Cumulative hrf of NMWD model for Renin in patients with Depression

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Abstract:
Hypercortisolism as a sign of hypothamamus-pituitary-adrenocortical (HPA) axis over activity and sleep EEG changes are frequently observed in depression. Closely related to the HPA axis is the renin-angiotensin-aldosterone system (RAAS) as 1. adrenocorticotropic hormone (ACTH) is a common stimulus for cortisol and aldosterone, 2. cortisol release is suppressed by mineralocorticoid receptor (MR) agonists 3. angiotensin II (ATII) releases CRH and vasopressin from the hypothalamus. Here difference of sleep related activity of the RAAS between depressed patients and healthy controls and also we found the analysis of survival function and cumulative hazard rate function for renin.

Keywords: Cumulative hrf, NMWD, depression

I. Introduction:
Hypercortisolism as well as a reduced feedback inhibition of the hypothalamus-pituitary-adrenocortical (HPA) system are frequently observed in depression [1]. Further, a decreased ability of dexamethasone to suppress adrenocorticotropic hormone (ACTH) and cortisol secretion is found in depressed patients, but appears to depend on the clinical characteristics, especially "typical" vegetative signs, as sleep disturbances and weight loss [2,3] and reproductive state in females. A reduced plasma renin activity (PRA) is observed in depressed patients [4] and the increase in resting PRA accompanied by a blunted renin response to posture in bipolar patients. The latter study described a normal aldosterone concentration, which was interpreted to be inappropriate in relation to the increased PRA. A possible link between possible changes of the RAAS and the HPA system in depression was further demonstrated by the significant increase in aldosterone secretion after administration of the glucocorticoid receptor agonist dexamethasone in healthy female subjects, whereas depressed females aldosterone showed a trend to a decrease.

Methods: We studied nocturnal plasma concentration and sleep EEG in 7 patients with depression (1) male, 6 females, age: 53.3 ± 14.4 (mean ± SD), range 34 – 70 years) and 7 age-matched controls (2) males, 5 females, age: 54.7 ± 19.5, range 27 – 76 years). The data from three of the controls were derived from the control condition of an earlier study [5] and four were newly recruited. Both patients and controls were free of medication for at least 10 days and fluoxetine for at least 4 weeks with the exception of 1 patient receiving 500 mg chloral hydrate at the two study nights and one subject receiving metoclopramide 10mg once the day of the examination. However, even after exclusion of these subjects the main findings of the study were unchanged (data not shown). No substances for blood pressure regulation, especially beta-receptor blockers or angiotensin-converting enzyme inhibitors or diuretics were used by any of the subjects. Further, no relevant comorbidity, especially no cardiovascular, renal or hepatic disorder was present in the patients or controls, as asessed by clinical examination and a standard clinical laboratory examination including serum creatinine and liver enzyme levels. The personal and family history of the controls was free of psychiatric disorders.
II. Mathematical Model:

Acronyms

hrf — Hazard Rate Function
NMWD — New modified Weibull distribution
sf — Survival Function

Notations

a,b — NMWD parameters a > 0, b > 0 for renin
h(t) — hrf of renin
H(t) — cumulative hrf of renin
t — time of renin
F(t) — sf of renin

Here, The usual 2-parameter Weibull distribution can be specified through its sf:

\[ F(t; \beta, \lambda) = \exp \left[-(\lambda \cdot t)^\beta\right] \]  \hspace{1cm} (1)

The lifetime distribution for NMWD arises from taking appropriate limits on the Beta integrated distribution from [6] The sf is

\[ F(t; \beta, \lambda) = \exp \left[- a \cdot t^b \cdot (\lambda \cdot t)\right] \]  \hspace{1cm} (2)

With parameters a > 0, b ≥ 0 and \( \lambda > 0 \).

The pdf and hrf are:

f(t) = a \cdot (b + \lambda \cdot t) \cdot t^{-b-1} \cdot \exp (\lambda \cdot t) \cdot \exp [-a \cdot t^b \cdot \exp (\lambda \cdot t)] \hspace{1cm} (3)

h(t) = a \cdot (b + \lambda \cdot t) \cdot t^{-b-1} \cdot \exp (\lambda \cdot t) \hspace{1cm} (4)

The derivative of \( h(t) \) intersects the t axis only once, at \( t^* \) for \( t^* > 0 \), h(t) is decreasing for \( t < t^* \), and is increasing for \( t > t^* \) which is given by

\[ t^* = \frac{\sqrt{b - \lambda}}{\lambda} \]  \hspace{1cm} (5)

The interesting feature is that \( t^* \) decreases as \( \lambda \) increases.

For \( \lambda = 0 \) in (2) NMWD reduces to \( F(t) = \exp [-a \cdot t^b] \) \hspace{1cm} (6)

Which is a common 2-parameter Weibull distribution.

The beta-integrated model was first introduced in [6]. The model’s cumulative hrf and sf are:

\[ H(t) = a \cdot t^b \cdot (1 - d \cdot t)^c, \hspace{1cm} 0 < t < 1/d; \]  \hspace{1cm} (7)

\[ F(t) = \exp[-H(t)]; \hspace{1cm} a, b, d > 0; c < 0 \]  \hspace{1cm} (8)

Set \( d = 1/n, c = \lambda, n \), For \( n \to \infty \),

\( \left(1 - \frac{t}{n}\right)^{\lambda n} \to \exp(\lambda \cdot t) \).

and this yields
H(t) = a . t^b . \exp(\lambda . t).

-------------(9)

Which is the cumulative hrf for NMWD.

The data is fitted with the distribution and the corresponding values for case:1 and case:2 are obtained as follows

Case 1: Depressive
If \( a=2.6887, \ b=10.2527, \ t=0.5 \) then \( H(t) = 0, \ F(t) = 0.9978 \)

Case 2: Control
If \( a=2.3572, \ b=18.8723, \ t=0.5 \) then \( H(t) = 0, \ F(t) = 0.9999 \)

### III. Conclusion:
There was no difference of sleep related activity of the RAAS between depressed patients and healthy controls and also we found the analysis of survival function and cumulative hazard rate function for renin.

### References:


