TRAINING ALTERATIONS IN ELITE CYCLISTS MAY CAUSE TRANSIENT CHANGES IN GLOMERULAR FILTRATION RATE

Chad D. Touchberry, Mark Ernsting, Greg Haff and J. Lon Kilgore ✉

Department of Kinesiology, College of Health Sciences and Human Services, Midwestern State University, USA.

ABSTRACT

Training alterations in elite cyclists may cause transient changes in glomerular filtration rate. To these authors' knowledge, no biochemical investigation of chronic renal function in athletes during a training cycle exists. The purpose of the present archival study was to evaluate the effects of training on homeostatic renal function, evaluated predicted glomerular filtration rate (GFR). Eight male competitive college cyclists (mean ± SD: age: 22.2 ± 3.8 yrs, height: 1.80 ± 0.06 m, mass: 76.6 ± 7.9 kg, and body fat was 7 ± 2%) volunteered to undergo 12 weeks of training, and were required to undergo blood sampling at timed intervals to calculate GFR. Homeostatic GFR was altered significantly during various points in the investigation. Volume and average cycling speed were found to have moderate correlations to alterations in GFR. In addition to these findings, 7 of the 8 subjects had GFR's below normal physiological ranges during some point in the experiment. The duration, intensity, and volume of cycling appear to have an influence on renal function. This influence is pronounced during periods when the athletes are unaccustomed to the training load.

KEY WORDS: Cycling, glomerular filtration rate, renal, kidney.

INTRODUCTION

The kidneys constitute less than 0.5% of the body mass, yet they receive almost one fourth (22%) of the cardiac output at rest (Johnson and Byrne, 1998; Garrett and Kirkendall, 2000; Guyton and Hall, 2000). During exercise, renal blood flow to the kidney is greatly reduced, as the muscles demand for oxygen and blood flow is increased (Johnson and Byrne, 1998; Mueller et al., 1998; Garrett and Kirkendall, 2000). Since the kidneys do not consume large amounts of oxygen, demand only a small portion of the cardiac output, and do not contribute to performance during exercise, there has been very little investigation into the physiology of the kidney during and after exercise.

Approximately a dozen investigations have attempted to explore renal function with regard to physical activity in a healthy human population (Knochel et al., 1974; Melamed et al., 1982; Irving et al., 1986; 1989; 1990a; 1990b; Poortmans, 1967; 1984; 1988; 1995; Poortmans et al., 1988; 1989; 1990; 1991; 1996; 1997a; 1997b; 1998; 2001; Poortmans and Vancalck, 1978; Poortmans and Haralambie, 1979; Poortmans and Henrist, 1989; Poortmans and Labilloy, 1988; Taverner et al., 1991; Poortmans and Vanderstraeten, 1994). A thorough search of the literature revealed that no studies have investigated the effects of chronic exercise on long-term homeostatic markers of renal function. There have been a few studies that looked at renal function and glomerular filtration rate (GFR) in the acute stage (1-72 hours) following exercise (Poortmans, 1984; 1985; 1995; Irving et al., 1986; 1990a; Poortmans et al., 1988; 1990; 1996; 1997b; Poortmans and Vanderstraeten, 1994).

Very few studies have calculated GFR as an indicator of renal function in the exercising human (Knochel et al., 1974; Melamed et al., 1982; Poortmans et al., 1990; 1997b; Irving et al., 1990a; Taverner et al., 1991; Averbukh et al., 1992; Poortmans et al., 1996; Neumayr et al., 2003). Of those that have calculated the GFR, two shortcomings in the research can be noted. One, renal function was only examined in an acute phase, and two, conflicting results were determined.

Poortmans and colleagues (Poortmans et al., 1996; 1997b; Poortmans and Vancalck, 1978) found that running at different intensities and durations resulted in short-term renal dysfunction, including major decreases in GFR post exercise. The work of Poortmans and colleagues suggests that training intensity is the key factor in depressing GFR. This decrease may be a reflection of the decreased renal blood flow associated with high intensity exercise.

Knochel et al. (1974) reported a significant increase in GFR in a group of soldiers who were subjected to an increase in physical training. Irving et al. (1990a) suggested that running and ultramarathon running caused an increase in GFR post-exercise, as reflected by an increase in creatinine clearance. The results of these studies suggest that low intensity, long duration exercise causes an increase in GFR following exercise.

Conversely, Averbukh et al. (1992) did not support this trend in healthy mice. They noted that GFR did not alter significantly after training. However, mice with varying degrees of renal mass reduction did show an increase in GFR. This suggests that the acute renal response to endurance exercise may be different for healthy individuals, as opposed to those with known disease.

Several other authors have found that long distance endurance training may reduce GFR. Melamed et al. (1982) were one of the first to find that repeated physical exercise decreased GFR in a human population. Poortmans et al. (1996) demonstrated a 40% decline in renal function during long distance runs. In 1997, Poortmans and colleagues also demonstrated a 17% decline in GFR in an exercising control group when comparing the renal responses of healthy subjects versus those who have had heart and/or kidney transplantation (Poortmans et al., 1997). Gleadhill et al. (2000) found that GFR fell by over 40% in elite Olympic equestrian horses, even during mild exercise. Taverner et al. (1991) found that there was a significant decline in GFR in patients with moderate impairment of renal function. There is currently conflicting information on what effects endurance exercise has on renal function. None of these studies looked at the alterations in GFR over a chronic period of time (weeks or months) or during a

competitive training cycle in an athletic population. Therefore, the purpose of this investigation was to explore the effects of a long-term training program on chronic homeostatic renal function in an athletic population.

Table 1. Training program. Data are means (±SD).

Week	Training Hours (h)	Training Kilometers (km)	Training Kilometers per hour (km·h⁻¹)
1	11.81 (3.38)	359.81 (96.63)	32.30 (14.35)
2	12.09 (4.39)	363.69 (146.55)	31.72 (15.00)
3	7.28 (4.71)	203.37 (137.00)	26.89 (11.42)
4	12.84 (5.35)	350.66 (185.69)	25.95 (7.95)
5	16.12 (4.32)	454.97 (168.17)	25.92 (7.95)
7	14.06 (3.03)	408.51 (91.17)	29.73 (8.84)
9	9.13 (4.52)	273.73 (140.55)	26.07 (9.95)
10	12.71 (5.43)	373.21 (171.63)	44.24 (50.91)
11	9.40 (4.55)	306.66 (184.96)	28.81 (9.34)
12	12.97 (5.78)	408.63 (177.89)	25.72 (11.02)

* Weeks 6 and 8 were race weeks.

METHODS

An evaluation of archival blood samples was conducted. Subjects consisted of 8 collegiate cyclists with similar training histories. Subjects completed a health history questionnaire, informed consent, and were screened according to American College of Sports Medicine (ACSM) guidelines prior to inclusion in this study (Franklin et al., 2000). The testing methodology and protocol received approval from the Human Subject Research Committee at Midwestern State University (# 03091101).

Experimental Design
A repeated measures design was employed with each subject's pre-season recovery measurements serving as matched control. All subjects had trained and competed in collegiate level cycling for at least one year prior to participation in this study. The subjects participated in a 12-week training cycle, and were instructed to complete the assigned training program as written. Table 1 presents the training program means. Performance measures were recorded prior to and following the completion of the study. This included both pre- and post-test

maximal cycle ergometry to assess VO$_{2max}$, and Wingate tests to assess anaerobic power. VO$_{2max}$ testing was conducted using a Monark cycle ergometer and a ParvoMedics TrueMax 2400 metabolic cart. Workloads were manually adjusted and began at 175W at 70 rpm. The workload was increased every two minutes by 50W and the test ended when the subject could no longer maintain 70 rpm or reached volitional fatigue. Thirty second duration Wingate testing was conducted on a Monark Wingate cycle ergometer with a 7.5% body mass resistance. Blood draws were acquired during the course of this investigation at weeks 1, 3, 5, 7, 11, and 12 to determine concentrations of serum creatinine, serum urea nitrogen, and serum albumin.

Blood Chemistry
Venipunctures were taken on the same hour and day for the duration of the study. Blood samples were obtained after an 8-hour fast and followed one day of recovery and reduced training. All samples were obtained on Friday mornings between 7:30 – 8:30 a.m. A standard 10 mL serum tube (Vacutainer SST, Becton-Dickinson, Franklin Lakes, NJ) of blood was acquired from the antecubital vein. Each subject and sample was assigned an identification code and the code key remained confidential until all analyses were completed. The samples were separated by centrifuge at 2750 g·s^{-1}, 4° C for 30 minutes (IEC Centra MP4, Needham Heights, MA) into packed cell and serum components. The serum was extracted using disposable transfer pipettes, placed in labeled microtubes, and stored at -84° C until analysis of the serum was performed. Chemistry assays were performed on known markers of renal function to determine the glomerular filtration rate. Samples were analyzed colormertically for serum urea nitrogen (SUN) (Cat. No. 47381), albumin (Cat. No. 3034607), and creatinine (Cat. No. 47003), using a COBAS Mira^{TM} analyzer (Hoffmann-La Roche, Ltd., Basel Switzerland).

Glomerular filtration rate was then calculated by using the formula proposed by Levey and colleagues (Levey, 1990; Levey et al., 1993; 1999). The formula based on age, gender, serum creatinine concentration (Scr), serum urea nitrogen (SUN), serum albumin (Alb), and whether or not the subject is African American (Levey, 1990; Levey et al., 1993; 1999). The equation is expressed in mL·min^{-1}·1.73 m^{-2} and is as follows (Levey, 1990; Levey et al., 1993; 1999):

GFR = 170•(Scr$^{-0.999}$)•(Age$^{-0.176}$)•(0.762 if subject is female)•(1.180 if subject is black)•(SUN$^{0.170}$)•(Alb$^{0.318}$)

The validity of this equation has been well documented ($R^2 = 90.3\%$) (Levey et al., 1999). By contrast, creatinine clearance measured by 24-hour urine collections or predicted by the Cockcroft-Gault equation overestimated GFR by 19% and 16%, respectively (Levey, 1990). Even after adjustment for the overestimation of GFR by creatinine clearance, the correlation of a direct measure of creatinine clearance and estimated creatinine clearance using the Cockcroft-Gault equation was lower ($R^2 = 86.6\%$ and 84.2%, respectively)(Levey, 1990). Several other studies have attested to the accuracy of the Levey - MDRD GFR prediction equation and some researchers have spoken out against using creatinine clearance (Shemesh et al., 1985; Levey et al., 1988; Lacour, 1992; Broekroelofs et al., 2000; Garrett and Kirkendall, 2000; Manjunath et al., 2001; Coresh et al., 2002; Lin et al., 2003; Rodrigo et al., 2003;). Furthermore, the formula chosen meets the recommendations made by the National Kidney Foundation concerning the assessment of kidney function (Manjunath et al., 2001).

Statistics
All serum chemistry results are reported as mean $\pm$ SD, and were analyzed with a repeated measures analysis of variance (ANOVA) using SPSS, version 12.1 (Chicago, Illinois, USA). The null hypothesis was rejected when $p < 0.05$. Paired comparisons were made using a Holms-Bonferroni adjustment in order to control for Type I error (Holm, 1979; Tarone, 1990). Spearmans r correlations were performed between selected variables using StatPlus, version 2.5 (La Jolla, California, USA). Correlation coefficients were computed among training volume variables and training intensity, and then compared to the GFR. A p-value of less than 0.05 was considered significant.

RESULTS

The mean age of the subjects was 22.2 ± 3.8 yrs, (18-29 years). The mean height was 1.80 ± 0.06 m. Mean body mass was not significantly different over the course of the experiment with an entry mass of 76.6 ± 7.9 kg and a final mass of 73.9 ± 9.6 kg. Mean body fat at entry was 7 ± 2%.

Data obtained from the testing of performance throughout the study were as follows: A non-significant increase from the pre-test value of VO$_{2max}$ 66.7 ± 4.4 ml·kg^{-1}·min^{-1} to a post-test value of 68.6 ± 5.6 ml·kg^{-1}·min^{-1} was noted. Additionally, a non-significant increase in pre-test Wingate average power of 867.2 ± 118.7 W to a post-test average power of 875.9 ± 175.3 W was determined. Table 2 presents individual performance testing data.

Table 2. Performance testing data.

Subject #	Pre VO$_2$ $_{max}$ (ml·kg^{-1}·min^{-1})	Post VO$_2$ $_{max}$ (ml·kg^{-1}·min^{-1})	Pre Wingate (W)	Post Wingate (W)
1	73.0	76.4	810.5	832.0
2	71.2	73.1	796.5	843.3
3	64.5	63.3	860.8	847.7
4	60.6	63.3	860.8	847.7
5	64.6	72.5	1090.1	1027.7
6	71.2	72.8	793.2	606.7
7	64.0	64.7	994.0	1148.4
8	64.7	62.8	729.0	705.4
Mean	66.7	68.6	867.2	875.9
SD	4.4	5.6	118.7	175.3

Blood Chemistry
Over the 12-week training period there were statistically significant changes noted in the serum chemistry markers for renal function. Statistically significant changes were also noted between the measures of GFR, [$F_{(5,3)}$ = 22.53, p = 0.014, partial η^2 = 0.97]. Post hoc T-tests revealed a significant decline in GFR when week 3 was compared to week 5, [$T_{(7)}$ = 5.348, p = 0.001, partial η^2 = 0.82] and when week 5 was compared to week 7 [$T_{(7)}$ = 4.219, p = 0.004, partial η^2 = 0.74]. Changes in GFR by sampling week are presented in Table 3.

Normal values, standard deviations, and means for the chemistry and equation results with laboratory norms for the 12-week cycle are presented in Table 4. Significant differences for plasma albumin were observed [$F_{(5,3)}$ = 13.434 p = 0.029, partial η^2 = 0.96]. Follow up T-tests revealed significance when comparing week 3 to baseline [$T_{(7)}$ = -5.198, p = 0.001, partial η^2 = 0.79]. No significant difference was found for creatinine (p < 0.083) and SUN (p = 0.119) over the course of this investigation, indicating that GFR remained at levels adequate to maintain normal renal homeostastis.

Training volume, when measured in terms of hours, showed a large correlation (r = 0.69, R^2 = 0.48, p = 0.058), however, it was not significant.

Finally, the effect of training intensity (Km·h^{-1}) was found to have a very large, statistically significant negative correlation to GFR (r = -0.77, R^2 = 0.58, p = 0.027).

DISCUSSION

The purpose of this investigation was to determine if manipulations in training volume and intensity altered renal function as measured by GFR. In this investigation, markers of renal function commonly used in clinically evaluating renal function (Levey, 1990; 1993; 1999; Manjunath et al., 2001) showed variations from normal serum levels, specifically during the onset of training (Table 4). The mean GFR during training was outside of the clinical norms on two instances. During weeks 7 and 11, GFR dropped below normal physiological ranges for healthy subjects, reaching 97.24 ± 11.22 mL·min^{-1}·1.73 m^{-2} and 101.21 ± 16.48 mL·min^{-1}·1.73 m^{-2} respectively. Close inspection of the data reveals that at one time or another, 7 of the 8 subjects had GFR's that were depressed below clinical norms. However, these changes in filtration rate cannot totally be explained by the training variables alone (Figures 1, 2, and 3).

Table 3. Individual glomerular filtration rates.

Subject	Week 1	Week 3	Week 5	Week 7	Week 11	Week 12
1	124.85	138.92	110.33	107.92	117.09	145.69
2	130.48	119.76	102.93	92.90	103.28	104.30
3	133.71	117.28	103.46	96.32	88.00	107.69
4	127.82	122.78	102.80	90.82	117.51	105.99
5	81.09	98.49	84.61	83.41	80.14	88.64
6	135.95	124.61	120.76	104.90	108.01	127.10
7	110.45	97.88	90.26	86.15	79.27	110.93
8	138.21	115.72	106.33	100.83	97.74	100.17
Mean	128.82	120.20	105.76	97.24	101.21	111.24
SD	29.28	16.15	15.07	11.22	16.48	22.69

All data is presented in ml·min^{-1} 1.73 m^{-2}.
* Clinical norms fall between 105 – 132 ml·min^{-1} 1.73 m^{-2}. (Guyton and Hall, 1996).

Table 4. Chemistry means with laboratory norms.

Chemistry	Norms		Week 1	Week 3	Week 5	Week 7	Week 11	Week 12
Albumin	3.8 - 5.2 g·dL^{-1}	Mean	4.49	5.03	4.76	4.74	4.96	4.61
		SD	0.31	0.28	0.27	0.21	0.36	0.15
SUN	7 - 26 mg·dL^{-1}	Mean	16.60	14.89	18.34	19.00	16.34	18.56
		SD	4.34	2.02	3.49	2.95	2.84	3.16
Creatinine	0.6 - 1.5 mg·dL^{-1}	Mean	0.83	0.90	0.98	1.04	0.94	0.96
		SD	0.14	0.07	0.11	0.097	0.13	0.13

Changes in GFR, in some instances, were correlated to changes in the training program. Hours of training (Figure 2) during week one showed a large correlation (r = 0.69), however, it did not reach significance (p > 0.05). Due to the relationship between training volume in kilometers and hours, it is thought that with a larger subject pool the data would have been significant.

Finally, the effect of training intensity (Km·h^{-1}) (Figure 3) was found to have a very large, statistically significant, negative correlation to GFR during week 3 (r = -0.77, R^2 = 0.58, p = 0.027). Due to the timing of these changes, and its correlation to alterations in training volume, the data suggests that homeostatic GFR is moderately related to an increase in the training load at the start of the training cycle. Because we did not perform weekly analyses, it is difficult to say if there is a direct effect of load on GFR. Further analysis of the cumulative effects of volume (Km) on GFR and the accumulating volume did not show an effect on renal function.

Elevations of serum creatinine and urea nitrogen are indicators of protein catabolism, and serve as markers of renal function. Creatinine and SUN deviated very little over the training period and reflect the low catabolic nature of cycling. Previous research in the acute phase of renal function also noted no alterations in creatinine or SUN concentrations, either in renal clearance or from serum (Poortmans and Vancalck, 1978; Irving et al., 1986; Poortmans and Labilloy, 1988; Poortmans et al., 1997). These data suggest that extensive chronic endurance cycling may not alter these markers of renal function. Therefore, these data support the contention that serum creatinine and SUN may not be an effective means for monitoring renal changes when used independently in sports with low rates of catabolism. Further studies on these markers (Creatinine and SUN) are needed to confirm similar findings in cycling and other minimally catabolic, low weight bearing sports such as swimming.
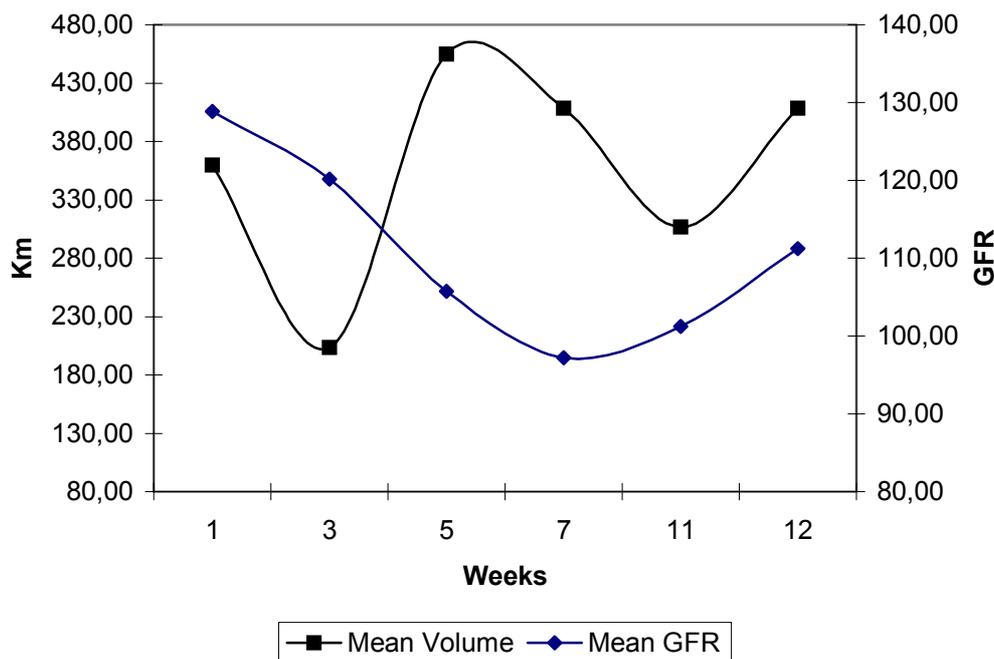
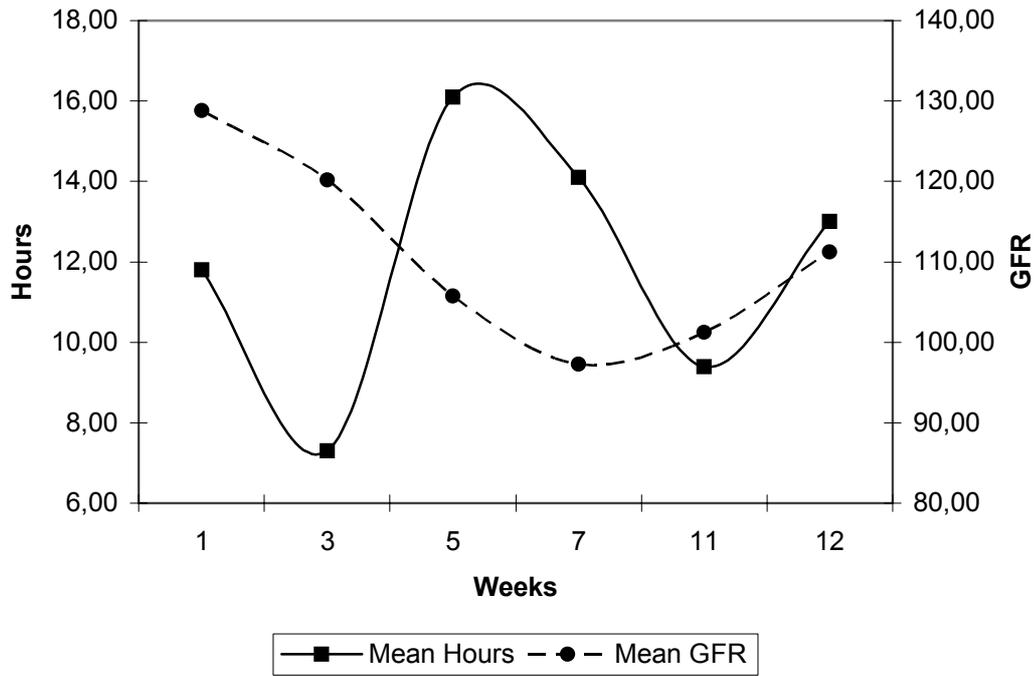**Figure 1.** Glomerular filtration rate and training kilometers. Volume in Km; GFR in mL·min^{-1} 1.73 m^{-2}.

Figure 2. Glomerular filtration rate and training hour. GFR in mL·min^{-1} 1.73 m^{-2}.

Moreover, further study is needed in higher catabolic, weight bearing sports such as weightlifting. Few, if any studies have addressed the issue of catabolism and renal function. During such activities, alterations in homeostatic renal function may be more apparent and lend insight into the chronic homeostatic changes in renal function. Previous research demonstrated an increase in plasma creatinine after an ultra marathon, supporting the contention that increased catabolism increases plasma creatinine concentrations (Irving et al., 1990a). In addition, the increased creatinine clearance was accompanied by an increase in GFR (Irving et al., 1990a). Since GFR was calculated by creatinine clearance, it is difficult to determine if the increase in GFR was due to an alteration in renal function, or due to the catabolism of creatine involved in distance running.

CONCLUSIONS

The present study examined the effects of endurance cycling on known markers of renal function in the homeostatic state. The authors do not know of any studies that have attempted to look at the effects of an exercise modality on basal GFR in human populations, nor in an elite athletic population.
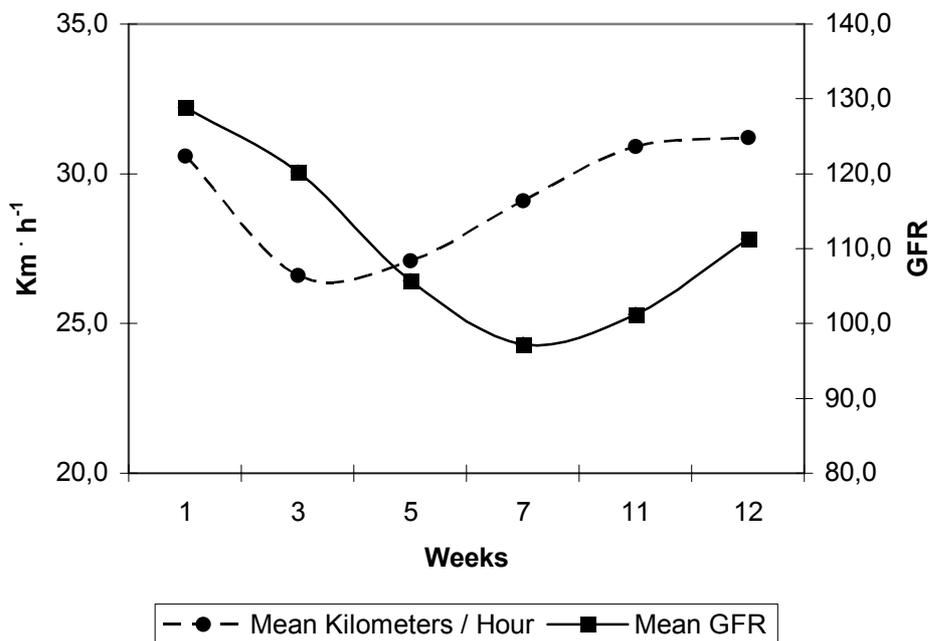

Figure 3. Glomerular filtration rate and intensity. GFR in mL·min^{-1} 1.73 m^{-2}.

Therefore, it was important to complete this investigation in order to provide some insight on the effects of chronic physical exercise on basal renal function, in a healthy population.

The evidence presented in this article may be the only experimental view of exercise on homeostatic renal function in an elite athletic population. Data from this experiment weakens the contention that intense exercise training does not alter renal function in healthy subjects. The results of blood analysis shows that specific chemistries that assess renal function (creatinine, SUN) are not significantly altered in an endurance oriented, low catabolic sport. However, GFR is decreased significantly below baseline, indicating that there may be a non-favorable, or adaptation oriented, change in renal function associated with the onset of high volume exercise in this population. This data is supported by studies of renal function in the acute phase, demonstrating a decrease in GFR both during and soon after physical activity (Gleadhill et al., 2000; Melamed et al., 1982; Neumayr et al., 2003; Poortmans et al., 1996; 1997b).

This study provides evidence that intense endurance exercise training may cause a transient change in homeostatic renal function in a healthy elite athletic population where the extremes of exercise are often seen. It is clear that more data are needed in this area, as well as in other sporting arenas, where the physiological stresses differ. It is possible that the low catabolic nature of cycling differs from other sports such as long-distance running or weightlifting. These other activities and their corresponding rates of catabolism may present a different strain on renal function. This study was exploratory in nature and future research should be directed at determining the mechanisms for such alterations in GFR.

REFERENCES

Averbukh, Z., Marcus, E., Berman, S., Shiloah, E., Horn, T., Weissgarten, J., Golik, A., Cohn, M., Rosenmann, E. and Modai, D. (1992) Effect of exercise training on glomerular filtration rate of mice with various degrees of renal mass reduction. *American Journal of Nephrology* **12,** 174-178.

Broekroelofs, J., Stegeman, C.A., Navis, G.J., de Haan, J., van der Bij, W., de Boer, W.J., de Zeeuw, D. and de Jong, P.E. (2000) Creatinine-based estimation of rate of long term renal function loss in lung transplant recipients. Which method is preferable? *The Journal of Heart and Lung Transplantation* **19,** 256-262.

Coresh, J., Astor, B.C., McQuillan, G., Kusek, J., Greene, T., Van Lente, F. and Levey A.S. (2002) Calibration and random variation of the serum creatinine assay as critical elements of using equations to estimate glomerular filtration rate. *American Journal of Kidney Diseases* **39,** 920-929.

Franklin, B. A., Whaley, M. H. and Howley, E. T. (2000) *ACSM's guidelines for exercise testing and prescription.* 6th edition. Philadelphia: Lippincott Williams & Wilkins.

Garrett, W.E. and Kirkendall, D.T. (2000) *Exercise and sport science.* Philadelphia: Lippincott Williams & Wilkins.

Gleadhill, A., Marlin, D., Harris, P.A. and Michell, A.R. (2000) Reduction of renal function in exercising horses. *Equine Veterinary Journal* **32,** 509-514.

Guyton, A.C. and Hall, J.E. (1996) *Textbook of medical physiology.* 9th edition. Philadelphia: W.B. Saunders.

Guyton, A.C. and Hall, J.E. (2000) *Textbook of medical physiology.* 10th edition. Philadelphia: W.B. Saunders.

Holm, S. (1979) A simple sequentially rejective multiple test procedure. *Scandinavian Journal of Statistics* **6,** 65-70.

Irving, R.A., Noakes, T.D., Burger, S.C., Myburgh, K. H., Querido, D. and van Zyl Smit, R. (1990a). Plasma volume and renal function during and after ultramarathon running. *Medicine and Science in Sports and Exercise* **22,** 581-587.

Irving, R.A., Noakes, T.D., Irving, G.A. and Van Zyl-Smit, R. (1986) The immediate and delayed effects of marathon running on renal function. *The Journal of Urology* **136,** 1176-1180.

Irving, R.A., Noakes, T.D., Raine, R.I. and Van Zyl Smit, R. (1990b) Transient oliguria with renal tubular dysfunction after a 90 km running race. *Medicine and Science in Sports and Exercise* **22,** 756-761.

Irving, R.A., Noakes, T.D. and van Zyl Smit, R. (1989) Metabolic and renal changes in two athletes during a world 24 hour relay record performance. *British Journal of Sports Medicine* **23,** 227-232.

Johnson, L.R. and Byrne, J.H. (1998) *Essential medical physiology.* 2nd edition. Philadelphia: Lippincott-Raven.

Knochel, J.P., Dotin, L.N. and Hamburger, R.J. (1974). Heat stress, exercise, and muscle injury: effects on urate metabolism and renal function. *Annals of Internal Medicine* **81,** 321-328.

Lacour, B. (1992) Creatinine and renal function. *Nephrologie* **13,** 73-81.

Levey, A.S. (1990) Measurement of renal function in chronic renal disease. *Kidney International* **38,** 167-184.

Levey, A.S., Bosch, J.P., Lewis, J.B., Greene, T., Rogers, N. and Roth, D. (1999) A more accurate method to estimate glomerular filtration rate from serum creatinine: a new prediction equation. Modification of Diet in Renal Disease Study Group. *Annals of Internal Medicine* **130,** 461-470.

Levey, A.S., Greene, T., Schluchter, M.D., Cleary, P.A., Teschan, P.E., Lorenz, R.A., Molitch, M.E., Mitch, W.E., Siebert, C. and Hall, P.M. (1993) Glomerular filtration rate measurements in clinical trials. Modification of Diet in Renal Disease Study Group and the Diabetes Control and Complications

Trial Research Group. *Journal of the American Society of Nephrology* **4,** 1159-1171.

Levey, A.S., Perrone, R.D. and Madias, N.E. (1988). Serum creatinine and renal function. *Annual Review of Medicine* **39,** 465-490.

Lin, J., Knight, E.L., Hogan, M.L. and Singh, A.K. (2003) A comparison of prediction equations for estimating glomerular filtration rate in adults without kidney disease. *Journal of The American Society of Nephrology* **14,** 2573-2580.

Manjunath, G., Sarnak, M.J. and Levey, A.S. (2001) Estimating the glomerular filtration rate. Dos and don'ts for assessing kidney function. *Postgraduate Medicine* **110,** 55-62, quiz 11.

Melamed, I., Romem, Y., Keren, G., Epstein, Y. and Dolev, E. (1982) March myoglobinemia: a hazard to renal function. *Archives of Internal Medicine* **142,** 1277-1279.

Mueller, P.J., O'Hagan, K.P., Skogg, K.A., Buckwalter, J. B. and Clifford, P.S. (1998) Renal hemodynamic responses to dynamic exercise in rabbits. *Journal of Applied Physiology* **85,** 1605-1614.

Neumayr, G., Pfister, R., Hoertnagl, H., Mitterbauer, G., Getzner, W., Ulmer, H., Gaenzer, H. and Joannidis, M. (2003) The effect of marathon cycling on renal function. *International Journal of Sports Medicine* **24,** 131-137.

Poortmans, J.R. (1967) Renal clearance of plasma proteins in man during rest and exertion]. *Archives Internationales de Physiologie et de Biochimie* **75,** 346-349.

Poortmans, J.R. (1984) Exercise and renal function. *Sports Medicine* **1,** 125-153.

Poortmans, J.R. (1985) Postexercise proteinuria in humans. Facts and mechanisms. *The Journal of The American Medical Association* **253,** 236-240.

Poortmans, J.R. (1988) Evidence of increased glomerular permeability to proteins during exercise in healthy men. *Contributions to Nephrology* **68,** 136-140.

Poortmans, J.R. (1995) Renal response to exercise in healthy and diseased patients. *Nephrologie* **16,** 317-324.

Poortmans, J.R., Blommaert, E., Baptista, M., De Broe, M.E. and Nouwen, E.J. (1997a) Evidence of differential renal dysfunctions during exercise in men. *European Journal of Applied Physiology and Occupational Physiology* **76,** 88-91.

Poortmans, J.R., Brauman, H., Staroukine, M., Verniory, A., Decaestecker, C. and Leclercq, R. (1988) Indirect evidence of glomerular/tubular mixed-type postexercise proteinuria in healthy humans. *The American Journal of Physiology* **254(2 Pt 2),** F277-283.

Poortmans, J.R., Engels, M.F., Sellier, M. Leclercq, R. (1991) Urine protein excretion and swimming events. *Medicine and Science in Sports and Exercise* **23,** 831-835.

Poortmans, J.R. and Francaux, M. (1998) Renal dysfunction accompanying oral creatine supplements. *Lancet* **352(9123),** 234.

Poortmans, J.R., Haggenmacher, C. and Vanderstraeten, J. (2001) Postexercise proteinuria in humans and its adrenergic component. *The Journal of Sports Medicine and Physical Fitness* **41,** 95-100.

Poortmans, J.R. and Haralambie, G. (1979) Biochemical changes in a 100 km run: proteins in serum and urine. *European Journal of Applied Physiology and Occupational Physiology* **40,** 245-254.

Poortmans, J.R. and Henrist, A. (1989) The influence of air-cushion shoes on post-exercise proteinuria. *The Journal of Sports Medicine and Physical Fitness* **29,** 213-217.

Poortmans, J.R., Hermans, L., Vandervliet, A., Niset, G. and Godefroid, C. (1997b) Renal responses to exercise in heart and kidney transplant patients. *Transplant International* **10,** 323-327.

Poortmans, J.R., Jourdain, M., Heyters, C. and Reardon, F.D. (1990) Postexercise proteinuria in rowers. *Canadian Journal of Sport Sciences* **15,** 126-130.

Poortmans, J.R. and Labilloy, D. (1988) The influence of work intensity on postexercise proteinuria. *European Journal of Applied Physiology and Occupational Physiology* **57,** 260-263.

Poortmans, J.R., Mathieu, N. and De Plaen, P. (1996) Influence of running different distances on renal glomerular and tubular impairment in humans. *European Journal of Applied Physiology and Occupational Physiology* **72,** 522-527.

Poortmans, J.R., Rampaer, L. and Wolfs, J.C. (1989) Renal protein excretion after exercise in man. *European Journal of Applied Physiology and Occupational Physiology* **58,** 476-480.

Poortmans, J.R. and Vancalck, B. (1978) Renal glomerular and tubular impairment during strenuous exercise in young women. *European Journal of Clinical Investigation* **8,** 175-178.

Poortmans, J.R. and Vanderstraeten, J. (1994). Kidney function during exercise in healthy and diseased humans. An update. *Sports Medicine* **18,** 419-437.

Rodrigo, E., Fernandez-Fresnedo, G., Ruiz, J. C., Pinera, C., Heras, M., de Francisco, A. L., Sanz de Castro, S., Cotorruelo, J.G., Zubimendi, J.A. and Arias, M. (2003) Assessment of glomerular filtration rate in transplant recipients with severe renal insufficiency by Nankivell, Modification of Diet in Renal Disease (MDRD), and Cockroft-Gault equations. *Transplantation Proceedings* **35,** 1671-1672.

Shemesh, O., Golbetz, H., Kriss, J.P. and Myers, B.D. (1985) Limitations of creatinine as a filtration marker in glomerulopathic patients. *Kidney International* **28,** 830-838.

Tarone, R.E. (1990) A modified Bonferroni method for discrete data. *Biometrics* **46,** 515-522.

Taverner, D., Craig, K., Mackay, I. and Watson, M.L. (1991) Effects of exercise on renal function in patients with moderate impairment of renal function compared to normal men. *Nephron* **57,** 288-292.

AUTHORS BIOGRAPHY

Chad D. TOUCHBERRY
Employment
Research Assistant, Department of Kinesiology, College of Health Sciences and Human Services, Midwestern State University **.**
Degree
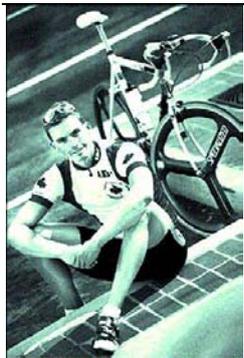BEd
Research interests
Exercise & renal function. The cellular and sub-cellular mechanisms responsible for muscle atrophy and muscle repair.
E-mail:
chadtouchberry@hotmail.com

Mark ERNSTING
Employment
Doctoral candidate, Warnborough University
Degree
MS
Research interests
Physiology of cycling performance
E-mail: raceya2004@yahoo.com

Greg HAFF
Employment
Assistant Professor, Department of Kinesiology, College of Health Sciences and Human Services, Midwestern State University
Degree
PhD
Research interests
Biochemical and biomechanical mechanisms of muscular strength, muscular hypertrophy and sport performance.
E-mail: greg.haff@mwsu.edu

Lon KILGORE
Employment
Associate Professor, Department of Kinesiology, College of Health Sciences and Human Services, Midwestern State University
Degree
PhD
Research interests
Chemical and physiological mechanisms of strength, hypertrophy, and performance.
E-mail: lon.kilgore@mwsu.edu

KEY POINTS

- Chronic cycling training is associated with alterations of glomerular filtration rate.
- Intensity of cycling exercise is associated with a reduction or resting glomerular filtration rate.
- Serum creatinine and serum urea nitrogen are not associated with changes in glomerular filtration rate in chronically exercising cyclists.

✉ **Dr. Lon Kilgore**
213 Ligon Hall, Department of Kinesiology, College of Health Sciences and Human Services, Midwestern State University, Wichita Falls, TX 76308