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ORIGINAL ARTICLE

Prostate Cancer

A population study of fasting time and serum prostate-specific antigen (PSA) level

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Prostate cancer is one of the most common cancers in men. Traditional screening and diagnostic methods include digital rectal examinations (DREs), biopsies and serum prostate-specific antigen (PSA) tests, with the latter being the more popular. PSA is a biomarker for prostate cancer; however, it is highly sensitive to external factors as well as other prostate diseases. As such, the reliability of of the serum PSA level as a sole screening and diagnostic tool for prostate cancer is controversial. Recently, it has been shown that fasting extremes can affect concentrations of serum chemistry analytes, thus raising the question of whether or not fasting has an effect on the highly sensitive PSA biomarker. Patients testing for serum PSA levels are often concomitantly submitting to other tests that require fasting, subjecting certain patients to a fasting PSA level while others not. The objective of this study was to investigate whether this discrepancy in fasting state translates into an effect on serum PSA levels. Serum PSA levels and fasting time records for 157 276 men who underwent testing at Calgary Laboratory Services (CLS; Calgary, Alberta, Canada) between 01 January 2010 and 31 March 2013 were accessed. Linear regression models of mean PSA levels and fasting times revealed a statistically important relationship at certain fasting times. Applying a dynamic mathematical model to explore the clinical effect of fasting suggests minimal impact on serum PSA result interpretation. Thus, patients can be tested for serum PSA levels regardless of their fasting state.

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INTRODUCTION

Prostate cancer is the most common form of cancer diagnosed in Canadian men.[1] The Canadian Cancer Society estimates that in the year 2013, there will be 23 569 new cases of prostate cancer.[2] As such, discovering and developing novel biomarkers for prostate cancer screening are of utmost importance.[3–5] Traditionally, digital rectal examination (DRE) has been the preferred assessment method for prostate cancer.[6] In the 1980s, the serum prostate-specific antigen (PSA) test was introduced as an additional method for screening, diagnosing and monitoring treatment of patients with prostate cancer.[7] PSA testing was not highly utilized in Canada until 1990.[8] As a result of its increased utilization, the incidence rate of prostate cancer detection increased rapidly and peaked in 1993.[9] Serum PSA testing is currently one of three common procedures: DRE, serum PSA test and transrectal ultrasound-guided biopsy, used to screen and diagnose prostate cancer.[10]

Serum PSA testing has, however, courted controversy because of its low specificity. Despite its shortcomings, PSA is still the most common and preferred screening method within the male population due to its convenience and accessibility. North American prostate cancer screening guidelines recommend that men with a family history of prostate cancer or of African descent should begin screening at 40 years of age.[7,11] For the rest of the male population, screening should commence at the age of 50 years.[7,11] Currently, physicians are responsible for informing patients of the availability of the different prostate cancer screening options and their associated risks to assist the patient in making informed screening decisions.[12] For prostate cancer screening, it is advocated that a DRE is performed in conjunction with serum PSA testing. Serum PSA levels, however, are highly sensitive and can be easily altered by prostate stimuli. PSA is a glycoprotein produced by prostate epithelial cells that line the ducts and acini of the prostate.[13] Any type of stimuli that causes a disruption of the innate glandular architecture of the prostate can facilitate PSA to enter systemic circulation, which leads to increased levels.[13] Apart from physical stimulation of the prostate, other conditions that elevate serum PSA concentrations from baseline include: benign prostatic hyperplasia (BPH), prostatitis, urethral instrumentation, prostate biopsy and prostate cancer.[13,14] Other causes of PSA elevation are vigorous DRE and recent ejaculation.[13,15]

Since the levels of serum PSA are highly responsive to its environment, we would like to investigate if diagnostic testing conditions, specifically fasting, can affect PSA results. Fasting is not required for serum PSA testing; however, serum PSA tests are often performed in combination with other diagnostic tests that do require fasting. Other common laboratory tests that have been examined for the relevance of fasting time have shown that at fasting extremes (such as less than 1 h or over 12 h) the effects of hemodilution/hemoconcentration are observed.[16,17] A previous limited study by Tuncel *et al.*[18] examined fasting time and serum PSA levels. In their study, 80 patients were asked to fast between 12 am and 8 am, after

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which they were provided breakfast and lunch. Serum samples were obtained periodically pre- and post-meals to monitor its effect on serum PSA levels.[18] They reported that there was no difference between pre- and post-prandial serum PSA levels.[18] Their findings however, are limited by a small sample size and limited fasting times examined.[18] Thus, the objective of our study is to revisit this topic by examining a wider range of fasting times and a larger data set: the community population of Calgary, Alberta. We hypothesize that fasting should not affect serum PSA levels except at fasting extremes where the effects of hemodilution/hemoconcentration may play a role.

MATERIALS AND METHODS

Ethics statement

This study was approved by the University of Calgary Conjoint Health Review Ethics Board (ID: E-25060).

Study population and data sources

This study was conducted using secondary data from the Laboratory Information System of Calgary Laboratory Services (CLS). CLS is the sole provider of laboratory services for Calgary, Alberta and its surrounding areas (approximately 1.4×10^6 people). The study population consisted of all men who underwent PSA testing at CLS between 01 January 2010 and 31 March 2013. All PSA tests were performed as part of routine patient care and were analyzed at CLS using a Roche Modular analyzer. Fasting time (hours) was obtained through patient self-reporting at the time of testing. In our analysis, fasting time was categorized into hourly intervals from 1 to 16. All reported fasting hours were rounded up to the next integral value. All individuals with a fasting time beyond 16 h were grouped into the 16 h category. PSA records were excluded for female patients, probable cancer patients (serum PSA level >20 μg l^{-1}) and if a fasting time was not recorded. As age-specific reference ranges are only commonly reported for men between the ages of 40 and 79 years, any records outside of this range were also removed. Finally, to avoid pseudoreplication we considered only the first PSA result from each patient. This resulted in a total of 157 276 PSA records and paired fasting times.

Statistical analysis

Fasting time for laboratory tests has been shown to vary as a function of age and sex.[19] As our study only consists of males, the effects of sex on fasting time are negated. The data were fitted to a linear regression model with serum PSA measurement as the dependent variable and fasting time (in hours) and age (10-year cohorts) as independent variables. Estimated marginal means were calculated based on 95% confidence intervals at each fasting time period, with age held constant at the group mean. Statistical significance was assessed through pairwise comparison of fasting hours and its corresponding serum PSA levels using the Tukey *post hoc* test. All statistical analyses were performed using the Statistical Package for Social Sciences (SPSS) software (Version 20 for Windows, SPSS Inc., Chicago, IL, USA).

Dynamic mathematical model

To explore the impact of fasting time on serum PSA levels through a clinical perspective, a dynamic mathematical model was developed to calculate the percentage of individuals expected to experience a change in PSA diagnostic interpretation (e.g. normal *vs* abnormal result) based on variations in fasting time. In this model, data were first parsed into age cohorts: 40–49, 50–59, 60–69 and 70 to 79-years-old, and the number of PSA values above of the normal range (2.5, 3.5, 4.5 and 6.5 μg l^{-1} respectively, based on age-dependent cutoffs) were calculated. In order to preclude ethnicity-related serum PSA effects, and since ethnicity data are not collected during PSA testing, only age-specific reference ranges for Caucasian males were used. PSA cutoff levels for an abnormal test were based on the Alberta Toward Optimized Practice 2010 guidelines.[11] We included only paired fasting time intervals that showed a statistically significant ($P < 0.05$) difference in mean PSA value (see Results section). For each of these paired fasting times, we calculated the expected difference in serum PSA value if the patient had fasted for the other period of time. These calculations were performed by adding (or subtracting) the differences in estimated marginal means to each individual result. For example, results for patients with a 4 h fasting interval were modified to simulate a 9 h fasting interval by subtracting 0.367 from every value (the mean difference attributable to fasting time; **Table 1**). Following this operation, we recalculated the number of abnormal PSA results for each simulated change in fasting time. These are presented as a projected percent change in abnormal results due to a change in fasting time and allow us to comment on the anticipated clinical significance of variations in fasting time.

RESULTS

A total of 157 276 PSA records were accessed from our Laboratory Information System. The mean PSA level for all records was 1.35 μg l^{-1}. The average age of patients tested was 57.0 years of age. The average fasting time of all records was 12.7 h with a minimum and maximum fasting time of 1 and 16 h, respectively. Mean PSA levels at different fasting times are shown in **Table 2**. **Table 3** illustrates the dependence of each independent variable (fasting time and age) on the dependent variable (serum PSA levels). These results indicate that fasting time and age have a statistical effect on serum PSA levels.

Table 1: Dynamic mathematical model results showing modeled effects of changes in fasting time on abnormal PSA (>upper limit of normal) classification rates

Starting fasting time	*Modeled fasting time*	*Mean difference*	*Starting abnormals (fasting group)*	*Model abnormals (same fasting group)*	*Total in fasting hour subgroup*	*Percent change (%)*
1	4	−0.346	35	48	796	1.63
9	15	−0.199	30	34	697	0.57
9	16	−0.199	30	34	697	0.57
10	12	−0.048	309	322	8193	0.16
10	13	−0.056	309	322	8193	0.16
10	14	−0.098	309	332	8193	0.28
10	15	−0.134	309	341	8193	0.39
10	16	−0.134	309	341	8193	0.39
11	12	−0.048	611	627	15483	0.10
11	13	−0.056	611	629	15483	0.12
11	14	−0.098	611	652	15483	0.26
11	15	−0.134	611	668	15483	0.37
11	16	−0.134	611	668	15483	0.37
12	14	−0.050	1841	1921	44265	0.18
12	15	−0.087	1841	1964	44265	0.28
12	16	−0.086	1841	1964	44265	0.28
13	14	−0.042	1469	1534	35040	0.19
13	15	−0.079	1469	1556	35040	0.25
13	16	−0.078	1469	1556	35040	0.25
4	9	0.367	26	22	374	−1.07
4	10	0.347	26	23	374	−0.80
4	11	0.302	26	23	374	−0.80

PSA: prostate-specific antigen

Table 4 shows the specific fasting hours that have an effect on serum PSA levels. At fasting times (>8 h), serum PSA levels are generally distinct from non-fasting times (1–8 h).

Table 2: Serum PSA levels by fasting time after adjustment for the effect of age

Fasting time (h)	*Mean PSA (ng ml^{-1})*	*Standard error*	*95% CI*	
			Lower bound	*Upper bound*
1	1.521	0.060	1.402	1.639
2	1.394	0.080	1.238	1.550
3	1.587	0.078	1.434	1.740
4	1.790	0.082	1.628	1.951
5	1.535	0.096	1.346	1.724
6	1.474	0.113	1.253	1.695
7	1.379	0.145	1.095	1.663
8	1.521	0.106	1.313	1.729
9	1.373	0.067	1.242	1.504
10	1.460	0.019	1.423	1.498
11	1.467	0.013	1.441	1.494
12	1.496	0.008	1.480	1.511
13	1.479	0.008	1.463	1.496
14	1.504	0.010	1.485	1.523
15	1.540	0.013	1.514	1.566
16	1.566	0.014	1.538	1.594

CI: confidence interval, PSA: prostate-specific antigen

Table 3: Univariate analysis of variance showing the effect of fasting time on serum PSA value

Source	*Type III sum of squares*	*df*	*Mean square*	*F*	*Significance*
Corrected model	28 621.3[a]	63	454.3	216.6	0.000
Intercept	14 702.4	1	14 702.4	7009.0	0.000
Fasting time (h)	139.8	15	9.3	4.4	0.000
Age group	1463.4	3	487.8	232.5	0.000
Fasting time age group	153.4	45	3.4	1.6	0.005
Error	329 774.2	157 212	2.1		
Total	647 006.0	157 276			
Corrected total	358 395.5	157 275			

df: degrees of freedom; PSA: prostate-specific antigen. [a]R^2 = 0.080 (adjusted R^2 = 0.079)

Although a number of variations in fasting time yielded statistically important results, we were also interested in assessing the potential clinical significance of these changes. A Dynamic Mathematical Model as described earlier showed that an increased fasting time would generally result in a modest increase in the number of patients with abnormal PSA values (**Table 1** and **4**). The overall changes in classification (normal to abnormal or vice versa) ranges from −1.07% to 1.63% (mean = 0.19%; **Table 1**).

DISCUSSION

Previous studies have shown that different fasting states can affect chemical analytes.[16,20] Specifically, it has been suggested that the ingestion of food can trigger different physiological responses that affect blood biochemical markers commonly used in laboratory tests.[20] For example, the serum concentration of liver biomarkers aspartate aminotransferase and alanine aminotransferase has been shown to notably increase 4 h after meal ingestion.[20] Apart from changes in the levels of liver biomarker enzymes, variations in red blood cell count, hematocrit and hemoglobin concentrations have also been noted.[16] These changes are attributed to hemodilution of the blood where an increase in blood volume alters the constituent.[16] Another analyte that has been examined for the effects of fasting is cholesterol.[17] Traditionally, testing of total lipids and lipid subclass levels were performed in a postprandial state of 8 or more hours.[21,22] However, recent work has suggested that fasting is largely unnecessary for routine lipid level.[17] However, at fasting extremes (14–16 h), statistically important differences exist.[17] In this study, we wanted to examine whether such fasting extremes have an effect on serum PSA levels, a prostate biomarker that is highly sensitive to environmental conditions. In particular, as serum PSA tests are often performed in conjunction with other fasting laboratory blood tests, the effects of fasting could potentially alter patient treatment depending on the variation in serum PSA concentrations. A previous study performed by Tuncel *et al.*[18] on a group of 80 individuals showed that fasting up to and including 8 h did not have an effect on serum PSA levels. To our knowledge, our study is the first to examine the effect of fasting time on serum PSA levels within a community-based population.

Figure 1 shows two regions of interest: 1–9 and 10–16 h. Between fasting times of 1–9 h, fluctuations exist; while at 10 h and beyond, there

Table 4: Multiple mean comparison between fasting hours and serum PSA values adjusting for age effects

Fasting time (h)	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*	*11*	*12*	*13*	*14*	*15*	*16*
1	0															
2	NS	0														
3	NS	NS	0													
4	–0.346	NS	NS	0												
5	NS	NS	NS	NS	0											
6	NS	NS	NS	NS	NS	0										
7	NS	NS	NS	NS	NS	NS	0									
8	NS	NS	NS	NS	NS	NS	NS	0								
9	NS	NS	NS	0.367	NS	NS	NS	NS	0							
10	NS	NS	NS	0.347	NS	NS	NS	NS	NS	0						
11	NS	NS	NS	0.302	NS	NS	NS	NS	NS	NS	0					
12	NS	NS	NS	NS	NS	NS	NS	NS	NS	–0.048	–0.048	0				
13	NS	NS	NS	NS	NS	NS	NS	NS	NS	–0.056	–0.056	NS	0			
14	NS	NS	NS	NS	NS	NS	NS	NS	NS	–0.098	–0.098	–0.050	–0.042	0		
15	NS	NS	NS	NS	NS	NS	NS	NS	–0.199	–0.134	–0.134	–0.087	–0.079	NS	0	
16	NS	NS	NS	NS	NS	NS	NS	NS	–0.199	–0.134	–0.134	–0.086	–0.078	NS	NS	0

NS: not significant; PSA: prostate-specific antigen. Only mean differences which were statistically significant at $P < 0.05$ are shown

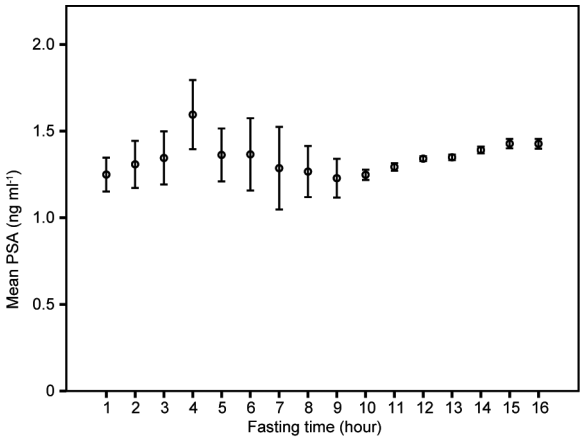

Figure 1: Mean serum PSA levels at different fasting time not accounting for age-variance; prepared by SPSS.

is a general increasing trend. A notable feature is the peak in serum PSA level observed at 4 h of fasting (**Figure 1**). We considered collection time (as opposed to fasting interval) as a possible confounding factor which could have biased the observed increase in PSA levels at 4 h of fasting. To remove this possible bias we reperformed the analysis with collection time as an additional variable. The overall trend, however was unchanged (data not shown). A potential reason for this increase may instead be related to diurnal variation in serum PSA levels. It has been suggested that PSA expression is controlled by hormones[23] and in particular, in cases of patients with BPH, testosterone has been shown to directly influence serum PSA levels[24] and exhibits a well-established circadian rhythm.[25] Thus, it is of interest to examine if serum PSA follows the same diurnal variation as testosterone. Diurnal variation of serum PSA has been previously examined and is controversial as some studies suggest its existence, while others disagree.[26–30] A major potential shortcoming of these studies is their small sample size; the study with the largest sample contained 44 men.[30]

The second region encompassing fasting hours of 10–16 shows a gradual increase in mean PSA values (**Figure 1**). Analyzing the mean differences in PSA values between the different fasting times shows that at fasting extremes (1 h or less or 9 or more hours), PSA values are significantly different (**Table 4**). This is similar to observations for other chemistry analytes.[16,17] Since the trend past 9 h is an increasing one (**Figure 1**), it should be noted that the longer the fasting time the greater the deviation in detected PSA values. Our statistical analysis suggests that the optimal time to obtain an accurate PSA level is fasting for 7–9 h, which avoids the increased PSA levels observed with fasting extremes as well as possible diurnal rhythm variations that may exist in shorter fasting times. It should be noted that the larger standard deviations observed in serum PSA levels for 1–9 h of fasting compared to 10 or more hours (**Figure 1**) are likely the result of smaller sample sizes for these time periods (160–800 men).

To explore the clinical effect of fasting time on serum PSA levels, a dynamic mathematical model was developed. Simulating the changes that would occur in PSA values if fasting times were altered revealed that the percentage change in the number of abnormal PSA values can range from −1.07% to 1.63% (**Table 1**). Therefore, despite the statistical significance of PSA variations with fasting time, our model predicts that this would translate into minimal clinical effect.

There were several limitations in this study that were unaccounted for in our analysis. The first is the possibility of patient recall error regarding fasting time. Another limitation that should be noted is our use of secondary data on individuals and not a random sample of individuals taken from the general population. As such, these findings can only be interpreted as representative of individuals presenting for testing and may be a biased representation of the overall population.

In conclusion, certain fasting states (1 or less hours or 4 h or 9 or more hours) have a statistically significant effect on serum PSA levels which substantiates both our hypothesis and other previous studies that have observed deviations in concentrations of serum analytes at fasting extremes.[16,17] However, when translating this to an anticipated clinical effect, we suggest this would have a minimal impact on overall classification rate of normal versus abnormal results. It appears that patients can be tested for serum PSA levels regardless of their fasting state. Future research should focus on identifying additional biomarkers and/or chemical analytes for prostate cancer that are robust and less sensitive to external stimuli. This will help reduce the ambiguity and increase the reliability associated with prostate cancer testing.

AUTHOR CONTRIBUTIONS

CKL, MG, JAZ and CTN participated in the conception and design of the study. JAZ acquired all relevant data. CKL, MG and CTN critically interpreted and analyzed the data. MG performed all statistical analyses. CKL and CTN drafted the manuscript. CKL, MG, JAZ and CTN all participated in the critical revision of the manuscript for intellectual content. CTN supervised the study. All authors read and approved the manuscript.

COMPETING INTERESTS

All authors declare no competing interests.

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