

Mathematical Model of WIE to Cortisol with Comparison between Obese Subjects with and Without BED

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ABSTRACT: In this paper, a generalized version of the inverted exponential distribution called the weighted inverted exponential distribution is introduced. Several properties of the weighted inverted exponential distribution are studied and derived. In application part the relationship between induced psychological stress, HPA axis and eating behaviour in Binge Eating Disorder (BED) has been discussed. The Trier Social Stress Test (TSST) was applied in obese participants with (n = 8) and without BED (n=8), and normal weight (NW) controls (n=8). At baseline desire to binge was significantly higher among the BED group. While the TSST induced an increase in Cortisol levels, a blunted Cortisol response was observed in the BED group.

KEY WORDS: WIE, cortisol, stress, obesity

I. INTRODUCTION

The gamma distribution and the generalized exponential distribution are different weighted versions of the exponential distribution. Gupta and Kundu [6] have followed a similar approach as of Azzalini [2], for introducing a shape parameter to the exponential distribution. They showed that by applying Azzalini's method to the exponential distribution, a new class of weighted exponential distribution denoted by WE distribution could be obtained. Furthermore, they showed that the WE distribution possess some good properties and can be used as a good fit to survival time data compared to other popular lifetime distributions. A similar way to introduce the two parameter weighted exponential distribution by simply modifying Azzalini's approach to introduce two shape parameters instead of one as the case in the weighted one parameter exponential and the weighted two parameter Weibull distributions. Most of distributional developments are made using the exponential distribution because of its simplicity and mathematical feasibility that made it the most widely used lifetime model in reliability theory. If a random variable X has an exponential distribution, $Y=1/X$ will have an inverted exponential (IE) distribution [3]. A shape parameter is introduced in the IE distribution to obtain the generalized inverted exponential (GIE) distribution [9, 10]. The IE distribution has the following cumulative distribution function (cdf) and probability density function (pdf) for $X > 0$.

$$F(x) = \exp\left(-\frac{\lambda}{x}\right),$$

With, $f(x) = \frac{\lambda}{x^2} \exp\left(-\frac{\lambda}{x}\right)$, where $\lambda > 0$ and $\alpha > 0$ are the scale and shape parameters respectively.

The main aim of this paper is to introduce a new generalization of the inverted exponential distribution based on a modified weighted version of Azzalini's approach is to introduce a shape parameter to a probability distribution function in the following way. Let $g_0(x)$ be a one-dimensional probability density function, and $G_0(x)$ is the corresponding one-dimensional distribution function such that $G_0(x)$ exists, then

$$f(x; \lambda, \alpha) = K g_0(x) G_0(\alpha x)$$

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WEIGHTED INVERTED EXPONENTIAL

A random variable T is said to have a weighted inverted exponential (WIE) distribution, if the cdf and pdf of T are given by

$$F(t; \lambda, \alpha) = (1 + \alpha) \left(\exp\left(-\frac{\lambda}{t}\right) - \frac{\alpha}{(1+\alpha)} \exp\left(-\frac{\lambda(1+\alpha)}{at}\right) \right), \text{And}$$

$$f(t; \lambda, \alpha) = (1 + \alpha) \frac{\lambda}{t^2} \exp\left(-\frac{\lambda}{t}\right) \left(1 - \exp\left(-\frac{\lambda}{at}\right) \right), \text{Where } \lambda \text{ and } \alpha \text{ are parameters.}$$

The WIE distribution is reduced to the inverted exponential (IE) distribution as $\alpha \rightarrow 0$. Furthermore, the distribution of $X = 1/T$ is the weighted exponential distribution and $\alpha \rightarrow 0$, it reduces to the one-parameter exponential distribution.

The failure rate function (FR), the reversed hazard rate (RHR) of the WIE distribution are given by,

$$r(t) = \frac{f(t)}{R(t)} = \frac{\lambda \exp\left(-\frac{\lambda}{t}\right) \left(1 - \exp\left(-\frac{\lambda}{at}\right) \right)}{t^2 \left(1 - (1 + \alpha) \exp\left(-\frac{\lambda}{t}\right) + \alpha \exp\left(-\frac{\lambda(1+\alpha)}{at}\right) \right)}$$

And the RHR (also known as Mills' ratio) is as follows

$$h(x) = \frac{f(t)}{1-R(t)} = \frac{\lambda \exp\left(-\frac{\lambda}{t}\right) \left(1 - \exp\left(-\frac{\lambda}{at}\right) \right)}{\left(\exp\left(-\frac{\lambda}{t}\right) - \frac{\alpha}{(1+\alpha)} \exp\left(-\frac{\lambda(1+\alpha)}{at}\right) \right)} \text{Where } x > 0 \text{ and } \lambda, \alpha > 0.$$

II. APPLICATION

The magnitude of this problem has resulted in substantial research, thus increasing our understanding of the biological factors involved in appetite and eating behaviour. A specific area in this field, the relationship between stress regulatory systems and eating behaviour has also received much attention lately. There is increasing evidence that central components of the reward system also play a role in the regulation of the stress response. Subsets of people who are obese suffer from Binge Eating Disorder (BED) [1, 4, 5], an eating disorder characterized by bouts of uncontrolled binges without the compensatory behaviours that characterize bulimic patients. About 30% of obese people are diagnosed with BED, while the prevalence within the general population is between 2–3% to about 6%. Negative emotions have been associated with both increased and decreased food intake and while this may be related to individual stress reactivity the mechanisms underlying such opposed behaviours have not been elucidated Under conditions of high emotional load, restrained and emotional eaters usually eat more food, specifically sweet and fat foods perhaps suggesting a lack of responsiveness to satiety signals while under stress.

Neuronal networks that interconnect the hypothalamus and the limbic system suggest the existence of a neural circuit in which mood states strongly influence eating behaviour. Activation of the HPA axis is involved in the onset of binge eating attacks relies on several lines of evidence: (1) Stress plays a definite role in the initiation of binge eating episodes.

(2) Cortisol is a mediator of increased caloric intake.

(3) High cortisol levels were positively related to food intake after laboratory stress stimuli.

Few studies have examined the HPA axis in participants with BED. Cortisol suppression after dexamethasone administration has been shown to be normal in this population Single measurement evening cortisol, as well as morning cortisol levels, have been reported to be normal both in obese as well as in non-obese women with BED. In contrast, overall cortisol secretion as reflected by repeated cortisol measurements during the day for two consecutive days were found to be significantly lower in obese women with BED in comparison to obese women without BED. Finally, Gluck and colleagues found higher basal cortisol levels in obese women with BED, and a nearly significant higher cortisol secretion following a cold stress test in comparison to the control group. In view of the contradictory results reported in these studies, no conclusions can be drawn as to the HPA axis function in BED subjects. An extensive review of HPA axis and stress involvement in eating disorders was published. This review concludes that different HPA axis abnormalities have been observed in BED and obese subjects, however these alterations are considered to be mainly due to excess weight. Thus, the specific association between BED and the HPA axis that is not secondary to obesity still merits study.

To further investigate the involvement of the HPA axis in the eating response to negative emotions, we chose to use a wellstandardized psychological stress test, the Trier Social Stress Test (TSST). Cortisol secretion and cognitions related

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to eating were measured prior and immediately after the psychological stress in obese participants with and without BED and a control group of normal weight participants. Our hypothesis was that the cortisol response to acute stress in obese BED patients will be higher than in obese non-BED patients [7, 8].

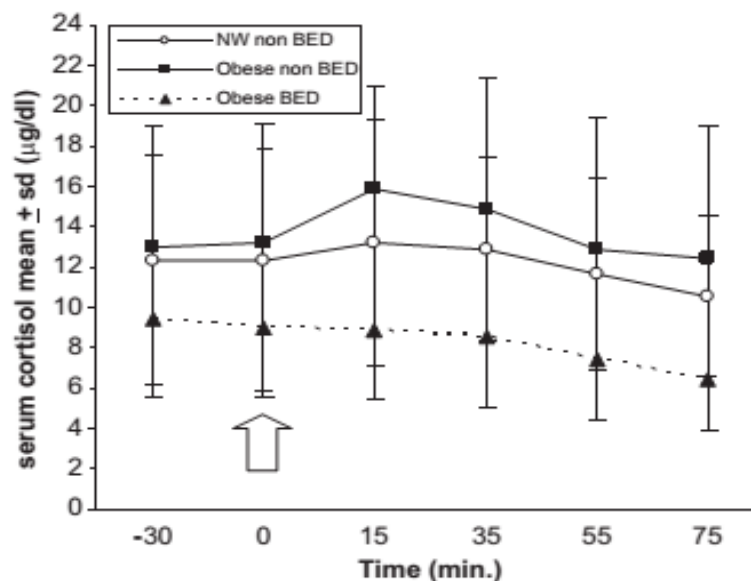
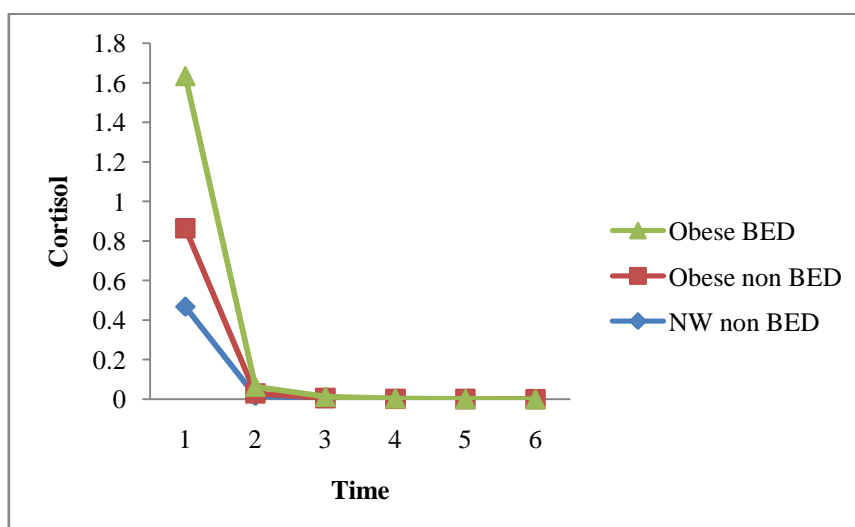


Fig 1: Cortisol response to the Trier Social Stress Test according to groups.

III. MATHEMATICAL RESULTS

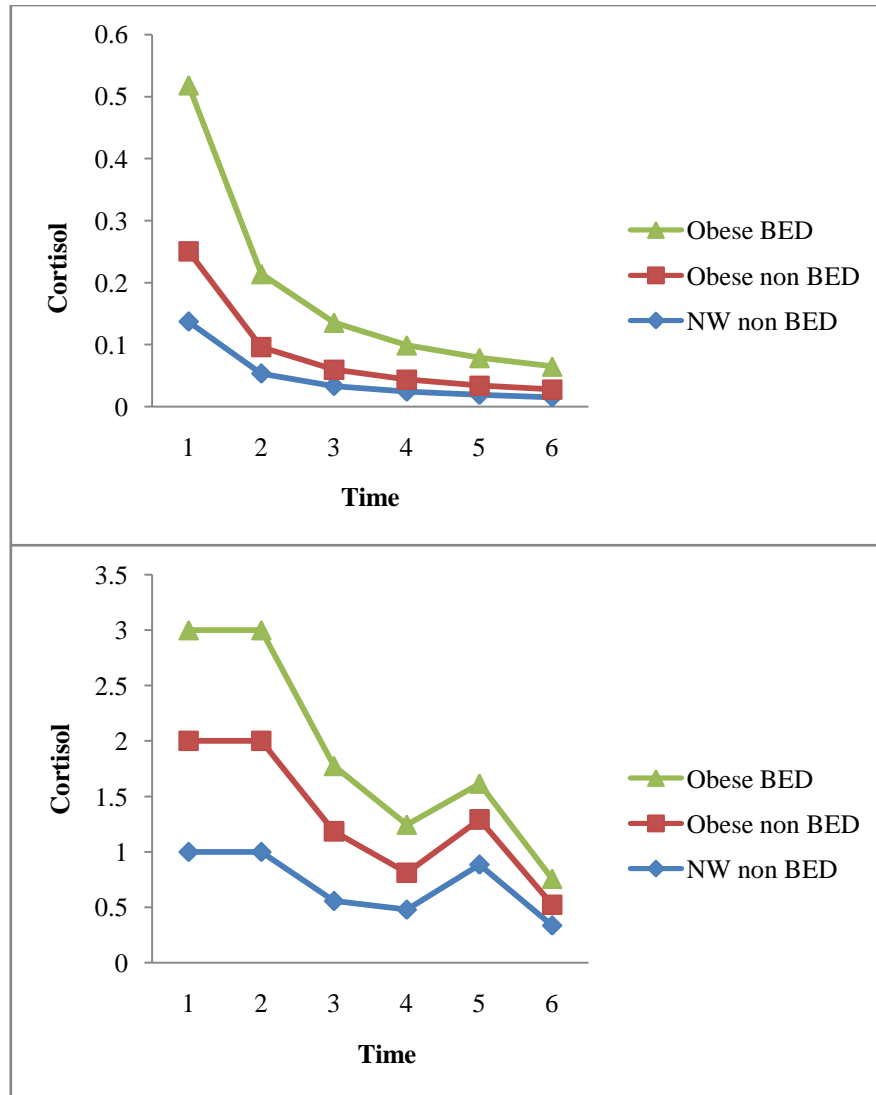
The mathematical results of WIE, RHR and HR of WIE are given below.



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IV. CONCLUSION

In this paper we discussed about the inverted exponential distribution which combined the kumaraswamy and the inverted exponential distributions to introduce the kumaraswamy inverted exponential distribution known as the generalized inverted exponential (GIE) distribution. The proposed distribution can be seen as another generalization to the inverted exponential distribution where, the idea is to use a modified Azzalini's approach to introduce the weighted inverted exponential distribution. In application part BED is discussed. Finally it is concluded from WIE basal cortisol levels were similar in all groups but blunted cortisol levels were observed in BED groups.

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