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A Bivariate Exponential distribution model for growth hormone response to repeated maximal cycle ergometer exercise at different pedaling rates.

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ABSTRACT
In this paper, we introduce the bivariate exponential distribution model approach to probability modeling. Our results leads to exponential model characterization of many well known life time model to the development of application part. Here we investigate the growth hormone (GH) response to repeated bout of maximal sprint cycling and the effect of cycling at different pedaling rates on post exercise serum GH concentrations by exponential model relative to the bivariate exponential distribution. The application part is well fitted with the mathematical model and conclusion is compared with the medical report.

Keywords: Cortisol, Generalized Exponential distribution, GH, Insulin.
Mathematical subject classification: 60Gxx, 62Hxx, 62Pxx.

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I. INTRODUCTION

Growth hormone (GH) is released in a pulsatile manner from the anterior pituitary gland. The release of GH is believed to be regulated by the antagonistic effects of the hypothalamic neuropeptides GH-releasing hormone (GHRH) and somatostatin, with modulation by feed-back mechanisms (6).A number of studies have shown exercise to stimulate increases in blood GH concentrations, although only a few have considered high-intensity or sprint exercise (9, 16). A single 30-s treadmill sprint produces a near-maximal GH response when compared with the results from pharmacological intervention studies, with GH levels remaining elevated for at least 60 min post exercise (16). However, the mechanisms controlling the magnitude of the GH response to exercise are not well understood. Intensity and duration of exercise have been suggested to influence the GH response (18), and the possible roles of blood lactate (4, 13), blood pH (9), and O_2 demand and availability (20) have also been considered. Repeated 30-min bouts of sub maximal exercise have been shown to elicit an augmented GH response (12). This finding is in contrast to studies showing that repeated administration of pharmacological stimuli results in an attenuated GH response in rats (14, 15) and humans (8). In addition, Cappon et al. (2) found that, in humans, three heavy 10-min exercise bouts resulted in progressive attenuation of the GH response to exercise. At present, therefore, the evidence regarding the GH response to repeated bouts of exercise is equivocal.

The changes in muscle metabolites after maximal cycling at fast (140 rpm) and slow (60 rpm) pedaling rates have been shown to be similar despite greater fatigue being evident with pedaling at 140 rpm (11). In addition, Cherry et al. (3) found no difference in the magnitude of the changes in blood or muscle metabolites with sprinting on a friction-loaded cycle ergometer against different applied resistance, although subjects performance appeared to recover more quickly after exercise involving fewer muscle actions.

Although it has recently been shown that plasma cortisol levels are elevated during sub maximal exercise at slow, but not fast, pedaling rates (5), no studies have yet considered the hormone responses to sprint exercise at different pedaling rates. However, if the metabolic response to exercise determines the magnitude of the GH response, it is likely that the GH response to exercise would be

unaffected by the applied resistance. Therefore, the aim of the present study was to test the hypothesis that repeated bouts of maximal sprint cycling result in an attenuation of the GH response to exercise. In addition, the present study was designed to assess the role of the metabolic response to sprinting in the regulation of the GH response by testing the hypothesis that sprint cycling at different pedaling rates results in similar changes in postexercise GH concentrations.

II. MATHEMATICAL MODEL AND ASSUMPTIONS

The random variable X is said to have a Weighted Exponential distribution with the shape and scale parameters $\alpha > 0$ and $\lambda > 0$ respectively, if the PDF of X is

$$f_X(x:\alpha,\lambda) = \frac{\alpha+1}{\alpha}\lambda e^{-\lambda x}\left(1-e^{-\alpha\lambda x}\right);\ x>0 \text{-----} \text{----------}(1)$$

and 0 otherwise. From now on a Weighted Exponential distribution with PDF (1) will be denoted by WE (α,λ).

The PDF of the Weighted Exponential distribution is always unimodel. The CDF and the hazard function (HF) can be expressed in explicit forms. The HF of the WE is always an increasing function, and WE family is a reverse rule of order two families. For different shapes of the PDFs of WE family, the readers are referred to the original work of Gupta and Kundu[10].It may be mentioned that the shapes of the PDFs of the WE distribution are very similar with the shapes of the PDFs of well knownWeibull,Gamma or generalized exponential distribution .Moreover ,in this model plays the role of a scale parameter and α plays the role of a shape parameter.

III. APPLICATIONS

Figure 3.1 shows the mean serum GH response to repeated maximal 30-s cycle ergometer sprints. The first sprint resulted in a distinct GH pulse with highest measured mean concentrations of 40.8 ± 8.2 and 20.8 ± 6.1 mU/1 30 min after the sprint in the fast and slow trails, respectively. Serum GH was still elevated 60 min after the first sprint, whereas there was no GH pulse after the second sprint. Serum GH did not show a trail effect (p=0.08), but there was a sprint effect (p<0.05), a time effect (p<0.05), and a sprint X time interaction (p<0.05). The peak GH response for sprint 1 (mean of individual peaks) during the fast trail was more than twice that during the slow trail (mean peak GH: 37.7 ± 6.0 vs. 17.6 ± 3.7 mU/1for fast vs. slow; p<0.05).

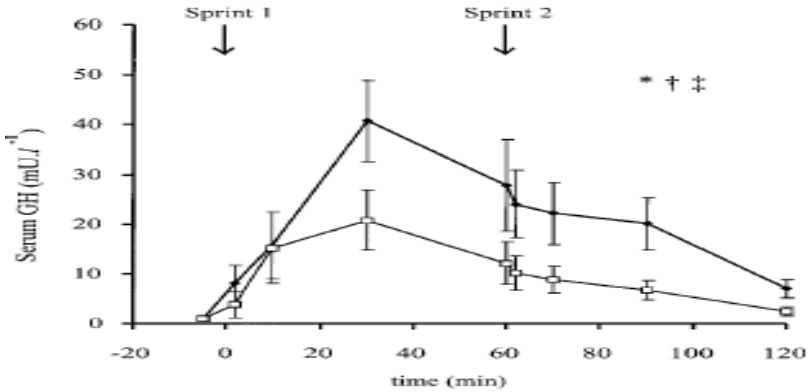


Fig.3.1 Serum growth hormone (GH) concentrations at rest and during 1h of recovery after two 30-s maximal cycle ergometer sprints for the fast () and slow () trails. Values are means ±SE. *Sprint main effect, P<0.05. Time main effect, P<0.01. Trail main effect, P=0.08 (not significant). Sprint x time interaction effect, P<0.01.

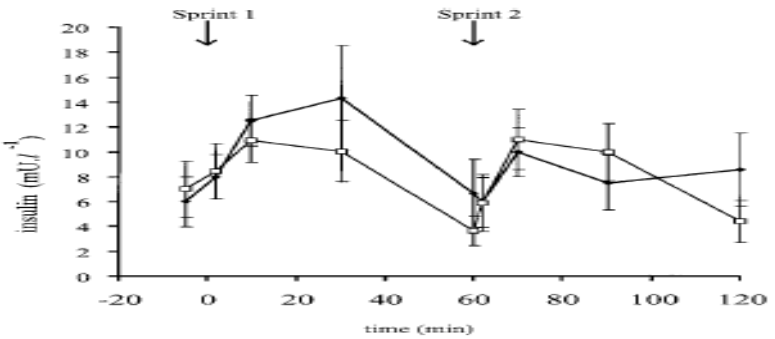


Fig 3.2 Serum insulin concentrations at rest and during 1 h of recovery after two 30-s maximal cycle ergo meter sprints for the fast and slow trails.

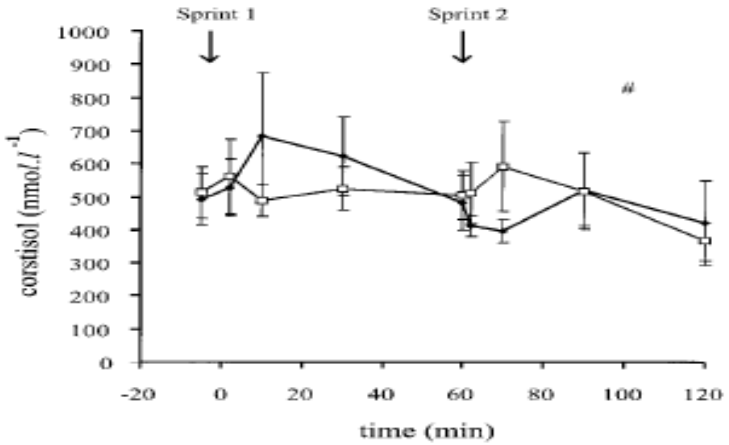


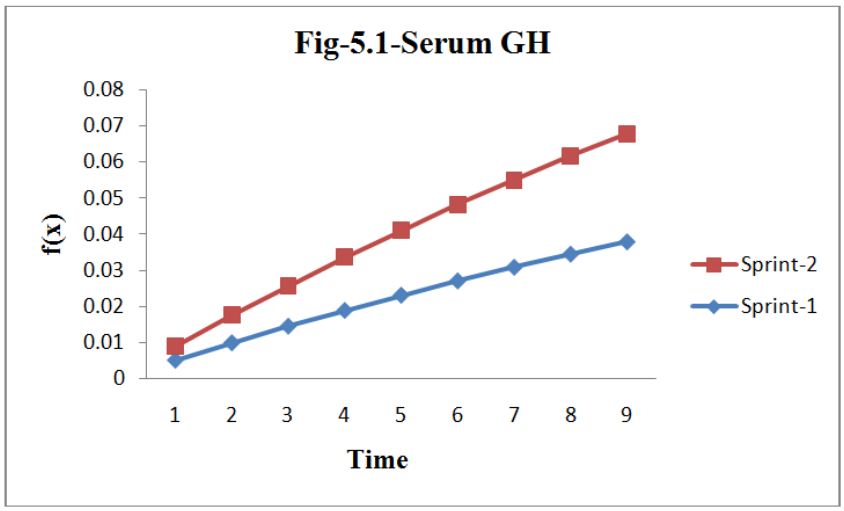
Fig.3.3 Serum cortisol concentrations at rest and during 1 h of recovery after two 30-s maximal cycle ergometer sprints for the fast and slow trails. Values are means ± SE. Sprint main effect, P<0.05.

Mean integrated GH concentrations (AUC) for the 1-h period after each sprint demonstrated a difference between sprints (mean GH AUC: 1,315 ± 243 vs. 729 ± 146 $min.mU^{-1}.1^{-1}$ for sprint 1 vs. sprint 2; p<0.01). There was also a trend for a lower GH AUC in the slow trail, with 9 of the 10 subjects following this pattern (mean GH AUC: 1,381 ± 231 vs. 663 ± 162 $min.mU^{-1}.1^{-1}$ for fast vs. slow; p=0.06). when the GH AUC for the 60 min of recovery after sprint 1 in the fast and slow trails was compared, there was also a trend for a smaller GH AUC in the slow trail (mean GH AUC after sprint 1: 1,697 ± 367 vs. 933 ± 306 $min.mU^{-1}.1^{-1}$ for fast vs. slow; p=0.05). The insulin (Fig.3.2) and cortisol (Fig.3.3) responses were similar in the two trails, although the cortisol response to the first sprint was different from that of the second (p<0.05).

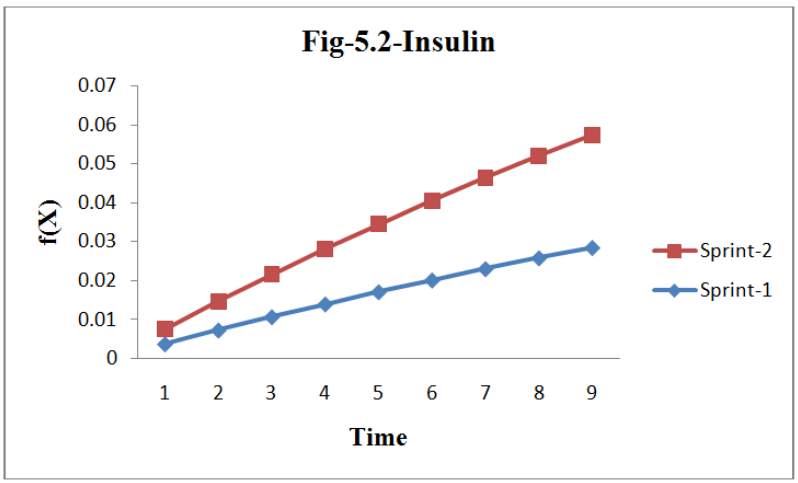
IV. DISCUSSION

GH can directly inhibit its own release, possibly at the pituitary gland (17). Because, in the present study, GH was still elevated at the start of the second sprint, GH auto inhibition may be responsible for preventing a GH response to the second sprint. Alternatively, it has been suggested that this auto-negative feedback occurs at the level of the hypothalamus, mediated by an increase in somatostatin release and/or a decrease in the release of GHRH. Lanzi and Tannenbaum (14) demonstrated that the immunoneutralization of somatostatin prevented the attenuation of spontaneous GH release after GH pretreatment in rats, thus obtaining strong support for role for somatostatin in GH auto regulation. In a further study, Lanzi and Tannenbaum (15) also demonstrated a role for somatostatin in the attenuation of exogenous GHRH-induced GH release. The understanding that GH-receptor mRNA is colocalized in somatostatin neurons in the rat hypothalamus (1) further supports these findings. Peripheral measurements of GHRH and somatostatin would probably not reflect hypothalamic secretion, but studies employing direct hypo physical-portal sampling in nonhuman species suggest that hypothalamic discharges of GHRH regulate GH pulses (7), whereas somatostatin may modulate the action of GHRH (19).In the present study, a GH response to the second bout of exercise was identified in two of the subjects. The GH response to the first sprint in both of these subjects was characterized by moderate increases in serum GH concentrations that had returned to near preexercise levels within 60 min of recovery. GH concentrations in two of the other subjects had, similarly, returned to near preexercise values 60 min after the first sprint, but the increases in serum GH concentrations in these individuals in response to both the first and second sprints were small. The other six subjects displayed moderate to large increases in serum GH concentrations after the first sprint, and GH concentrations were still elevated above preexercises levels after 60 min of recovery. In these six individuals, a second sprint completed 60 min after the first did not result in a further increase in serum GH concentrations. These results provide further evidence that elevated serum GH concentrations might result in the attenuation of the GH response to repeated bouts of exercise.

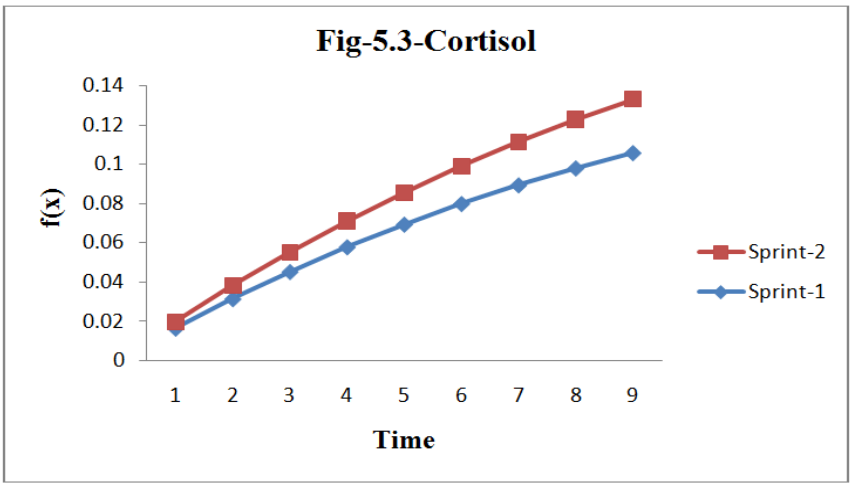
V. MATHEMATICAL RESULTS



Probability densities function for Serum GH concentrations for the fast and slow trials, corresponding to figure 5.1.



Probability densities function for Serum Insulin concentrations for the fast and slow trials, corresponding to figure 5.2.



Probability densities function for Serum GH concentrations for the fast and slow trials, corresponding to figure 5.3.

VI. CONCLUSION

Here we investigate the growth hormone (GH), Insulin, cortisol response to repeated bout of maximal sprint cycling and the effect of cycling at different pedaling rates on post exercise serum GH concentrations by exponential model relative to the bivariate exponential distribution. The present study also reveals that cycle ergo meter sprinting at faster pedaling rates elicits a greater GH response than pedaling at slower pedaling rates, despite similar blood lactate and pH responses. However, the plasma ammonia and serum GH responses followed similar trends after exercise at different pedaling rates, and it is possible that circulating ammonia, in addition to a proprioceptive mechanism, contributes to the regulation of GH release during sprint exercise. The application part is well fitted with the mathematical model and conclusion is compared with the medical report.

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