or As Bad As Old (ABAO) and perfect repair or As Good As New (AGAN). In the ABAO case, each repair leaves the system in the same state as it was before failure. In the AGAN case, each repair is perfect and leaves the system as if it were new. Obviously, reality is between these two extreme cases: standard maintenance reduces failure intensity but does not leave the system as good as new. This is sometimes known as imperfect or better-than-minimal repair.

Assumptions

The distribution of these processes is completely given by the failure intensity defined as:

T_i - be the time of stress effect.

N_t- be the number of stress effect in time t.

H_t - be the past stress effect in time t.

$$\forall t \geq 0, \quad \lambda_t = \lim_{dt \to 0} \frac{1}{dt} P(N_{t+dt} - N_t = 1 | \mathcal{H}_t)$$

We assume that before the first failure, the failure intensity is a deterministic and continuous function of time $\lambda(t)$ called the initial intensity. In addition, the considered system is supposed to wear out continuously, so the initial intensity is strictly increasing.

For any stochastic point process $\{E_t\}_{t \geq 0}$ wedefine E_{Ti}^{+} (resp. E_{Ti}^{-}) as the left (resp. right) limit, if it exists, of E_t when t tends to T_i.

Definition:

For a model with failure intensity λt, the minimal wear intensity is, if it exists, the deterministic function $\lambda \min(t)$ defined as:

$$\forall t \in \mathbb{R}^+,\ P(\lambda_t \geq \lambda_{min}(t)) = 1$$

$$\forall \epsilon > 0,\ \forall t \in \mathbb{R}^+,\ P(\lambda_t \leq \lambda_{min}(t) + \epsilon) > 0$$

(2) means thatλ_t is greater than λ_{min}(t)and (3) means that λ_t can be asclose as possible to λ_{min} (t).The minimal wear intensity can be viewed as the maximal lower bound for failure intensity. The wear out of all system with failure intensity λ_t is greater than that of a system with failure intensity λ_{min}(t).

The ARA$_\infty$ model

The ARA$_\infty$ assumption is that repair reduces the virtual age of the system of an amount proportional to its age just before repair:

$$A_{T_i^+} = A_{T_i^-} - \rho A_{T_i^-}$$

Then, by analogy with the ARI$_\infty$ model, the failure intensity of the Arithmetic Reduction of Age model with infinite memory (ARA$_\infty$) is:

$$A_{T_i^-} - A_{T_{i-1}^+} \text{ to } (1-\rho)[A_{T_i^-} - A_{T_{i-1}^+}]:$$

The minimal wear intensity is equal to zero.

This model appears to be the same as the one introduced by Brown-Mahoney-Sivazlian .

Property

If λ is convex (resp. concave), for the same parameter $\rho\epsilon$ [0; 1],the minimal wear intensity of the ARI$_1$ model is greater (resp. less) than thatof the ARA$_1$ model.

Proof

Since λ is convex and $\rho\epsilon$ [0; 1], $\forall t \geq 0$, $\lambda'(t) \geq \lambda'(1-\rho)t$).

By integrating the previous inequality on [0,t] we obtain:

$$(1-\rho)\lambda(t) \geq \lambda((1-\rho)t)$$

And the property is proved for λ convex. A similar proof holds for λ concave.

When the initial intensity is that of a Power Law Process (PLP: $\lambda(t) = \alpha\,\beta\,t^{\beta-1}$, there exists two parameters ρI_1 and ρA_1 such that the ARI_1model with parameters (α, β, ρI_1) and the ARA_1 model with parameters (α, β, ρA_1)have the same minimal wear intensity.

$$\rho_{I1} = 1 - (1-\rho_{A1})^{\beta-1}$$

The proof is immediate since:

$$(1-\rho_{I1})\alpha\beta t^{\beta-1} = \alpha\beta[(1-\rho_{A1})t]^{\beta-1} \Rightarrow \rho_{I1} = 1-(1-\rho_{A1})^{\beta-1}$$

Conclusion

Here we found the initial intensity, minimal wear intensity of failure process without repair for The occurrence of defects in HPA function in PTSD may be specific responses to particular combinations of trauma type, genetic susceptibility, and individual history.

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