

EXPONENTIATED WEIGHTED EXPONENTIAL
 DISTRIBUTION FOR HORMONAL AND METABOLIC RESPONSES TO EXERCISE

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ABSTRACT

In this paper we used Exponentiated Weighted Exponential distribution and it serves as an alternative to both the Weighted Exponential distribution and the Exponential distribution. Hormonal and metabolic responses to exercise across time of day and menstrual cycle phase. Two studies, each utilizing short-term treadmill exercise of a different intensity, assessed the metabolic and hormonal responses of women to exercise in the morning (AM) and late afternoon (PM). In study 1, plasma concentrations of growth hormone, arginine vasopressin, catecholamines, adrenocorticotrophic hormone, cortisol, lactate, and glucose were measured before, during, and after high-intensity exercise (90% maximal O₂ uptake) in the AM and PM. In study 2, plasma concentrations of adrenocorticotrophic hormone, cortisol, lactate, and glucose were measured before, during, and after moderate-intensity exercise (70% maximal O₂ uptake) in the AM and PM in the follicular (days 3–9), midcycle (days 10–16), and luteal (days 18–26) phases of the menstrual cycle. The results of studies 1 and 2 revealed no significant diurnal differences in the magnitude of responses for any measured variable. We use WE distribution to find the metabolic and hormonal responses to short-term, highintensity exercise can be assessed with equal reliability in the AM and PM and that there are subtle differences in blood glucose responses to moderate-intensity exercise across menstrual cycle phase.

Keywords: WE distribution, Exponential distribution, adrenocorticotrophic hormone, AVP.

MATHEMATICAL MODEL

Extreme Value distributions have been found to be useful in the fields of engineering, insurance and modern science. Gupta and Kundu developed a two-parameter Weighted Exponential (WE) distribution as a lifetime model and it has been widely used engineering and medicine. In addition, the we distribution has been discovered to be a competitor to the Weibull, Gamma and Generalized Exponential distributions. The probability density function (pdf) and the cumulative density function (cdf) of the we distribution are given respectively

$$g(x) = \left\{ \theta(\alpha + 1) / \alpha \right\} \lambda e^{-\lambda x} \left[1 - e^{-\lambda \alpha x} \right], \quad x > 0, \alpha > 0, \lambda > 0 \quad (1)$$

$$G(x) = \left[(\alpha + 1) / \alpha \right] \left[1 - e^{-\lambda x} - \left[1 / (1 + \alpha) \right] \left\{ \left(1 - e^{-\lambda x(1 + \alpha)} \right) \right\} \right]; \quad x > 0, \alpha > 0, \lambda > 0 \quad (2)$$

Where;

- α is the shape parameter
- λ is the scale parameter
- The aim of this article

THE EXPONENTIATED WEIGHTED EXPONENTIAL DISTRIBUTION

The Exponentiated family of distribution is derived by raising the cdf of an arbitrary parent distribution by a shape parameter say; $\theta > 0$. Its pdf is given by;

$$f(x) = \theta g(x) G(X)^{\theta - 1} \quad (3)$$

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Its corresponding pdf is given by;

$$f(x) = g(x)G(X)^{\theta-1} \tag{4}$$

With this understanding, we insert Equations (1) and (2) into Equation (3) to give the pdf of the Exp We as;

$$f(x) = \theta \left([\alpha + 1/\alpha] \lambda e^{-\lambda x} [1 - e^{-\lambda \alpha x}] \right) \left\{ (\alpha + 1/\alpha) \left[1 - e^{-\lambda x} - (1/(1 + \alpha)) (1 - e^{-\lambda x(1+\alpha)}) \right] \right\}^{\theta-1} \tag{5}$$

The corresponding cdf of the Exp. We distribution is given by;

$$F(X) = \left\{ [\alpha + 1/\alpha] \left[1 - e^{-\lambda x} - (1/(1 + \alpha)) (1 - e^{-\lambda x(1+\alpha)}) \right] \right\} \tag{6}$$

For $x > 0, \alpha > 0, \lambda > 0, \theta > 0$

Where;

α and θ are the shape parameters

λ is the scale parameter

RELIABILITY ANALYSIS

The reliability (or survival) function and the hazard function (or failure rate) of the proposed Exp. We distribution.

The survival function is mathematically represented by;

$$S(x) = 1 - F(x)$$

Therefore, the survival function of the Exp we distribution is given by;

$$S(X) = 1 - \left\{ \alpha + 1/\alpha \left[1 - e^{-\lambda x} - 1/1 + \alpha (1 - e^{-\lambda x}) \right] \right\} \theta \tag{7}$$

Survival Function of the Exponentiated Weighted Exponential Distribution

The probability that a system having age 'x' units of time will survive up to 'x + t' units of time for $X > 0, \alpha > 0, \lambda > 0, \theta > 0$.

$$S(t|x) = S(x+t)/S(x)$$

$$S(t|x) = \frac{1 - \left\{ (\alpha + 1/\alpha) \left[1 - e^{-\lambda(x+t)} - (1/(1 + \alpha)) \left[1 - e^{-\lambda(x+t)(1+\alpha)} \right] \right] \right\} \theta}{1 - \left\{ (\alpha + 1/\alpha) \left[1 - e^{-\lambda x} - 1/(1 + \alpha) \left[1 - e^{-\lambda(x+t)(1+\alpha)} \right] \right] \right\} \theta} \tag{8}$$

The hazard function is mathematically given by

$$H(x) = f(x)/1 - F(x)$$

Therefore, the expression for the hazard function of the Exp. We distribution is given by;

$$h(x) = \frac{\theta [\alpha + 1/\alpha] \lambda e^{-\lambda x} [1 - e^{-\lambda \alpha x}] \left\{ \alpha + 1/\alpha \left[1 - e^{-\lambda x} - 1/1 + \alpha (1 - e^{-\lambda x(1+\alpha)}) \right] \right\}^{\theta-1}}{1 - \left\{ \alpha + 1/\alpha \left[1 - e^{-\lambda x} - 1/1 + \alpha (1 - e^{-\lambda x(1+\alpha)}) \right] \right\} \theta} \tag{9}$$

APPLICATION

All women were eumenorrhic according to self-reports of regular menstrual cycles and normal menses. No subject with a history of menstrual irregularity or other gynecological problems was admitted to the study. All women were nonsmokers, medication free, and had not been on oral contraceptives for at least 6 mo. Before participation, each subject had a physical examination and an electrocardiogram. All subjects had normal results from laboratory screening tests including routine chemistries, thyroid function tests, urinalysis, and complete blood counts. Subjects were asked to refrain from caffeine and alcohol for 16 h before studies, from strenuous activity for 24 h, and from food for at least 6 h before testing. Resting hemoglobin (Hb) and hematocrit (Hct) determinations before all exercise tests were within the normal range for each subject. Electrocardiograms and heart rate were monitored continuously throughout exercise testing.

Study 1: High-Intensity Exercise

Nine healthy female subjects participated in this study. For exercise test sessions, each subject reported to the laboratory 1 h before the initiation of exercise, weighed herself, and drank 5 ml water/kg body weight to ensure uniform hydration. An indwelling catheter was then placed in an antecubital vein at least 20 min before the first basal blood sample. Samples were collected before exercise (220 and 210 min), midway through exercise (10 min), and immediately after exercise (20 min); subjects were kept in a standing position. Four postexercise blood samples were taken (30, 40, 60, and 80 min after the start of exercise) with the subject in a semirecumbent position. The total treadmill exercise lasted 20 min followed by a 5-min cool-down period.

Study 2: Moderate-Intensity Exercise

Eight healthy female subjects participated in this study. The exercise test session has already been described for *study 1*. In contrast with *study 1*, subjects remained standing for all recovery blood draws in *study 2*. The treadmill exercise test employed here was the same as described in *study 1*, except for the last 5 of the 20 min, at which time subjects in *study 2* continued exercising at 70% $\dot{V}O_{2max}$ instead of achieving 90% $\dot{V}O_{2max}$. A 5-min cool-down period (3 miles/h, 2% grade) again followed each test. Respiratory exchange ratios (RER) were continuously calculated from the $\dot{V}O_2$ and CO_2 production data collected during the exercise test. The patterns of ACTH responses to exercise in the AM and PM were similar, with peak concentrations achieved at the end of high-intensity exercise (*time 20*). The net integrated ACTH responses in the AM and PM were also similar. Although mean basal plasma cortisol levels (357.5 \pm 24.3 vs. 206.4 \pm 19.3 nM, AM vs. PM, respectively; $P < 0.001$) were significantly higher in the AM compared with the PM, analysis of AUC revealed no significant differences in the net integrated response in the AM and PM. Similar patterns of response were noted for both AVP and GH. Peak levels were attained at the end of high-intensity exercise and returned to resting levels 20 min after exercise. The changes in AVP and GH over time in the AM and PM were not significantly different. Additionally, analysis of AUC for AVP and GH revealed no significant differences in the magnitude of response across test sessions. Catecholamine responses to exercise in the AM and PM are shown in Fig. Relative to basal levels, the concentration of NE and Epi increased significantly during exercise but returned to resting levels 20 min after terminating exercise. The pattern of change over time in the AM and PM was also similar for both catecholamines.

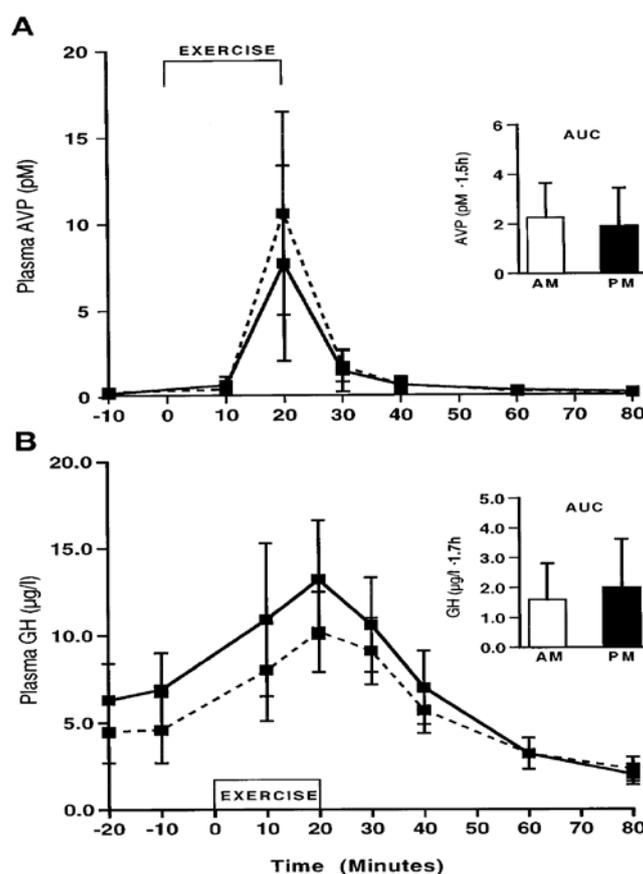


Figure 1: Plasma arginine vasopressin (AVP; A) and growth hormone (GH; B) responses to exercise at 90% $\dot{V}O_{2max}$ in AM (dashed line) and PM (solid line).

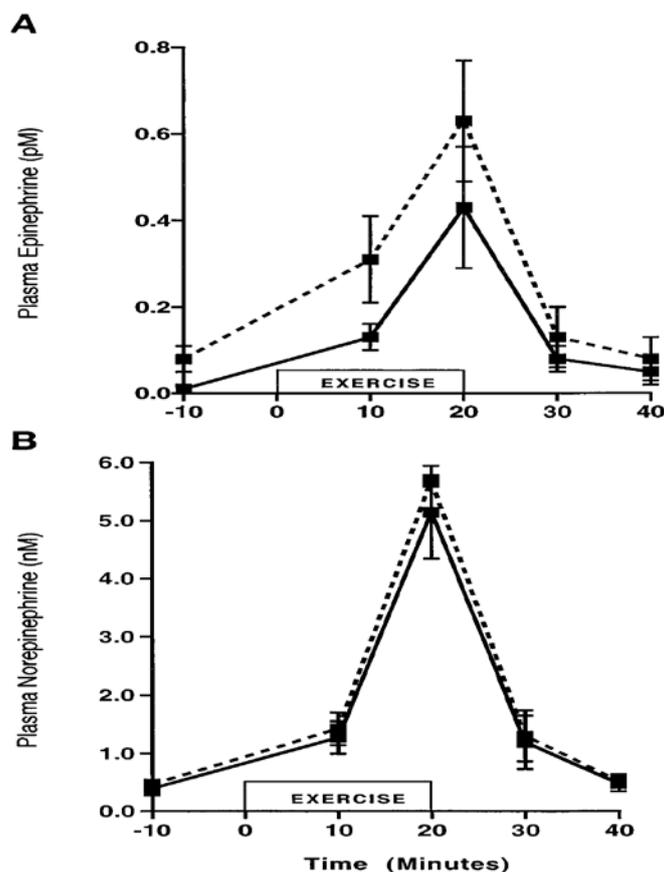


Figure 2: Plasma epinephrine (A) and norepinephrine (B) responses to exercise at 90% $\dot{V}O_{2max}$ in AM (dashed line) and PM (solid line).

Using AVP stimulation, Salata *et al.* reported higher net integrated ACTH responses in the AM compared with the PM. This diurnal response in ACTH is opposite of that for oCRH administration, for which higher responses are reported in the PM when levels of cortisol are low. Salata *et al.* (29) speculated that perhaps an AVP stimulus might show greater potentiation of ACTH release in the AM when CRH levels are at a maximum compared with PM when CRH levels are presumably low. This possibility is supported by the finding that AVP by itself is a weak secretagogue for ACTH but markedly potentiates the effects of CRH. Consistent with this model, oCRH stimulation in the AM, when endogenous levels are high, should have little appreciable effect on ACTH release, thus accounting for the previously reported finding of reduced responses to oCRH in the AM. Because the diurnal ACTH responses reported in the present study are more similar to those obtained by Salata *et al.* (29) for AVP and inconsistent with those obtained with oCRH, we speculate that AVP plays an important role in the acute pituitary-adrenal responses to exercise stress. Indeed, we have shown that short-term, high-intensity exercise is a potent stimulus for AVP release. Consistent with the model proposed by Salata *et al.*, exercise performed in the AM, when endogenous CRH levels are high, may produce a relatively greater stimulation of ACTH release, which is partially blunted by higher ambient cortisol levels. Conversely, the same exercise stimulus in the PM, when CRH levels are low, may produce less ACTH secretion, which is partially offset by reduced glucocorticoid negative feedback. Our finding of nearly identical ACTH and cortisol responses to intense exercise in the context of differing cortisol levels suggests that these responses are determined, in part, by the degree of hypothalamic CRH drive in combination with the degree of glucocorticoid negative feedback at the time of the stress. The author E. A. GALLIVEN, 1 A. SINGH, 1 D. MICHELSON, 3 S. BINA, 2 P. W. GOLD, 3 AND P. A. DEUSTERI measured basal and exercise-induced levels of plasma AVP in the AM and PM. Consistent with Altemus *et al.* (1), we found intense exercise to be a potent stimulus for AVP release in women, which resulted in a .35-fold increase over preexercise values. However, the patterns and magnitudes of change in AVP were essentially the same at both times of the day.

CONCLUSION

Using the mathematical model we found the probability density function and the cumulative density function of the WE distribution for the Plasma arginine vasopressin and growth hormone responses to exercise at 90% $\dot{V}O_{2max}$ in AM and PM

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