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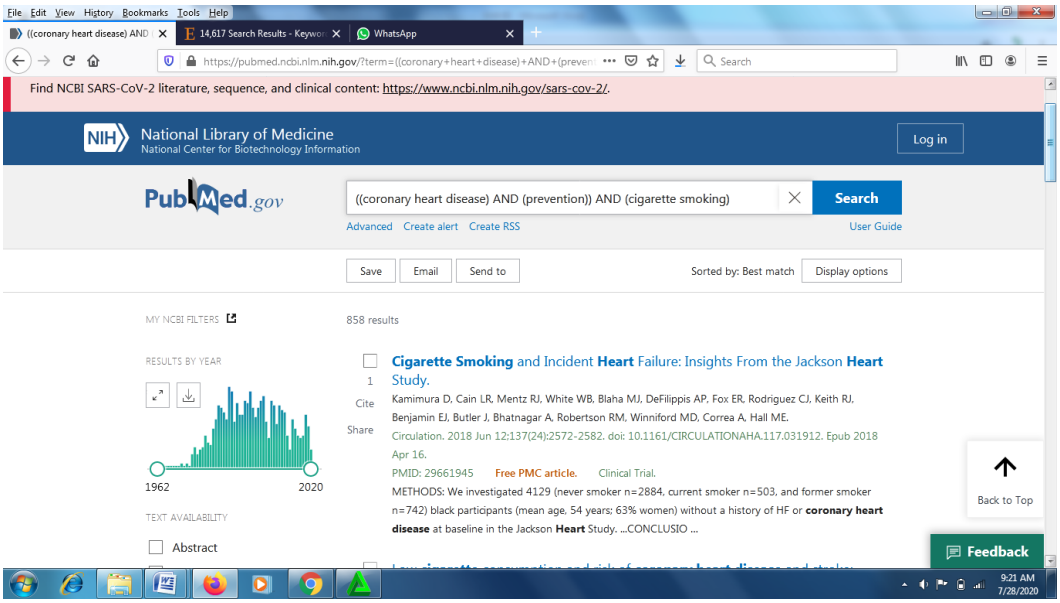

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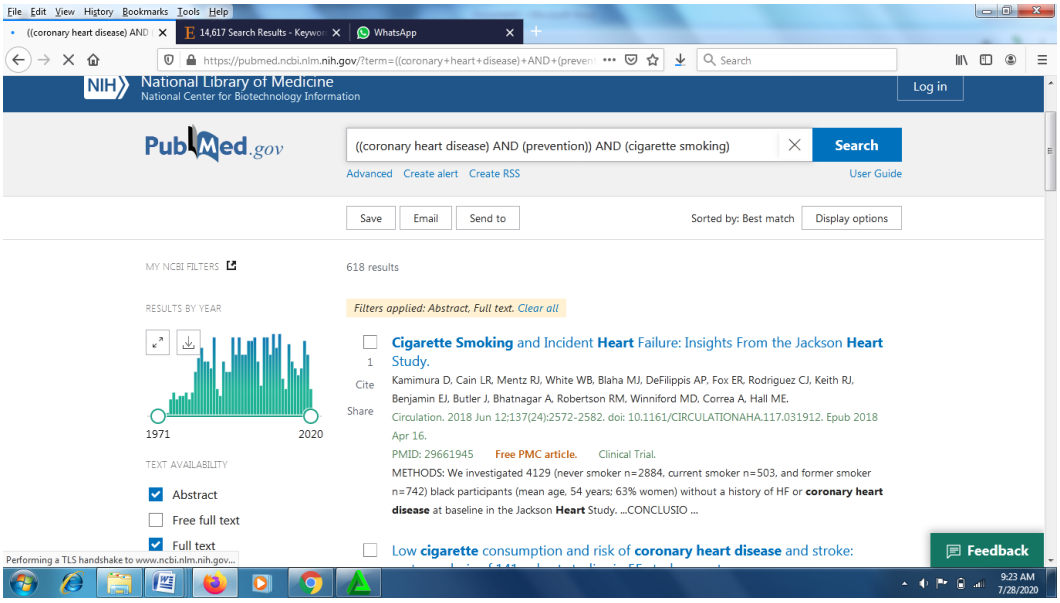

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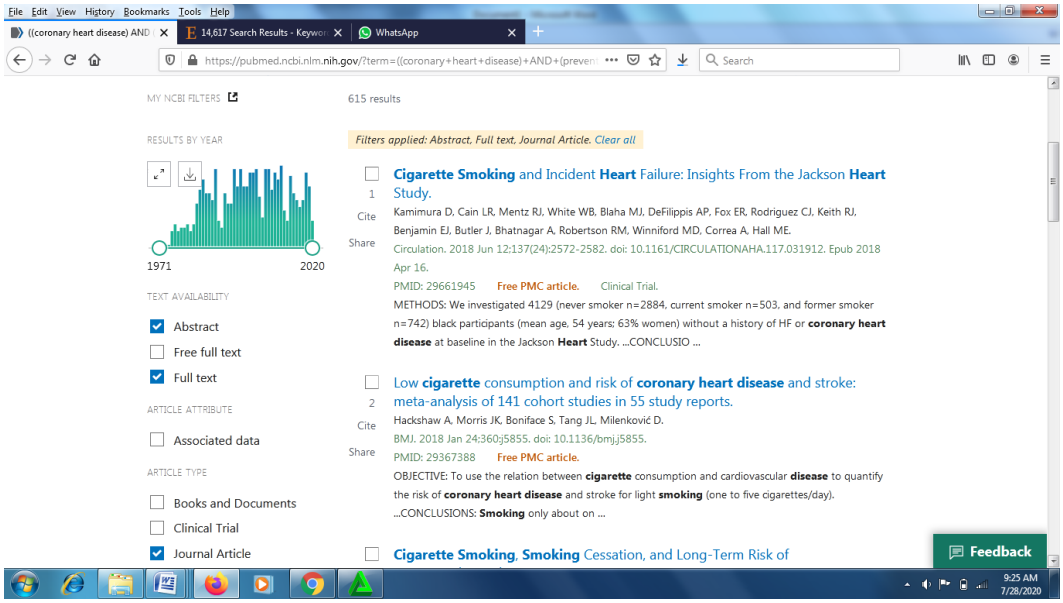

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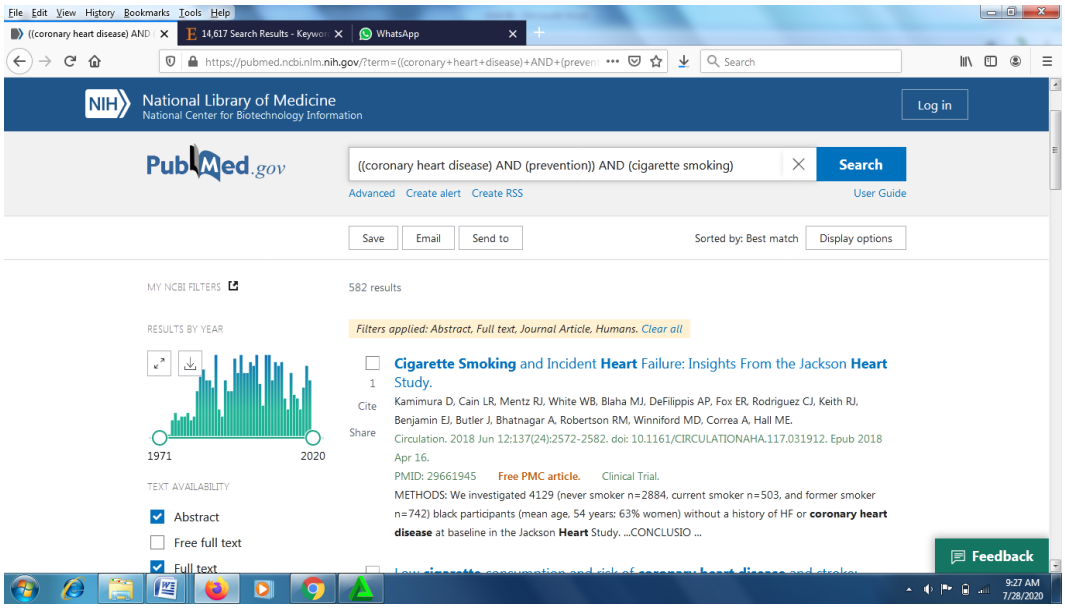

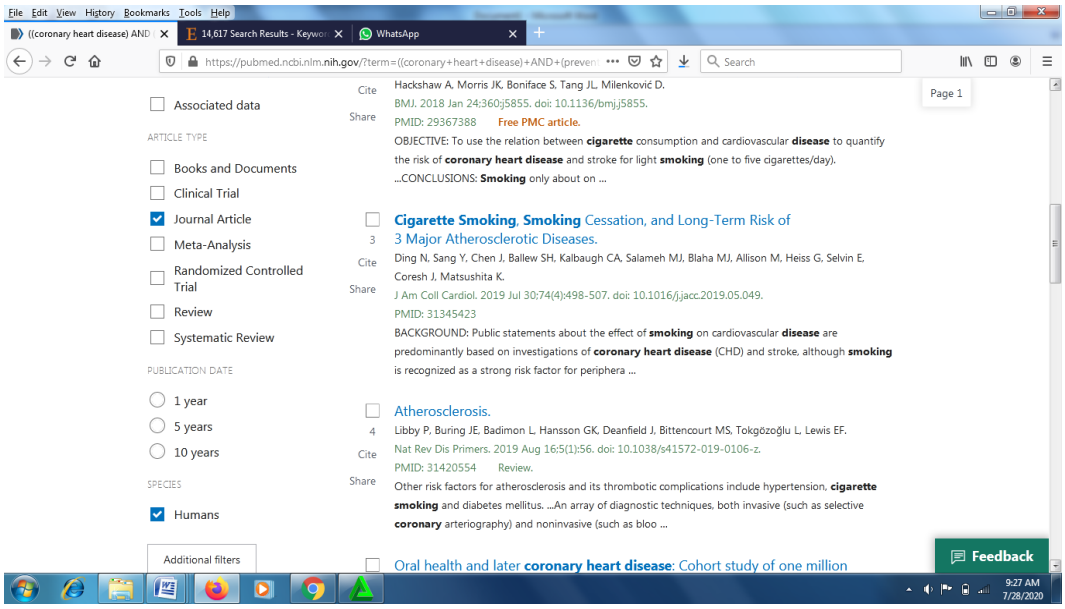

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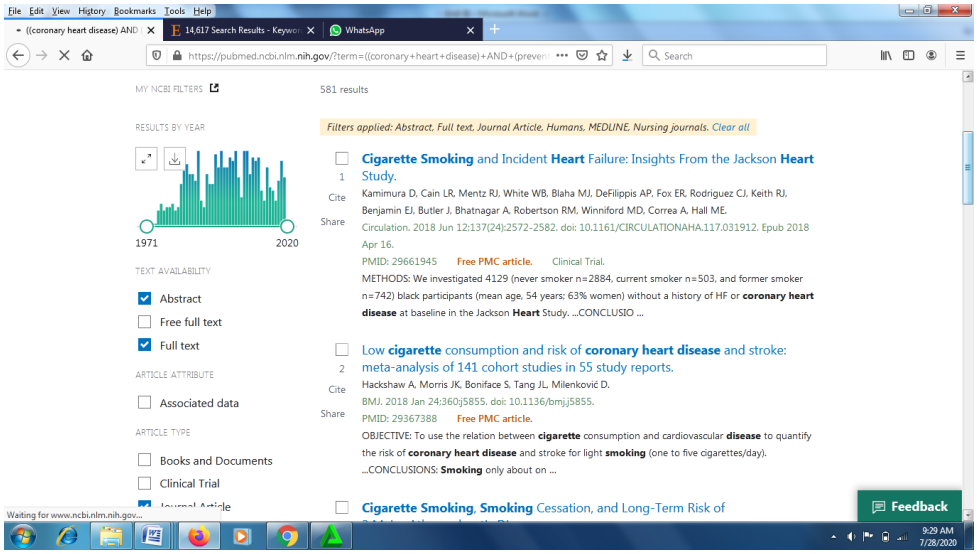

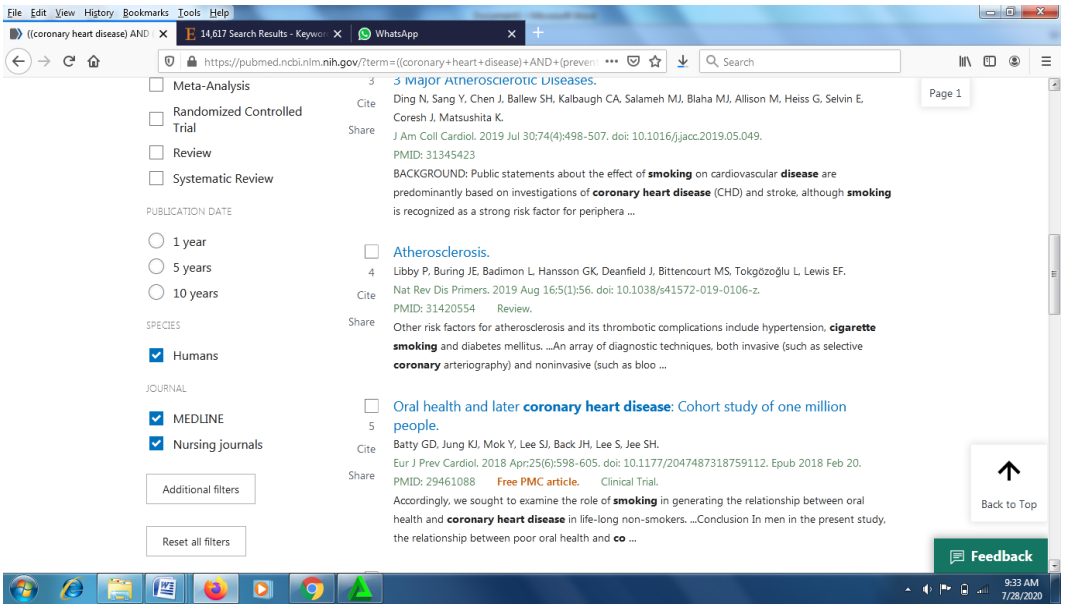

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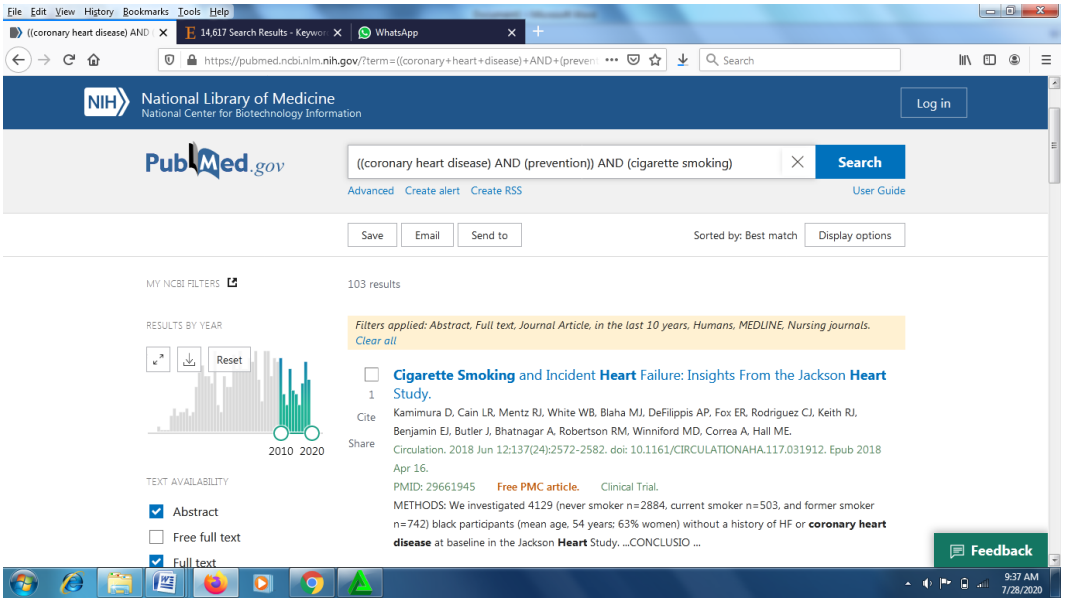

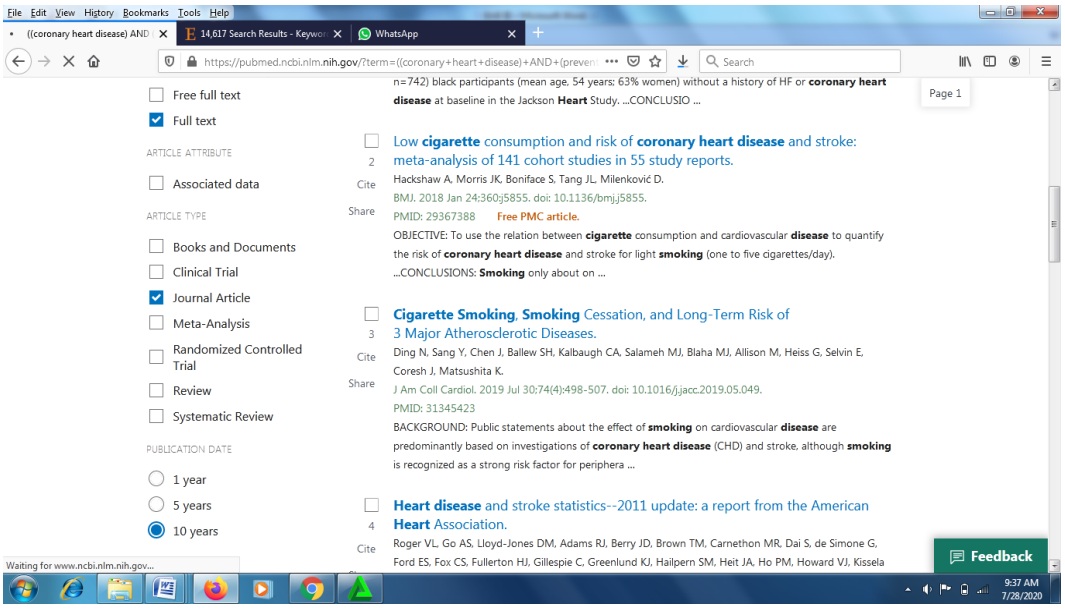

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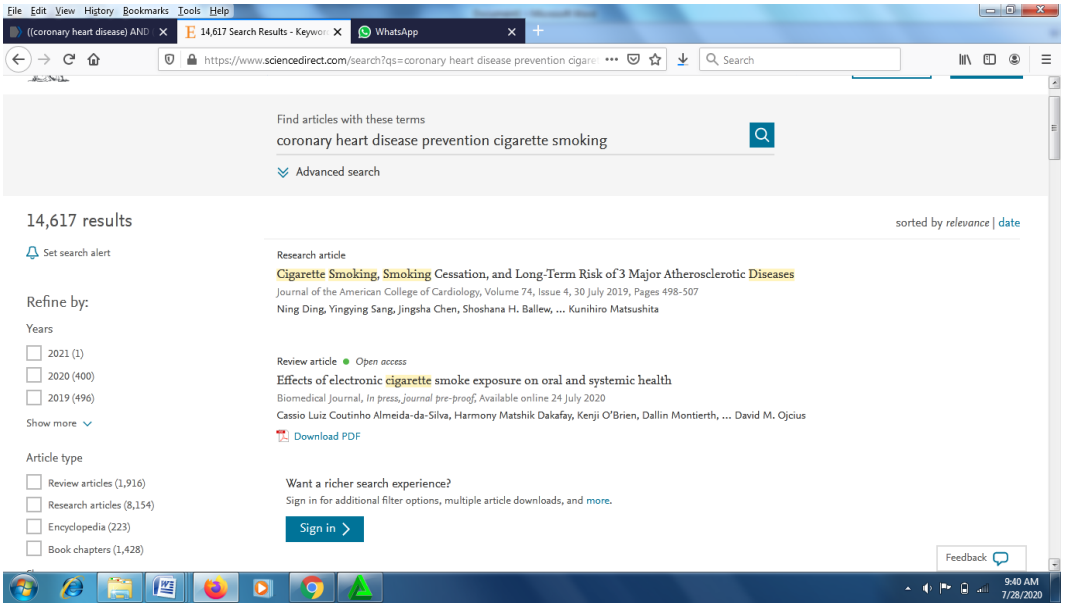

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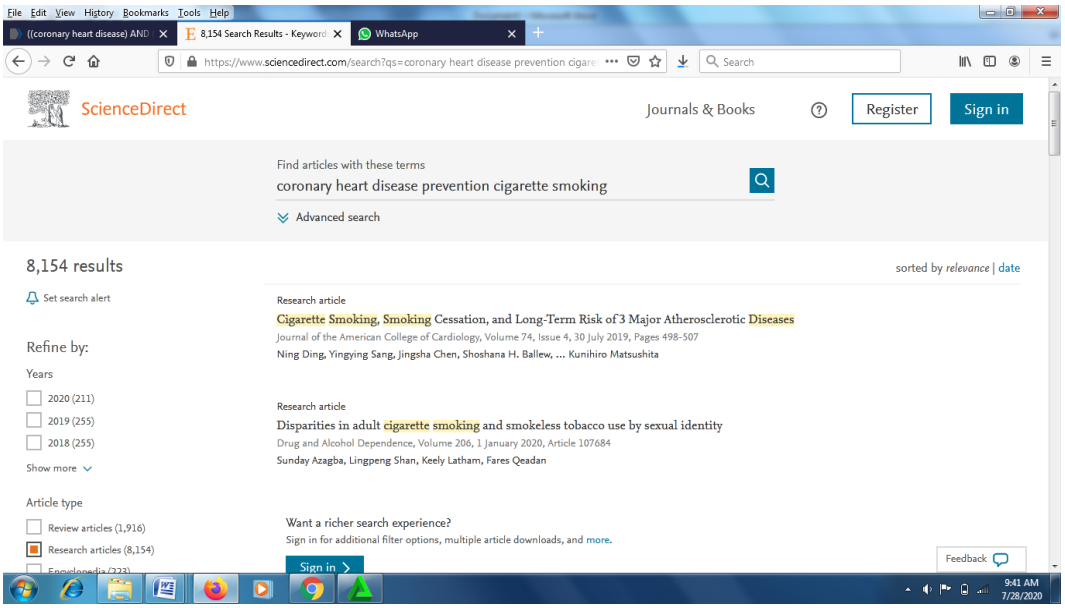

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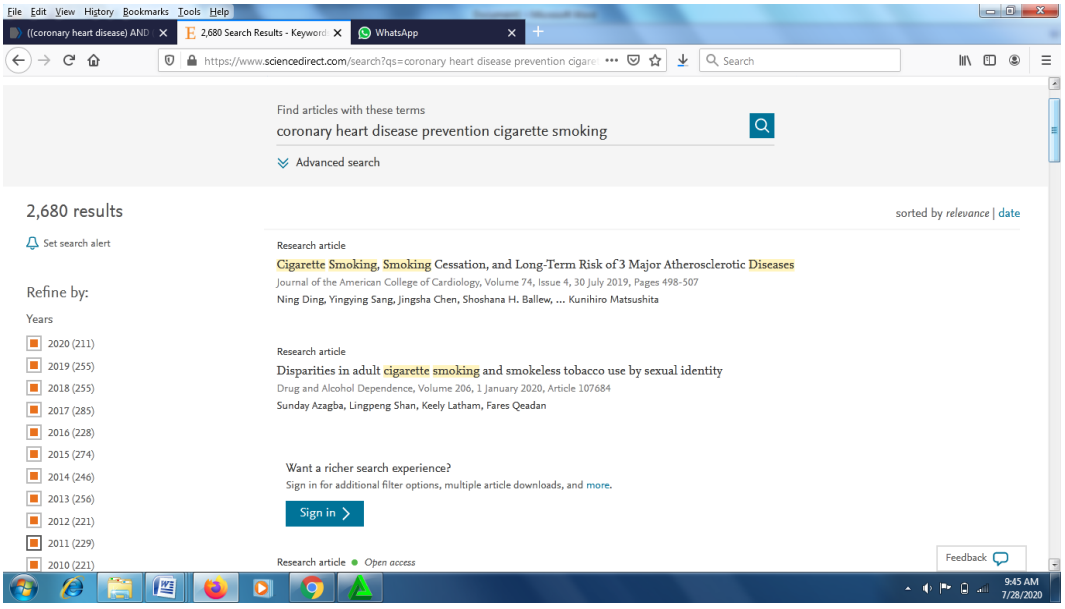

Lampiran 2

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Impact of Cigarette Smoking on Extent of Coronary Artery Disease and Prognosis of Patients With Non–ST-Segment Elevation Acute Coronary Syndromes

An Analysis From the ACUITY Trial (Acute Catheterization and Urgent Intervention Triage Strategy)

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Cleveland, Ohio; Los Angeles, California; New Haven, Connecticut; and New York, New York

Objectives This study sought to evaluate the short- and long-term outcomes for smokers with non–ST-segment elevation acute coronary syndromes (NSTE-ACS).

Background Smoking has been associated with the "paradox" of reduced mortality after acute myocardial infarction (MI). This is thought to be due to favorable baseline characteristics and less diffuse coronary artery disease (CAD) among smokers.

Methods In the ACUITY (Acute Catheterization and Urgent Intervention Triage Strategy) trial, 13,819 patients (29.1% smokers) with moderate- to high-risk NSTE-ACS underwent angiography and, if indicated, revascularization.

Results Smokers were significantly younger and had fewer comorbidities than nonsmokers. Incidence of death and MI were comparable at 30 days, although smokers had significantly reduced risks of 30-day major bleeding (hazard ratio [HR]: 0.80, 95% confidence interval [CI]: 0.67 to 0.96; p = 0.016) and 1-year mortality (HR: 0.797, 95% CI: 0.65 to 0.97; p = 0.027). After correction for baseline and clinical differences, smoking was no longer predictive of major bleeding (odds ratio: 1.06, 95% CI: 0.86 to 1.32; p = 0.56) and was associated with higher 1-year mortality (HR: 1.37, 95% CI: 1.07 to 1.7; p = 0.013). This pattern of reversed risk after multivariable correction held true for those smokers requiring percutaneous coronary intervention. Core laboratory angiographic analysis showed that smokers and nonsmokers were comparable in terms of the extent of CAD, Thrombolysis In Myocardial Infarction flow, myocardial blush, and the presence of thrombi.

Conclusions In contrast to the paradox previously described in ST-segment elevation MI, our analysis finds smoking to be an independent predictor of higher 1-year mortality in patients presenting with

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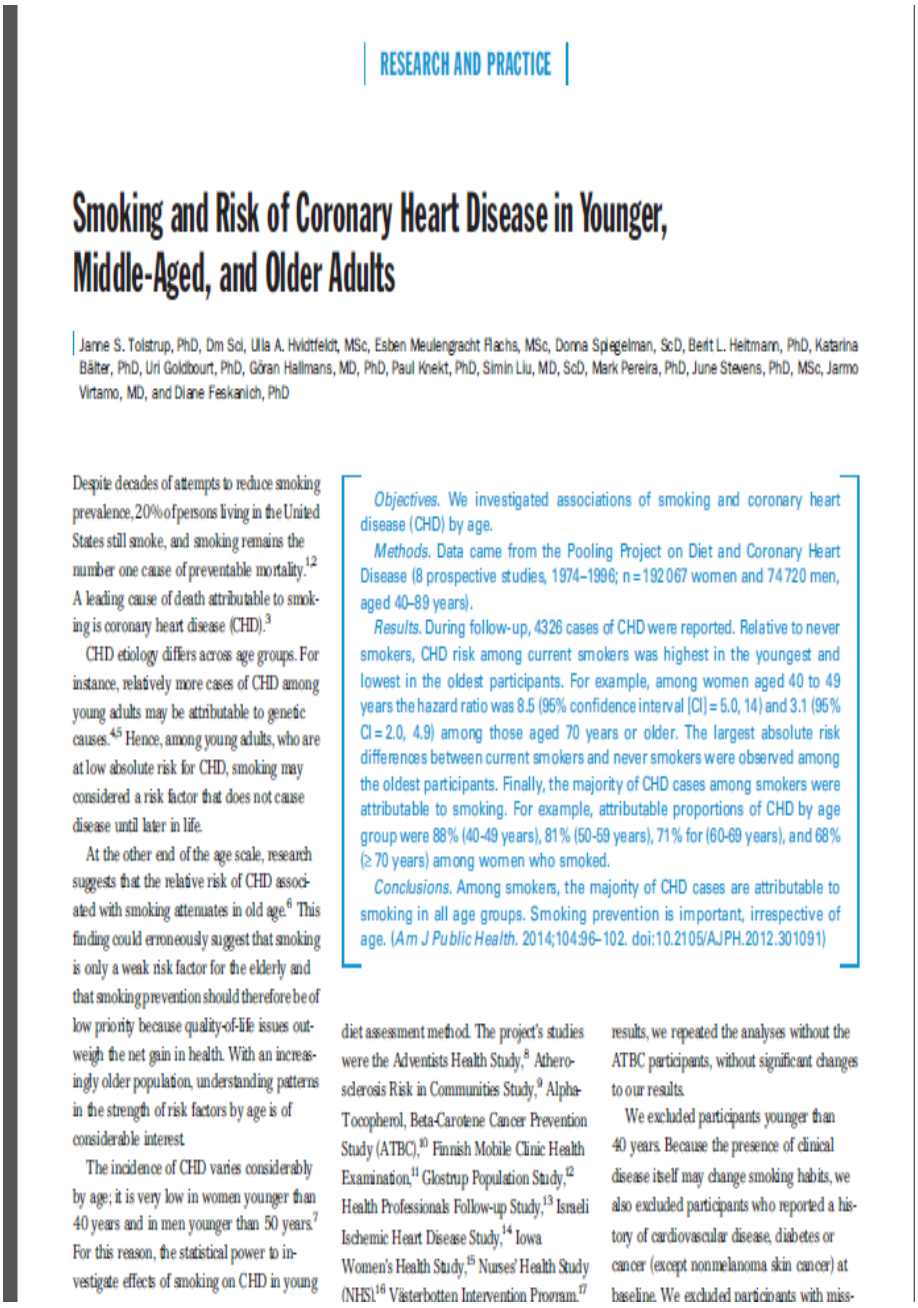

RESEARCH AND PRACTICE

Smoking and Risk of Coronary Heart Disease in Younger, Middle-Aged, and Older Adults

Janne S. Tolstrup, PhD, Dm Sci, Ulla A. Hvidtfeldt, MSc, Esben Meulengracht Flachs, MSc, Donna Spiegelman, ScD, Berit L. Heitmann, PhD, Katarina Bälter, PhD, Uri Goldbourt, PhD, Göran Hallmans, MD, PhD, Paul Knekt, PhD, Simin Liu, MD, ScD, Mark Pereira, PhD, June Stevens, PhD, MSc, Jarmo Virtamo, MD, and Diane Feskanich, PhD

Objectives. We investigated associations of smoking and coronary heart disease (CHD) by age.

Methods. Data came from the Pooling Project on Diet and Coronary Heart Disease (8 prospective studies, 1974–1996; n = 192 067 women and 74 720 men, aged 40–89 years).

Results. During follow-up, 4326 cases of CHD were reported. Relative to never smokers, CHD risk among current smokers was highest in the youngest and lowest in the oldest participants. For example, among women aged 40 to 49 years the hazard ratio was 8.5 (95% confidence interval [CI] = 5.0, 14) and 3.1 (95% CI = 2.0, 4.9) among those aged 70 years or older. The largest absolute risk differences between current smokers and never smokers were observed among the oldest participants. Finally, the majority of CHD cases among smokers were attributable to smoking. For example, attributable proportions of CHD by age group were 88% (40-49 years), 81% (50-59 years), 71% for (60-69 years), and 68% (≥ 70 years) among women who smoked.

Conclusions. Among smokers, the majority of CHD cases are attributable to smoking in all age groups. Smoking prevention is important, irrespective of age. (*Am J Public Health.* 2014;104:96–102. doi:10.2105/AJPH.2012.301091)

Despite decades of attempts to reduce smoking prevalence, 20% of persons living in the United States still smoke, and smoking remains the number one cause of preventable mortality.[1,2] A leading cause of death attributable to smoking is coronary heart disease (CHD).[3]

CHD etiology differs across age groups. For instance, relatively more cases of CHD among young adults may be attributable to genetic causes.[4,5] Hence, among young adults, who are at low absolute risk for CHD, smoking may considered a risk factor that does not cause disease until later in life.

At the other end of the age scale, research suggests that the relative risk of CHD associated with smoking attenuates in old age.[6] This finding could erroneously suggest that smoking is only a weak risk factor for the elderly and that smoking prevention should therefore be of low priority because quality-of-life issues outweigh the net gain in health. With an increasingly older population, understanding patterns in the strength of risk factors by age is of considerable interest.

The incidence of CHD varies considerably by age; it is very low in women younger than 40 years and in men younger than 50 years.[7] For this reason, the statistical power to investigate effects of smoking on CHD in young

diet assessment method. The project's studies were the Adventists Health Study,[8] Atherosclerosis Risk in Communities Study,[9] Alpha-Tocopherol, Beta-Carotene Cancer Prevention Study (ATBC),[10] Finnish Mobile Clinic Health Examination,[11] Glostrup Population Study,[12] Health Professionals Follow-up Study,[13] Israeli Ischemic Heart Disease Study,[14] Iowa Women's Health Study,[15] Nurses' Health Study (NHS),[16] Västerbotten Intervention Program.[17]

results, we repeated the analyses without the ATBC participants, without significant changes to our results.

We excluded participants younger than 40 years. Because the presence of clinical disease itself may change smoking habits, we also excluded participants who reported a history of cardiovascular disease, diabetes or cancer (except nonmelanoma skin cancer) at baseline. We excluded participants with miss-

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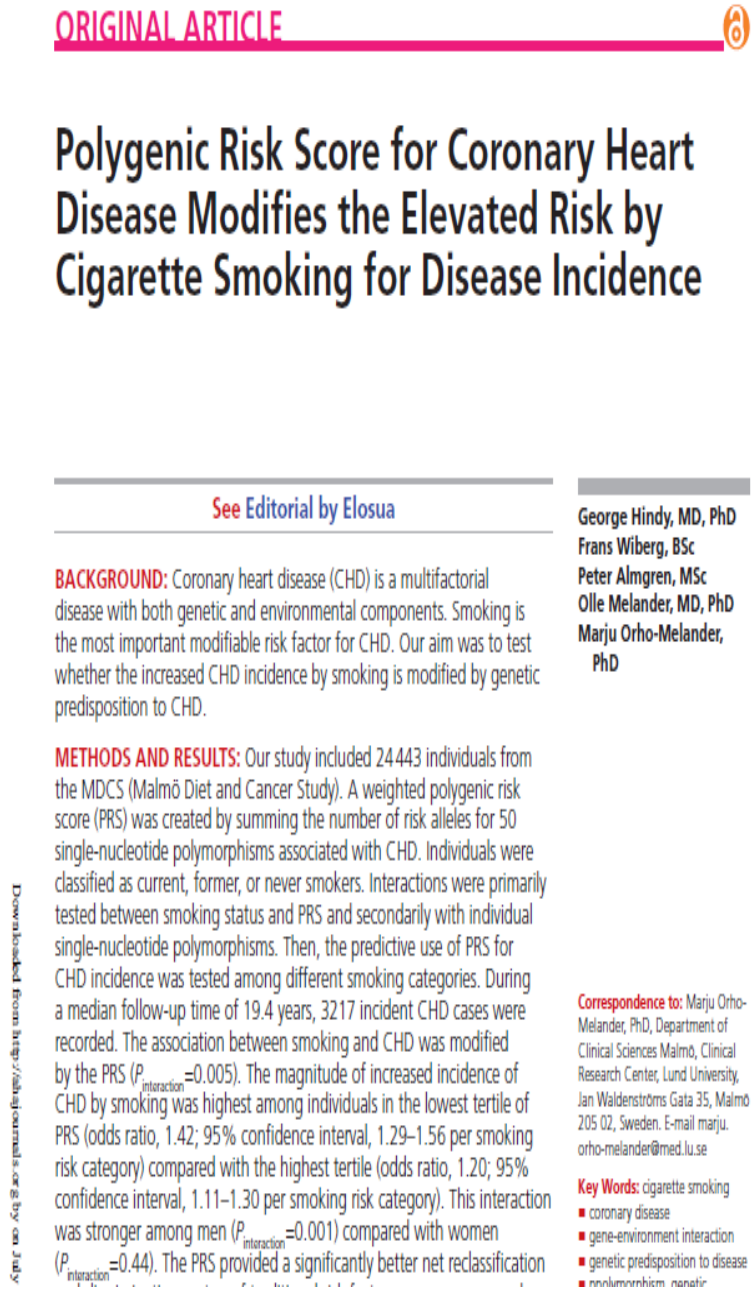

ORIGINAL ARTICLE

Polygenic Risk Score for Coronary Heart Disease Modifies the Elevated Risk by Cigarette Smoking for Disease Incidence

See Editorial by Elosua

BACKGROUND: Coronary heart disease (CHD) is a multifactorial disease with both genetic and environmental components. Smoking is the most important modifiable risk factor for CHD. Our aim was to test whether the increased CHD incidence by smoking is modified by genetic predisposition to CHD.

METHODS AND RESULTS: Our study included 24 443 individuals from the MDCS (Malmö Diet and Cancer Study). A weighted polygenic risk score (PRS) was created by summing the number of risk alleles for 50 single-nucleotide polymorphisms associated with CHD. Individuals were classified as current, former, or never smokers. Interactions were primarily tested between smoking status and PRS and secondarily with individual single-nucleotide polymorphisms. Then, the predictive use of PRS for CHD incidence was tested among different smoking categories. During a median follow-up time of 19.4 years, 3217 incident CHD cases were recorded. The association between smoking and CHD was modified by the PRS ($P_{interaction}$=0.005). The magnitude of increased incidence of CHD by smoking was highest among individuals in the lowest tertile of PRS (odds ratio, 1.42; 95% confidence interval, 1.29–1.56 per smoking risk category) compared with the highest tertile (odds ratio, 1.20; 95% confidence interval, 1.11–1.30 per smoking risk category). This interaction was stronger among men ($P_{interaction}$=0.001) compared with women ($P_{interaction}$=0.44). The PRS provided a significantly better net reclassification

George Hindy, MD, PhD
Frans Wiberg, BSc
Peter Almgren, MSc
Olle Melander, MD, PhD
Marju Orho-Melander, PhD

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Key Words: cigarette smoking
- coronary disease
- gene-environment interaction
- genetic predisposition to disease
- polymorphism, genetic

Gambar 3: artikel 3

Inflamm. Res. (2015) 64:415–422
DOI 10.1007/s00011-015-0821-1

Inflammation Research

ORIGINAL RESEARCH PAPER

Impact of inflammation, gene variants, and cigarette smoking on coronary artery disease risk

Mahmoud Merhi[1] · Sally Demirdjian[2] · Essa Hariri[1] · Nada Sabbah[1] · Sonia Youhanna[3] · Michella Ghassibe-Sabbagh[1] · Joseph Naoum[1] · Marc Haber[1] · Raed Othman[3] · Samer Kibbani[3] · Elie Chammas[1] · Roy Kanbar[1] · Hamid el Bayeh[1] · Youssef Chami[1] · Antoine Abchee[4] · Daniel E. Platt[5] · Pierre Zalloua[1,6] · Georges Khazen[7]

Received: 29 January 2015 / Revised: 15 April 2015 / Accepted: 16 April 2015 / Published online: 24 April 2015

Abstract

Background The role of inflammation in coronary artery disease (CAD) pathogenesis is well recognized. Moreover, smoking inhalation increases the activity of inflammatory mediators through an increase in leukotriene synthesis essential in atherosclerosis pathogenesis.

Aim The aim of this study is to investigate the effect of "selected" genetic variants within the leukotriene (LT) pathway and other variants on the development of CAD.

Methods CAD was detected by cardiac catheterization. Logistic regression was performed to investigate the association of smoking and selected susceptibility variants in the LT pathway including ALOX5AP, LTA4H, LTC4S, PON1, and LTA as well as CYP1A1 on CAD risk while controlling for age, gender, BMI, family history, diabetes, hyperlipidemia, and hypertension.

Results rs4769874 (ALOX5AP), rs854560 (PON1), and rs4646903 (CYP1A1 MspI polymorphism) are significantly associated with an increased risk of CAD with respective odds ratios of 1.53703, 1.67710, and 1.35520; the genetic variant rs9579646 (ALOX5AP) is significantly associated with a decreased risk of CAD (OR 0.76163). Moreover, a significant smoking-gene interaction is determined with CYP1A1 MspI polymorphism rs4646903 and is associated with a decreased risk of CAD in current smokers (OR 0.52137).

Conclusion This study provides further evidence that genetic variation of the LT pathway, PON1, and CYP1A1 can modulate the atherogenic processes and eventually increase the risk of CAD in our study population. Moreover, it also shows the effect of smoking-gene interaction on CAD risk, where the CYP1A1 MspI polymorphism

Responsible Editor: John Di Battista.

M. Merhi, S. Demirdjian, E. Hariri and N. Sabbah contributed equaly.

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✉ Georges Khazen

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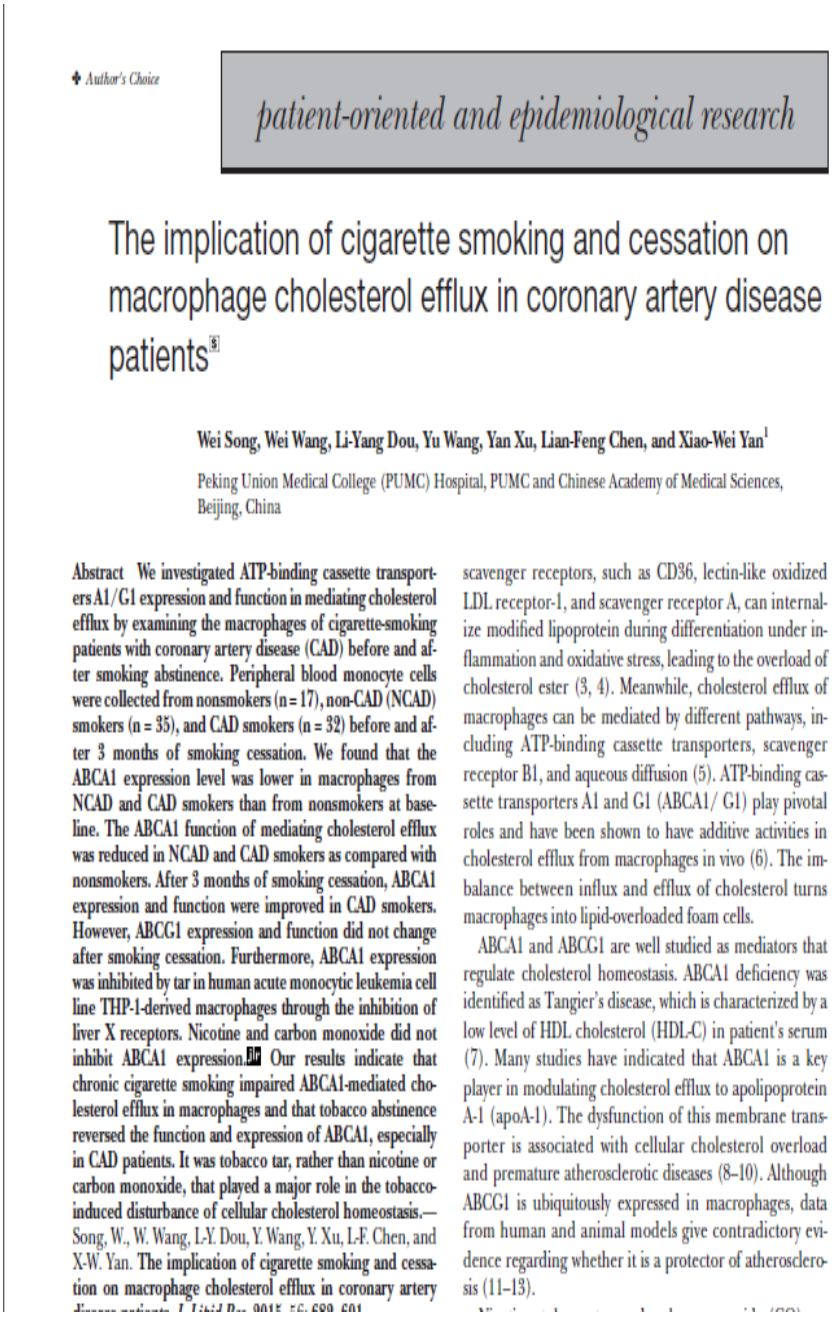

♦ Author's Choice

patient-oriented and epidemiological research

The implication of cigarette smoking and cessation on macrophage cholesterol efflux in coronary artery disease patients[S]

Wei Song, Wei Wang, Li-Yang Dou, Yu Wang, Yan Xu, Lian-Feng Chen, and Xiao-Wei Yan[1]

Peking Union Medical College (PUMC) Hospital, PUMC and Chinese Academy of Medical Sciences, Beijing, China

Abstract We investigated ATP-binding cassette transporters A1/G1 expression and function in mediating cholesterol efflux by examining the macrophages of cigarette-smoking patients with coronary artery disease (CAD) before and after smoking abstinence. Peripheral blood monocyte cells were collected from nonsmokers (n = 17), non-CAD (NCAD) smokers (n = 35), and CAD smokers (n = 32) before and after 3 months of smoking cessation. We found that the ABCA1 expression level was lower in macrophages from NCAD and CAD smokers than from nonsmokers at baseline. The ABCA1 function of mediating cholesterol efflux was reduced in NCAD and CAD smokers as compared with nonsmokers. After 3 months of smoking cessation, ABCA1 expression and function were improved in CAD smokers. However, ABCG1 expression and function did not change after smoking cessation. Furthermore, ABCA1 expression was inhibited by tar in human acute monocytic leukemia cell line THP-1-derived macrophages through the inhibition of liver X receptors. Nicotine and carbon monoxide did not inhibit ABCA1 expression. Our results indicate that chronic cigarette smoking impaired ABCA1-mediated cholesterol efflux in macrophages and that tobacco abstinence reversed the function and expression of ABCA1, especially in CAD patients. It was tobacco tar, rather than nicotine or carbon monoxide, that played a major role in the tobacco-induced disturbance of cellular cholesterol homeostasis.—Song, W., W. Wang, L-Y. Dou, Y. Wang, Y. Xu, L-F. Chen, and X-W. Yan. The implication of cigarette smoking and cessation on macrophage cholesterol efflux in coronary artery

scavenger receptors, such as CD36, lectin-like oxidized LDL receptor-1, and scavenger receptor A, can internalize modified lipoprotein during differentiation under inflammation and oxidative stress, leading to the overload of cholesterol ester (3, 4). Meanwhile, cholesterol efflux of macrophages can be mediated by different pathways, including ATP-binding cassette transporters, scavenger receptor B1, and aqueous diffusion (5). ATP-binding cassette transporters A1 and G1 (ABCA1/ G1) play pivotal roles and have been shown to have additive activities in cholesterol efflux from macrophages in vivo (6). The imbalance between influx and efflux of cholesterol turns macrophages into lipid-overloaded foam cells.

ABCA1 and ABCG1 are well studied as mediators that regulate cholesterol homeostasis. ABCA1 deficiency was identified as Tangier's disease, which is characterized by a low level of HDL cholesterol (HDL-C) in patient's serum (7). Many studies have indicated that ABCA1 is a key player in modulating cholesterol efflux to apolipoprotein A-1 (apoA-1). The dysfunction of this membrane transporter is associated with cellular cholesterol overload and premature atherosclerotic diseases (8–10). Although ABCG1 is ubiquitously expressed in macrophages, data from human and animal models give contradictory evidence regarding whether it is a protector of atherosclerosis (11–13).

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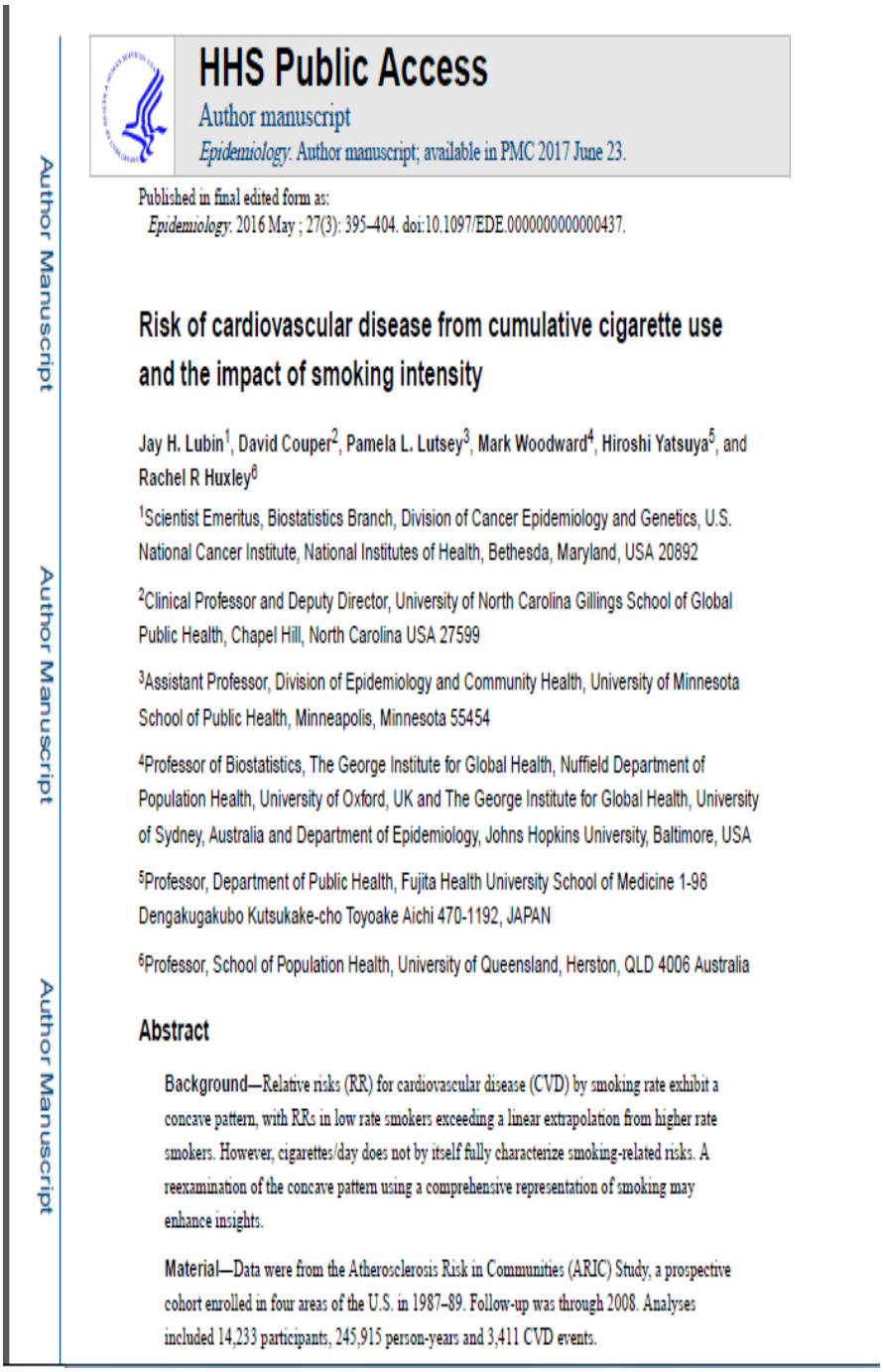

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Risk of cardiovascular disease from cumulative cigarette use and the impact of smoking intensity

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Abstract

Background—Relative risks (RR) for cardiovascular disease (CVD) by smoking rate exhibit a concave pattern, with RRs in low rate smokers exceeding a linear extrapolation from higher rate smokers. However, cigarettes/day does not by itself fully characterize smoking-related risks. A reexamination of the concave pattern using a comprehensive representation of smoking may enhance insights.

Material—Data were from the Atherosclerosis Risk in Communities (ARIC) Study, a prospective cohort enrolled in four areas of the U.S. in 1987–89. Follow-up was through 2008. Analyses included 14,233 participants, 245,915 person-years and 3,411 CVD events.

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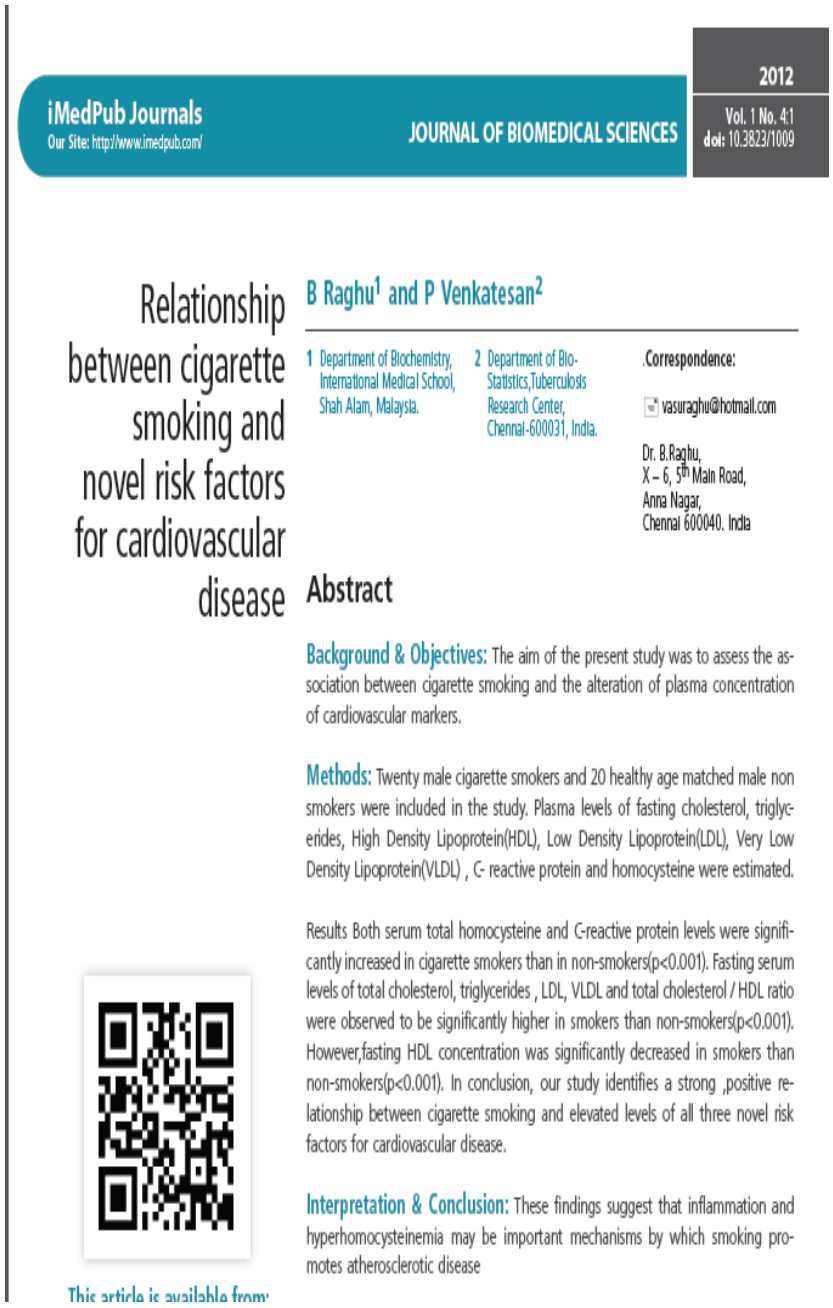

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2012
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Relationship between cigarette smoking and novel risk factors for cardiovascular disease

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Abstract

Background & Objectives: The aim of the present study was to assess the association between cigarette smoking and the alteration of plasma concentration of cardiovascular markers.

Methods: Twenty male cigarette smokers and 20 healthy age matched male non smokers were included in the study. Plasma levels of fasting cholesterol, triglycerides, High Density Lipoprotein(HDL), Low Density Lipoprotein(LDL), Very Low Density Lipoprotein(VLDL) , C- reactive protein and homocysteine were estimated.

Results Both serum total homocysteine and C-reactive protein levels were significantly increased in cigarette smokers than in non-smokers($p<0.001$). Fasting serum levels of total cholesterol, triglycerides , LDL, VLDL and total cholesterol / HDL ratio were observed to be significantly higher in smokers than non-smokers($p<0.001$). However,fasting HDL concentration was significantly decreased in smokers than non-smokers($p<0.001$). In conclusion, our study identifies a strong ,positive relationship between cigarette smoking and elevated levels of all three novel risk factors for cardiovascular disease.

Interpretation & Conclusion: These findings suggest that inflammation and hyperhomocysteinemia may be important mechanisms by which smoking promotes atherosclerotic disease

Gambar 7: artikel 7

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Lifetime cumulative exposure to waterpipe smoking is associated with coronary artery disease

Abla M. Sibai[a], Rania A. Tohme[a,b], Mohamad M. Almedawar[c,d], Taha Itani[e], Sara I. Yassine[a], Eden A. Nohra[f], Hussain A. Isma'eel[c,d,*]

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ABSTRACT

Objective: Globally, waterpipe (WP) smoking is becoming a more prevalent form of tobacco consumption. Whilst research so far has demonstrated a significant link between WP use and a number of health outcomes, little is known of its association with heart disease. We examine in this study the association of WP smoking with angiographically confirmed coronary artery disease (CAD).

Methods: A total of 1210 patients, aged 40 years and over and free from smoking-associated illnesses or history of cardiovascular procedures, admitted for coronary angiography at four major hospitals in Lebanon, were included. The extent of CAD was summarized in two ways, firstly as diseased (≥50% and ≥70% occlusion in at least one main coronary artery) versus non-diseased (entirely normal coronaries), and secondly, as CAD cumulative score based on Duke CAD Prognostic Index. A score of WP-years, capturing intensity and lifetime duration of exposure, was estimated for each individual.

Results: Lifetime exposure exceeding 40 WP-years was associated with a threefold significant increase in the odds of having severe stenosis (≥70%) compared to non-smokers (OR = 2.94, 95% CI 1.04–8.33) as well as with the CAD Index (β = 7.835, p-value = 0.027), net of the effect of socio-demographic characteristics, health behaviors and co-morbidity. A dose–response relationship between WP-years and percent stenosis was also established. WP smoking status (never, past and current) did not associate with CAD.

Conclusions: Cumulative exposure to WP smoking is significantly associated with severe CAD. There is a need to monitor WP use among cardiac patients and include this information in their medical charts in the same manner cigarettes smoking is documented. This is likely to increase awareness of the hazards of WP smoking and prompt physicians to target WP tobacco control by providing advice to their patients on WP smoking cessation.

Gambar 8: artikel 8

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Clinical Science

Cigarette Smoking and Oxidative Stress in Patients with Coronary Artery Disease

Gordana Kamceva[1*], Zorica Arsova-Sarafinovska[2], Tatjana Ruskovska[3], Milka Zdravkovska[3], Lidija Kamceva-Panova[4], Elisaveta Stikova[2]

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Keywords: smoking; oxidative stress; coronary artery disease; Republic of Macedonia.

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Competing Interests: The authors have declared that no competing interests exist.

Abstract

AIM: To determine whether cigarette smoking, as a risk factor for CAD, affects (anti)oxidant status.

MATERIAL AND METHODS: The study included patients with CAD, divided according to their smoking status and the number of cigarettes smoked during a day. Biological markers of oxidative stress (concentration of oxidants and activity of antioxidant enzymes) were measured in all subjects.

RESULTS: The study included 300 patients with CAD, (average age of 63 ± 11 years), predominantly males. Of the total, 34.0% were active smokers, 23.0% were former smokers, and 43.0% were non-smokers. Most of the active smokers smoked 1-20 cigarettes/day. In terms of concentration of oxidants (MDA and HP) there was not a significant difference between smokers versus non-smokers. As for the activity of antioxidant enzymes (SOD, CAT and GPX), a statistically significant difference was found in the activity of GPX among the active smokers with CAD and the non-smokers with CAD ($p = 0.039$).

CONCLUSION: Smoking as a risk factor for CAD is closely associated with increased oxidative stress, and the number of cigarettes smoked plays an important role in increasing the level of oxidative damage and reducing antioxidant defence.

Gambar 9: artiekel 9

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Interaction effects between *Paraoxonase 1* variants and cigarette smoking on risk of coronary heart disease in a Singaporean Chinese population

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Abstract

Objective—Paraoxonase 1 (PON1) plays an important role in reducing the risk of coronary heart disease (CHD). Smoking is known to reduce PON1 activity. We aimed to investigate the effects of interactions between *PON1* variants and smoking on CHD in the Singaporean Chinese population.

Methods—In a case-control study nested within Singapore Chinese Health Study (N = 1914),

Gambar 10: artikel 10

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No	PROPOSAL SKRIPSI	HAL	PERBAIKAN
1.	Judul	1	Analisis Analisis Pengurangan Konsumsi Rokok Sebagai Upaya Pencegahan Penyakit Jantung Koroner menjadi Analisis Konsumsi Rokok Sebagai Upaya Pencegahan Penyakit Jantung Koroner
2.	Latar Belakang (Solusi)	4	Penambahan solusi atau opini dari peneliti
3.	Latar Belakang (Konsep)	1	Pematangan konsep

Surabaya, 11 Agustus 2020

Penguji

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No	SEMINAR HASIL SKRIPSI	HAL	PERBAIKAN
1.	Abstrak	viii	Revisi abstrak
2.	BAB 4 (Pembahasan)	36	Penambahan opini peneliti dan kesimpulan dari 10 artikel yang dilakukan *Literatur Review*
3.	BAB 5 (Kesimpulan)	38	Penambahan kesimpulan
4.	BAB 5 (Saran)	38	Penambahan saran kepada pengguna atau masyarakat

Surabaya, 01 September 2020
Penguji

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No	SEMINAR HASIL SKRIPSI	HAL	PERBAIKAN
1.	BAB 5 (Saran)	38	Penambahan saran kepada pengguna maupun masyarakat dan penggunaan kata-kata yg sesuai

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Pembimbing 1

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No	SEMINAR HASIL SKRIPSI	HAL	PERBAIKAN
1.	BAB 4 (Hasil)	20	Perbaikan tabel hasil dan kesimpulan harus lebih informatif dan lengkap
2.	BAB 5 (Kesimpulan)	38	Kesimpulan ditambahi dan sesuai dengan artikel yang didapat

Surabaya, 01 September 2020
Pembimbing 2

Ratna Agustin S.Kep.,Ns., M.Kep

LEMBAR KONSULTASI PROPOSAL/SKRIPSI

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Judul Skripsi : Analisis Konsumsi Rokok Sebagai Upaya Pencegahan Penyakit Jantung Koroner

No	Hari, Tanggal	Catatan Pembimbing hal yang direvisi	Hasil Revisian	Tanda Tangan
1	12 Nov 2019	Konsul judul	Acc judul	
2.	18 Des 2019	Konsul Bab 1	Revisi Bab 1	
3.	20 Des 2019	Konsul Bab 1	Revisi Bab 1	
4.	26 Des 2019	Konsul Bab 1	Acc bab 1	
5.	06 Jan 2020	Konsul Bab 2	Acc bab 2	
6.	10 Jan 2020	Konsul Bab 3	Revisi Bab 3	
7.	05 Feb 2020	Konsul Bab 3	Revisi Bab 3	
8.	19 Maret 2020	Konsul Bab 3	Revisi Bab 3	

No	Hari, Tanggal	Catatan Pembimbing hal yang direvisi	Hasil Revisian	Tanda Tangan
9.	06 Juli 2020	Konsul Bab 1-3 literartur review	Revisi Bab 1-3	
10.	13 Juli 2020	Konsul Bab 1-3 literartur review	Revisi Bab 1-3	
11.	23 Juli 2020	Konsul Bab 1-3 literartur review	Revisi Bab 1-3	
12.	05 Agu 2020	Konsul Bab 1-3 literartur review	Acc ujian proposal	
13.	13 Agu 2020	Konsul Bab 4	Revisi Bab 4	
14.	18 Agu 2020	Konsul Bab 4	Revisi Bab 4	
15.	24 Agu 2020	Konsul Bab 4 & 5, Abstrak	Revisi Bab 5 dan abtrak	
16.	27 agu 2020	Konsul Bab 4 & 5, Abstrak	Acc ujian seminar hasil	

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Judul Skripsi : Analisis Konsumsi Rokok Sebagai Upaya Pencegahan Penyakit Jantung Koroner

No	Hari, Tanggal	Catatan Pembimbing hal yang direvisi	Hasil Revisian	Tanda Tangan
1.	15 Nov 2019	Konsul Judul	Acc Judul	
2.	20 Des 2019	Konsul Bab 1	Revisi Bab 1	
3.	08 Jan 2020	Konsul Bab 1	Revisi Bab 1	
4.	13 Feb 2020	Konsul Bab 1	Revisi Bab 1	
5.	26 Mar	Konsul bab 1-3	Revisi Bab 1-3	
6.	06 Juli 2020	Konsul Bab 1-3 literartur review	Revisi Bab 1-3	
7.	13 Juli 2020	Konsul Bab 1-3 literartur review	Revisi Bab 1-3	
8.	23 Juli 2020	Konsul Bab 1-3 literartur review	Revisi Bab 1-3	

No	Hari, Tanggal	Catatan Pembimbing hal yang direvisi	Hasil Revisian	Tanda Tangan
10.	05 Agu 2020	Konsul Bab 1-3 literartur review	Acc ujian proposal	
11.	13 Agu 2020	Konsul Bab 4	Revisi Bab 4	
12.	18 Agu 2020	Konsul Bab 4	Revisi Bab 4	
13.	20 Agu 2020	Konsul Bab 4 & 5, Abstrak	Revisi Bab 4 & 5, Abstrak	
14.	24 Agus 2020	Konsul Bab 4 & 5, Abstrak	Revisi Bab 4 & 5, Abstrak	
15.	26 agu 2020	Abstrak	Revisi Bab 5 dan abtrak	
16.	27 agu 2020	Konsul Bab 4 & 5, Abstrak	Acc ujian seminar hasil	
		Konsul Bab 4 & 5, Abstrak		

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Pada tanggal : 1 September 2020

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