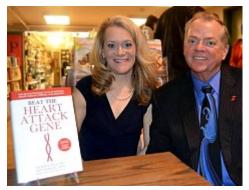
# Heart FALK

Heart-healthy and Stroke-free Living with Amy L. Doneen, MSN, ARNP

Vol 26 April 2014



# From Amy Doneen

Amy Doneen, MSN, ARNP

Happy Spring, everyone! I wanted to share our manuscript which was published in the Journal of Clinical Experimental Cardiology April 2014. This landmark publication is the first of its kind, suggesting that clinicians step up and stand behind their work. Our aim is to improve the medical system by holding ourselves accountable for our work by being optimal scholars of the science and study of arterial wall health. It remains a true honor and pleasure to work with you to insure that your health is maintained so you can enjoy a life of happiness and complete wellness.

I am happy to report that I successfully ran in this year's Boston Marathon. See last page for photos and a few notes about this historic event!

Finally, a quick update on our book, *Beat the Heart Attack Gene*. Feedback from patients, the public and the healthcare community has been terrific. We are into our second running and remain #1 on Amazon in Genetics. We are pleased it is now available via hardback, kindle, or audio. Thank you for your continued support in sharing our message of hope, survival, and optimal health.

Aту



# A Guarantee of Arterial Wellness: New Era of Cardio-vascular Medicine

Bale and Doneen, J Clin Exper Cardiol 2014,5:3. http://dx.doi.org/10.4112/2155-9880.1000298.
Bradley Field Bale1,2\* and Amy Lynn Doneen 2,3
1Heart Health Program, Grace Clinic, Lubbock, TX, USA
2Texas Tech Health Science Centre School of Nursing, USA
3Heart Attack, Stroke and Diabetes Prevention Clinic, Spokane, WA, USA
\*Corresponding author: Bradley Field Bale, 113 Shadowhaven Way N., Hendersonville, TN 37075, 1-509-951-9346, USA, Tel: 1-615-265-8998; Fax: 1-615-265-8998; Rec date: Mar 03, 2014, Acc date: Mar 27, 2014, Pub date: Apr 05, 2014

Copyright:  $\bigcirc$  2014 Bale, BF, and Doneen, AL. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

## Abstract

The door is open to change the United States platform of healthcare for Cardiovascular Disease (CVD) in terms of maintaining arterial wellness. Accomplishments in this arena of healthcare have been phenomenal, but the vast majority of them deal with end-stage arterial disease. The price tag has been substantial in terms of financial resources and quality of life. Current projections indicate CVD driven mainly by arterial disease will bankrupt our country. Those estimates are based on the current system of treating end-stage arterial disease. We can avoid fiscal insolvency and deteriorating quality of life by shifting to a platform that prevents arterial disease or, at minimum, treats it before it is evident. We live in an era of technology sufficient to identify subclinical arterial disease and knowledge necessary to halt atherosclerosis. For example, safe, inexpensive, painless, and reliable imaging techniques now exist that detect silent non-obstructing arterial plaque, or atheroma; a prerequisite for a majority of cardiovascular events. Additionally, numerous health issues are known to produce inflammation of the arteries, which is causal of atherosclerosis. Each can be managed effectively to extinguish the inflammation. When this is accomplished the disease will halt, which significantly mitigates risk for an acute obstructive cardiovascular event. CV healthcare can now enter a new era of maintaining arterial wellness. We must seize this opportunity for fiscal and humanitarian reasons.

See pages that follow for the complete article and references.

Heart Attack & Stroke Prevention Center

507 S. Washington, Suite 170 Spokane, Washington 99204 (509) 747-8000 www.TheHASPC.com



Cardiovascular (CV) healthcare was established on a platform that treats end-stage disease. At the time, existent technology and knowledge could only manage evident arterial disease. The financial and intellectual commitment in this area of healthcare have resulted in astonishing accomplishments such as coronary bypass grafting, angioplasty, stents, acute antithrombotic therapies, left ventricular assistant devices, transplants, and stem cell research. The consequence has been dramatic reduction in CV mortality. Unfortunately, the incidence of end-stage arterial disease has not been significantly impacted. In addition, the price tag for these phenomenal advances in therapy has been devastating. Based on the current system of healthcare, projections for the incidence and cost of CV disease over the next decade are dismal [1]. The current CV healthcare system is not financially sustainable [2]. Our system must enter a new era, anchored on a platform of maintaining arterial wellness. Fortunately, we now possess the technology and knowledge to do so. Traditional methods of dealing with Cardiovascular Events (CVE) are reactionary and often inefficient. For example, predicting CVEs on the basis of risk factors frequently fails to identify individuals that actually experience a heart attack or stroke [3]. Identifying arterial disease is a superior predictor of CVEs compared to the presence of a conventional risk factor because the presence of arterial disease in any arterial bed elevates event potential regardless of risk factors [4-6]. According to an important CAFES-CAVE study, individuals who harbor such disease are high risk without treatment for a cardiovascular event [7]. The issue has been the difficulty of detecting arterial disease prior to an event. This is in part due to the fact that while the presence of an atheroma is an essential ingredient for a cardiovascular event, the majority are non-obstructing prior to an atherothrombotic event [8-10]. Waiting for a CVE to prove the presence of arterial disease is no longer necessary. There are safe inexpensive means to detect subclinical arterial plaques [11].

The current era of medicine possesses several non-invasive, innocuous, low-cost means to detect atheroma. X-rays that demonstrate calcification in arterial beds documents the presence of arterial disease. This type of incidental finding is associated with higher CVE risk [12,13]. Ankle Brachial Index (ABI) testing for peripheral artery disease is simple and inexpensive. An abnormal ABI portends significant increased CVE risk [14]. Coronary Calcification (CAC) via Computerized Tomography (CT) is widely available, painless, and involves little radiation. A positive score documents the presence of atherosclerosis [15]. CAC has been associated with increased risk of CVEs [16,17]. Carotid intimal media thickness (CIMT) via B-mode ultrasound is safe, valid, reliable and lowpriced [18]. CIMT is associated with increased CVE risk [19]. This imaging technique can also identify carotid plaque, which is associated with increased CVE risk [20,21]. We have multiple reasonable modalities to utilize clinically to evaluate each patient for the

The CAFES-CAVE study referenced earlier demonstrated that patients with atherosclerosis who do not receive treatment have increased risk for CVEs. When arterial disease is identified, these individuals should be treated. Therapy should be directed to the cause of their arterial disease. Evidence shows that inflammation is causal of atherosclerosis and CVEs [22,23]. Numerous pathologies can contribute to arterial inflammation, which include lipids, smoking, hypertension, insulin resistance, vitamin D deficiency, obstructive sleep apnea, obesity, diet, physical inactivity, psychosocial issues, oral health issues, systemic inflammatory conditions such as rheumatoid arthritis, lupus, genetic influences, and systemic infectious disease [24-37]. Thankfully, therapies for all of these conditions are available, and evaluation of all potential "root" pathologies contributing to arterial inflammation can be performed in an economical manner with each patient.

A priori, halting arterial disease requires extinguishing arterial inflammation. There are objective inflammatory biomarkers that allow for clinical monitoring of arterial inflammation. The acute phase reactants fibrinogen and high sensitivity C Reactive Protein (hs-CRP) can indicate endothelial inflammation. These are as predictive for CVEs as high-density lipoprotein and total cholesterol [38]. Urinary microalbumin-creatinine ratio is an excellent biomarker of endothelial wellness and an independent predictor of CVEs [39,40]. Lipoprotein associated phospholipase A2 (Lp-PLA2) indicates inflammation in the arterial wall [41,42]. Oxidative stress can drive arterial inflammation, and the urinary biomarker F2-isoprostane is an excellent test for this [43]. Myeloperoxidase can result in inflammation of the endothelium and/or arterial wall [44]. The effectiveness of reducing arterial inflammation by managing the "root" inflammatory pathologies can be determined objectively by these serum and urinary biomarkers.

Emphasis upon preventative care has gained momentum over the past few years. The concept of clinically utilizing the aforementioned screening tests for subclinical atherosclerosis was promoted during a recent American Heart Association conference [45]. Subsequent to this publication, we now have appropriate use documents for CAC and CIMT [46-48]. In June 2009, the state of Texas passed the Texas Heart Attack Prevention Bill, which mandates health-benefit plans to provide coverage for CAC and CIMT testing [49]. Additionally, there is an increase in publications that discuss the merit of basing an individual's CVE risk on the detection of subclinical atherosclerosis with such tools as CAC [50]. Recent publications have also elucidated the importance of reducing arterial inflammation as measured by biomarkers discussed above [51-54]. Yet policy change has not kept pace. Despite these endorsements,

national health care policy does not yet mandate this type of arterial disease care. This is not surprising, as it is well known that translation of excellent science into the clinical arena can take decades [55]. Green and Seifert demonstrated there is too much delay in the utilization of innovative information by health care systems and providers. As a consequence, patients' outcomes suffer. They suggested health policies should support expert physicians who integrate novel evidence-based knowledge into practice [56].

Fortunately, healthcare providers do not need to wait for a national directive. In the arena of arterial disease, which is so costly from a humanitarian and financial standpoint, healthcare providers may choose, as the authors have, to enter this new era of arterial disease care now. It is exciting to practice medicine in an era where we possess clinical tools and knowledge allowing us to migrate to a more superior platform of healthcare. The platform that focuses on managing end-stage arterial disease is too expensive and devastating to an individual's wellness. We now have the opportunity to shift to a platform designed to prevent disease, or at minimum treating it before it is evident. We do not need to wait for huge randomized double blind prospective outcome studies to prove such a platform will be superior. We have no choice. We have proven that the current platform leads to insolvency. It is possible with a personalized, comprehensive, and holistic approach to determine the causes of the arterial disease in each patient. There are effective therapies for all the inflammatory conditions and adequate biomarkers to judge the effectiveness of the treatment. Arterial inflammation can be extinguished. The new era of CV healthcare can guarantee arterial wellness. The authors have been guaranteeing their patients' arterial wellness for years with the method articulated in their book, Beat the Heart Attack Gene: The Revolutionary Plan to Prevent Heart Disease, Stroke, and Diabetes [57]. We invite you to join us in this satisfying endeavor.

#### References

1.Go AS, Mozaffarian D, Roger VL, Benjamin EJ, Berry JD, et al. (2014)Heart disease and stroke statistics--2014 update: a report from the american heart association. Circulation 129:e28-e292.

 Weintraub WS, Daniels SR, Burke LE, Franklin BA, Goff DC Jr, et al. (2011) Value of primordial and primary prevention for cardiovascular disease: a policy statement from the American Heart Association. Circulation 124: 967-990.

3.Cook NR, Paynter NP, Eaton CB, Manson JE, Martin LW, et al. (2012) Comparison of the Framingham and Reynolds Risk scores for global cardiovascular risk prediction in the multiethnic Women's Health Initiative. Circulation 125: 1748-1756.

4. Martin SS, Blaha MJ, Blankstein R, Agatston AS, Rivera JJ, et al. (2014) Dyslipidemia, Coronary Artery Calcium, and Incident Atherosclerotic Cardiovascular Disease: Implications for Statin Therapy from the Multi-Ethnic Study of Atherosclerosis. Circulation 129: 77-86.

5.Sirimarco G, Lavallée PC, Labreuche J, Meseguer E, Cabrejo L, et al. (2013) Overlap of diseases underlying ischemic stroke: the ASCOD phenotyping. Stroke 44: 2427-2433.

6.Sirimarco G, Amarenco P, Labreuche J, Touboul PJ, Alberts M, et al. (2013) Carotid atherosclerosis and risk of subsequent coronary event in outpatients with atherothrombosis. Stroke 44: 373-379.

7.Belcaro G, Nicolaides AN, Ramaswami G, Cesarone MR, De Sanctis M, et al. (2001) Carotid and femoral ultrasound morphology screening and cardio-vascular events in low risk subjects: a 10-year follow-up study (the CAFES-CAVE study(1)). Atherosclerosis 156: 379-387.

8.Arbab-Zadeh A, Nakano M, Virmani R, Fuster V (2012) Acute coronary events. Circulation 125: 1147-1156.

9.Aldrovandi A, Cademartiri F, Arduini D, Lina D, Ugo F, et al. (2012) Computed tomography coronary angiography in patients with acute myocardial infarction without significant coronary stenosis. Circulation 126: 3000-3007.

10.Falk E, Shah PK, Fuster V (1995) Coronary plaque disruption. Circulation 92: 657-671.

11. Criqui MH, Alberts MJ, Fowkes FGR, Hirsch AT, O'Gara PT, et al. (2008) Atherosclerotic Peripheral Vascular Disease Symposium II: Screening for Atherosclerotic Vascular Diseases: Should Nationwide Programs Be Instituted? Circulation 118:2830-2836.

12.Rotter MA, Schnatz PF, Currier AA Jr, O'Sullivan DM (2008) Breast arterial calcifications (BACs) found on screening mammography and their association with cardiovascular disease. Menopause 15: 276-281.

13.Friedlander AH, Cohen SN (2007) Panoramic radiographic atheromas portend adverse vascular events. Oral Surg Oral Med Oral Pathol Oral RadiolEndod 103: 830-835.

14.Hirsch AT, Criqui MH, Treat-Jacobson D, Regensteiner JG, Creager MA, et al. (2001) Peripheral arterial disease tection, awareness, and treatment in primary care. JAMA 286: 1317-1324.

15.Budoff MJ, Achenbach S, Blumenthal RS, Carr JJ, Goldin JG, Greenland P, et al. (2006) Assessment of Coronary Artery Disease by Cardiac Computed Tomography: A Scientific Statement From the American Heart Association Committee on Cardiovascular Imaging and Intervention, Council on Cardiovascular Radiology and Intervention, and Committee on Cardiac Imaging, Council on Clinical Cardiology. Circulation 114:1761-1791.

16.Yeboah J, McClelland RL, Polonsky TS, Burke GL, Sibley CT, et al. (2012) Comparison of novel risk markers for improvement in cardiovascular risk assessment in intermediate-risk individuals. JAMA 308: 788-795.

17.Hermann DM, Gronewold J, Lehmann N, Moebus S, Jöckel KH, et al. (2013) Coronary artery calcification is an independent stroke predictor in

detection, awareness, and treatment in primary care. JAMA 286: 1317-1324.

18.Smith SC Jr, Greenland P, Grundy SM (2000) AHA Conference Proceedings. Prevention conference V: Beyond secondary prevention: Identifying the high-risk patient for primary prevention: executive summary. American Heart Association. Circulation 101: 111-116.

19.Baldassarre D, Hamsten A, Veglia F, de Faire U, Humphries SE, Smit AJ, et al. (2012) Measurements of carotid intima-media thickness and of interadventitia common carotid diameter improve prediction of cardiovascular events: results of the IMPROVE (Carotid Intima Media Thickness [IMT] and IMT-Progression as Predictors of Vascular Events in a High Risk European Population) study. J Am CollCardiol 60:1489-1499.

20.Nambi V, Chambless L, Folsom AR, He M, Hu Y, et al. (2010) Carotid intima-media thickness and presence or absence of plaque improves prediction of coronary heart disease risk: the ARIC (Atherosclerosis Risk In Communities) study. J Am CollCardiol 55: 1600-1607.

21.Roquer J, Segura T, Serena J, Cuadrado-Godia E, Blanco M, et al. (2011) Value of carotid intima-media thickness and significant carotid stenosis as markers of stroke recurrence. Stroke 42:3099-3104.

22.IL6R Genetics Consortium Emerging Risk Factors Collaboration, Sarwar N, Butterworth AS, Freitag DF, Gregson J, et al. (2012) Interleukin-6 receptor pathways in coronary heart disease: a collaborative meta-analysis of 82 studies. Lancet 379:1205-1213.

23.Interleukin-6 Receptor MendelianRandomisation Analysis (IL6R MR) Consortium, Hingorani AD, Casas JP (2012) The interleukin-6 receptor as a target for prevention of coronary heart disease: a mendelianrandomisation analysis. Lancet 379: 1214-1224.

24. Weinberg EO, Genco CA (2012) Directing TRAF-ic: cell-specific TRAF6 signaling in chronic inflammation and atherosclerosis. Circulation 126: 1678-1680.

25.Csiszar A, Podlutsky A, Wolin MS, Losonczy G, Pacher P, et al. (2009) Oxidative stress and accelerated vascular aging: implications for cigarette smoking. Front Biosci (Landmark Ed) 14: 3128-3144.

26.Boos CJ, Lip GY (2005) Elevated high-sensitive C-reactive protein, large arterial stiffness and atherosclerosis: a relationship between inflammation and hypertension? J Hum Hypertens 19: 511-513.

27.Kim TN, Kim S, Yang SJ, Yoo HJ, Seo JA, et al. (2010) Vascular Inflammation in Patients With Impaired Glucose Tolerance and Type 2 Diabetes: Analysis With 18F-Fluorodeoxyglucose Positron Emission Tomography. CircCardiovasc Imaging 3:142-148.

28.Lavie CJ, Lee JH, Milani RV (2011) Vitamin D and cardiovascular disease will it live up to its hype? J Am CollCardiol 58: 1547-1556.

29. Jelic S, Lederer DJ, Adams T, Padeletti M, Colombo PC, et al (2010) Vascular inflammation in obesity and sleep apnea. Circulation 121: 1014-1021.

30.Keaney JF Jr, Larson MG, Vasan RS, Wilson PW, Lipinska I, et al.

(2003) Obesity and systemic oxidative stress: clinical correlates of oxidative stress in the Framingham Study. ArteriosclerThrombVascBiol 23: 434-439.

31. Tang WH, Wang Z, Levison BS, Koeth RA, Britt EB, et al. (2013) Intestinal microbial metabolism of phosphatidylcholine and cardiovascular risk. N Engl J Med 368: 1575-1584.

32.Mora S, Cook N, Buring JE, Ridker PM, Lee IM (2007) Physical activity and reduced risk of cardiovascular events: potential mediating mechanisms. Circulation 116: 2110-2118.

33.Steptoe A, Molloy GJ, Messerli-Bürgy N, Wikman A, Randall G, et al. (2011) Fear of dying and inflammation following acute coronary syndrome. Eur Heart J 32: 2405-2411.

34.Fifer KM, Qadir S, Subramanian S, Vijayakumar J, Figueroa AL, et al. (2011) Positron emission tomography measurement of periodontal 18F-fluorodeoxyglucose uptake is associated with histologically determined carotid plaque inflammation. J Am CollCardiol 57: 971-976.

35.Fischer LM, Schlienger RG, Matter C, Jick H, Meier CR (2004) Effect of rheumatoid arthritis or systemic lupus erythematosus on the risk of first-time acute myocardial infarction. Am J Cardiol 93: 198-200.

36.Harismendy O, Notani D, Song X, Rahim NG, Tanasa B, et al. (2011) 9p21 DNA variants associated with coronary artery disease impair interferon- $\hat{I}^3$  signalling response. Nature 470: 264-268.

37.Dalager-Pedersen M, Søgaard M, Schønheyder HC, Nielsen H, Wernich Thomsen R (2014) Risk for Myocardial Infarction and Stroke after Community-Acquired Bacteremia: A 20-Year Population-Based Cohort Study. Circulation .

38.Emerging Risk Factors Collaboration, Kaptoge S, Di Angelantonio E, Pennells L, Wood AM, et al. (2012) C-reactive protein, fibrinogen, and cardiovascular disease prediction. N Engl J Med 367: 1310-1320.

39.Wang TJ, Wollert KC, Larson MG, Coglianese E, McCabe EL, et al. (2012) Prognostic utility of novel biomarkers of cardiovascular stress: the Framingham Heart Study. Circulation 126: 1596-1604.

40.Arnlöv J, Evans JC, Meigs JB, Wang TJ, Fox CS, et al. (2005) Lowgrade albuminuria and incidence of cardiovascular disease events in nonhypertensive and nondiabetic individuals: the Framingham Heart Study. Circulation 112: 969-975.

41.Oestvang J, Johansen B (2006) PhospholipaseA2: a key regulator of inflammatory signalling and a connector to fibrosis development in atherosclerosis. BiochimBiophysActa 1761: 1309-1316.

42. Triggiani M, Granata F, Frattini A, Marone G (2006) Activation of human inflammatory cells by secreted phospholipases A2. BiochimBiophysActa 1761: 1289-1300.

43.Montuschi P, Barnes PJ, Roberts LJ 2nd (2004) Isoprostanes: markers and mediators of oxidative stress. FASEB J 18: 1791-1800.

44. Nicholls SJ, Hazen SL (2005) Myeloperoxidase and cardiovascular dis-

ease. ArteriosclerThrombVascBiol 25: 1102-1111.

45. Bale, Bradley, Amy Doneen, Lisa Collier Cool (2014). Beat the Heart Attack Gene: A Revolutionary Plan to Prevent Heart Disease, Stroke, and Diabetes. John Wiley & Sons.45. Criqui MH, Alberts MJ, Fowkes FGR, Hirsch AT, O'Gara PT, Olin JW, et al. Atherosclerotic Peripheral Vascular Disease Symposium II: Screening for Atherosclerotic Vascular Diseases:: Should Nationwide Programs Be Instituted? Circulation 118: 2830-6.

46.Taylor AJ, Cerqueira M, Hodgson JM, Mark D, Min J, et al. (2010) ACCF/ SCCT/ACR/AHA/ASE/ASNC/NASCI/SCAI/SCMR 2010 Appropriate Use Criteria for Cardiac Computed TomographyA Report of the American College of Cardiology Foundation Appropriate Use Criteria Task Force, the Society of Cardiovascular Computed Tomography, the American College of Radiology, the American Heart Association, the American Society of Echocardiography, the American Society of Nuclear Cardiology, the North American Society for Cardiovascular Imaging, the Society for Cardiovascular Angiography and Interventions, and the Society for Cardiovascular Magnetic Resonance. Journal of the American College of Cardiology 56: 1864-94.

47.Shah NR1, Coulter SA (2012) An evidence-based guide for coronary calcium scoring in asymptomatic patients without coronary heart disease. Tex Heart Inst J 39: 240-242.

48. Society of Atherosclerosis Imaging and Prevention Developed in collaboration with the International Atherosclerosis Society (2011) Appropriate use criteria for carotid intima media thickness testing. Atherosclerosis 214: 43-46.

49.H.B. 1290, 81st Reg. Sess. (Tex. 2009).

50.Blaha MJ, Silverman MG, Budoff MJ (2014) Is There a Role for Coronary Artery Calcium Scoring for Management of Asymptomatic Patients at Risk for Coronary Artery Disease?: Clinical Risk Scores Are Not Sufficient To Define Primary Prevention Treatment Strategies Among Asymptomatic Patients. Circulation: Cardiovascular Imaging 7:398-408.

51.Ridker PM, Rifai N, Rose L, Buring JE, Cook NR (2002) Comparison of C-reactive protein and low-density lipoprotein cholesterol levels in the prediction of first cardiovascular events. N Engl J Med 347: 1557-1565.

52.Nissen SE, Tuzcu EM, Schoenhagen P, Crowe T, Sasiela WJ, et al. (2005) Statin therapy, LDL cholesterol, C-reactive protein, and coronary artery disease. N Engl J Med 352: 29-38.

53.Ridker PM, Danielson E, Fonseca FA, Genest J, Gotto AM Jr, et al. (2009) Reduction in C-reactive protein and LDL cholesterol and cardiovascular event rates after initiation of rosuvastatin: a prospective study of the JUPITER trial. Lancet 373: 1175-1182.

54. White HD, Simes J, Stewart RA, Blankenberg S, Barnes EH, et al. (2013) Changes in lipoprotein-associated phospholipase A2 activity predict coronary events and partly account for the treatment effect of pravastatin: Results from the long-term intervention with Pravastatin in ischemic disease study. J Am Heart Assoc. 2:e000360.

55.Morris ZS, Wooding S, Grant J (2011) The answer is 17 years, what is the question: understanding time lags in translational research. J R Soc Med 104: 510-520.

56.Green LA, Seifert CM (2005) Translation of research into practice: why we can't "just do it". J Am Board FamPract 18: 541-545.

57.Bale, Bradley, Amy Doneen, Lisa Collier Cool (2014). Beat the Heart Attack Gene: A Revolutionary Plan to Prevent Heart Disease, Stroke, and Diabetes. John Wiley & Sons.

\* \* \*

#### Boston Strong! Boston Marathon — April 21st, 2014

Finished my third Boston Marathon with a smile! So grateful to run the world's oldest annual marathon in such a historic city. Boston pride is evident. Boston Strong!

Here is a recap of posts from my Facebook page about this year's Boston Marathon.

4/18 In route to Boston! Just got on plane.... Yahoo!! Thanks for all of your well wishes and kind thoughts. I'm running this year celebrating good health and running for those who cannot. It is an honor.

4/19 At Boston picked up number and race bag. Lots of memorials by the finish line. It is very special to be here. Boston Strong.

4/21 Finished Boston. Slow but smart run. Feel great. Heat and hills are unforgiving. Crowd was amazing and Boston is Strong. Feeling healthy and grateful.

