Published in final edited form as:

BJU Int. 2008 February ; 101(3): 313–318. doi:10.1111/j.1464-410X.2007.07332.x.

Lipids, Lipoproteins, and Risk of Benign Prostatic Hyperplasia in Community Dwelling Men

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Abstract

Objective—We examined associations of serum lipids and lipoproteins with benign prostatic hyperplasia (BPH) in a cohort of community dwelling men.

Subjects and Methods—This analysis was conducted in the Rancho Bernardo Study, a prospective, community-based cohort study. BPH was defined as a history of non-cancer prostate surgery or a medical diagnosis of BPH. Logistic regression modeling—with adjustments for age and stratification by diabetes diagnosis—was utilized to estimate the odds ratio (OR) of BPH associated with fasting serum concentrations of total cholesterol, HDL cholesterol, LDL cholesterol, triglycerides, and the triglyceride to HDL ratio.

Results—Among 531 eligible participants, 259 (48%) reported BPH and 272 (52%) reported no BPH. Men with BPH (75.8 years, range 76.1 to 80.1 years) were older than men without BPH (72.7 years, range 72.4 to 74.0 years). There were no significant associations of total cholesterol (p-trend = 0.52), HDL cholesterol (p-trend = 0.56), triglycerides (p-trend = 0.30), or triglyceride to HDL ratio (p-trend = 0.13) with BPH risk. In a subset analysis in men with diabetes, those in the highest tertile (>133 mg/dL) of LDL cholesterol, compared with those in the lowest tertile (<110 mg/dL), were 4-times more likely to have BPH (OR 4.00, 95% CI 1.27–12.63, p-trend=0.02). These results were not explained by statin use.

Conclusions—In this cohort of community dwelling men, higher serum LDL was associated with increased risk of BPH among diabetics. These data suggest that diabetic men with increased LDL cholesterol are at increased risk for BPH. This observation is consistent with the concept that cardiac risk factors are involved with BPH pathogenesis.

Keywords

benign prostatic hyperplasia; dyslipidemia; cholesterol; triglyceride; risk factor; lower urinary tract symptoms

Introduction

Benign prostatic hyperplasia (BPH) is a highly prevalent disease among older men and a substantial public health problem. In 2000, the most recent year for which comprehensive U.S. data are available, BPH accounted for over 4.4 million office visits, 117,000 emergency room

visits, and 105,000 hospitalizations [1]. The prevalence among U.S. men aged 60 to 69 years is estimated to be over 70%, with approximately 6.5 million white men aged 50 to 79 years affected [1].

Despite its widespread prevalence, the etiology of BPH is not well understood. Previous causal models have focused primarily on sex steroid hormones. However, while androgens and genetic predisposition play important roles, accumulating evidence indicates that modifiable risk factors of cardiovascular disease may also increase the risk of BPH and potentially contribute to BPH development. Obesity [2–7], elevated fasting plasma glucose [2,8], diabetes [2,5,9,10], and the metabolic syndrome [9] have been associated with an increased risk of BPH and male lower urinary tract symptoms (LUTS).

Abnormal concentrations of lipids and lipoproteins are well-described risk factors for cardiovascular disease and include elevated serum low density lipoprotein (LDL) cholesterol (usually defined as ≥ 130 – 140 mg/dL), decreased serum high density lipoprotein (HDL) cholesterol (< 40 mg/dL), and increased serum triglycerides (≥ 150 mg/dL). These factors are components of the metabolic syndrome and frequently occur in association with other cardiovascular risk factors, including diabetes. This observation raises the possibility that abnormal lipids and lipoproteins may also be connected to BPH pathogenesis. There are laboratory data to support the biological plausibility of this concept: in one study, rats fed cholesterol-rich diets exhibited both altered blood lipid profiles and hyperplastic changes in the prostate [11].

There are relatively few clinical studies, however, and the results have been inconsistent. In a cohort of Swedish men with BPH, lower HDL cholesterol, higher LDL cholesterol, and higher triglycerides were associated with increased prostate volume [12], while in a case control study in India, men undergoing BPH surgery were more likely to have lower HDL and higher LDL cholesterol compared to controls [13]. However, there were no associations of patient-reported hyperlipidemia with histological BPH in a case control analysis of Italian men [14]; no associations of serum lipids or lipoproteins with International Prostate Symptom Score or prostate volume in a cohort of Turkish men [15]; and no associations of serum lipids or lipoproteins with ICD-9 coded BPH diagnosis in a cohort of U.S. Air Force Veterans [8].

Limitations of these prior observational studies include imprecise definitions of abnormal lipid and lipoprotein levels and use of populations with potentially restricted external validity for the general population (i.e. hospital-based cases/controls and select groups of combat veterans). Moreover, since most of these studies did not consider diabetes and other cardiovascular risk factors for BPH in their analyses, it remains unclear as to how these variables may possibly interact with lipids and lipoproteins to alter BPH risk.

Further study of lipids, lipoproteins, and diagnosis of BPH in community-dwelling men may elucidate mechanisms of BPH pathogenesis and suggest novel methods of prevention and treatment. Therefore, we examined associations of serum lipid and lipoprotein concentrations with the risk of clinical BPH in a cohort of community-dwelling U.S. men.

Subjects and Methods

Participants

The Rancho Bernardo Study, established in 1972, is a cohort of white, middle to upper middle class, community-dwelling adults in southern California, U.S.A. Between 1992 and 1996, 699 surviving, locally-dwelling men attended a clinic visit designed to study chronic diseases. Personal history regarding hypertension, diabetes, current cigarette smoking, daily alcohol consumption, and regular exercise (≥ 3 times per week) were determined by standardized

questionnaire and interview. Height and weight were measured with participants wearing lightweight clothing without shoes; body mass index (BMI) was calculated as weight (kg)/height² (m²). Waist and hip girth were measured in centimeters over single-thickness clothing with the participant standing in an erect position with feet together. Waist circumference was used as an integrated measure of obesity and fat distribution. Lipids and lipoproteins were measured in a Lipid Research Clinic Laboratory certified by the Center for Disease Control. Total cholesterol and triglycerides were measured by enzymatic methods with an ABA-200 biochromatic analyzer (Abbott), HDL cholesterol was measured by precipitation according to Lipid Research Clinic protocol, and LDL cholesterol was calculated by means of the formula of Friedewald and colleagues. Men aged 60 years and older with complete information, including prostate health history, were included in the analysis.

Definition of BPH

Prior observational studies have utilized different definitions of BPH. In this analysis, BPH was defined as a history of non-cancer surgery of the prostate or physician-diagnosed BPH. Both of these definitions, which emphasize clinical diagnosis, have been used in several large, population-based studies [3,16–18].

Statistical analysis

This was a cross-sectional analysis. Bivariate associations between BPH prevalence and baseline characteristics and lipid values were evaluated with a Student's *t* test for continuous variables and a χ^2 test for categorical variables. Because triglycerides showed a skewed distribution, analyses were performed using log-transformed data. Geometric mean values of all variables are presented; all probability values were based on log-transformed data. Logistic regression analyses were used to assess the independent association of lipid levels (serum total cholesterol, HDL cholesterol, LDL cholesterol and triglyceride concentrations and triglyceride to HDL ratio) with prevalent BPH, with adjustments for age and statin use. Lipid and lipoprotein levels were analyzed continuously as standardized variables, binary as dichotomous variables based on standard cut-offs and as tertiles.

Results

Among 531 men without known prostate cancer, 259 (48%) reported a diagnosis of BPH or compatible non-cancer prostate surgery and 272 (52%) reported no BPH. Mean ages for the BPH and no BPH groups were 75.8 years (range 76.1 to 80.1 years) and 72.7 years (range 72.4 to 74.0 years), respectively ($p < 0.001$).

As shown in Table 1, most factors potentially associated with abnormal lipids and lipoproteins—including known cardiovascular disease, diabetes, body size, hypertension, use of lipid-lowering medications, and lifestyle (smoking, alcohol use, physical activity)—did not differ significantly in men with versus those without BPH. The exceptions were a lower mean fasting plasma glucose concentration, a slightly lower mean BMI, and trends toward a lower waist circumference, waist-to-hip ratio, and lower prevalence of diabetes among men with BPH. There were no significant differences in obesity by BMI, hypertension prevalence, history of TIA or stroke, use of lipid-lowering medications, alcohol consumption, smoking, or physical activity by BPH status.

There were no significant differences in mean total cholesterol concentrations or the proportions of men with total cholesterol ≥ 200 between the no BPH and BPH groups (Table 1). Compared to men with total cholesterol < 200 , the odds ratio (OR) for BPH among men with total cholesterol ≥ 200 mg/dl was 1.06 (95% CI 0.74–1.50, $p = 0.76$).

There were no significant differences in mean LDL cholesterol concentrations or the proportions of men with LDL cholesterol ≥ 130 between the no BPH and BPH groups (Table 1). Compared to men with LDL cholesterol <130 , the OR for BPH for men with LDL cholesterol ≥ 130 mg/dl was 1.20 (95% CI 0.84–1.71, $p=0.33$). There were no significant differences in mean HDL cholesterol concentrations or the proportions of men with HDL cholesterol <40 between the no BPH and BPH groups (Table 2). Compared to men with HDL cholesterol ≥ 40 , the OR for BPH for men with HDL cholesterol <40 mg/dl was 0.78 (95% CI 0.52–1.15, $p=0.33$). There were no significant differences in mean triglyceride concentrations or the proportions of men with triglyceride ≥ 150 between the no BPH and BPH groups. Compared to men with triglyceride <150 , the OR for BPH for men with triglyceride ≥ 150 mg/dl was 0.94 (95% CI 0.62–1.42, $p=0.75$). Among all participants combined, there were no significant associations of BPH with any lipid or lipoprotein or by triglyceride:HDL ratio by tertile (Table 2).

In a pre-planned subset analysis of lipids and lipoproteins by diabetes status, all analyses were repeated stratifying by diagnosis of diabetes. Similar, non-significant results were obtained for associations of BPH with total and HDL cholesterol and triglyceride concentrations (mean concentrations, clinically relevant cut-points, and tertiles) for both the non-diabetes and diabetes groups (data not shown).

The only significant associations of BPH with lipid and lipoproteins were observed for LDL levels in diabetics, in whom higher LDL cholesterol was associated with a significantly increased risk of BPH. Each standard deviation increase in serum LDL in diabetics corresponded to a 67% increase in BPH risk (OR 1.67, 95% CI 1.04 to 2.70, $p\text{-trend}=0.04$). Those with clinically elevated LDL cholesterol (≥ 130 mg/dl), compared to those with non-elevated cholesterol (<130 mg/dl), were 2-times more likely to have BPH (OR 2.17, 95% CI 0.88–5.38, $p\text{-trend}=0.09$). Those in the highest tertile (>133 mg/dL) of LDL cholesterol, compared with those in the lowest tertile (<110 mg/dL), were 4-times more likely to have BPH (OR 4.00, 95% CI 1.27–12.63, $p\text{-trend}=0.02$). These associations were not explained by age or statin use (Table 4). There was no difference in age- and statin-adjusted LDL in non-diabetics (mean 123.0, 95% CI 120.1 to 125.9) versus diabetics (mean LDL 120.2, 95% CI 113.8 to 126.7, $p\text{-value}=0.44$). There was no difference in the proportion of statin users among non-diabetics ($N=46$, 10.38%) versus diabetics ($N=10$, 11.36%, $p\text{-value}=0.78$).

Discussion

In this cross-sectional study, serum lipid and lipoprotein concentrations did not differ significantly in men with or without clinical BPH. The previously described associations of BPH with obesity and diabetes did not extend to triglycerides or HDL-cholesterol, the lipid and lipoprotein most strongly associated with obesity and diabetes [19]. The absent association may suggest that an alternative mechanism, distinct from abnormal HDL and triglyceride profiles, drives the reported associations of BPH with obesity and diabetes. On the other hand, there was little difference in BMI, waist girth, or BPH status in the present study, although there was a small but significant difference in fasting plasma glucose.

Although there were no overall associations of BPH with lipids and lipoproteins, there was a four-fold higher risk of BPH in men who had both diabetes and elevated levels of LDL cholesterol compared to men who had just diabetes. These results are surprising in that, although diabetics have unfavorable total and LDL cholesterol particle size and density, serum elevations in total or LDL cholesterol are not typically characteristic of diabetes [20,21] and were not observed in an earlier study of this cohort [22]. Moreover, the mean LDL concentrations in the men with and without diabetes were substantially lower than the US average as measured in the third National Health and Nutrition Examination Survey [23].

Compared to the general population, diabetic patients typically have higher triglyceride levels and lower HDL levels (the classic dyslipidemia of diabetes and the metabolic syndrome) and no difference in total or LDL cholesterol [20,21].

To our knowledge, this study is one of the largest community-based studies of lipids, lipoproteins, and BPH risk to date and the first to investigate potential interactions of these factors with a diabetes diagnosis. Prior, larger studies have observed an increased risk of BPH and LUTS among patients with diabetes [2,5,9,10]. Patients with diabetes have decreased risk of prostate cancer, observed in this cohort and elsewhere [24,25]. This lower risk of prostate cancer makes it less likely that the higher risk of BPH reported here among diabetes is due to diagnostic detection bias.

Although BPH was not associated with HDL cholesterol or triglycerides, the null results could reflect non-differential bias introduced by our case definition for BPH. Because case definitions for BPH in clinical research vary considerably, measurement and detection biases are acknowledged limitations of observational BPH research. BPH definitions in prior studies include histological analysis of prostate tissue, radiographically-determined prostate enlargement, acute urinary retention, decreased urinary flow rate, pressure-flow studies consistent with bladder outlet obstruction, history of non-cancer surgery of the prostate, clinical BPH, and American Urological Association or International Prostate Symptom Score using investigator-designated cut-offs for BPH [26].

In this study we utilized two of these well-established definitions. Nevertheless, because clinical diagnosis and surgical history are self-reported, a non-differential observational bias may have been introduced into our analysis that biased associations of BPH with HDL and triglycerides toward the null, attenuating and thus masking any true associations. Another limitation in this regard is the absence of validation of the BPH diagnosis in this cohort. To our knowledge, there are no published studies validating or correlating patient-reported or physician-diagnosed BPH with other BPH measures. Still, the prevalence of BPH in this cohort is similar to the end-of-study prevalence in another cohort that utilized physician-diagnosed BPH [8]. Validations of other self-reported diagnoses in this cohort range from 85% (cardiovascular disease) to 95% (cancers).

BPH was associated with lower BMI and trends toward lower waist circumference and waist-to-hip ratio; however, these differences were small (Table 1) and thus of uncertain clinical significance. Moreover, we observed no significant associations of BMI-measured obesity with BPH, an observation that contrasts with those of several prior studies of BPH and LUTS [2–7] and thus also raises the possibility of bias. The possibility of non-differential bias in our study emphasizes the need for consensus regarding BPH case definitions in future observational and clinical studies.

Associations of another major risk factor for cardiac disease (and a component of the metabolic syndrome), hypertension, with BPH and LUTS remain unclear. Hypertension was associated with increased risk of BPH surgery in the Physician's Health Study [27] and increased risk of LUTS in 3 other population-based observational studies [5,9,10]. However, there were no significant associations of hypertension with clinical BPH in the present study or among participants in the Massachusetts Male Aging Study [17] or with histological BPH in a case control analysis [14]. In addition, since BPH normally presents with LUTS, our results may have been confounded by the frequent use of anti-hypertensive medications in this cohort.

Nevertheless, these results could be important. If confirmed, they intimate that maintenance of low LDL cholesterol with statins might prevent or attenuate BPH and its symptoms in diabetic men. In one study, lovastatin induced apoptosis in prostate stromal cells in vitro [28]. A randomized clinical trial of 319 patients, however, showed no differences in IPSS,

prostate volume, urinary flow rate, PSA, or disease-specific quality of life among BPH patients treated for 6 months with atorvastatin compared to controls [29]. This trial excluded men with diabetes. Based on our data, however, it is possible that statins may prove more useful for preventing BPH in diabetic men; perhaps a synergistic interaction of high LDL cholesterol with insulin resistance exists which predisposes diabetic men to BPH. This concept is plausible and consistent with cardiovascular disease among diabetes patients: elevated LDL significantly increases the already considerable risks of cardiovascular morbidity and mortality among diabetics, and maintenance of lower LDL concentrations with statins decreases these risks in this population [30].

In summary, these results suggest that higher LDL cholesterol might be a risk factor for clinical BPH among diabetic men. This observation is consistent with the concept that cardiac risk factors are involved with BPH pathogenesis, and raises the possibility that modulation of LDL cholesterol may possibly delay the development of BPH in diabetic men. Further studies of dyslipidemia and other acquired risk factors for BPH and LUTS are needed to define these relationships and externally validate our results.

Acknowledgements

Funding: This study was supported by research grants from the National Institutes of Health on Aging: AG07181 and the National Institute of Diabetes and Digestive and Kidney Diseases: DK31801. Dr. Parsons was supported in part by UCSD Faculty Senate Grant #RF821H.

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Table 1

Age and age-adjusted characteristics (95% confidence intervals) by BPH status among 531 men in the Rancho Bernardo Study 1992 to 1996

	NO BPH (N=272)	BPH (N=259)	p-value
Means			
Fasting plasma glucose (mg/dl) [†]	103.9 (101.5–106.3)	99.2 (96.8–101.6)	<0.01
Waist (cm)	95.5 (94.3–96.7)	94.1 (92.9–95.3)	0.10
Hip (cm)	102.7 (101.8–103.6)	102.0 (101.1–103.0)	0.31
Waist-to-hip-ratio	0.93 (0.923–0.937)	0.92 (0.915–0.929)	0.10
BMI (kg/m ²)	26.6 (26.2–27.0)	26.0 (25.5–26.4)	0.05
Total cholesterol (mg/dl)	195.1 (190.9–199.4)	196.1 (191.7–200.5)	0.76
LDL cholesterol (mg/dl)	121.0 (117.3–124.7)	124.1 (120.3–127.9)	0.25
HDL cholesterol (mg/dl)	49.0 (47.3–50.6)	49.1 (47.4–50.8)	0.93
Triglycerides (mg/dl) [†]	103.6 (96.5–111.1)	98.0 (91.2–105.4)	0.23
Proportions			
Total cholesterol ≥ 200 (mg/dl)	43.2 (37.3–49.2)	44.7 (38.6–50.9)	0.73
LDL cholesterol ≥ 130 mg/dl	36.4 (30.8–42.3)	40.6 (34.7–46.8)	0.33
HDL cholesterol < 40 mg/dl	29.6 (24.4–35.4)	24.7 (19.7–30.4)	0.21
Triglycerides ≥ 150 mg/dl	23.5 (18.7–29.0)	22.8 (18.0–28.5)	0.86
Obese (BMI > 30 kg/m ²)	12.7 (9.1–17.4)	11.0 (7.7–15.5)	0.55
Hypertension	34.6 (29.1–40.5)	38.2 (32.4–44.3)	0.40
Diabetes	19.4 (15.1–24.6)	13.6 (9.9–18.3)	0.08
History of stroke	5.1 (3.0–8.7)	2.7 (1.3–5.4)	0.11
History of TIA	4.1 (2.3–7.2)	6.1 (3.7–9.8)	0.30
On lipid lowering medication	8.4 (5.6–12.4)	11.3 (7.9–15.8)	0.26
Drinking alcohol ≥3 times/week	53.2 (47.2–59.2)	53.7 (47.6–59.8)	0.91
Ever smoked	69.7 (63.9–74.9)	67.0 (61.0–72.5)	0.52
Exercise ≥3 times/week	74.2 (68.6–79.1)	78.4 (73.0–83.1)	0.25

[†]geometric means (95% CI [Cox method]); p-value based on log-transformed data

Table 2

Odds ratio (with 95% confidence intervals) of BPH by tertile of serum lipid concentration among 531 men in the Rancho Bernardo Study 1992 to 1996[†]

	Tertile			p-trend
	1	2	3	
Total cholesterol				
Range (mg/dL)	< 180	180–207	>207	
OR (95% CI)	1	1.38 (0.90–2.12)	1.15 (0.75–1.77)	0.52
HDL-cholesterol				
Range (mg/dL)	>53	42–53	<42	
OR (95% CI)	1	1.15 (0.75–1.76)	0.88 (0.57–1.36)	0.56
Triglycerides				
Range (mg/dL)	<78	78–125	>125	
OR (95% CI)	1	0.83 (0.54–1.27)	0.80 (0.52–1.23)	0.30
Triglyceride/HDL				
Range	<1.53	1.53–2.93	>2.93	
OR (95% CI)	1	0.81 (0.53–1.24)	0.71 (0.46–1.10)	0.13

[†] models adjusted for age and lipid-lowering medication

Table 3

Odds ratio (with 95% confidence intervals) of BPH by tertile of serum LDL cholesterol concentrations with stratification by diabetes in the Rancho Bernardo Study 1992 to 1996[‡]

	Total (n=531)		No Diabetes (n=443)	Diabetes (n=88)
Tertile	Range (mg/dL)	OR (95% CI)	OR (95% CI)	OR (95% CI)
1	<110	1	1	1
2	110–133	1.10 (0.72–1.69)	0.96 (0.60–1.54)	2.46 (0.79–7.67)
3	>133	1.32 (0.86–2.02)	1.07 (0.67–1.71)	4.00 (1.27–12.63)
p-trend		0.20	0.77	0.02

[‡] model adjusted for age and use of lipid-lowering medication