patients, respectively, were as follows: PSA (ACS:180), 0.89, 0.54, 0.39, 0.27, 0.26, and 0.15 μg/L (concentrations in healthy females were undetectable at <0.04 μg/L); PSA (Yu assay), undetectable; PGDS, 798, 281, 294, 480, 366, and 705 mg/L. There was no correlation between the serum samples that exhibited the highest PSA concentrations and those with higher PGDS. In fact, the PGDS concentrations, although variable, were all within the reference interval. Of interest was the inability of the monoclonal-based Yu assay to detect PSA.

From these preliminary data, it appears that PGDS expression is not increased in RCC and that PGDS is unlikely to be the source of the cross-reacting antigen detected previously in the serum of women with RCC. In keeping with these findings, we have determined that PGDS does not cross-react with several commercially available PSA antibodies and assay systems (data not shown). Although PSA could not be detected here with a more specific and sensitive PSA assay, PSA expression was detected recently in some RCC cell lines (10) and a cDNA library (11). These findings are yet to be extrapolated to human tissue. Other potential PSA-related antigens are currently being examined as candidates for the cross-reacting protein.

References

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Plasma Cardiac Troponin Concentrations after Extreme Exercise

To the Editor:
The New Zealand Ironman competition is an international ultradistance triathlon in which each athlete swims 3.8 km, cycles 180 km, and runs 42.2 km on the same day, completing the event in a time ranging from 9 to 16 h. In 1998, the race was held on March 15. A summary of the medical complications of the race and their treatment has been published separately (1). During and immediately after the race, 134 of the 650 starting athletes presented to the race medical facility for advice and treatment. Of these, 64 underwent venipuncture for measurement of plasma electrolytes because of clinical suspicion of acute hyponatremia (2). The residual blood from these tests was used in the study reported here.

Athletes withdrew from the race because of injury or exhaustion when necessary. Those who presented for medical treatment were asked for informed consent, either at presenta-
tion or after recovery, for use of their clinical records and use of residual blood for assays of markers of myocardial injury. The study protocol was approved by the North Health Ethics Committee.

The first blood specimen was drawn ~11 h, and the last ~16 h after the race starting time. Whole blood drawn into lithium heparin evacuated tubes was used for urgent measurement of sodium, potassium, and glucose. After these tests, and within 20 min of venipuncture, the residual blood was centrifuged and the plasma separated and stored at 0 °C for further analysis within 18 h.

Total creatine kinase (CK) was measured with a Boehringer-Hitachi 717 analyzer. Cardiac troponin T (cTnT) was measured with a Boehringer Elecsys 1010 analyzer using the “second-generation” dual monoclonal antibody assay. Cardiac troponin I (cTnI) was measured with an Abbott AxSYM analyzer. In all cases, the reagents and analytical protocols provided by the equipment manufacturers were used. Correlation analysis was performed with Microsoft Excel. In the statistical analysis, cTnT concentrations reported as <0.01 µg/L were represented as 0.005 µg/L.

A total of 64 athletes underwent venipuncture. One sample had insufficient residual blood for analysis, and one athlete had two tests 1 h apart. There thus were 64 samples from 63 athletes analyzed for CK, cTnI, and cTnT. The plasma CK activities were increased, consistent with skeletal muscle damage. The median CK was 1515 U/L (range, 328–23 500 U/L; reference interval, 0–2.0 U/L). The scattergram for cTnT (reference interval, 0–0.10 µg/L) and cTnI (reference interval, 0–2.0 µg/L) is shown in Fig. 1. Regression analysis confirmed significant correlation between cTnI and cTnT (r = 0.870; P <0.01). Reanalysis with the results above the reference interval excluded still showed strong correlation (r = 0.822; P <0.01). Both cTnI and cTnT were above the reference interval in four athletes, and cTnT but not cTnI was above the reference interval in one. Of these five athletes, three were available for follow-up 3–6 days after the race. Clinical review was conducted, and assays for CK, cTnI, and cTnT were repeated. The review gave no cause to suspect myocardial damage or disease.

Although there are reports on cTnI and cTnT after a marathon (3–5), we have no prior experience of, and could find no published accounts of their assay after physiological stress of the magnitude induced in the Ironman race. Since conducting this study, we have noted similar results published from the Hawaii Ironman competition (6), where mildly increased cTnI and cTnT in some athletes were associated with echocardiographic evidence of cardiac wall hypokinesia.

It is apparent that all of the athletes that we tested had suffered skeletal muscle damage, as evidenced by their increased plasma CK activities. The plasma cTnI and cTnT concentrations were within the reference intervals in most athletes, consistent with the belief that these markers are relatively unaffected by skeletal muscle injury. In five athletes, however, cTnT and/or cTnI were above the reference interval, suggestive of myocardial injury. These increases were relatively minor, to at most 2.5 times the upper limit of the reference interval; in our experience, larger increases are common in myocardial infarction with increased CKMB. In athletes who were available for follow-up at 3–6 days, the increases were not sustained, as is usual with myocardial infarction (7).

It remains undetermined whether the increases in troponin concentrations were indicative of significant acute myocardial damage in these five athletes. However, the overall correlation of plasma cTnI and cTnT concentrations leads us to hypothesize that the extreme exertion had produced some minor transient myocardial injury or “stunning”, as described previously (3, 6), in many of the athletes and that this injury was sufficient to increase the troponin concentrations above the reference intervals in a small group.

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References
3. Siegel AJ, Sholar M, Yang J, Dhanak E, Lewandrowski KB. Elevated serum cardiac markers in asymptomatic marathon runners after competi-


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