Atherosclerosis imaging and the Canadian Atherosclerosis Imaging Network.

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Language: English

Publication Type: Article
Abstract:
Atherosclerosis exacts a large toll on society in the form of cardiovascular morbidity, mortality, and resource use and is exacerbated by the epidemics of obesity and diabetes. Consequently, there is a critical need for more-effective methods of diagnosis, treatment, and prevention of the complications of atherosclerosis. Careful and well-conducted large population studies are needed in order to truly understand the natural history of the disease, its imaging biomarkers, and their links to patient outcomes. The Canadian Atherosclerosis Imaging Network (CAIN) is a unique research network funded by the Canadian Institutes of Health Research and the Canada Foundation for Innovation and designed to address these needs and to enable large population-based imaging studies. The central objective of CAIN is to move innovations in imaging toward their broad application in clinical research and clinical practice for the improved evaluation of cardiac and neurologic vascular disease. CAIN is established as an international resource for studying the natural history, progression, and regression of atherosclerosis, as well as novel therapeutic interventions aimed at atherosclerosis. The network represents Canada's leading atherosclerosis imaging experts, embodying both basic imaging science and clinical imaging research. The network is improving methods of detection and treatment of atherosclerosis and, through a better understanding of the underlying disease itself, improving strategies for disease prevention. The benefits are expected to appear in the next 2 to 3 years. CAIN will drive innovation in imaging technology within the field of cardiology and neurology and improve health outcomes in Canada and worldwide.
Abstract:
Subjects with type 2 diabetes are at an increased risk of vascular complications. The use of carotid ultrasound remains an attractive, non-invasive method to monitor atherosclerotic disease progression and/or response to treatment in patients with type 2 diabetes, with intima-media thickness routinely used as the gold standard to detect pathology. However, alternative measurements, such as plaque area or volume, may represent a potentially more powerful approach. Thus, the objective of this study was to compare the traditional intima-media thickness measurement against the novel total plaque volume measurement in analyzing carotid atherosclerosis development in individuals with type 2 diabetes.

The case-control study included 49 Oji-Cree adults with diabetes or impaired glucose tolerance, aged 21-69, and 49 sex- and age-matched normoglycemic subjects. At baseline, metabolic variables were measured, including body mass index, waist circumference, total cholesterol: high density lipoprotein ratio, plasma triglycerides, plasma glucose, and serum insulin. Carotid ultrasound measurements, 7 years later, assessed carotid arterial intima-media thickness and total plaque volume.

At baseline, the two groups were well matched for smoking habits, hypertension, body mass index, and waist circumference. Differences were noted in baseline measurements of total cholesterol: high density lipoprotein (P = 0.0006), plasma triglycerides (P 0.70 when comparing intima-media thickness measurements for diabetics versus non-diabetics, thousands of study subjects are required. For comparing total plaque volume measurements, only hundreds of study subjects are required.

The development of atherosclerotic plaque is greater in subjects with diabetes/impaired glucose tolerance. Total plaque volume appears to capture the atherosclerotic disease burden more effectively in subjects with type 2 diabetes, and would be an appropriate outcome measure for studies aimed at changing the diabetic milieu.

Notes:
Cites: Diabetologia. 2000 Feb;43(2):156-6410753036
Cites: Am J Cardiol. 2002 Feb 21;89(4A):10B-15B; discussion 15B-16B11879661
Cites: Arterioscler Thromb Vasc Biol. 2003 Jun 1;23(6):1035-4112702517
Cites: Stroke. 2005 Sep;36(9):1904-916081857
Cites: Diabetes. 1995 Apr;44(4):369-747698502
Cites: Atherosclerosis. 2005 Feb;178(2):319-2515694940
Cites: Stroke. 2004 Apr;35(4):864-915017019

PubMed ID: 15958169 View in PubMed
Complications of Type 2 Diabetes Among Aboriginal Canadians: prevalence and associated risk factors.

https://arctichealth.org/en/permalink/ahliterature173653

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Keywords: Albuminuria - epidemiology
Blood Glucose - metabolism
C-Reactive Protein - metabolism
Diabetes Mellitus, Type 2 - complications
Diabetic Angiopathies - epidemiology
Diabetic Neuropathies - epidemiology
Diabetic Retinopathy - epidemiology
Female
Humans
Indians, North American
Male
Middle Aged
Ontario
Prevalence
Risk factors
Smoking
Triglycerides - blood

PubMed ID: 16043760 View in PubMed
Differences between carotid wall morphological phenotypes measured by ultrasound in one, two and three dimensions.

https://arctichealth.org/en/permalink/ahliterature176293

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Keywords: Adult
Arteriosclerosis - ultrasonography
Carotid Arteries - pathology - ultrasonography
Carotid Stenosis - ultrasonography
Female
Humans
Indians, North American
Male
Middle Aged
Multivariate Analysis
Phenotype
Risk factors
Smoking - adverse effects
Tunica Intima - anatomy & histology - pathology - ultrasonography

Abstract: Ultrasound measurements are both surrogate markers and risk factors for atherosclerosis end points. Carotid intima-media thickness (IMT) is most commonly used, but ultrasound can also define structures in higher spatial dimensions, such as total plaque area (TPA) and total plaque volume (TPV). Because there are minimal data regarding the relationship between IMT, TPA and TPV, we measured these variables in 272 Oji-Cree subjects. We found pairwise correlations for IMT:TPA, IMT:TPV and TPA:TPV of 0.507, 0.588 and 0.846, respectively (transformed variables, all P


PubMed ID: 15694940 View in PubMed
Cytosolic phosphoenolpyruvate carboxykinase (PEPCK; EC 4.1.1.32), encoded by PCK1, catalyzes the first committed step in gluconeogenesis. We previously showed that a -232C>G promoter polymorphism within a cis-acting element required for basal and cAMP-mediated PCK1 gene transcription results in loss of negative regulation by insulin, contributing to worsened metabolic control in the context of insulin resistance. We hypothesized that this polymorphism would be associated with carotid atherosclerosis in a sample of 150 aboriginal Canadians.

Dependent variables were 2 distinct carotid traits, namely intima-media thickness (IMT) assessed using B-mode ultrasound and total carotid plaque volume (TPV) assessed using 3D ultrasound.

Multivariate analysis showed significant but opposite associations of PCK1 genotype with these traits. Specifically, subjects with the PCK1-232G/G genotype had more carotid IMT (0.80+/-.02 versus 0.73+/-.03 mm; P=0.007) but less TPV (0.10+/-.09 versus 0.38+/-.13; P=0.03) than subjects with other genotypes.

The findings connect the key enzyme in gluconeogenesis with atherosclerosis. The meaning of the opposing associations of PCK1 genotype with IMT and TPV is unclear; more work is required to confirm whether these might be distinct quantitative traits with different biological determinants.

PubMed ID: 16282543 View in PubMed
Increasingly the potential harm from high cholesterol intake, and specifically from egg yolks, is considered insignificant. We therefore assessed total plaque area (TPA) in patients attending Canadian vascular prevention clinics to determine if the atherosclerosis burden, as a marker of arterial damage, was related to egg intake. To provide perspective on the magnitude of the effect, we also analysed the effect of smoking (pack-years).

Consecutive patients attending vascular prevention clinics at University Hospital had baseline measurement of TPA by duplex ultrasound, and filled out questionnaires regarding their lifestyle and medications, including pack-years of smoking, and the number of egg yolks consumed per week times the number of years consumed (egg-yolk years).

Data were available in 1262 patients; mean (SD) age was 61.5 (14.8) years; 47% were women. Carotid plaque area increased linearly with age after age 40, but increased exponentially with pack-years of smoking and with egg-yolk years. Plaque area in patients consuming...
Abstract: Peroxisome proliferator-activated receptor gamma is a crucial molecule in atherogenesis because it is associated with metabolic risk factors such as obesity and diabetes and also plays a key role in subcellular metabolism of arterial wall macrophage foam cells. Genetic variation in PPARG has been associated with metabolic and cardiovascular end points.

We investigated the relationship between 2 common PPARG polymorphisms, namely P12A and c.1431C>T, and carotid atherosclerosis in a sample of 161 Canadian aboriginal people. Dependent variables were carotid intima media thickness (IMT), assessed using B-mode ultrasonography, and total carotid plaque volume (TPV), assessed using 3D ultrasound.

Using multivariate analysis, we found that subjects with > or =1 PPARG A12 allele had less carotid IMT than others (0.72+/−0.03 versus 0.80+/−0.02 mm; P=0.0045), with no between-genotype difference in TPV. In contrast, subjects with the PPARG c.1431T allele had greater TPV than others (124+/−18.4 versus 65.1+/−23.7 mm3; P=0.0079), with no between-genotype difference in IMT.

The findings show an association between PPARG genotypes and carotid arterial phenotypes, and further reflect the prevailing view that the PPARG A12 allele protects against deleterious phenotypes. Also, whereas IMT and TPV are somewhat correlated with each other, they might also represent distinct traits with discrete determinants representing different stages of atherogenesis.

PubMed ID: 15284449 View in PubMed
Lessons from Africa: the importance of measuring plasma renin and aldosterone in resistant hypertension.

https://arctichealth.org/en/permalink/ahliterature127486

Author: J David Spence
Source: Can J Cardiol. 2012 May;28(3):254-7
Date: May-2012
Language: English
Publication Type: Article
Keywords: Africa
African Americans
Aldosterone - blood - metabolism
Antihypertensive Agents - therapeutic use
Drug resistance
Female
Humans
Hyperaldosteronism - diagnosis - drug therapy - ethnology
Hypertension - blood - drug therapy - ethnology
Male
Ontario
Renin - blood - metabolism
Renin-Angiotensin System - drug effects - physiology
Risk assessment
Severity of Illness Index
Treatment Outcome
PubMed ID: 22289470 View in PubMed
Letter by Spence Regarding Article, "Serum Potassium Is Positively Associated With Stroke and Mortality in the Large, Population-Based Malmö Preventive Project Cohort".

https://arctichealth.org/en/permalink/ahliterature290349

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Source: Stroke. 2018 01; 49(1):e22
Date: 01-2018
Language: English
Publication Type: Letter
Comment
Keywords: Cohort Studies
Humans
Potassium
Risk factors
Stroke
Sweden
Notes: CommentOn: Stroke. 2017 Nov;48(11):2973-2978 PMID 28974633
CommentIn: Stroke. 2018 Jan;49(1):e23 PMID 29222230
PubMed ID: 29222226 View in PubMed

Lipoprotein lipase (LPL) gene variation and progression of carotid artery plaque.

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Source: Stroke. 2003 May;34(5):1176-80
Date: May-2003
Language: English
Publication Type: Article
Abstract:
Coding single nucleotide polymorphisms (cSNPs) in the lipoprotein lipase (LPL) gene have been associated with lipoprotein phenotypes and vascular disease risk. We studied the association between LPL cSNPs and a novel noninvasive measure of disease, namely, cross-sectional carotid plaque area (CPA) on B-mode ultrasound.

Four hundred fifty-two patients from an atherosclerosis prevention clinic had determinations of baseline and total CPA. Traditional atherosclerosis risk factors were recorded, and the LPL D9N, N291S, and S447X cSNPs were genotyped. Multiple regression analysis was used to identify determinants of CPA.

Minor allele frequencies for LPL D9N, N291S, and S447X were 2.8%, 0.9%, and 4.4%, respectively. There were no significant between-genotype differences in treated fasting lipids. The LPL D9N genotype was a significant predictor of both baseline CPA (P=0.008) and plaque progression from baseline to 1 year later (P=0.001). Heterozygotes for the N9 allele had higher mean baseline CPA and plaque progression than did LPL D9/D9 homozygotes.

LPL D9N genotype may be a determinant of atherosclerosis as estimated by static baseline CPA and by progression of CPA.