HIGH SENSITIVITY CARDIAC TROPONIN-T IS ASSOCIATED WITH INCIDENT HYPERTENSION

by

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ABSTRACT

Background: Hypertension is often preceded by cardiac structural abnormalities. Thus, we assessed whether high-sensitivity cardiac troponin-T (hs-cTNT), a marker of chronic subclinical myocardial damage, can identify persons at risk for hypertension or its representative complication, left ventricular hypertrophy (LVH).

Methods: We studied 6,516 ARIC Study participants, free of prevalent hypertension and cardiovascular disease at baseline (1990-1992). We examined the association of baseline hs-cTNT categories with incident diagnosed hypertension (defined by self-report of a diagnosis or medication use during a maximum of 19.9 years of follow-up) and with incident visit-based hypertension (defined by self-report, medication use, or measured BP >140/90 mmHg over 6 years).

Results: Relative to hs-cTNT <5 ng/L, adjusted hazard ratios for incident diagnosed hypertension were 1.16 (95% CI 1.08, 1.25) for persons with hs-cTNT 5-8 ng/L, 1.29 (1.14, 1.47) for hs-cTNT 9-13 ng/L, and 1.31 (1.07, 1.61) for hs-cTNT ≥14 ng/L (p-trend <0.001). Associations were stronger for incident visit-based hypertension. These associations were driven by higher relative hazard in normotensive persons (relative to those with prehypertension, p-interaction=0.001). Baseline hs-cTNT was also strongly associated with incident LVH by electrocardiography over 6 years (e.g. adjusted HR 5.19 [1.49-18.08] for hs-cTNT ≥14 ng/L vs <5 ng/L). Findings were not appreciably changed

after accounting for competing deaths or adjustment for baseline BP levels or N-terminal prohormone of brain natriuretic peptide.

Conclusion: In an ambulatory population with no history of cardiovascular disease, hs-cTNT was associated with incident hypertension and risk of LVH. Further research is needed to determine whether hs-cTNT can identify persons who may benefit from ambulatory BP monitoring or hypertension prevention lifestyle strategies.

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INTRODUCTION

Hypertension remains a major cause of heart disease and stroke, with approximately 1 in 3 U.S. adults currently diagnosed as hypertensive and a further 6-10% of Americans estimated to have undiagnosed hypertension. [1] In addition, a significant proportion of those with diagnosed hypertension have poorly controlled blood pressure (BP). [2, 3] Thus, there is a need to identify persons at risk either for progressing to clinically overt hypertension or developing complications of this disease. In particular, BP can be improved by more aggressive lifestyle and dietary changes in those with both prehypertension and overt hypertension. [4]

The onset of hypertension is an insidious process that often occurs over many years and is often preceded by altered diurnal BP patterns and prehypertension. [5, 6] While hypertension is diagnosed using defined BP thresholds, risks associated with elevated BP occur on a continuous spectrum. Indeed, even early, pre-hypertensive, BP abnormalities can induce structural heart changes[7], potentially affording the opportunity to both detect persons at risk for overt hypertension and to initiate timely preventive strategies. [8] Prior studies have focused on the association between subclinical structural heart disease, particularly left ventricular hypertrophy (LVH), and subsequent development of hypertension. While these studies have confirmed that baseline LVH, measured by both

echocardiography[9, 10] and by cardiac MRI[11], is associated with incident hypertension, these imaging modalities are often impractical and too costly for routine screening.

New highly sensitive cardiac troponin T (hs-cTNT) assays have great potential as noninvasive laboratory-based markers of subclinical myocardial damage. These assays can detect subclinical myocardial damage in a significant proportion of persons who are free of known cardiovascular disease in the community. [12, 13] Interestingly, detectable levels of hs-cTNT appear to be more strongly linked to structural heart disease and its risk factors than to epicardial coronary artery disease. [13-15] Thus, hs-cTNT screening may be of particular use in persons at risk for clinically apparent hypertension and other cardiovascular endpoints. Hs-cTNT may also help identify individuals who are at risk of developing hypertension and/or hypertensive end-organ damage, such as LVH.

Thus, we sought to determine if hs-cTNT could identify persons at risk for subsequent hypertension in a large U.S. community-based cohort, the Atherosclerosis Risk in Communities (ARIC) Study.

METHODS

Study Population

The ARIC Study is a prospective cohort of 15,792 participants enrolled between 1987 and 1989 from four U.S. communities (Forsyth Country, North Carolina; Jackson, Mississippi; Minneapolis, Minnesota; and Washington County, Maryland). [16] We measured hs-cTNT in samples collected from all participants at ARIC visit 2, which took place from 1990 to 1992 and represents the baseline for this current analysis. Of the 14,348 persons who attended visit 2, we excluded all persons with prevalent diagnosed hypertension (n=6,334), those who had coronary heart disease (CHD, including silent myocardial infarction detected by electrocardiogram [ECG]), stroke, or heart failure at or prior to visit 2 (n=177), or those who were missing variables of interest (n=1,321) (Supplemental Figure 1, Appendix). Thus, 6,516 persons were included in the analysis evaluating our primary outcome of incident diagnosed hypertension (self-report of diagnosis or medication use during annual telephone follow-up). In our secondary analysis of incident visit-based hypertension, we further excluded 599 persons with measured BP at baseline >140/90 mmHg (undiagnosed hypertension) and 242 persons without available BP measurement data, for an analytic study sample of 5,675 persons. In our analysis of incident LVH, we further excluded 44 persons with baseline LVH by ECG for an analytic sample of 5,631 (Supplemental Table 1, Appendix). Institutional review boards at each clinical site reviewed the study and informed consent was obtained from all participants.

Measurement of hs-cTNT and other exposure variables

We measured hs-cTNT in stored serum samples, collected at visit 2, using a Roche Elecys 2010 Analyzer (Roche Diagnostics, Indianapolis, Indiana) at the University of Minnesota in 2012-2013. Coefficient of variation for hs-cTNT was 6.0% at a concentration of 25 ng/L. Participants self-reported smoking status. Plasma lipid concentrations and body weight and height for body mass index (BMI) were determined using standardized protocols. Glucose was measured using the hexokinase method.[17] Diagnosed diabetes was defined as a self-reported physician diagnosis of diabetes or current use of diabetic medications. Glomerular filtration rate was estimated using serum creatinine and the CKD-EPI 2009 equation.[18] N-terminal prohormone-brain natriuretic peptide (NT-proBNP) was measured in stored serum samples from visit 2 on a Roche Elecsys 2010 Analyzer using a sandwich immunoassay method (Roche Diagnostics, Indianapolis, Indiana).

Follow-up for outcomes of interest

Participants were contacted annually via telephone, with follow-up currently available through March 2012. Incident diagnosed hypertension was assessed during these annual telephone calls using the following questions: "Has a doctor ever said you had high blood pressure?", or "Since we last contacted you has a doctor said you had high blood pressure?", and "Did you take any medications during the past two weeks for high blood pressure?". After visit 2, ARIC participants also completed two further follow-up study examinations, visit 3 (1992-1995) and visit 4 (1996-1998). During these follow-up study

visits, BP was recorded as the mean of at least 2 seated measurements using a manual random-zero sphygmomanometer.

We also evaluated the association between hs-cTNT and a well-known sequela of hypertension, LVH. This secondary outcome was assessed at visits 3 and 4, in persons free of both hypertension and LVH at visit 2, using resting 12-lead electrocardiograms and defined by Cornell criteria. [19]

Statistical Analyses

Characteristics for the study population were presented according to categories of hs-cTNT (<5, 5-8, 9-13, or ≥14 ng/L) at baseline (visit 2, 1990-1992). While the hs-cTNT assay used can measure concentrations of troponin T as low as 3 ng/L[13], values <5 ng/L are measured with reduced precision and represent the typical lower cut-point in categorical analyses. Values ≥14ng/L represent approximately the 90th percentile of the ARIC population and correspond to the 99th percentile value for a "healthy" reference group of persons aged 20-70 years. [20]

The primary outcome of interest was incident diagnosed hypertension during a maximum of 19.9 and a median of 12 years of follow-up. Incident cases were identified at ARIC study visits 3 and 4 and, thereafter, by annual telephone contact with all participants. Time of incident diagnosed hypertension was the date when participants first reported diagnosis of or treatment for hypertension. We evaluated visit-based hypertension during a maximum of 8.7 and a median of 6 years follow-up as a secondary outcome. Visit-based cases of hypertension were identified by mean systolic BP ≥140mmHg or mean

diastolic BP ≥90mmHg, self-reported physician diagnosis, or medication use at visits 3 (1993-1995) or 4 (1996-1998). For both hypertension endpoints, we also conducted analyses stratified by baseline BP category (normotensive [BP ≤120 mmHg systolic and ≤80 mmHg diastolic] or pre-hypertensive [BP >120 mmHg <140 mmHg systolic and/or >80 mmHg <90 mmHg]). Finally, we evaluated the association between baseline hs-cTNT and incident LVH at visit 3 (1993-1995) or visit 4 (1996-1998) among persons free from both hypertension and LVH at visit 2 (1990-1992).

We used Cox proportional hazards regression models for the primary outcome of diagnosed hypertension and discrete proportional hazards (cloglog) regression models for both secondary outcomes (visit-based incident hypertension or incident LVH) to estimate adjusted hazard ratios (HRs) for the association between baseline hs-cTNT and these outcomes. We modeled hs-cTNT as a categorical (<5 [reference], 5-8, 9-13, or ≥14 ng/L) and continuous exposure (log-transformed). In analyses with hs-cTNT as a continuous exposure, hs-cTNT was modeled as untransformed or log-transformed and either truncated at <3ng/L (the limit of measurement) or with unmeasurable levels assigned a value of half the lower limit of measurement (1.5ng/L). [21] Using Martingale residuals to assess fit, the log-transformed visit 2 hs-cTNT without truncating fit the continuous models best. P-values for linear trend among hs-cTNT categories were obtained by assigning the median hs-cTNT value in the above categories of hs-cTNT and modeling this ordinal variable continuous. For all models, we verified the proportionality of the hazards with Schoenfeld residuals.

Models were adjusted for age (years), race-center (whites-Washington County; whites-Minneapolis; blacks-Jackson; blacks-Forsyth County, whites-Forsyth County), sex (male

or female), body mass index (kg/m^2), smoking (current; former; never), LDL-cholesterol (mg/dL), HDL-cholesterol (mg/dL), triglycerides (mg/dL), estimated glomerular filtration rate (mL/min/1.73 m^2), current lipid-lowering medication use (yes or no), LVH (yes or no), and diagnosed diabetes (yes or no). We tested for interactions by age, sex, and race. In sensitivity analyses we additionally adjusted for baseline (visit 2) blood pressure or NT-proBNP levels. Because baseline hs-cTNT is associated with all-cause mortality, cardiovascular death, and sudden cardiac death[13, 15, 22-24], we also conducted sensitivity analyses with a competing risk regression method (Fine-Gray approach) using a cumulative incidence function to account for intervening deaths. [25]

Finally, we also modeled hs-cTNT using linear splines in fully-adjusted Cox models for both incident diagnosed and incident visit-based hypertension, with knots at hs-cTNT concentrations of 5, 8 and 13 ng/L (figures truncated at the 99th percentile), These models are shown graphically and overlaid on histograms showing the distribution of hs-cTNT in the study population. All analyses were performed using Stata version 13.0 (College Station, TX: StataCorp LP).

RESULTS

In our community-based study population of subjects without a history of cardiovascular disease and free from baseline hypertension, persons with higher baseline hs-cTNT were more likely to be older, male, black, pre-hypertensive, obese, have higher HbA1c, and have reduced kidney function (Table 1).

During a maximum of 19.9 and a median 12 years of follow-up, 68% (n=4421) of the study sample developed our primary endpoint; incident diagnosed hypertension. Of these, 70% (n=3108) had hs-cTNT <5 ng/L at baseline, 21% (n=936) had hs-cTNT 5-8 ng/L, 6% (n=281) had hs-cTNT 9-13 ng/L, and 2% (n=96) had hs-cTNT ≥14 ng/L (Table 2). Crude incidence rates (per 1,000 person/years) of diagnosed hypertension were 54 in those with hs-cTNT <5 ng/L, 64 with hs-cTNT 5-8 ng/L, 73 with hs-cTNT 9-13 ng/L, and 74 in persons with baseline hs-cTNT ≥14 ng/L.

After multivariable adjustment, baseline hs-cTNT remained significantly associated with incident diagnosed hypertension (Table 2). Specifically, relative to those with undetectable hs-cTNT at baseline, persons in the higher categories of hs-cTNT had a higher adjusted risk of hypertension. Similar results were found for log-hs-cTNT modeled as a continuous variable. A continuous and roughly linear association between baseline hs-cTNT and incident diagnosed hypertension is shown graphically in Figure 1a.

The association between baseline hs-cTNT (both by category and as a continuous exposure) and our secondary outcome of incident visit-based hypertension was stronger than that for incident diagnosed hypertension (Table 2, Figure 1b). Further, in analyses stratified by baseline blood pressure category (normotensive or prehypertensive), we

observed that the overall association between hs-cTNT and both incident hypertension outcomes appeared to be largely driven by robust associations in persons who were normotensive at baseline (p-for-interaction=0.04 for diagnosed hypertension and p-for-interaction=0.001 for visit-based hypertension) (Table 3 and Supplemental Table 2 in the Appendix). Associations between hs-cTNT and both incident diagnosed and visit-based hypertension were largely unchanged after further adjustment for baseline blood pressure or NT-proBNP (Supplemental Table 3 in the Appendix). Interactions for the association of hs-cTNT and risk of hypertension by age, gender, and race were all were non-significant (all p-values-for-interaction >0.10).

There were 532 competing deaths for the primary outcome of incident diagnosed hypertension over 12-years follow-up, but only 57 competing deaths for the secondary outcome of incident visit-based hypertension over 6-years. In competing risk models, the association between hs-cTNT and incident diagnosed hypertension remained significant but was somewhat weakened in the hs-cTNT 5-8 ng/L and 9-13 ng/L categories and was no longer significant in those with baseline hs-cTNT ≥ 14 ng/L (Table 2). In contrast, results for the 6-year incident visit-based hypertension were similar in the Fine-Gray competing-risk models

Finally, we found that, relative to hs-cTNT of <5ng/L, persons in higher categories of hs-cTNT had a highly significant 6-year risk of incident LVH by ECG: HR, 2.29 (95% CI 1.24-4.26) for hs-cTNT 5-8ng/L, HR, 2.94 (95% CI 1.14-7.58) for hs-cTNT 9-13ng/L and HR, 5.19 (95% CI 1.49-18.08) for hs-cTNT ≥14ng/L (Table 4).

DISCUSSION AND CONCLUSIONS

In this large community-based study population of U.S. adults, free of baseline hypertension and cardiovascular disease, we found that hs-cTNT was independently associated with subsequent development of hypertension, defined either by diagnosed cases (self-reported diagnosis or medication use) or by objective office measurement of BP (which also captured undiagnosed hypertension), and incident LVH by ECG. Our findings were similar by age, race and gender. Our results suggest that chronic subclinical myocardial damage, detected by elevated hs-cTNT, may precede the development of hypertension in the general population and that this novel biomarker of cardiac damage may have utility for identifying persons at future risk for hypertension and hypertensive end-organ damage.

Prior studies have reported the cross-sectional association between established hypertension and prevalent hs-cTNT elevations[15, 26-28]. However, to our knowledge, this is the first study to evaluate the association between preceding subclinical myocardial damage, as measured by hs-cTNT, and subsequent risk of hypertension over prospective follow-up. Notably, this association was strongest for persons who had baseline normal BP readings (in contrast to those with pre-hypertension). Notably, the vast majority of individuals with prehypertension go on to develop hypertension, irrespective of baseline hs-cTNT. Therefore, the absolute risk of hypertension is very high in this entire sub-group, helping to explain why the relative impact of hs-cTNT was non-significant in those with prehypertension.

While the exact pathway linking hs-cTNT to incident hypertension is unclear, a number of considerations are worth discussing. Elevated BP develops over time along a spectrum from normotension, to abnormal diurnal patterns of BP, to pre-hypertension, and then, ultimately, to clinical hypertension[5]. It is known that abnormalities in cardiac structure can occur long before the diagnosis of overt hypertension is made in the clinic[5, 9, 29]. Based on our results, it is possible that these early BP aberrations cause myocardial damage, leading to elevated hs-cTNT, before clinically overt hypertension is recognized. Compatible with this hypothesis, it has been shown elsewhere that "non-dippers" (an abnormal diurnal BP pattern where sleep systolic BP fails to decrease >10% from daytime systolic BP and a known risk-factor for subsequent hypertension[6]), are more likely to have prevalent detectable troponin levels by hs-cTNT assays[30].

In addition, BP is highly variable and its true hemodynamic load cannot easily be captured by single office measurements (which are subject to misclassification error). Thus, it is also possible that the association between hs-cTNT and subsequent hypertension in ARIC could be a manifestation of the well-known phenomenon of "masked hypertension," the clinical condition in which a patient's office BP level is <140/90 mm Hg but home or ambulatory readings are in the hypertensive range[31]. Masked hypertension occurs in over 10% of the adult population[31, 32] and is known to result in end organ damage[33, 34].

Further, we found that elevated hs-cTNT was also a risk factor for the development of LVH in persons without baseline hypertension or clinical cardiovascular disease (including silent MI). This suggests that hs-cTNT may also predict future sequelae of hypertension and is consistent with our hypothesis that elevated hs-cTNT may reflect

occult BP abnormalities in some individuals. Indeed, prior case-control data suggest that hs-cTNT is also associated with subsequent albuminuria in hypertensive patients. [35] It is also worth noting that hs-cTNT could potentially be contributing more directly to the development of hypertension. For example, elevated hs-cTNT has been linked with arterial stiffness in diabetics[36].

Our results were robust to multiple sensitivity analyses. Standard Cox proportional-hazards models assume that persons censored (including those censored for death) prior to the end of follow-up have the same probability of incident hypertension as similar persons who remain under-observation. However, in persons who die, the development of hypertension cannot be observed. In this context, the Fine-Gray regression results, which account for interval deaths, are important to determine the 'real-world' impact of hs-cTNT screening in persons at risk for hypertension. In these Fine-Gray models, we found that the association between hs-cTNT and incident diagnosed hypertension (an endpoint with longer follow-up and increased interval accrual of deaths) was attenuated, particularly in the ≥14 ng/L category.

Our findings may have implications for future clinical practice. Specifically, normotensive persons with elevated hs-cTNT are at risk for subsequent hypertension and may benefit from evaluation for masked hypertension and from more intensive lifestyle BP interventions to reduce the likelihood of developing this morbid disease. Further research is necessary to confirm whether hs-cTNT could be useful in screening those at risk for hypertension.

This analysis has some limitations. This study was observational and, thus, may be subject to residual confounding. We do not have ambulatory BP recordings to determine whether or not masked hypertension or abnormal diurnal values were present in persons with elevated hs-cTNT. Cases of diagnosed hypertension were identified by self- report of diagnoses or medication used during annually follow-up; however, similar results were obtained in our analyses of visit-based hypertension (incorporating undiagnosed cases identified by elevated blood pressured obtained during the clinical visit). Strengths of the study include the large sample size, bi-racial population, and rigorous measurement of cardiovascular risk factors.

In conclusion, elevated hs-cTNT in the general population, particularly among persons with normal blood pressure, identifies persons at risk for subsequent hypertension or LVH. Further studies are necessary to determine whether subclinical myocardial damage in these settings is due to masked hypertension or abnormal diurnal variability in BP. Elevated hs-cTNT in low-risk ambulatory populations may prove to be clinically useful in identifying persons at risk for hypertension, affording the opportunity both to consider ambulatory blood pressure monitoring and to initiate more intensive preventive strategies.

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Table 1. Characteristics of the Study Population (ARIC participants without cardiovascular disease or diagnosed hypertension): Overall and According to categories of high sensitivity cardiac troponin T (ng/L) at baseline (1990-1992)

	Overall	Stratified by hs-cTNT			
		<5 ng/L	5-8 ng/L	9-13 ng/L	≥14 ng/L
Number (%)	6516 (100%)	4681 (71.8%)	1317 (20.2%)	384 (5.9%)	134 (2.1%)
Age, years	56.1 (5.6)	55.3 (5.4)	57.5 (5.7)	59.2 (5.6)	58.5 (5.7)
Male %	44.1	35.8	61.4	74.2	76.1
Black %	17.3	16.4	17.5	25.3	23.9
Current smoker %	22.8	24.8	17.8	16.9	20.1
Systolic blood pressure mmHg	115.2 (15.1)	113.8 (14.6)	117.6 (15.5)	120.4 (14.9)	124.0 (19.4)
Diastolic blood pressure mmHg	69.7 (9.1)	69.3 (9.0)	70.7 (9.4)	70.8 (9.1)	70.7 (9.6)
Hypertension categories (%)					
Normotension (BP <120/80 mmHg)	66.3	69.6	59.6	55.5	48.5
Prehypertension (BP 120-139/80-89 mmHg)	27.4	25.2	32.2	34.6	35.1
Undiagnosed hypertension (BP >140/90mmHg)*	6.3	5.2	8.2	9.9	16.4
LVH %	0.9	0.7	0.9	2.3	3.0
BMI, kg/m^2					
Normal weight % (< 25)	39.0	41.3	33.4	32.6	29.9
Overweight % (25 - 30)	41.1	39.9	44.6	42.7	42.5
Obese % (> 30)	20.0	18.8	22.0	24.7	27.6
Total cholesterol, mg/dL	207.4 (37.6)	208.1 (37.7)	206.4 (36.8)	202.8 (38.1)	205.8 (40.6)
LDL-cholesterol, mg/dL	131.8 (35.9)	131.5 (36.3)	132.9 (34.7)	131.1 (35.7)	131.2 (37.1)
HDL-cholesterol, mg/dL	51.5 (16.9)	52.8 (17.0)	48.4 (15.8)	47.1 (15.6)	49.3 (18.9)
Triglyceride, mg/dL	120.6 (61.0)	118.8 (59.4)	125.5 (63.9)	123.1 (66.6)	126.5 (67.9)
Lipid Medicines %	3.3	3.4	3.5	2.1	1.5
Diagnosed diabetes %	4.2	3.4	4.6	9.6	14.9
eGFR <60 mL/min/1.73m^2 (%)	0.5	0.4	0.6	0.5	4.5

Estimates are mean (SD) or %, unless otherwise indicated. Hs-cTNT= high-sensitivity Troponin-T, BP= Blood Pressure, LVH= left ventricular hypertrophy, BMI= body mass index, LDL= Low Density Lipoprotein, HDL= High Density Lipoprotein, eGFR= estimated Glomerular Filtration Rate.

*This group was excluded for the visit-based hypertension outcome analysis

Table 2. Crude incidence rates and adjusted* hazard ratios (95% confidence intervals) for incident hypertension outcomes, according to baseline high sensitivity cardiac troponin T

				Proportional Hazards Regression†	Competing Risks Regression‡
Baseline hs-cTnT	**N**	**Events (n)**	**Incidence rate, per 1,000 person years (95% CI)**	**HR (95% CI)**	**HR (95% CI)**
Incident Diagnosed Hypertension					
Categories					
<5 ng/L	4,681	3,108	54.0 (52.2-56.0)	1 (reference)	1 (reference)
5-8 ng/L	1,317	936	64.0 (60.0-68.3)	1.16 (1.08-1.25)	1.15 (1.06-1.24)
9-13 ng/L	384	281	72.6 (64.6-81.6)	1.29 (1.14-1.47)	1.21 (1.05-1.38)
≥14 ng/L	134	96	73.9 (60.5-90.2)	1.31 (1.07-1.61)	1.15 (0.91-1.44)
p-value for linear trend				*<0.001*	*<0.001*
Continuous					
Log(hs-cTnT)	6,516	4421	57.2 (55.5-58.9)	1.14 (1.09-1.19)	1.11 (1.06-1.16)
Incident Visit-based Hypertension					
Categories					
<5 ng/L	4,139	1,059	47.1 (44.4-50.0)	1 (reference)	1 (reference)
5-8 ng/L	1,130	351	58.6 (52.8-65.1)	1.15 (1.01-1.30)	1.14 (1.00-1.29)
9-13 ng/L	306	120	74.9 (62.6-89.5)	1.32 (1.08-1.61)	1.38 (1.13-1.67)
≥14 ng/L	100	41	77.8 (57.3-105.6)	1.49 (1.09-2.05)	1.47 (1.07-2.01)
p-value for linear trend				*<0.001*	*<0.001*
Continuous					
Log(hs-cTnT)	5,675	1,571	51.3 (48.9-53.9)	1.14 (1.06-1.22)	1.15 (1.07-1.24)

* Adjusted for age (years), race-center (whites-Washington County; whites-Minneapolis; blacks-Jackson; blacks-Forsyth County, whites-Forsyth County), sex (male or female), body mass index (kg/m2), smoking (current; former; never), LDL-cholesterol (mg/dL), HDL-cholesterol (mg/dL), triglycerides (mg/dL), estimated glomerular filtration rate (mL/min/1.73m2), lipid-lowering medications (yes or no), left ventricular hypertrophy (yes or no), diagnosed diabetes (yes or no).
†Cox regression for diagnosed hypertension outcome, cloglog regression for visit-based hypertension
‡Fine-Gray regression model. [25] There were 532 interval deaths for the diagnosed hypertension outcome and 57 interval deaths for the visit-based hypertension outcome prior to administrative censoring.

Table 3. Crude incidence rates and adjusted* hazard ratios (95% confidence intervals) for incident diagnosed hypertension, according to baseline categories of high sensitivity cardiac troponin T: further stratified by baseline blood pressure (N=6,516)

				Proportional Hazards Regression†	Competing Risks Regression‡
Baseline hs-cTNT	**N**	**Events (n)**	**Incidence rate, per 1,000 person years (95% CI)**	**HR (95% CI)**	**HR (95% CI)**
NORMOTENSIVE SUBGROUP (BP ≤120 mmHg systolic and ≤80 mmHg diastolic)					
Categorical					
<5 ng/L	3,259	1,892	42.4 (40.6-44.4)	1 (reference)	1 (reference)
5-8 ng/L	785	480	47.9 (43.8-52.3)	1.13 (1.02-1.25)	1.12 (1.01-1.25)
9-13 ng/L	213	134	53.1 (44.8-62.9)	1.24 (1.03-1.48)	1.18 (0.97-1.43)
≥14 ng/L	65	41	54.1 (39.9-73.5)	1.39 (1.01-1.90)	1.29 (0.93-1.79)
p-value for linear trend				*<0.001*	*0.006*
Continuous					
Log(hs-cTnT)	4,322	2,547	44.0 (42.3-45.7)	1.13 (1.06-1.20)	1.11 (1.04-1.18)
PREHYPERTENSION SUBGROUP (BP >120 <140 mmHg systolic and/or >80<90 mmHg diastolic)					
Categorical					
<5 ng/L	1,179	988	87.4 (82.1-93.0)	1 (reference)	1 (reference)
5-8 ng/L	424	356	90.6 (81.7-100.6)	1.11 (0.98-1.26)	1.11 (0.98-1.25)
9-13 ng/L	133	113	99.7 (82.9-119.9)	1.28 (1.05-1.58)	1.20 (0.97-1.48)
≥14 ng/L	47	33	78.6 (55.9-110.6)	0.88 (0.61-1.25)	0.72 (0.48-1.08)
p-value for linear trend				*0.125*	*0.667*
Continuous					
Log(hs-cTnT)	1,783	1,490	88.8 (84.4-93.4)	1.07 (0.99-1.15)	1.03 (0.96-1.11)
UNDIAGNOSED HYPERTENSION SUBGROUP (BP ≥140 mmHg systolic and/or ≥90mmHg diastolic)					
Categorical					
<5 ng/L	243	228	138.1 (121.3-157.3)	1 (reference)	1 (reference)
5-8 ng/L	108	100	150.1 (123.4-182.6)	1.14 (0.89-1.46)	1.10 (0.84-1.44)
9-13 ng/L	38	34	158.5 (113.2-221.8)	1.31 (0.90-1.93)	1.12 (0.76-1.65)
≥14 ng/L	22	22	180.1 (118.6-273.5)	1.45 (0.91-2.32)	1.53 (1.04-2.26)
p-value for linear trend				*0.045*	*0.088*
Continuous					
Log(hs-cTnT)	411	384	144.7 (130.9-159.9)	1.14 (1.00-1.30)	1.10 (0.96-1.26)

* Adjusted for age (years), race-center (whites-Washington County; whites-Minneapolis; blacks-Jackson; blacks-Forsyth County, whites-Forsyth County), sex (male or female), body mass index (kg/m2), smoking (current; former; never), LDL-cholesterol (mg/dL), HDL-cholesterol (mg/dL), triglycerides (mg/dL), estimated glomerular filtration rate (mL/min/1.73m2), current lipid-lowering medication use (yes or no), left ventricular hypertrophy (yes or no), diagnosed diabetes (yes or no). Abbreviations as per Table 1.

†Cox regression for diagnosed hypertension outcome

‡Fine-Gray regression model. [25] There were 532 interval deaths for the diagnosed hypertension outcome prior to administrative censoring.

Table 4. Adjusted* Hazard Ratios (95% confidence intervals) for the secondary outcome of incident left ventricular hypertrophy over 6 years of follow-up by baseline high sensitivity cardiac troponin T among persons free of baseline cardiovascular disease or hypertension (N=5,631)

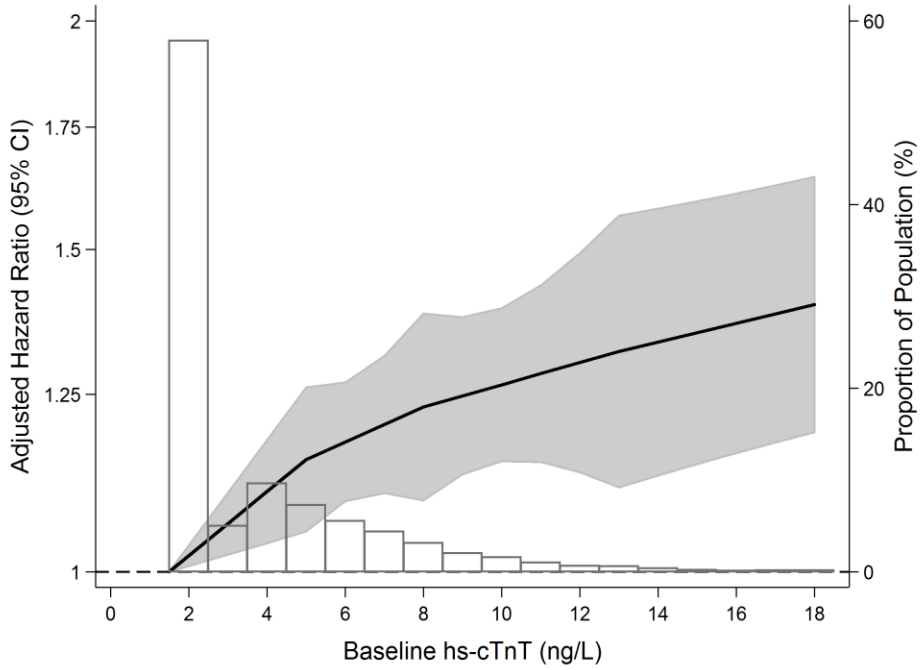
			Proportional Hazards Regression†
Baseline hs-cTnT	**N**	**Events (n)**	**HR (95% CI)**
Categorical			
<5 ng/L	4109	30	1 (reference)
5-8 ng/L	1121	17	2.29 (1.24-4.26)
9-13 ng/L	302	6	2.94 (1.14-7.58)
≥14 ng/L	99	3	5.19 (1.49-18.08)
p-value for trend			*<0.001*
Continuous			
Log(hs-cTnT)	5,631	56	1.79 (1.27-2.52)

* Adjusted for age (years), race-center (whites-Washington County; whites-Minneapolis; blacks-Jackson; blacks-Forsyth County, whites-Forsyth County), sex (male or female), body mass index (kg/m2), smoking (current; former; never), LDL-cholesterol (mg/dL), HDL-cholesterol (mg/dL), triglycerides (mg/dL), estimated glomerular filtration rate (mL/min/1.73m2), current lipid-lowering medication use (yes or no), diagnosed diabetes (yes or no). Abbreviations as per Table 1.

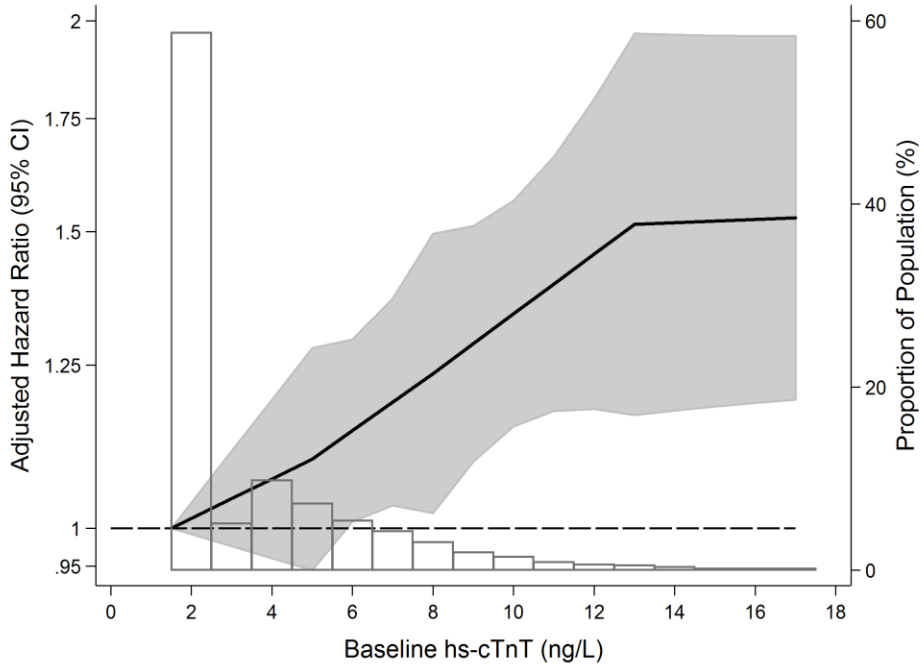
†Cloglog regression for incident LVH by ECG

Figure 1. Adjusted* hazard ratio (95% confidence interval) of A) incident diagnosed hypertension and B) incident visit-based hypertension, according to baseline hs-cTnT modeled as linear splines (knots at 5, 8 and 13 ng/L) with background histogram of hs-cTNT in the study sample of ARIC subjects without baseline cardiovascular disease or hypertension

A)



B)

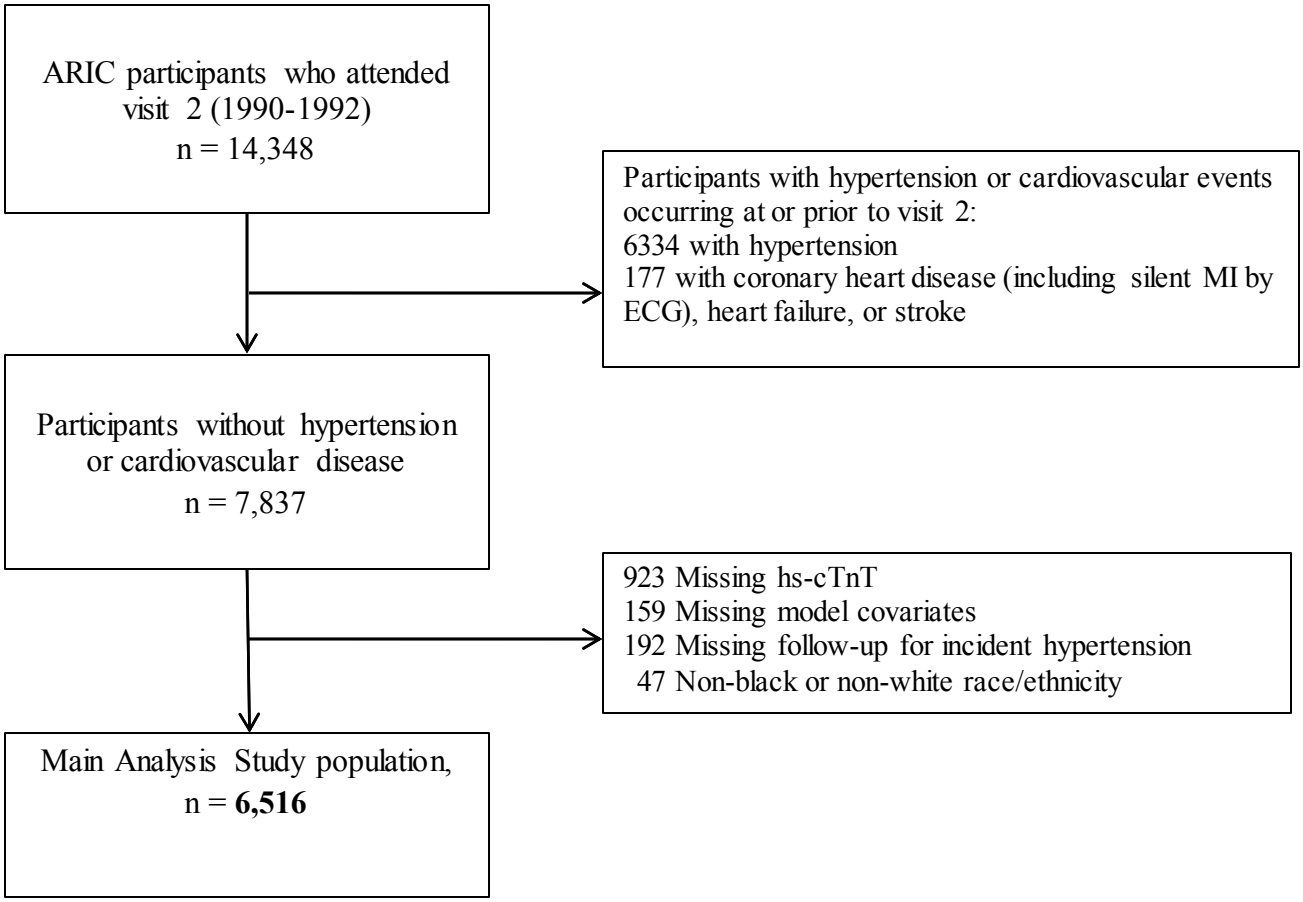


* Adjusted for age (years), race-center (whites-Washington County; whites-Minneapolis; blacks-Jackson; blacks-Forsyth County, whites-Forsyth County), sex (male or female), body mass index (kg/m2), smoking (current; former; never), LDL-cholesterol (mg/dL), HDL-cholesterol (mg/dL), triglycerides (mg/dL), estimated glomerular filtration rate (mL/min/1.73m2), current lipid-lowering medication use (yes or no), left ventricular hypertrophy (yes or no), diagnosed diabetes (yes or no). Abbreviations as per Table 1.

The distribution was truncated at the 99th percentile of hs-cTnT (18 ng/L)

APPENDIX

Supplemental Figure 1. Flow Chart of exclusion process for the main study sample used to assess the primary outcome of Diagnosed Hypertension, drawn from ARIC subjects free of baseline hypertension or cardiovascular disease



Supplemental Table 1- Study Sample Exclusion Process for Secondary Outcomes of interest

Exclusion Process	Exclusion	Population
1- Visit-Based Hypertension		
Attended visit 2		14348
Missing hs-cTnT data at visit 2	923	13426
Prevalent diagnosed hypertension at or before visit 2	6334	7091
Prevalent undiagnosed hypertension at or before visit 2	599	6492
Prevalent CHD, HF, stroke	177	6315
Missing follow-up hypertension information after visit 2	192	6123
Missing follow-up BP information at visits 2 and 4	242	5881
Missing visit 2 variables of interest	159	5722
Non black or white, race-center exclusion	47	**5675**
2- 6-year risk of incident LVH		
Attended visit 2		14348
Missing hs-cTnT data at visit 2	923	13426
Prevalent diagnosed hypertension at or before visit 2	6334	7091
Prevalent undiagnosed hypertension at or before visit 2	599	6492
Prevalent CHD, HF, stroke	177	6315
Missing follow-up hypertension information after visit 2	192	6123
Missing follow-up BP information at visits 2 and 4	242	5881
Missing visit 2 variables of interest	159	5722
Non black or white, race-center exclusion	47	5675
Prevalent LVH by EKG (visit 1 or 2)	44	**5631**

Supplemental Table 2. Crude incidence rates and adjusted* hazard ratios (95% confidence intervals) for incident visit-based hypertension, according to baseline categories of high sensitivity cardiac troponin T: further stratified by baseline blood pressure (N=5,479)

				Proportional Hazards Regression†	Competing Risks Regression‡
Baseline hs-cTNT	**N**	**Events (n)**	**Incidence rate, per 1,000 person years (95% CI)**	**HR (95% CI)**	**HR (95% CI)**
NORMOTENSIVE SUBGROUP (BP ≤120 mmHg systolic and ≤80 mmHg diastolic)					
Categorical					
<5 ng/L	3,088	515	29.8 (27.4-32.5)	1 (reference)	1 (reference)
5-8 ng/L	753	156	37.5 (32.0-43.8)	1.17 (0.97-1.41)	1.16 (0.96-1.41)
9-13 ng/L	200	57	52.4 (40.4-68.0)	1.45 (1.09-1.93)	1.56 (1.17-2.09)
≥14 ng/L	62	20	57.6 (37.1-89.2)	1.86 (1.18-2.94)	1.72 (1.11-2.69)
p-value for linear trend				*<0.001*	*<0.001*
Continuous					
Log(hs-cTnT)	4,103	748	32.7 (30.5-35.2)	1.20 (1.08-1.33)	1.22 (1.09-1.35)
PREHYPERTENSION SUBGROUP (BP >120 <140 mmHg systolic and/or >80<90 mmHg diastolic)					
Categorical					
<5 ng/L	1,051	544	104.2 (95.8-113.3)	1 (reference)	1 (reference)
5-8 ng/L	377	195	107.0 (93.0-123.2)	1.03 (0.87-1.22)	1.05 (0.88-1.25)
9-13 ng/L	106	63	122.2 (95.5-156.5)	1.18 (0.90-1.56)	1.18 (0.91-1.54)
≥14 ng/L	38	21	116.9 (76.2-179.3)	1.19 (0.76-1.87)	1.29 (0.83-2.02)
p-value for linear trend				*0.226*	*0.133*
Continuous					
Log(hs-cTnT)	1,572	823	106.4 (99.3-113.9)	1.03 (0.93-1.14)	1.05 (0.95-1.15)

* Adjusted for age (years), race-center (whites-Washington County; whites-Minneapolis; blacks-Jackson; blacks-Forsyth County, whites-Forsyth County), sex (male or female), body mass index (kg/m2), smoking (current; former; never), LDL-cholesterol (mg/dL), HDL-cholesterol (mg/dL), triglycerides (mg/dL), estimated glomerular filtration rate (mL/min/1.73m2), current lipid-lowering medication use (yes or no), left ventricular hypertrophy (yes or no), diagnosed diabetes (yes or no). Abbreviations as per Table 1.

†Cox regression for diagnosed hypertension outcome, cloglog regression for visit-based hypertension ‡Fine-Gray regression model. [1] There were 532 interval deaths for the diagnosed hypertension outcome and 57 interval deaths for the visit-based hypertension outcome prior to administrative censoring.

Supplemental Table 3. Adjusted* Cox Hazard Ratios (HRs) for incident diagnosed hypertension (N=6,516) and for visit-based incident hypertension (N=5675) by baseline hs-cTnT status, with additional adjustment for either baseline mean BP or NT-proBNP

Additional Correction for Baseline (Visit 2) Mean BP									
Diagnosed hypertension					**Visit Based hypertension**				
Visit 2 hs-cTnT	**N**	**Events (n)**	**HR (95% CI)**	**p-value**	**Visit 2 hs-cTnT**	**N**	**Events (n)**	**HR (95% CI)**	**p-value**
<5 ng/L	4,681	3,108	1 (ref.)		<5 ng/L	4,139	1,059	1 (ref.)	
5-8 ng/L	1,317	936	1.12 (1.04-1.21)	0.004	5-8 ng/L	1,130	351	1.16 (1.07-1.25)	<0.001
9-13 ng/L	384	281	1.27 (1.12-1.45)	<0.001	9-13 ng/L	306	120	1.29 (1.13-1.46)	<0.001
≥14 ng/L	134	96	1.20 (0.98-1.48)	0.083	≥14 ng/L	100	41	1.26 (1.02-1.55)	0.032
p-value for trend			*<0.001*		*p-value for trend*			*<0.001*	
Log(v2 hs-cTnT)	6,516	4,421	1.11 (1.07-1.16)	<0.001	Log (hs-cTnT)	5,675	1,571	1.13 (1.08-1.18)	<0.001
Additional Correction for Baseline (Visit 2) NT-proBNP									
Diagnosed hypertension					**Visit Based hypertension**				
Visit 2 hs-cTnT	**N**	**Events (n)**	**HR (95% CI)**	**p-value**	**Visit 2 hs-cTnT**	**N**	**Events (n)**	**HR (95% CI)**	**p-value**
<5 ng/L	4,678	3,107	1 (ref.)		<5 ng/L	4,138	1,058	1 (ref.)	
5-8 ng/L	1,316	936	1.09 (0.96-1.24)	0.187	5-8 ng/L	1,129	351	1.11 (0.98-1.26)	0.099
9-13 ng/L	383	280	1.34 (1.10-1.64)	0.003	9-13 ng/L	305	119	1.33 (1.09-1.62)	0.006
≥14 ng/L	134	96	1.41 (1.02-1.93)	0.036	≥14 ng/L	100	41	1.34 (0.97-1.84)	0.075
p-value for trend			*<0.001*		*p-value for trend*			*0.002*	
Log(hs-cTnT)	6,511	4,419	1.12 (1.04-1.20)	0.002	Log (hs-cTnT)	5,672	1,569	1.13 (1.05-1.21)	<0.001

*Adjusted for age (years), race-center (whites-Washington County; whites-Minneapolis; blacks-Jackson; blacks-Forsyth County, whites-Forsyth County), sex (male or female), body mass index (kg/m2), smoking (current; former; never), LDL-cholesterol (mg/dL), HDL-cholesterol (mg/dL), triglycerides (mg/dL), estimated glomerular filtration rate (mL/min/1.73m2), current lipid-lowering medication use (yes or no), diagnosed diabetes (yes or no).

CURRICULUM VITAE

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Date of Preparation: March 26th, 2015

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Birth Place	Cork, Ireland
Citizenship	Irish
U.S. Visa Status	J1

Education

B.Med.Sc.	National University of Ireland, University College Cork campus 1999-2003 Bachelor of Medical Science 1st Class Honors, Mode A
MB BCh BAO	National University of Ireland, University College Cork campus 1999-2004 Bachelor of Medicine, Surgery and Obstetrics 1st Class Honors, Mode A
M.H.S. Candidate	Johns Hopkins University, Bloomberg School of Public Health 2013-2015 (pending) Master of Health Science, Cardiovascular Epidemiology concentration Thesis: High-sensitivity Troponin-T is Associated with Incident Hypertension

Academic Appointments

July 2004- June 2005	Intern in Medicine and Surgery	Cork University Hospital, Cork, Ireland
July 2005- January 2006	Visiting Resident, Internal Medicine	Mayo Clinic, Mayo Medical School, USA
July 2005- June 2007	Senior House Officer, Internal Medicine	Mater Misericordiae University Hospital, Dublin, Ireland
July 2007- June 2008	Registrar, Cardiology	Mater Misericordiae University Hospital, Dublin, Ireland
July 2008- June 2011	Osler Residency, Internal Medicine- Intern, Junior Assistant, Senior Assistant	Johns Hopkins Hospital, Baltimore, USA
July 2011- June 2014	Cardiovascular Medicine Fellowship	Johns Hopkins Hospital, Baltimore, USA
July 2014- present	Pollin Cardiovascular Prevention Fellowship	Johns Hopkins Hospital, Baltimore, USA
July 2014- present	Chief Fellow in Cardiology	Johns Hopkins Hospital, Baltimore, USA

Professional Certification

MRCPI	Certified Membership 2007
Royal College of Physicians in Ireland	
ABIM- Internal Medicine	Board Certified 2011
American Board of Internal Medicine	
ABIM- Cardiovascular Diseases	Board Certified 2014
American Board of Internal Medicine	
SCCT- Cardiovascular Computed Tomography	Board Certified 2014
Society of Cardiovascular CT Board certification	

Professional Affiliations

American Heart Association	2011- present
American Heart Association Council on Epidemiology and Prevention	2012- present
American College of Cardiology	2011- present
European Society of Cardiology	2011- present

Honors/Awards

September 2001	Title of College Scholar	University College Cork
September 2002	Undergraduate Scholarship, 1st place in class	University College Cork
September 2003	Undergraduate Scholarship, 1st place in class	University College Cork
September 2003	Pfizer Psychiatry Prize, 1st place in class	University College Cork
September 2003	Dr. H.H. Stewart Psychiatry Scholarship, 1st place in Ireland	National University of Ireland
September 2004	W. Kearney Obstetrics & Gynecology Prize, 1st place in class	University College Cork
September 2004	Dr. H.H. Stewart Surgery Scholarship, 2nd place in Ireland	National University of Ireland
June 2005	Intern of the Year Award	Cork University Hospital
June 2013	Johns Hopkins Fellows Teaching Award	Johns Hopkins Department of Medicine
August 2014	Mayo Clinic CVBR Fellows Scholarship	Mayo Medical School
October 2014	Dr. Brian Maurer Young Investigator Finalist	Irish Cardiac Society
November 2014	Northwestern National Cardiovascular Young Investigator Award, 2nd place	Northwestern University Chicago

Peer Reviewed Publications

Peer Reviewed Articles Published

McEvoy JW, Blaha MJ, Defilippis AP, Budoff MJ, Nasir K, Blumenthal RS, Jones SR. Coronary artery calcium progression: an important clinical measurement? A review of published reports. *J Am Coll Cardiol. 2010 Nov 9;56(20):1613-22.*

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Tota-Maharaj R, Blaha MJ, **McEvoy JW**, Blumenthal RS, Muse ED, Budoff MJ, Shaw LJ, Berman DS, Rana JS, Rumberger J, Callister T, Rivera J, Agatston A, Nasir K. Coronary artery calcium for the prediction of mortality in young adults <45 years old and elderly adults >75 years old. *Eur Heart J. 2012 Dec;33(23):2955-62.*

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Martin SS, Blaha MJ, Elshazly MB, Brinton EA, Toth PP**, McEvoy JW**, Joshi PH, Kulkarni KR, Mize PD, Kwiterovich PO, Defilippis AP, Blumenthal RS, Jones SR. Friedewald-estimated versus directly measured low-density lipoprotein cholesterol and treatment implications. *J Am Coll Cardiol. 2013 Aug 20;62(8):732-9.*

Chrispin J, Martin SS, Hasan RK, Joshi PH, Minder CM, **McEvoy JW**, Kohli P, Johnson AE, Wang L, Blaha MJ, Blumenthal RS. Landmark lipid-lowering trials in the primary prevention of cardiovascular disease. *Clin Cardiol. 2013 Sep;36(9):516-23.*

Elshazly MB, Martin SS, Blaha MJ, Joshi PH, Toth PP, **McEvoy JW**, Al-Hijji MA, Kulkarni KR, Kwiterovich PO, Blumenthal RS, Jones SR. Non-high-density lipoprotein cholesterol, guideline targets, and population percentiles for secondary prevention in 1.3

million adults: the VLDL-2 study (very large database of lipids). *J Am Coll Cardiol. 2013 Nov 19;62(21):1960-5.*

Martin SS, Blaha MJ, Toth PP, Joshi PH, **McEvoy JW**, Ahmed HM, Elshazly MB, Swiger KJ, Michos ED, Kwiterovich PO, Kulkarni KR, Chimera J, Cannon CP, Blumenthal RS, Jones SR. Very large database of lipids: rationale and design. *Clin Cardiol. 2013 Nov;36(11):641-8.*

Shaharyar S, Warraich H, **McEvoy JW**, Oni E, Ali SS, Karim A, Jamal O, Blaha MJ, Blumenthal RS, Fialkow J, Cury R, Budoff MJ, Agatston AA, Nasir K. Subclinical cardiovascular disease in plaque psoriasis: association or causal link? *Atherosclerosis. 2014 Jan;232(1):72-8.*

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Selvin E, Lazo M, Chen Y, Shen L, Rubin J, **McEvoy JW**, Hoogeveen RC, Sharrett AR, Ballantyne CM, Coresh J. Diabetes mellitus, prediabetes, and incidence of subclinical myocardial damage. *Circulation. 2014 Oct 14;130(16):1374-82.*

Kohli P, Whelton SP, Hsu S, Yancy CW, Stone NJ, Chrispin J, Gilotra NA, Houston B, Ashen MD, Martin SS, Joshi PH, **McEvoy JW**, Gluckman TJ, Michos ED, Blaha MJ, Blumenthal RS. Clinician's guide to the updated ABCs of cardiovascular disease prevention. *J Am Heart Assoc. 2014 Sep 22;3(5):e001098.*

Hung RK, Al-Mallah MH, **McEvoy JW**, Whelton SP, Blumenthal RS, Nasir K, Schairer JR, Brawner C, Alam M, Keteyian SJ, Blaha MJ. Prognostic value of exercise capacity in patients with coronary artery disease: the FIT (Henry Ford ExercIse Testing) project. *Mayo Clin Proc. 2014 Dec;89(12):1644-54*

McEvoy JW, Blaha MJ, DeFilippis AP, Lima JA, Bluemke DA, Hundley WG, Min JK, Shaw LJ, Lloyd-Jones DM, Barr RG, Budoff MJ, Blumenthal RS, Nasir K. Cigarette Smoking and Cardiovascular Events: Role of Inflammation and Subclinical Atherosclerosis from the Multiethnic Study of Atherosclerosis. *Arterioscler Thromb Vasc Biol. 2015 Jan 8. pii: ATVBAHA.114.304562. [Epub ahead of print]*

Peer Reviewed Commentaries and Letters Published

McEvoy JW, Margey R, Blake GJ. Long-term follow-up of the West of Scotland Coronary Prevention Study. *N Engl J Med. 2008 Jan 10;358(2):193; author reply 194-5.*

McEvoy JW. Evolutionary game theory: lessons and limitations, a cancer perspective. *Br J Cancer. 2009 Dec 15;101(12):2060-1; author reply 2062-3.*

McEvoy JW. Defibrillator implantation early after myocardial infarction. *N Engl J Med. 2010 Jan 21;362(3):269-70; author reply 270.*

McEvoy JW. Biventricular pacing. *N Engl J Med. 2010 Mar 11;362(10):956-7; author reply 958-9.*

McEvoy JW, Blaha MJ. Progression of coronary artery calcification: not down & out. *Arch Intern Med. 2010 Apr 26;170(8):735-6; author reply 736.*

McEvoy JW. Statins and risk of incident diabetes. *Lancet. 2010 Jun 19;375(9732):2139; author reply 2141-2.*

McEvoy JW. What is the prognostic value of a zero calcium score? Ask Bayes! *J Am Coll Cardiol. 2010 Aug 10;56(7):612; author replies 614-5.*

McEvoy JW. Coronary artery calcium score and cardiovascular event prediction. *JAMA. 2010 Aug 18;304(7):741-2; author reply 742.*

McEvoy JW, Blaha MJ, Blumenthal RS. Caveat emptor: the coronary calcium warranty. *J Am Coll Cardiol. 2010 Sep 14;56(12):997; author reply 997-8.*

McEvoy JW, Blaha MJ, Nasir K. "Metabolically benign" obesity: a wolf in sheep's clothing. *Atherosclerosis. 2011 Jul;217(1):74-6.*

McEvoy JW. Letter by McEvoy regarding article, "Pathogenesis of sudden unexpected death in a clinical trial of patients with myocardial infarction and left ventricular dysfunction, heart failure, or both". *Circulation. 2011 May 10;123(18):e585; author reply e586-7.*

McEvoy JW, Nasir K, Blumenthal RS. Calcium score reclassification: how should baseline risk be measured? *J Am Coll Cardiol. 2011 Jun 14;57(24):2456-7; author reply 2457.*

McEvoy JW. Statin therapy dose and risk of new-onset diabetes. *JAMA. 2011 Sep 28;306(12):1325-6; author reply 1326.*

McEvoy JW. Lifetime risks of cardiovascular disease. *N Engl J Med. 2012 Apr 26;366(17):1642; author reply 1642-3.*

McEvoy JW, Blaha MJ, Blumenthal RS. Rhinotillexis: a possible heuristic to reduce inappropriate noninvasive cardiac imaging? *J Am Coll Cardiol. 2012 Jul 31;60(5):432; author reply 433.*

McEvoy JW, Blumenthal RS, Blaha MJ. Statin therapy for hyperlipidemia. *JAMA. 2013 Sep 18;310(11):1184-5.*

McEvoy JW. The Turing test and a call to action to improve electronic health record documentation. *Am J Med. 2014 Jul;127(7):572-3.*

McEvoy JW, Nasir K. ABI and stroke: action at a distance and a call to action. *Atherosclerosis. 2014 May;234(1):73-4.*

McEvoy JW, Blaha MJ. Coronary artery calcium testing: exploring the need for a randomized trial. *Circ Cardiovasc Imaging. 2014 Jul;7(4):578-80*

McEvoy JW. The Turing test and EHR Documentation: The reply. *Am J Med. 2014 Dec;127(12):e23.*

Peer-reviewed Electronic Publications (online-only publications)

McEvoy JW. Frontiers. *University College Cork Medical Research Newsletter. Feb 2011.*

McEvoy JW. The Expert Is In: New Algorithm for Patients with LBBB and Suspected MI. *Cardioexchange: an NEJM practice community. July 2012.*

McEvoy JW. 2013: The Year of the Guideline. *Cardioexchange: an NEJM practice community. January 2014.*

McEvoy JW. Article of the Month: A review of the STAMPEDE trial. *ACC Cardiosource: Cardiometabolic Disease Community. May 2014.*

McEvoy JW. The Risk-Prediction Conundrum: Individual Risk vs. Population Risk. *Cardioexchange: an NEJM practice community. May 2014.*

McEvoy JW. Do Doctors Need a Better Way to Take Notes? *Cardioexchange: an NEJM practice community. August 2014.*

McEvoy JW. Debating the Lipid Guidelines. *American College of Cardiology Early Career Newsletter. Fall 2014, Vol 1, No. 9.*

McEvoy JW. Cardiometabolic Disease "Hot Topic": My thoughts regarding the article, "Further Insight Into the Cardiovascular Risk Calculator: The Roles of Statins, Revascularizations, and Underascertainment in the Women's Health Study" by Cook et al. *ACC Cardiosource: Cardiometabolic Disease Community. February 2015.*

Books and Book Chapters Published

McEvoy JW, Tota-Maharaj R, Blaha MJ. Metabolic Syndrome: Understanding the Epidemic in India and South Asia. Book. *MacMillan Medical Communications. 2013*

McEvoy JW, Blumenthal RS, Michos ED. Pathophysiology and Pharmacotherapy of Cardiovascular Disease. "Cardiac Prevention Guidelines". Chapter. *Springer Publications, Heidelberg, Germany. 2015.*

Selected Presentations

Oral Presentations

McEvoy JW, Blaha MJ, DeFilippis AP, Lima JA, Bluemke DA, Hundley WG, Min JK, Shaw LJ, Lloyd-Jones DM, Barr RG, Budoff MJ, Blumenthal RS, Nasir K. Cigarette Smoking: Association Between Inflammation, Subclinical Atherosclerosis and Cardiovascular Events. The Multi-Ethnic Study of Atherosclerosis (MESA). *American Heart Association Scientific Sessions, Oral Abstract 9726, November 2011.*

McEvoy JW, Blaha MJ, DeFilippis AP, Lima JA, Bluemke DA, Hundley WG, Min JK, Shaw LJ, Lloyd-Jones DM, Barr RG, Budoff MJ, Blumenthal RS, Nasir K. Cigarette Smoking: Association Between Inflammation, Subclinical Atherosclerosis and Cardiovascular Events. The Multi-Ethnic Study of Atherosclerosis (MESA). *Irish Cardiac Society Annual Conference Oral Presentation. October 2012.*

McEvoy JW. Screening for Coronary Artery Disease: A State of the Art. *Cork University Hospital Medical Grand Rounds, May 2013.*

McEvoy JW, Blaha MJ, DeFilippis AP, Lima JA, Bluemke DA, Hundley WG, Min JK, Shaw LJ, Lloyd-Jones DM, Barr RG, Budoff MJ, Blumenthal RS, Nasir K. Cigarette Smoking: Relationship with Inflammation, Arterial Stiffness, and Subclinical Atherosclerosis. The Multi-Ethnic Study of Atherosclerosis (MESA). *Irish Cardiac Society Annual Conference, Brian Maurer Young Investigator Finalist Oral Presentation. October 2014.*

McEvoy JW, Lazo M, Shen L, Nambi V, Hoogeveen RC, Ballantyne C, Blumenthal RS, Coresh J, Selvin E. Cardiac Risk Factors and 6-Year Change in high-sensitivity Cardiac Troponin-T from ARIC. *Northwestern University National Cardiovascular Young Investigator Finalist Oral Presentation. November 2014.*

McEvoy JW. High sensitivity troponin and hypertension- post hoc or propter hoc? *University of Maryland Cardiology Grand Rounds, January 2015.*

Poster Presentations

McEvoy JW, Blaha MJ, DeFilippis AP, Lima JA, Bluemke DA, Hundley WG, Min JK, Shaw LJ, Lloyd-Jones DM, Barr RG, Budoff MJ, Blumenthal RS, Nasir K. Cigarette Smoking: Relationship with Inflammation, Arterial Stiffness, and Subclinical Atherosclerosis. The Multi-Ethnic Study of Atherosclerosis (MESA). *American Heart Association Scientific Sessions, Poster Abstract 9713, November 2011.*

McEvoy JW, Lazo M, Shen L, Nambi V, Hoogeveen RC, Ballantyne CM, Blumenthal RS, Coresh J, Selvin E. Cardiac Risk Factors and 6-Year Change in high-sensitivity Cardiac Troponin-T from ARIC. *American Heart Association: Epidemiology and Prevention, Lifestyle and Cardiometabolic Health Scientific Sessions, Poster Abstract, March 2014.*

McEvoy JW, Martin SS, Blaha MJ, Joshi PH, Manning S, Blumenthal RS, Jones SR. The Relationship between Thyroid dysfunction and Advanced Lipoprotein Cholesterol Subfractions in a Contemporary Sample of US Adults: The Very Large Database of Lipids Thyroid Substudy. *American College of Cardiology Scientific Session, Poster Abstract, March 2014.*

McEvoy JW, Chen Y, Nambi V, Ballantyne CM, Sharrett AR, Appel LJ, Post WS, Blumenthal RS, Matsushita K, Selvin E. Baseline High-sensitivity Cardiac Troponin-T is independently associated with Incident Hypertension. *American Heart Association: Epidemiology and Prevention, Lifestyle and Cardiometabolic Health Scientific Sessions, Moderated Poster Abstract, March 2015 (accepted).*

Editorial Commitments

Editorial Board

Editorial Fellow, British Medical Journal
April 2013- present.
Supervisor: Jose Merino MD, BMJ U.S. Clinical Research Editor

Journal Peer Reviewer

British Medical Journal
New England Journal of Medicine
Journal of the American Medical Association
Archives of Internal Medicine (JAMA-Internal Medicine)
Journal of the American College of Cardiology
Journal of the American College of Cardiology: Cardiovascular Imaging
Circulation
Circulation: Cardiovascular Imaging
Atherosclerosis

Fellowship and Grant Support

Pollin Cardiovascular Prevention Fellowship. Annual Stipend and Tuition support for MHS Degree.

Co-investigator. *Patient Centered Outcomes Research Institute. Demonstrating respect and acceptable consent strategies: What matters to patients in PCOR?* Principle Investigator: Nancy Kass ScD. Funding amount: $1,678,250

Co-investigator. *Magic That Matters Fund, Johns Hopkins Division of Cardiology. Pathogenic HDL: An Alternative Lox-1 Receptor Ligand Induces Endothelial Dysfunction.* $15,000 Salary Support. Principle Investigator: Dan Berkowitz MD PhD.

Mentorship

Original Investigation Mentorship

Jaideep Patel, MD. Assistant Clinical Professor, Department of Internal Medicine, Virginia Commonwealth Medical Center

Amer I. Aladin, MD. Ciccarone Center for the Prevention of Heart Disease, Johns Hopkins School of Medicine, Baltimore, MD

Teaching Experience

Graduate Medical Education

Graduate Medical Education
Activity description: Lecture, "Cardiac Hemodynamics Lecture".
Role: Teacher/Presenter
Learners: 50 /year (undergraduate medical students and residents)
Time Frame: 2014-present

Graduate Medical Education
Activity description: Lecture, "Vasoactive Medications: 101".
Role: Teacher/Presenter
Learners: 50 /year (undergraduate medical students and residents)
Time Frame: 2014-present

Graduate Medical Education
Activity description: Lecture, "Anti-arrhythmic Medications".
Role: Teacher/Presenter
Learners: 50 /year (undergraduate medical students and residents)
Time Frame: 2014-present

Graduate Medical Education
Activity description: SIMULATION- Medical resident vascular access.
Role: Teacher/Presenter
Learners: 40 /year (Medical residents)
Time Frame: 2014-present

Graduate Medical Education
Activity description: Fellow's Noon Conference Didactic Lecture Series
Role: Director/ Organizer/ Facilitator
Learners: 30 /year (cardiology fellows)
Time Frame: 2014-2015. Two lectures per week over the academic year

Graduate Medical Education
Activity description: Fellow's Case Conference Series
Role: Contributor
Learners: 30 /year (cardiology fellows)
Time Frame: 2011-present. One conference per week over academic year

Summary of Teaching Activities:

- 500 hours/year contact time with students
- Lead sessions on varied topics in cardiology, discussed above.
- Provided clinical precepting for students and residents on both the General Cardiology and CCU clinical service