INSTITUT DE CARDIOLOGIE DE MONTRÉAL

Université de Montréal

RÉADAPTATION CARDIAQUE et PRÉVENTION SECONDAIRE: NOUVELLES DONNÉES

MARTIN JUNEAU MPs MD FRCP
DIRECTEUR de la PRÉVENTION
INSTITUT DE CARDIOLOGIE de MONTRÉAL
CENTRE ÉPIC

SSQV

22 NOVEMBRE 2014

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Divulgation de conflits d'intérêts potentiels

Société des sciences vasculaires du Québec (SSVQ)

CONGRÈS SSVQ 2014

21-22 novembre 2014

Dr Martin Juneau, Cardiologue, I.C.M., Montréal

Subvention à nos cliniques	Banque de Montréal	2008 – 2018
Subvention à nos cliniques	Banque Scotia	2011 – 2016
Subvention à nos cliniques	Cascades	2010 – 2015
Subvention à nos cliniques	Boehringer-Ingelheim	2012 - 2014

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ACTIVITÉS PHYSIQUES
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SPINNER
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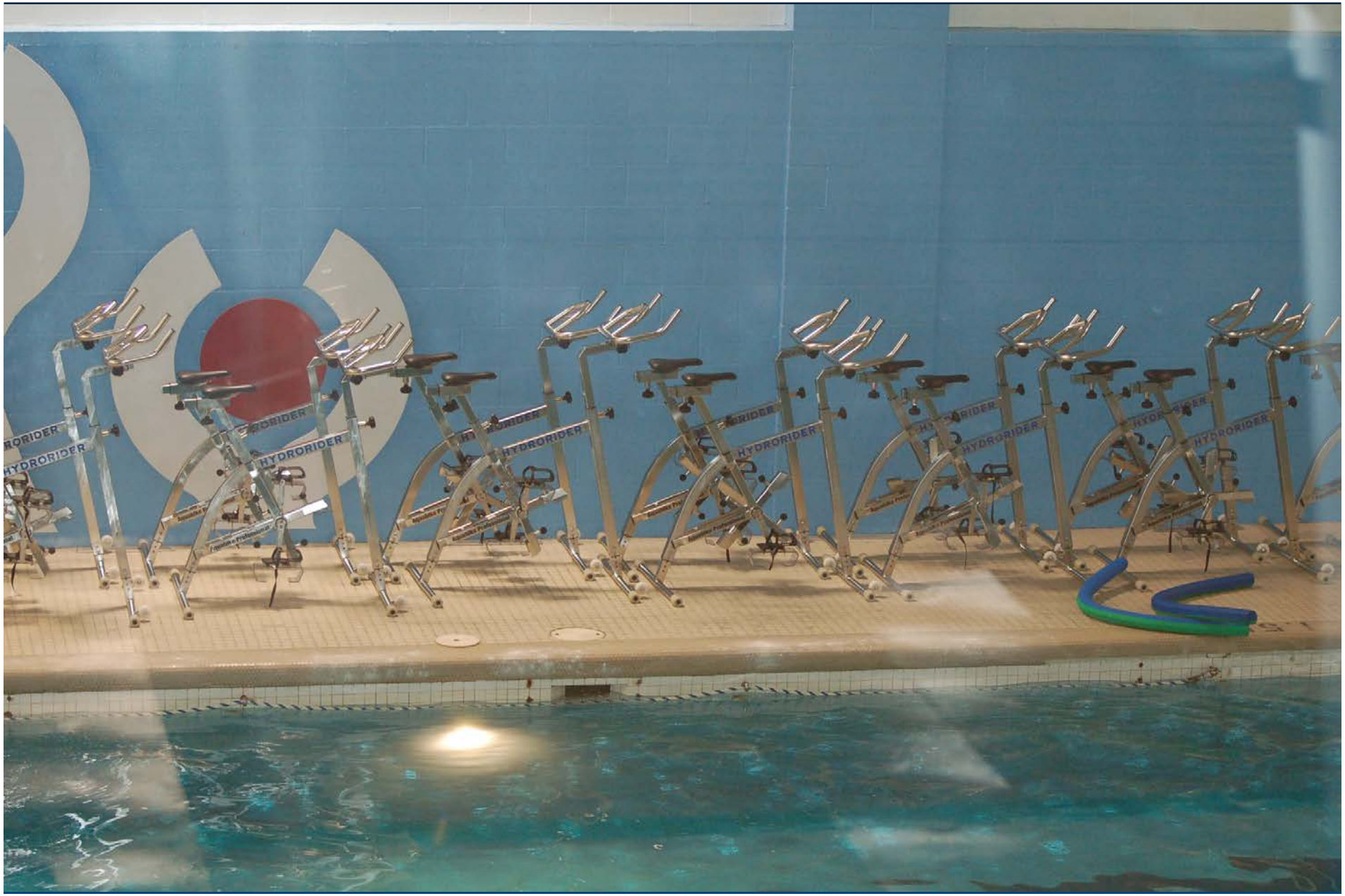
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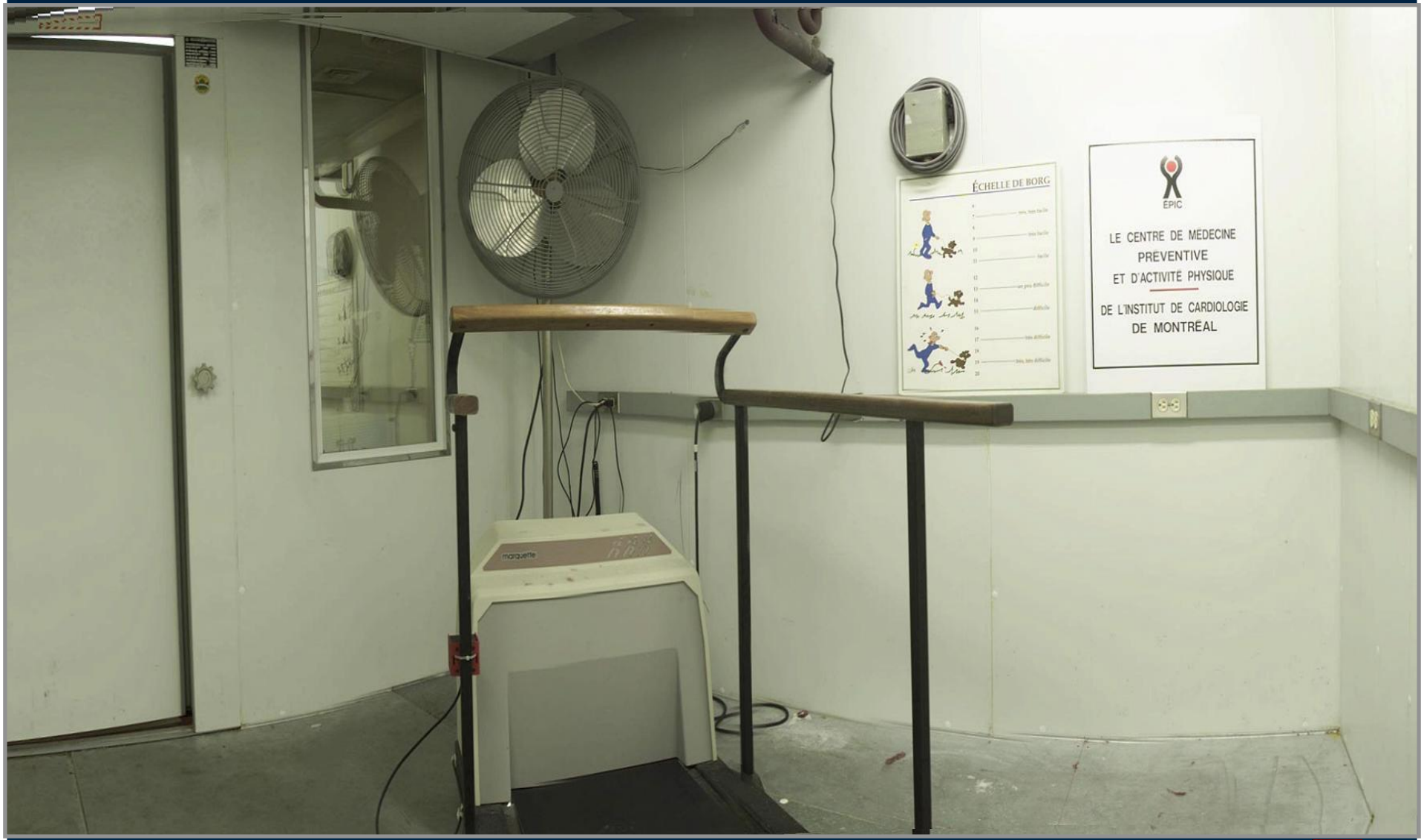
ÉCHELLE DE BORG
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ET D'ACTIVITÉ PHYSIQUE
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Promesse
promesse
Bon appé

Intensive versus Moderate Lipid Lowering with Statins after Acute Coronary Syndromes

Christopher P. Cannon, M.D., Eugene Braunwald, M.D., Carolyn H. McCabe, B.S., Daniel J. Rader, M.D., Jean L. Rouleau, M.D., Rene Belder, M.D., Steven V. Joyal, M.D., Karen A. Hill, B.A., Marc A. Pfeffer, M.D., Ph.D., Allan M. Skene, Ph.D. and the Pravastatin or Atorvastatin Evaluation and Infection Therapy-Thrombolysis in Myocardial Infarction 22 Investigators

N Engl J Med
Volume 350;15:1495-1504
April 8, 2004

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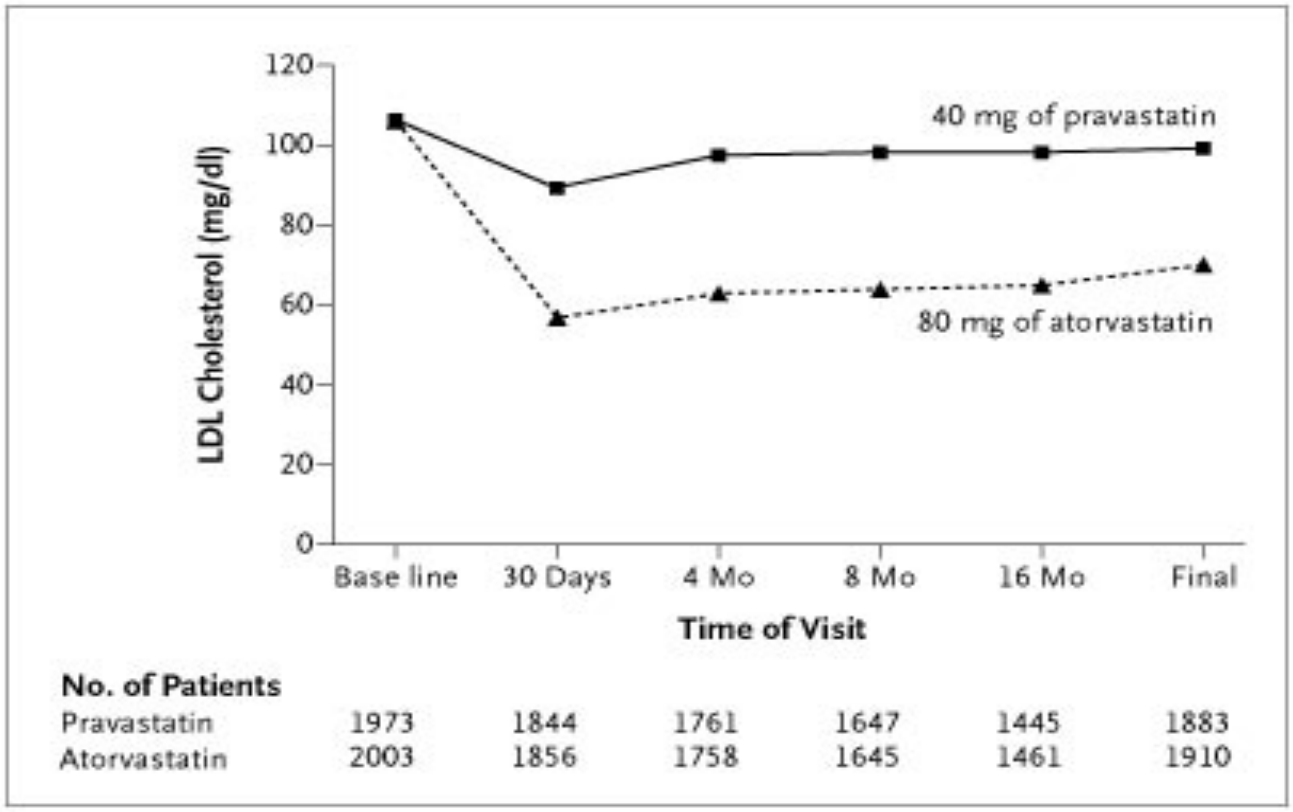
Median Low-Density Lipoprotein (LDL) Cholesterol Levels during the Study



No. of Patients						
Pravastatin	1973	1844	1761	1647	1445	1883
Atorvastatin	2003	1856	1758	1645	1461	1910

Cannon, C. et al. N Engl J Med 2004;350:1495-1504

The NEW ENGLAND JOURNAL of MEDICINE

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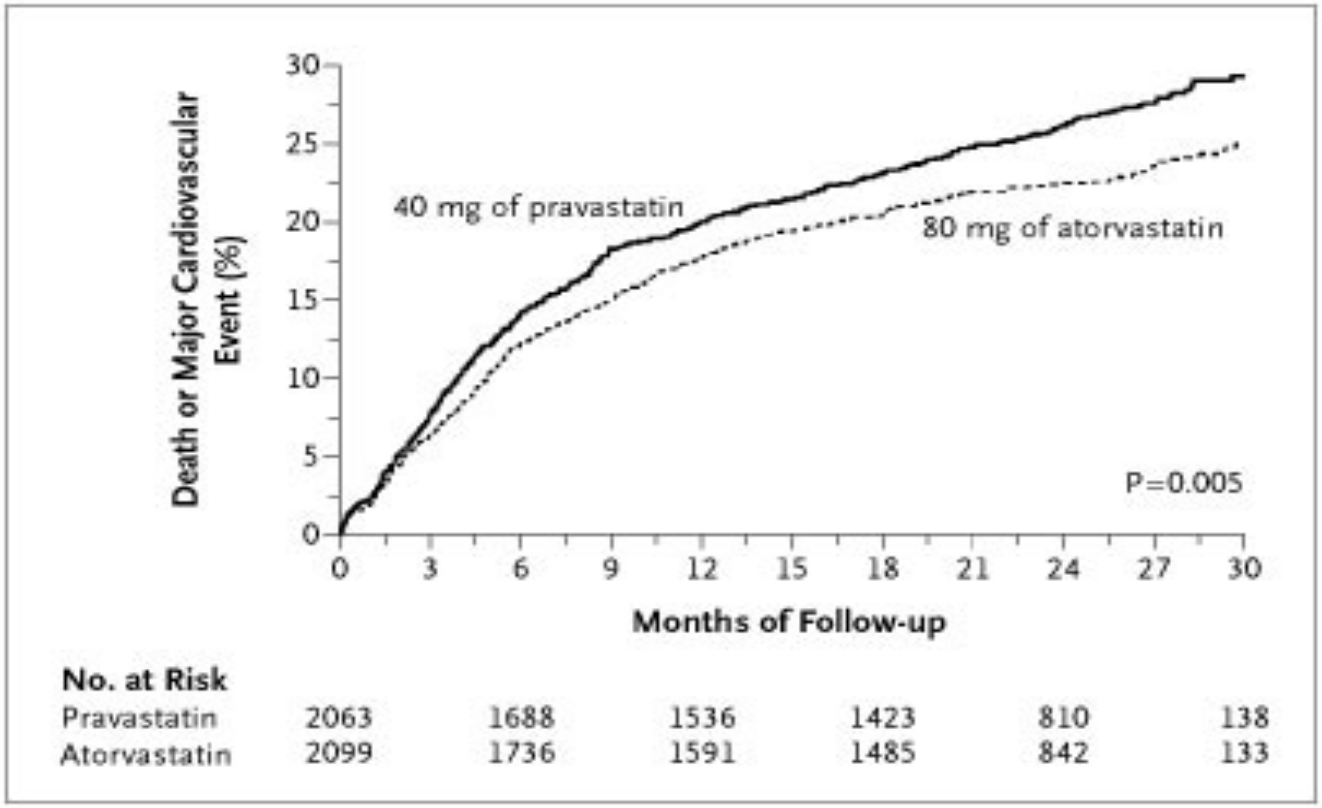
Kaplan-Meier Estimates of the Incidence of the Primary End Point of Death from Any Cause or a Major Cardiovascular Event



Cannon, C. et al. N Engl J Med 2004;350:1495-1504

The NEW ENGLAND JOURNAL of MEDICINE

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Epidemiology and Prevention

Association of Diet, Exercise, and Smoking Modification With Risk of Early Cardiovascular Events After Acute Coronary Syndromes

Clara K. Chow, MBBS, FRACP, PhD; Sanjit Jolly, MD, MSc, FRCPC; Purnima Rao-Melacini, MSc; Keith A.A. Fox, BSc (Hons), MB, ChB, FRCP, FESC, FMedSci; Sonia S. Anand, MD, PhD, FRCPc; Salim Yusuf, DPhil, FRCPC, FRSC

Background—Although preventive drug therapy is a priority after acute coronary syndrome, less is known about adherence to behavioral recommendations. The aim of this study was to examine the influence of adherence to behavioral recommendations in the short term on risk of cardiovascular events.

Methods and Results—The study population included 18 809 patients from 41 countries enrolled in the Organization to Assess Strategies in Acute Ischemic Syndromes (OASIS) 5 randomized clinical trial. At the 30-day follow-up, patients

(*Circulation*. 2010;121:750-758.)

Table 6. Relationship Between Diet/Exercise Modification and Repeat Cardiovascular Events in Patients With ACS

	Risk of MI		Risk of Stroke		Risk of Death		Risk of Death/MI/Stroke	
Category	OR (95% CI)	*P*	OR (95% CI)	*P*	OR (95% CI)	*P*	OR (95% CI)	*P*
No diet/exercise	Reference		Reference		Reference		Reference	
Diet only	0.93 (0.74–1.16)	0.5137	0.84 (0.54–1.31)	0.4357	0.91 (0.73–1.13)	0.3986	0.91 (0.77–1.07)	0.2605
Exercise only	0.78 (0.56–1.1)	0.1547	0.94 (0.52–1.68)	0.8256	0.61 (0.42–0.88)	0.0091	0.69 (0.54–0.89)	0.0037
Both diet and exercise	0.52 (0.40–0.69)	<0.0001	0.46 (0.26–0.82)	0.0079	0.45 (0.33–0.60)	<0.0001	0.46 (0.38–0.57)	<0.0001

Models were adjusted for age; sex; region; history of hypertension, diabetes, and prior MI; body mass index; creatinine; PCI/CABG before 30 days; and use of β-blockers, statins, antiplatelets, and ACE/ARB drugs at 30 days.

Relationship Between Healthy Diet and Risk of Cardiovascular Disease Among Patients on Drug Therapies for Secondary Prevention

A Prospective Cohort Study of 31 546 High-Risk Individuals From 40 Countries

Mahshid Dehghan, PhD; Andrew Mente, PhD; Koon K. Teo, PhD; Peggy Gao, MSc; Peter Sleight, DM; Gilles Dagenais, MD; Alvaro Avezum, MD; Jeffrey L. Probstfield, MD; Tony Dans, MD; Salim Yusuf, DPhil; on Behalf of the Ongoing Telmisartan Alone and in Combination With Ramipril Global End Point Trial (ONTARGET)/Telmisartan Randomized Assessment Study in ACEI Intolerant Subjects With Cardiovascular Disease (TRANSCEND) Trial Investigators

Background—Diet quality is strongly related to cardiovascular disease (CVD) incidence, but little is known about its impact on CVD events in older people at high risk of CVD and receiving effective drugs for secondary prevention. This study assessed the association between diet quality and CVD events in a large population of subjects from 40 countries with CVD or diabetes mellitus with end-organ damage receiving proven medications.

Methods and Results—Overall, 31 546 women and men 66.5±6.2 years of age enrolled in 2 randomized trials, the Ongoing Telmisartan Alone and in Combination With Ramipril Global End Point Trial (ONTARGET) and the Telmisartan Randomized Assessment Study in ACEI Intolerant Subjects With Cardiovascular Disease (TRANSCEND),

(*Circulation*. 2012;126:2705-2712.)

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Table 2. HRs and 95% CIs of Composite Outcome for Individuals With Different Types of Medication Use and Quintiles of Modified Alternative Healthy Eating Index (Quintile 5 Versus 1, Healthiest Versus Unhealthiest)

	mAHEI				*P* for Trend
	Q2 vs Q1	Q 3 vs Q1	Q4 vs Q1	Q5 vs Q1	
Aspirin use					
Yes (n=23 828)	0.96 (0.88–1.06)	0.86 (0.77–0.95)	0.80 (0.71–0.89)	0.79 (0.70–0.89)	<0.001
No (n=7718)	0.92 (0.78–1.08)	0.92 (0.78–1.10)	0.85 (0.71–1.02)	0.72 (0.60–0.87)	<0.001
β-blocker use					
Yes (n=18 036)	1.00 (0.89–1.12)	0.85 (0.75–0.96)	0.83 (0.72–0.95)	0.75 (0.66–0.87)	<0.001
No (n=13 510)	0.91 (0.80–1.02)	0.91 (0.80–1.04)	0.80 (0.70–0.92)	0.81 (0.71–0.93)	<0.001
Statin use					
Yes (n=19 055)	0.96 (0.86–1.07)	0.85 (0.74–0.97)	0.80 (0.70–0.92)	0.76 (0.66–0.87)	<0.001
No (n=12 491)	0.95 (0.83–1.07)	0.91 (0.80–1.04)	0.83 (0.72–0.96)	0.81 (0.71–0.94)	<0.001
Combination of any drugs					
Any 1 drug (n=28 721)*	0.95 (0.87–1.04)	0.86 (0.78–0.95)	0.81 (0.73–0.90)	0.77 (0.70–0.86)	<0.001
Any 2 drugs (n=11 192)	0.94 (0.82–1.08)	0.87 (0.75–1.00)	0.81 (0.70–0.94)	0.77 (0.65–0.90)	<0.001
Any 3 drugs (n=10 503)	1.02 (0.87–1.20)	0.84 (0.70–0.99)	0.79 (0.66–0.96)	0.77 (0.63–0.93)	<0.001

HR indicates hazard ratio; CI, confidence interval; mAHEI, modified Alternative Healthy Eating Index; and Q, quintile. All HRs are adjusted for age; sex; region; trial enrollment allocation; education; smoking; physical activity; body mass index; systolic and diastolic blood pressures; history of hypertension, diabetes mellitus, and stroke/transient ischemic attack; β-blockers; calcium channel blockers; antiplatelets; and statin. Categories of covariate adjustments were as follows. Regions: West region versus South America,

Beyond Restenosis

Five-Year Clinical Outcomes From Second-Generation Coronary Stent Trials

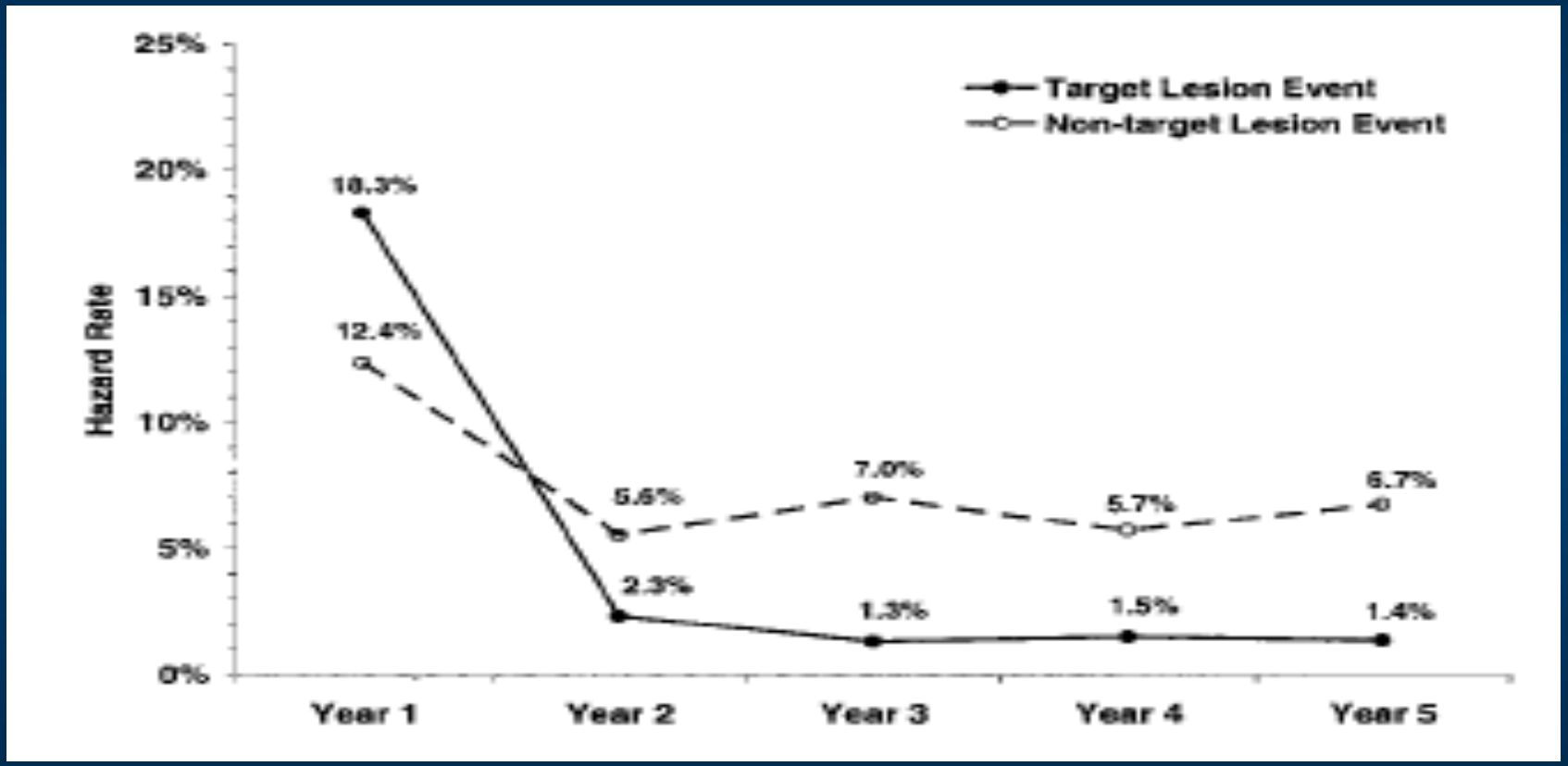
Donald E. Cutlip, MD; Amit G. Chhabra, MBBS, MPH; Donald S. Baim, MD; Manish S. Chauhan, MD; Sachin Marulkar, MBBS, MPH; Joseph Massaro, PhD; Ameet Bakhai, MD; David J. Cohen, MD, MSc; Richard E. Kuntz, MD, MSc; Kalon K.L. Ho, MD, MSc

Background—In the first year after coronary stent implantation, clinical failures are driven mainly by procedural complications and restenosis, but the subsequent relative contributions of restenosis and disease progression to late failures are less clear.

Methods and Results—We observed 1228 patients for 5 years after the implantation of stents as part of pivotal second-generation coronary stent trials. Clinical events of death, myocardial infarction, repeat revascularization, and repeat hospitalization for acute coronary syndrome or congestive heart failure were attributed to the index stented (target) lesion or other distinct sites (either in the target or other coronary vessels) and further classified as procedural, restenosis, or nonrestenosis. During the first year the hazard rate was 18.3% for target-lesion events and 12.4% for events unrelated to the target lesion. After the first year the average annual hazard rate was 1.7% for target-lesion events and 6.3% for nontarget-lesion events. By the fifth year, restenosis events occurred in 20.3% of patients, whereas 30-day procedural complications or later nonrestenosis events occurred in 37.9%, including 11.4% who also experienced a restenosis event, for a combined cumulative event rate of 46.4%. Diabetes mellitus and multivessel disease were independently associated with increased risk for both restenosis and nonrestenosis events.

Conclusion—In a low-risk clinical trial population, the clinical outcome beyond 1 year after stenting is determined by a high rate of events related to disease progression in segments other than the stented lesion, which itself remains relatively stable. **(*Circulation*. 2004;110:1226-1230.)**

Five-Year Clinical Outcomes From Second-Generation Coronary Stent Trials *Circulation*. 2004;110:1226-1230



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ORIGINAL INVESTIGATIONS

Low-Risk Diet and Lifestyle Habits in the Primary Prevention of Myocardial Infarction in Men

A Population-Based Prospective Cohort Study

Agneta Åkesson, PhD, Susanna C. Larsson, PhD, Andrea Discacciati, MSc, Alicja Wolk, DMSc

TABLE 3 Effect of Combined Low-Risk Behaviors in Relation to Risk of Myocardial Infarction*

Group	Low-Risk Group No. of Events (% Men)	Age-Standardized Incidence Rate† (95% CI)	Compared With High-Risk Group‡ RR (95% CI)	Compared With the Remainder of the Study Population RR (95% CI)	Population Attributable Risk§ % (95% CI)
1 low-risk factor: healthy diet (RFS top quintile)	177 (18)	495 (417-572)	0.74 (0.58-0.96)	0.82 (069-0.96)	16 (4-35)
2 low-risk factors‖: healthy diet (RFS top quintile), alcohol consumption 10-30 g/day	74 (8.7)	429 (321-537)	0.65 (0.48-0. 87)	0.75 (0.59-0.95)	23 (4-39)
3 low-risk factors¶: healthy diet (RFS top quintile), alcohol consumption 10-30 g/day, no smoking	36 (5.4)	321 (208-433)	0.36 (0.25-0.53)	0.54 (0.39-0.76)	44 (23-49)
4 low-risk factors#: healthy diet (RFS top quintile), alcohol consumption 10-30 g/day, no smoking, physically active (≥40 min/day of walking/ bicycling and ≥1 h/week of exercise)	9 (1.7)	218 (73-363)	0.24 (0.12-0.47)	0.36 (0.19-0.69)	64 (30-81)
5 low-risk factors: healthy diet (RFS top quintile), alcohol consumption 10-30 g/day, no smoking, physically active (≥40 min/day of walking/ bicycling and ≥1 h/week of exercise), waist circumference <95 cm	3 (1.0)	131 (0-279)	0.14 (0.04-0.43)	0.21 (0.07-0.66)	79 (34-93)

*All relative risks were adjusted for age (continuous), educational achievement (≤9, 10 to 12, >12 years), family history of myocardial infarction (yes/no), use of aspirin (yes/no), marital status (unmarried, married, divorced, widowed), non-Recommended Food Score (quintiles), and total energy intake (continuous). †Per 100,000 person-years. ‡The high-risk group (8.3% of the study population and 166 cases of myocardial infarction [age-standardized incidence rate 979 cases per 100,000 person-years]) included men with no low-risk factors and was characterized by the following: median 2.9 servings/day of vegetables and fruit, 3.0 servings/day of whole grains, and 1.4 servings/week of fish; 24 pack-years of tobacco smoking (55% reported to be current smokers); 36% reported neither ≥40 min of daily walking/bicycling nor ≥1 h per week of exercise; and a median waist circumference 101 cm. §Estimated compared with the remainder of the total study population, representing 91.3%, 94.6%, 98.3%, and 99%, respectively, for each additional low-risk factor. ‖The model was also adjusted for smoking, physical activity, and waist circumference. ¶The model was also adjusted for physical activity and waist circumference. #The model was also adjusted for waist circumference.

RFS = Recommended Food Score; other abbreviations as in Table 2.

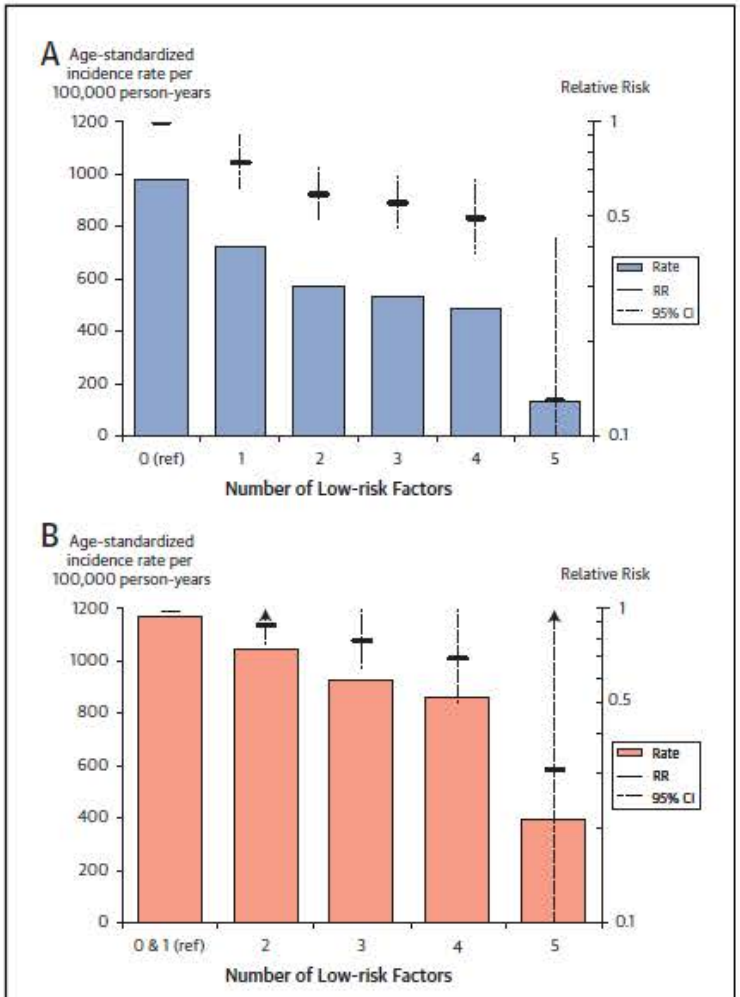
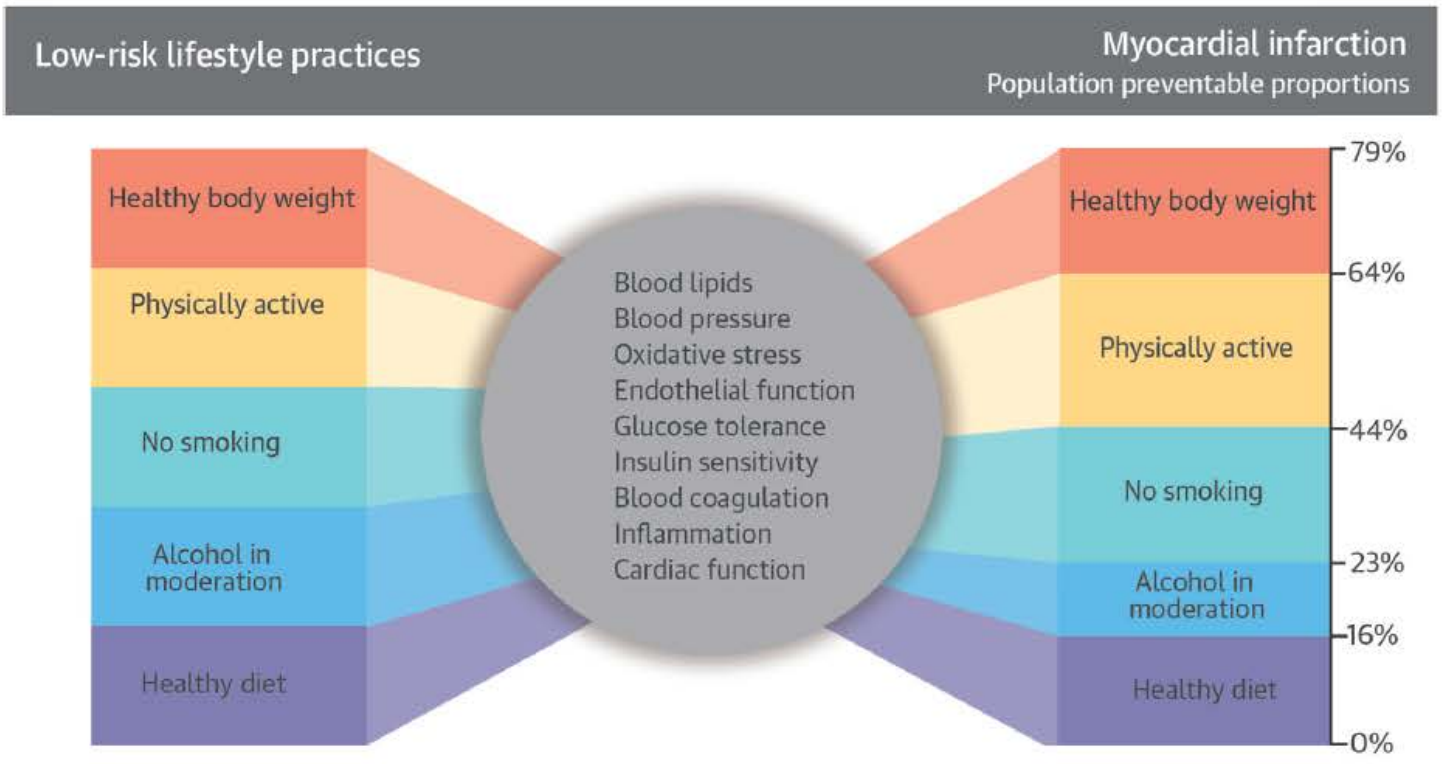


FIGURE 1 **MI Incidence for the Addition of Any Low-Risk Behavior**

Age-standardized incidence rates and multivariable-adjusted relative risks of MI for the addition of any single low-risk factor compared with the high-risk group for men without hypertension and high cholesterol (n = 20,721), p for trend < 0.001 **(A)**, and men with hypertension and high cholesterol (n = 7,139), p for trend = 0.002 (3 and 4 statistically



CENTRAL ILLUSTRATION **5 Combined Low-Risk Behaviors and the Population Preventable Proportions of MI**

The combination of the 5 low-risk dietary and lifestyle factors, the proposed intermediate biological factors, and the population preventable proportions of myocardial infarction.

EDITORIAL COMMENT

The Promise of Lifestyle for Cardiovascular Health

Time for Implementation*

Dariush Mozaffarian, MD, DrPH

and of abdominal obesity to 4 in 5 MIs. These findings highlight the primacy of healthy lifestyle. For both individual patients and populations, lifestyle goals should not be formulated solely for control of weight or blood pressure, cholesterol, and glucose levels. Although lifestyle has major benefits on these physiological factors, a healthier diet, greater activity, and nonsmoking influence numerous other pathways of risk and produce substantial additional benefits for cardiovascular and noncardiovascular health (5). For example, in the present investigation among >20,000 Swedish men, the combination of a healthier diet

an end and instead recognizing the relevance of lifestyle as a primary target for health. It is time for medical educators, clinicians, health administrators, and insurance providers to follow suit by designing and implementing a comprehensive, ambitious agenda to incorporate measures of and targets for dietary quality, physical activity, smoking, and central obesity into every aspect of the health system (9). Patients should enter their doctor's office and not simply ask "How are my blood pressure, cholesterol, and glucose levels?" but also ask "How are my dietary habits, physical activity level, smoking, and waist measurement?"

The New England
Journal of Medicine

VOLUME 346 MARCH 14, 2002 NUMBER 11

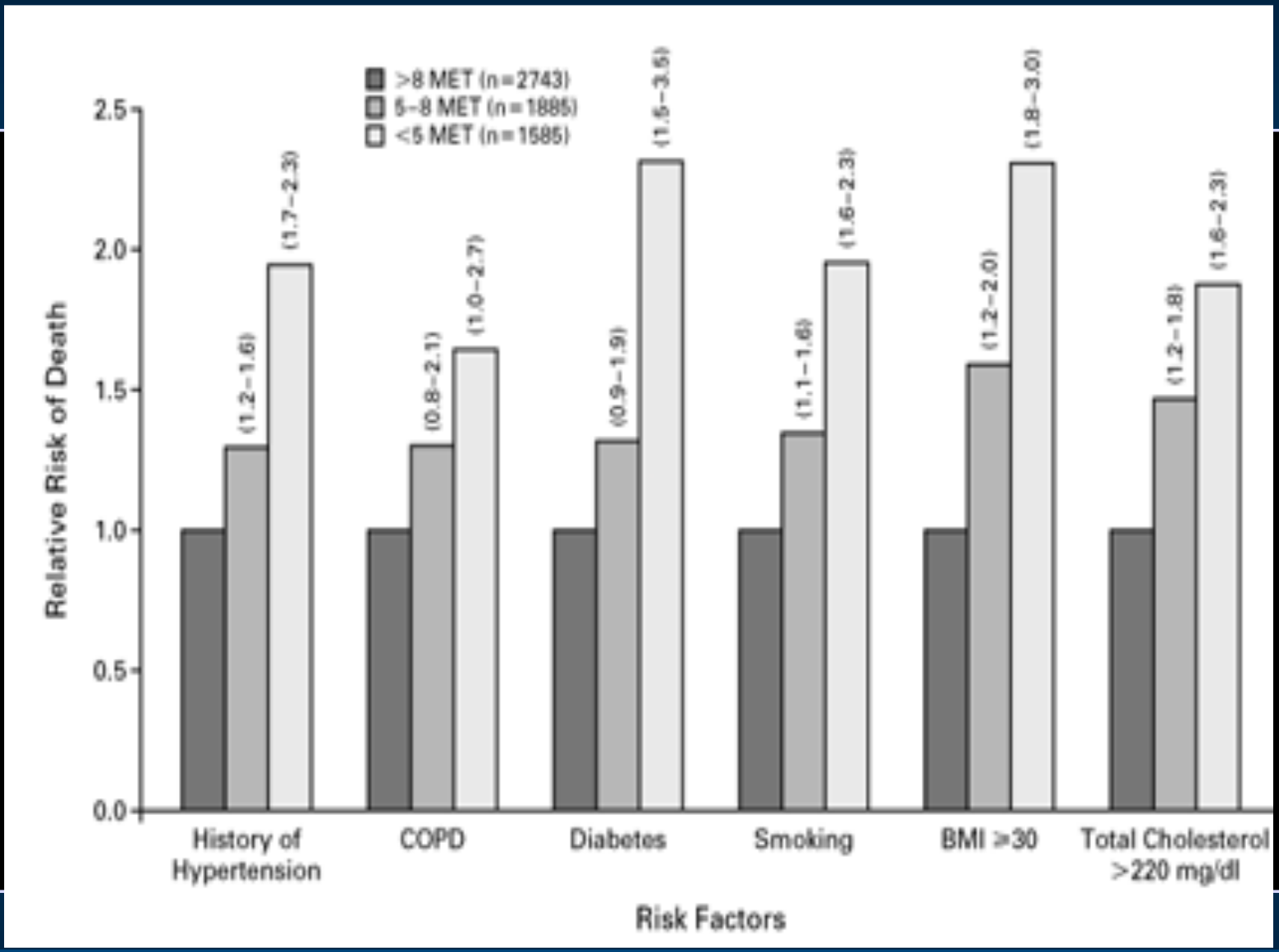
EXERCISE CAPACITY AND MORTALITY AMONG MEN REFERRED FOR EXERCISE TESTING

JONATHAN MYERS, PH.D., MANISH PRAKASH, M.D., VICTOR FROELICHER, M.D., DAT DO, M.D., SARA PARTINGTON, B.SC., AND J. EDWIN ATWOOD, M.D.

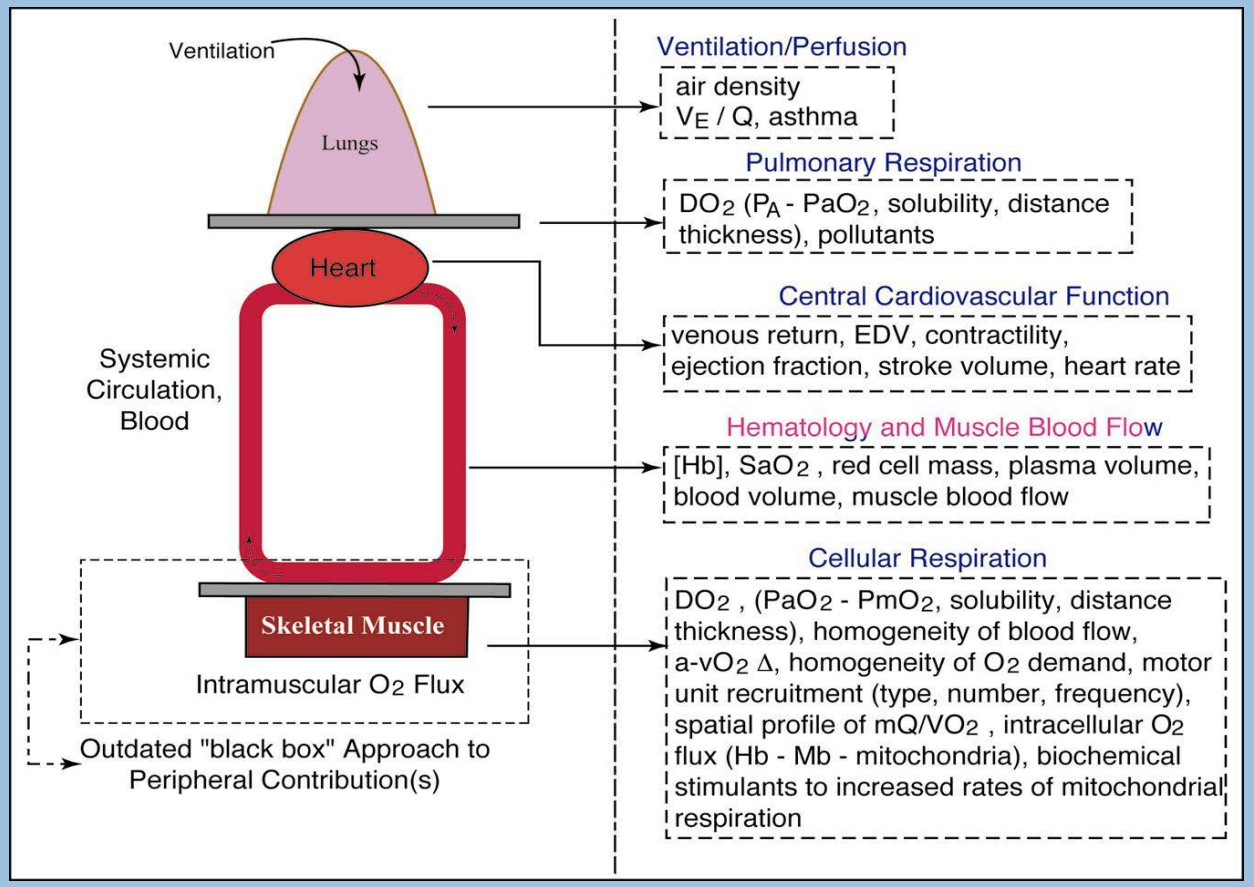
ABSTRACT

Background Exercise capacity is known to be an important prognostic factor in patients with cardiovascular disease, but it is uncertain whether it predicts mortality equally well among healthy persons. There is also uncertainty regarding the predictive power of

DURING the past two decades, exercise capacity and activity status have become well-established predictors of cardiovascular and overall mortality.[1,2] The fact that exercise capacity is a strong and independent predictor of outcomes supports the value of the exercise test as a clin-


>8 MET (n=2743)
5–8 MET (n=1885)
<5 MET (n=1585)
Relative Risk of Death
2.5
2.0
1.5
1.0
0.5
0.0
(1.2–1.6)
(1.7–2.3)
(0.8–2.1)
(1.0–2.7)
(0.9–1.9)
(1.5–3.5)
(1.1–1.6)
(1.6–2.3)
(1.2–2.0)
(1.8–3.0)
(1.2–1.8)
(1.6–2.3)
History of Hypertension
COPD
Diabetes
Smoking
BMI ≥30
Total Cholesterol >220 mg/dl
Risk Factors

Limitations de la capacité maximale:



(Wasserman et al 2005, Robergs 2001, ATS/ACCP Statement CPET 2003)

Basic Science for Clinicians

Running Forward

New Frontiers in Endurance Exercise Biology

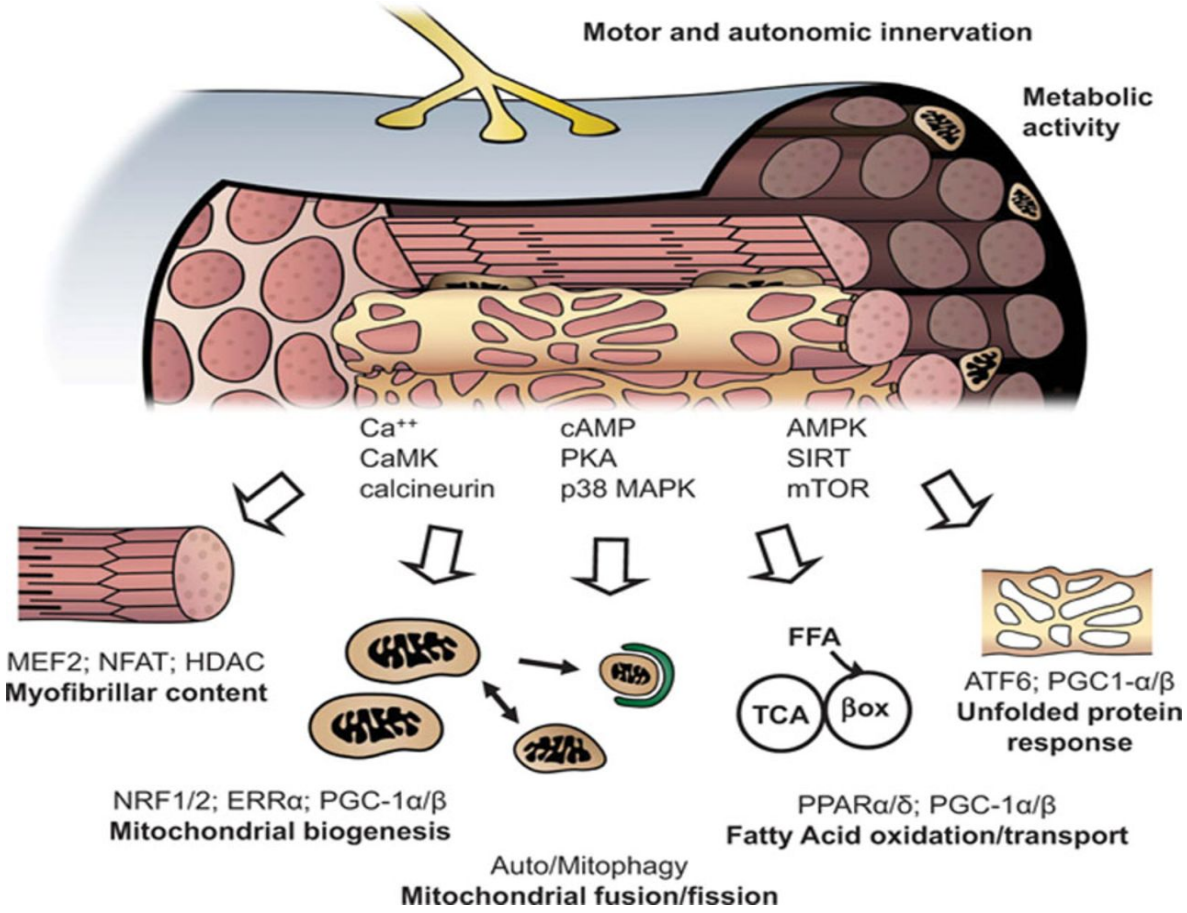
Glenn C. Rowe, PhD; Adeel Safdar, PhD; Zolt Arany, MD, PhD

The many long-term benefits of regular endurance exercise.

Rowe G et al. Circulation 2014;129:798-810

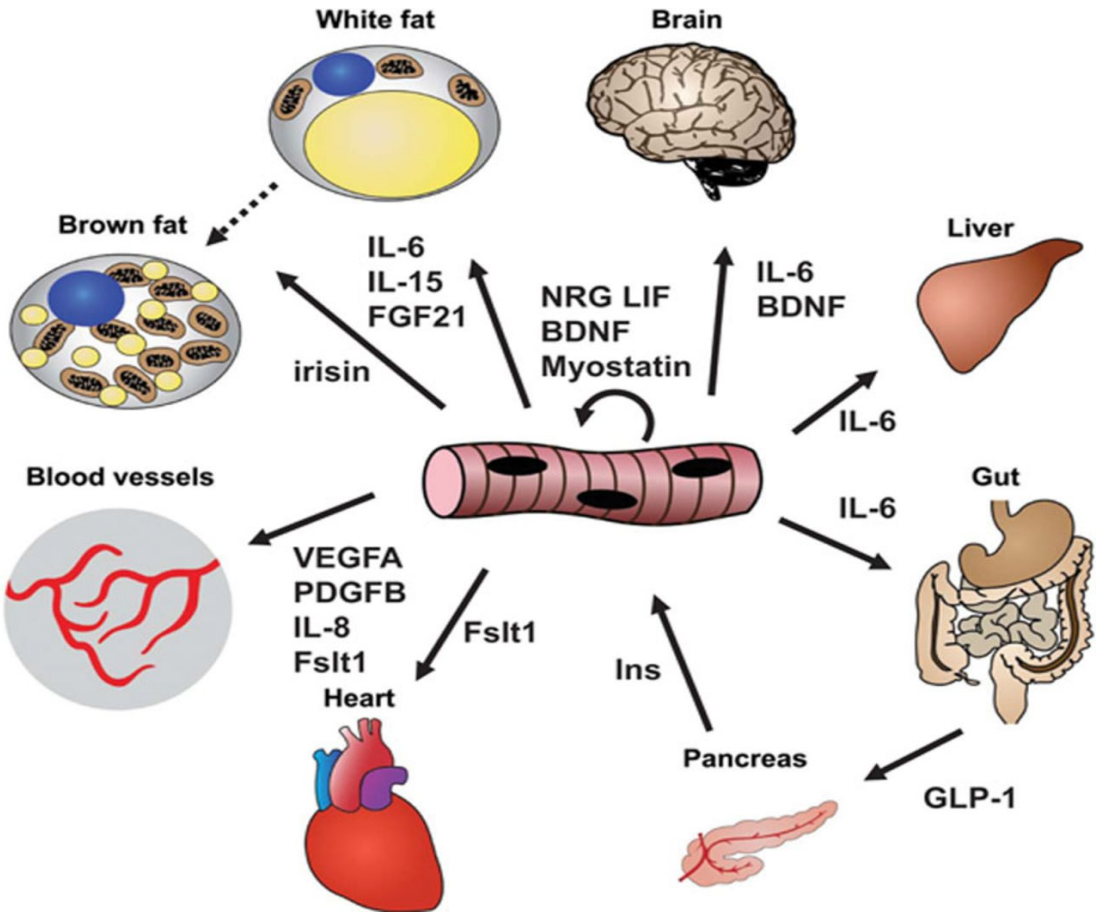
American Heart Association.

Modular signaling pathways that underpin muscular adaptations to endurance exercise.



Rowe G et al. Circulation 2014;129:798-810

Muscle as an endocrine organ.



Rowe G et al. Circulation 2014;129:798-810

American Heart Association.

PHYSIOLOGY 28: 330–358, 2013; doi:10.1152/physiol.00019.2013

Exercise is the Real Polypill

The concept of a "polypill" is receiving growing attention to prevent cardiovascular disease. Yet similar if not overall higher benefits are achievable with regular exercise, a drug-free intervention for which our genome has been haped over evolution. Compared with drugs, exercise is available at low cost and relatively free of adverse effects. We summarize epidemiological evidence on the preventive/therapeutic benefits of exercise and on the main biological mediators involved.

Carmen Fiuza-Luces,[1,2]
Nuria Garatachea,[3]
Nathan A. Berger,[4] and
Alejandro Lucia[1,2]

[1]Universidad Europea Madrid, Madrid, Spain; [2]Instituto de Investigación, Hospital 12 de Octubre, Madrid, Spain; [3]Facultad de Ciencias de la Salud y del Deporte, Universidad de Zaragoza, Huesca, Spain; and [4]Center for Science, Health and Society, Case Western Reserve University, School of Medicine, Cleveland, Ohio
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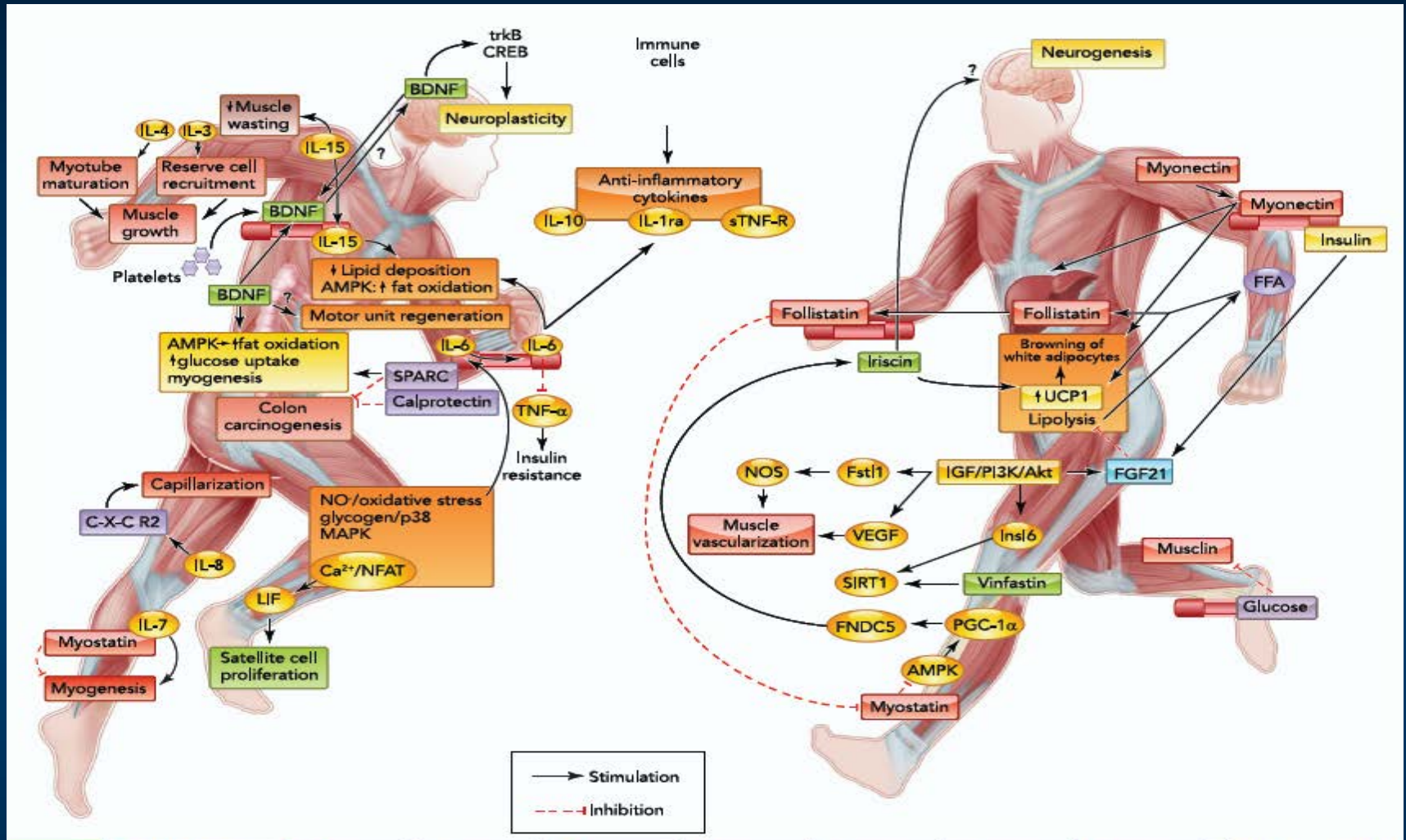

FIGURE 2. **Summary of the main myokines, their putative effects, and the molecular signals/pathways involved**
AMPK, AMP-activated protein kinase; BDNF, brain-derived neurotrophic factor; CREB, cAMP response-element-binding protein; C-X-C R2, C-X-C receptor 2; FFA, free-fatty acid; FGF21, fibroblast growth factor 21; Fndc5, fibronectin type III domain-containing 5 protein; Fstl1, follistatin-like 1; IGF, insulin-like growth factor; IL-1ra, IL-1 receptor antagonist; Insl6, insulin-like 6; LIF, leukemia inhibitory factor; $NO^{\cdot}$, nitric oxide; NOS, nitric oxide synthase; PGC-1α, peroxisome proliferator-activated receptor-γ coactivator 1α; PI3K, phosphatidylinositol 3-kinase; SIRT1, sirtuin 1; SPARC, secreted protein acidic and rich in cysteine; sTNF-R, soluble TNF receptors; trkB, tropomyosin receptor kinase; UCP1, uncoupling protein 1.

Minimum amount of physical activity for reduced mortality and extended life expectancy: a prospective cohort study

Chi Pang Wen, Jackson Pui Man Wai*, Min Kuang Tsai, Yi Chen Yang, Ting Yuan David Cheng, Meng-Chih Lee, Hui Ting Chan, Chwen Keng Tsao, Shan Pou Tsai, Xifeng Wu*

Summary

Background The health benefits of leisure-time physical activity are well known, but whether less exercise than the recommended 150 min a week can have life expectancy benefits is unclear. We assessed the health benefits of a range of volumes of physical activity in a Taiwanese population.

Methods In this prospective cohort study, 416 175 individuals (199 265 men and 216 910 women) participated in a standard medical screening programme in Taiwan between 1996 and 2008, with an average follow-up of 8·05 years

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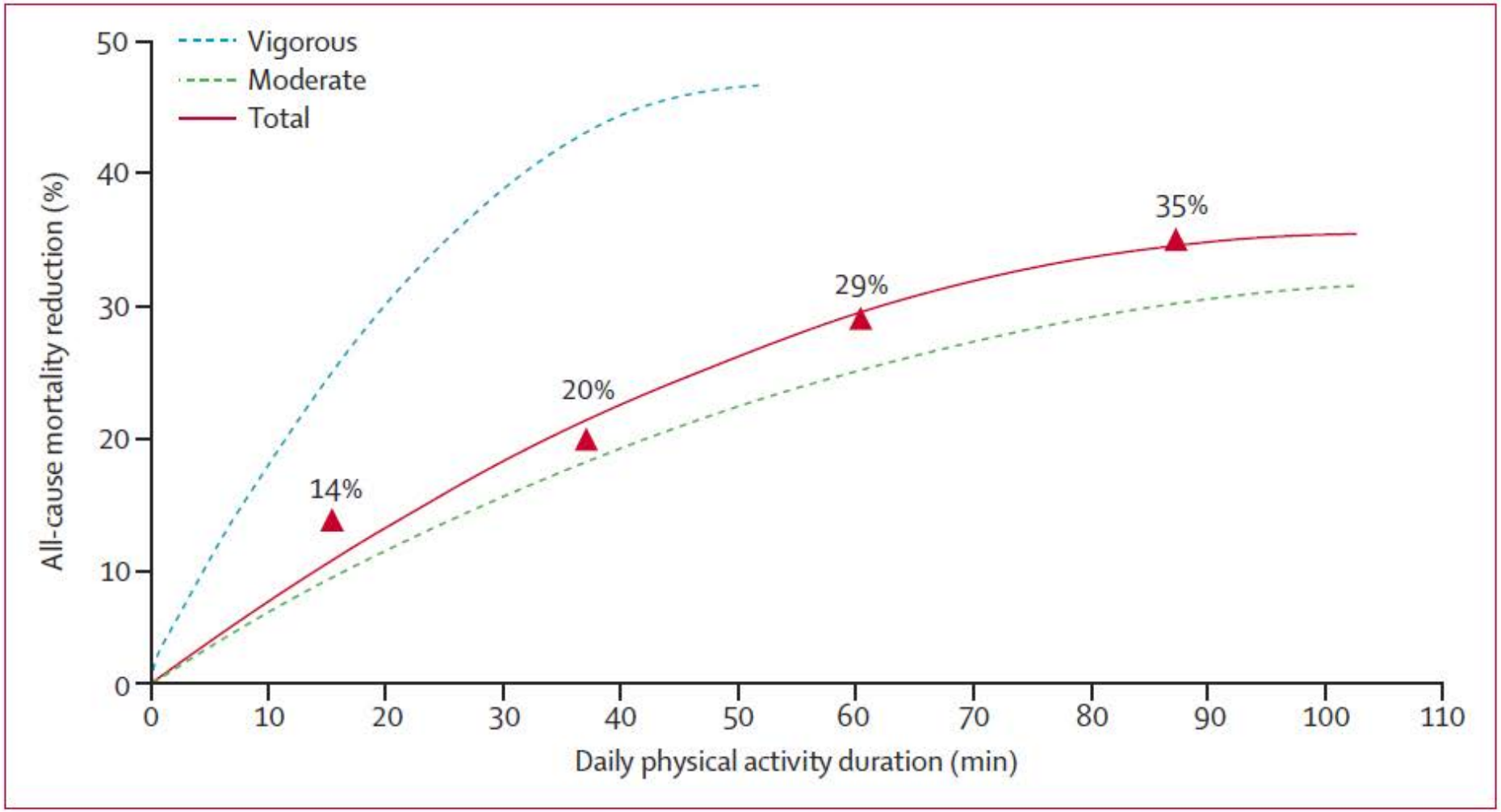
See Online/Comment
DOI:10.1016/S0140-6736(11)61029-5


Vigorous
Moderate
Total
All-cause mortality reduction (%)
50
40
30
20
10
0
14%
20%
29%
35%
0
10
20
30
40
50
60
70
80
90
100
110
Daily physical activity duration (min)

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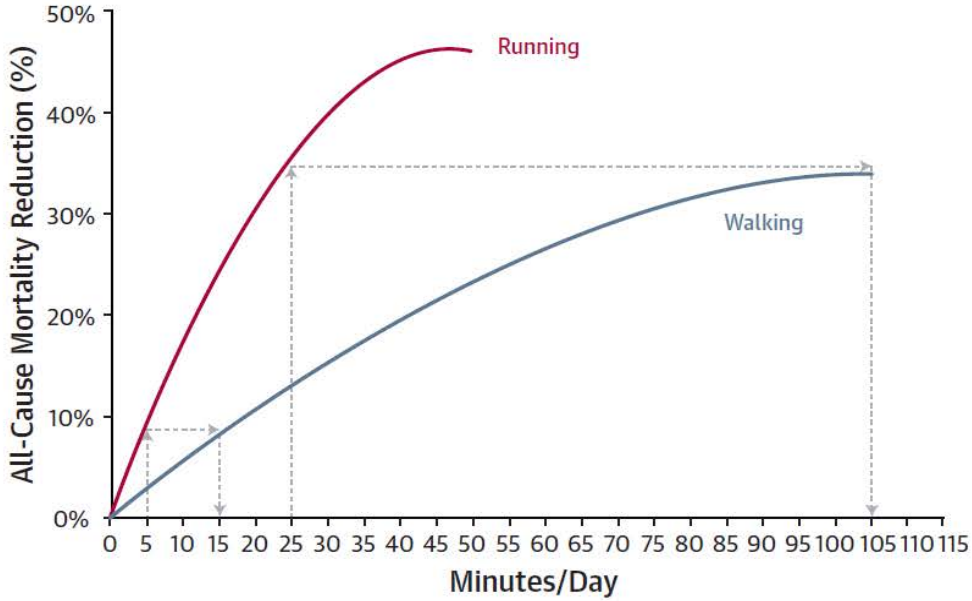

FIGURE 1 Comparison of Benefits Between Walking and Running

A 5-min run generates the same benefits as a 15-min walk, and a 25-min run is equivalent to a 105-min walk.

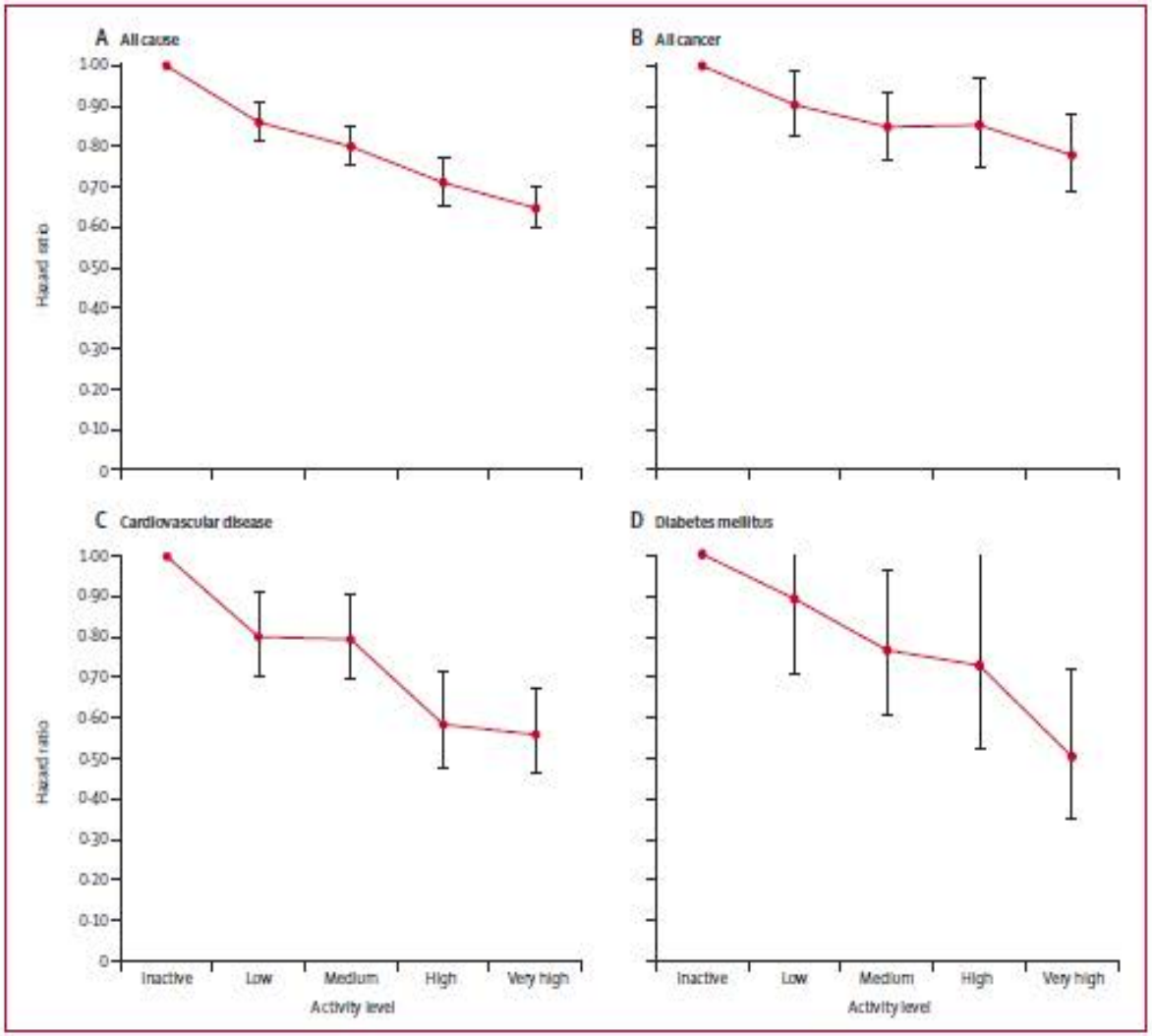

Figure 1: Relation between physical activity volume and mortality reduction compared with individuals in the inactive group
Bars show 95% CIs.

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Montreal Heart Institute and Université de Montréal,
5000 Bélanger Street, Montreal, Quebec H1T 1C8, Canada

Survival benefit associated with low-level physical activity

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DOI:10.1016/S0140-6736(11)61029-5
See Online/Articles
DOI:10.1016/S0140-6736(11)60749-6

In *The Lancet*, Chi Pang Wen and colleagues[1] report their findings from a very large observational study, showing that a small amount of leisure-time physical activity reduces total mortality, mortality from cardiovascular disease, and mortality from cancer. Although the ability of physical activity in moderate amounts to reduce mortality from all causes has been well documented,[2-4] the public-health recommendation in most countries is to do the equivalent of at least 30 min per day of walking, most days of the week—ie, 150 min per week.[5,6] Wen and colleagues' study shows that half this amount of physical activity (15 min per day for 6 days a week) reduces all-cause mortality by 14%, cancer mortality by 10%, and mortality from cardiovascular disease by 20%. To our knowledge, this is the first observational study of this size to report important and global health benefits at such a low volume of leisure-time physical activity with this degree of precision. The benefits of physical activity follow a dose-response curve (figure), which clearly shows that although a little amount of physical activity is good, more is better. In an ideal world, people would benefit greatly from 300 min of moderate-intensity physical activity per week, but data from most countries show that this amount of physical activity is achieved by only a small proportion of the population.[5,7] The reason for this reality is multifactorial and complex, and individual, psychosocial, and environmental factors all play a part.[8] Repeated, simple advice from a physician—as Wen and colleagues suggest—is one of many interventions that can effectively contribute to increased physical activity.[9] We agree that this advice is very simple and probably easily achievable.

Because of its observational nature, Wen and colleagues' study cannot establish causality, but their results are entirely consistent with the findings of prospective randomised trials in secondary cardiovascular prevention that show a clear mortality benefit from regular exercise.[10,11] As such, the direct health benefits of exercise are irrefutable. Exercise can reduce cardiovascular mortality and, in particular, coronary mortality by many mechanisms, including improvements in endothelial function, autonomic tone, inflammation, and risk-factor control. The final common pathways of cardiovascular risk reduction presumably operate through both improved endothelial function and improved autonomic regulation of cardiovascular function.[12] Improved endothelial function leads to the prevention and stabilisation of coronary atherosclerosis, thereby reducing the risk of acute coronary syndromes. Improved autonomic function leads to a reduced risk of sudden cardiac death. Cancer, like coronary heart disease, is also to an extent preventable and shares several common risk factors such as poor nutrition, obesity, inflammation, and physical inactivity. Therefore, improvements in some of these risk factors with regular exercise could plausibly explain the cancer mortality benefits recorded by Wen and colleagues. The oncoprotective effects of exercise are certainly an expanding topic of research in cancer.[13]

Finally, noteworthy from a public health perspective, 54% of individuals in the Taiwanese cohort studied were inactive, with another 22% doing low levels of leisure-time physical activity only. Rural-to-urban migration across the Asia-Pacific region through rapid economic growth and industrialisation during the past few decades could explain these low levels of physical activity and the concomitant epidemics of obesity and diabetes that are being witnessed.[14] The knowledge that as little as 15 min per day of exercise on most days of the week can substantially reduce an individual's risk of dying could encourage many more individuals to incorporate a small amount of physical activity into their busy lives. Governments and health professionals both have major roles to play to spread this good news story and convince people of the importance of being at least minimally active.

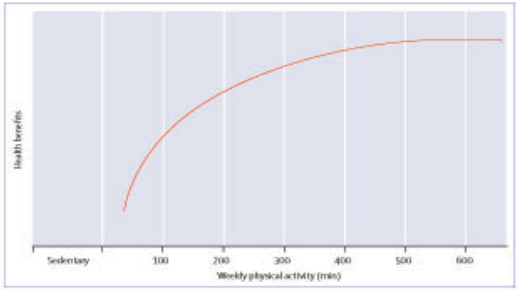

Figure: Relation between health benefits and physical activity

MJ

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Leisure-Time Running Reduces All-Cause and Cardiovascular Mortality Risk

Duck-chul Lee, PhD,* Russell R. Pate, PhD,† Carl J. Lavie, MD,‡§ Xuemei Sui, MD, PhD,† Timothy S. Church, MD, PhD,§ Steven N. Blair, PED‖

ABSTRACT

BACKGROUND Although running is a popular leisure-time physical activity, little is known about the long-term effects of running on mortality. The dose-response relations between running, as well as the change in running behaviors over time, and mortality remain uncertain.

OBJECTIVES We examined the associations of running with all-cause and cardiovascular mortality risks in 55,137 adults, 18 to 100 years of age (mean age 44 years).

METHODS Running was assessed on a medical history questionnaire by leisure-time activity.

RESULTS During a mean follow-up of 15 years, 3,413 all-cause and 1,217 cardiovascular deaths occurred. Approximately 24% of adults participated in running in this population. Compared with nonrunners, runners had 30% and 45% lower adjusted risks of all-cause and cardiovascular mortality, respectively, with a 3-year life expectancy benefit. In dose-response analyses, the mortality benefits in runners were similar across quintiles of running time, distance, frequency, amount, and speed, compared with nonrunners. Weekly running even <51 min, <6 miles, 1 to 2 times, <506 metabolic equivalent-minutes, or <6 miles/h was sufficient to reduce risk of mortality, compared with not running. In the analyses of change in running behaviors and mortality, persistent runners had the most significant benefits, with 29% and 50% lower risks of all-cause and cardiovascular mortality, respectively, compared with never-runners.

CONCLUSIONS Running, even 5 to 10 min/day and at slow speeds <6 miles/h, is associated with markedly reduced risks of death from all causes and cardiovascular disease. This study may motivate healthy but sedentary individuals to begin and continue running for substantial and attainable mortality benefits. (J Am Coll Cardiol 2014;64:472–81)

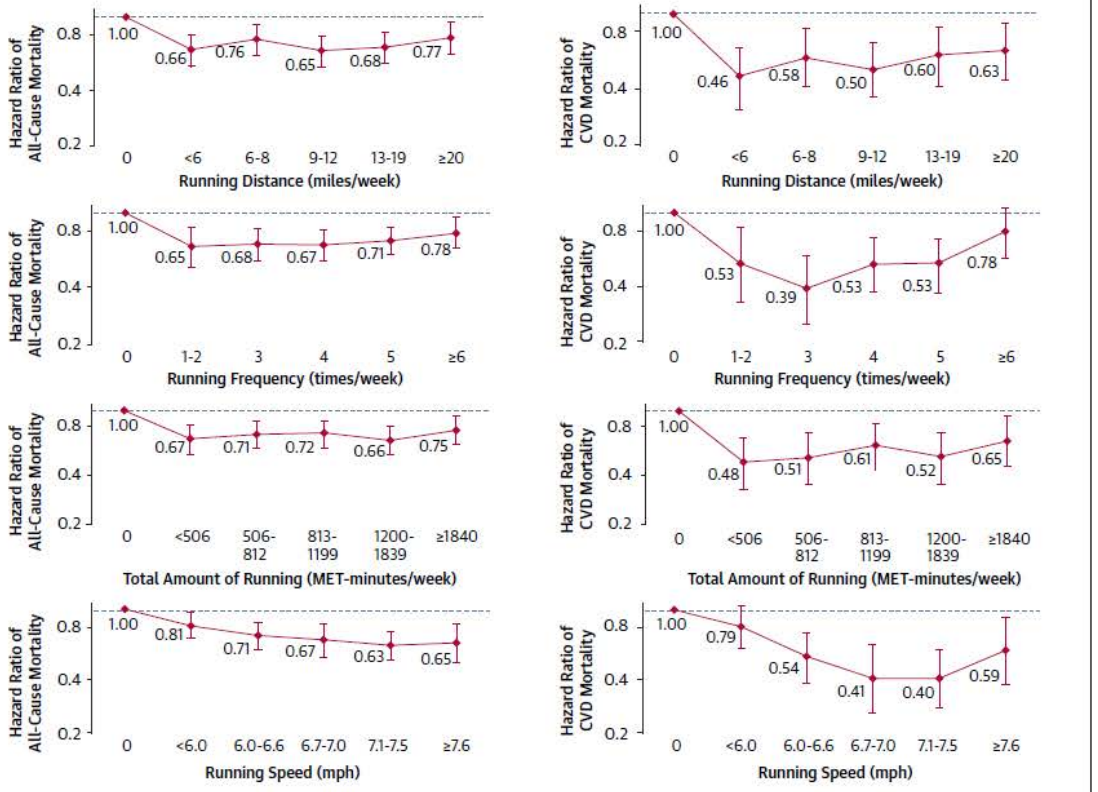
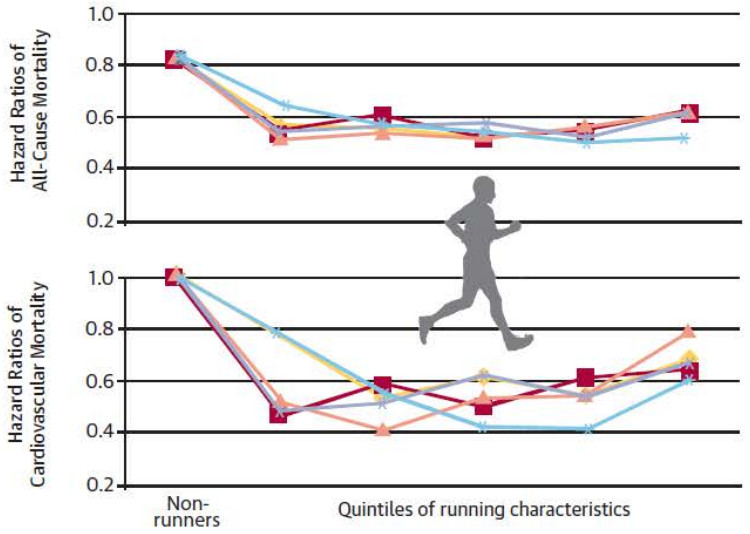


FIGURE 2 **HRs of All-Cause and Cardiovascular Mortality by Running Distance, Frequency, Total Amount, and Speed**

Participants were classified into 6 groups: nonrunners and 5 quintiles of each running distance, frequency, total amount, and speed. All hazard ratios (HRs) were adjusted for baseline age (years), sex, examination year, smoking status (never, former, or current), alcohol consumption (heavy drinker or not), other physical activities except running (0, 1 to 499, or ≥500 MET-min/week), and parental cardiovascular disease (CVD) (yes or no). The **bars** indicate 95% CI, and HRs are shown next to the **bars**. MET = metabolic equivalent.



Time (min/wk)	0	<51	51-80	81-119	120-175	≥176
Distance (miles/wk)	0	<6	6-8	9-12	13-19	≥20
Frequency (times/wk)	0	1-2	3	4	5	≥6
Total amount (MET-min/wk)	0	<506	506-812	813-1199	1200-1839	≥1840
Speed (mph)	0	<6.0	6.0-6.6	6.7-7.0	7.1-7.5	≥7.6

CENTRAL ILLUSTRATION **Leisure-Time Running Reduced All-Cause and Cardiovascular Mortality Risk**

Hazard ratios (HRs) of all-cause and cardiovascular mortality by running characteristic (weekly running time, distance, frequency, total amount, and speed). Participants were classified into 6 groups: nonrunners (reference group) and 5 quintiles of each running characteristic. All HRs were adjusted for baseline age (years), sex, examination year, smoking status (never, former, or current), alcohol consumption (heavy drinker or not), other physical activities except running (0, 1 to 499, or ≥500 MET-minutes/week), and parental history of cardiovascular disease (yes or no). All p values for HRs across running characteristics were <0.05 for all-cause and cardiovascular mortality except for running frequency of ≥6 times/week ($p = 0.11$) and speed of <6.0 miles/h ($p = 0.10$) for cardiovascular mortality. Abbreviation as in Figure 2.

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Relationship of Sedentary Behavior and Physical Activity to Incident Cardiovascular Disease

Results From the Women's Health Initiative

Andrea K. Chomistek, ScD,* JoAnn E. Manson, MD, DrPH,† Marcia L. Stefanick, PhD,‡ Bing Lu, MD, DrPH,† Megan Sands-Lincoln, PhD,§ Scott B. Going, PhD,|| Lorena Garcia, PhD,¶ Matthew A. Allison, MD,# Stacy T. Sims, PhD,‡ Michael J. LaMonte, PhD,** Karen C. Johnson, MD,†† Charles B. Eaton, MD‡‡§§

Boston, Massachusetts; Stanford, Davis, and San Diego California; Philadelphia, Pennsylvania; Tucson, Arizona; Buffalo, New York; Memphis, Tennessee; and Providence and Pawtucket, Rhode Island

Objectives The aim of this study was to examine the independent and joint associations of sitting time and physical activity with risk of incident cardiovascular disease (CVD).

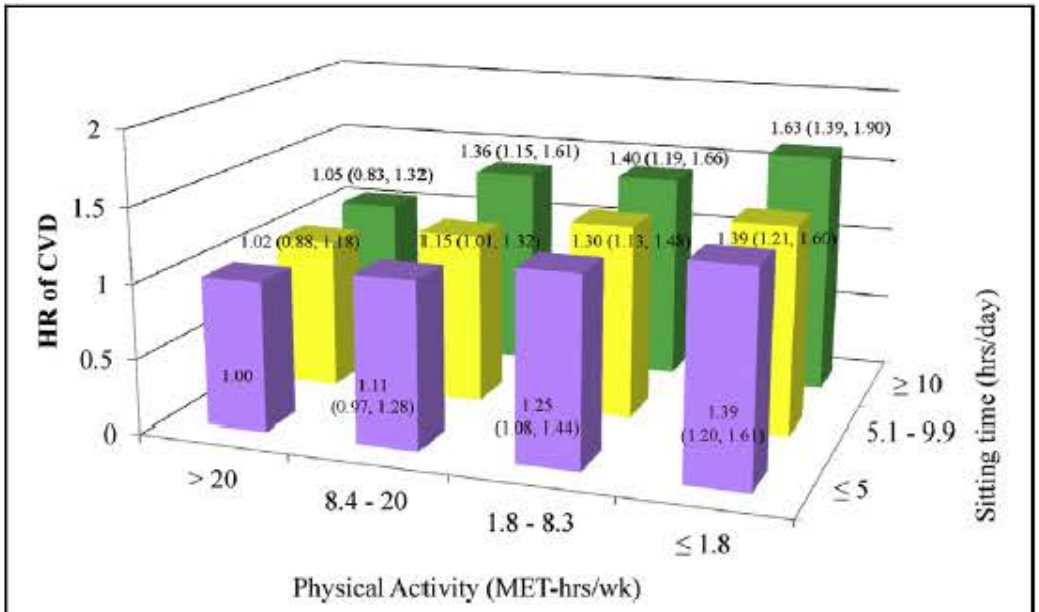
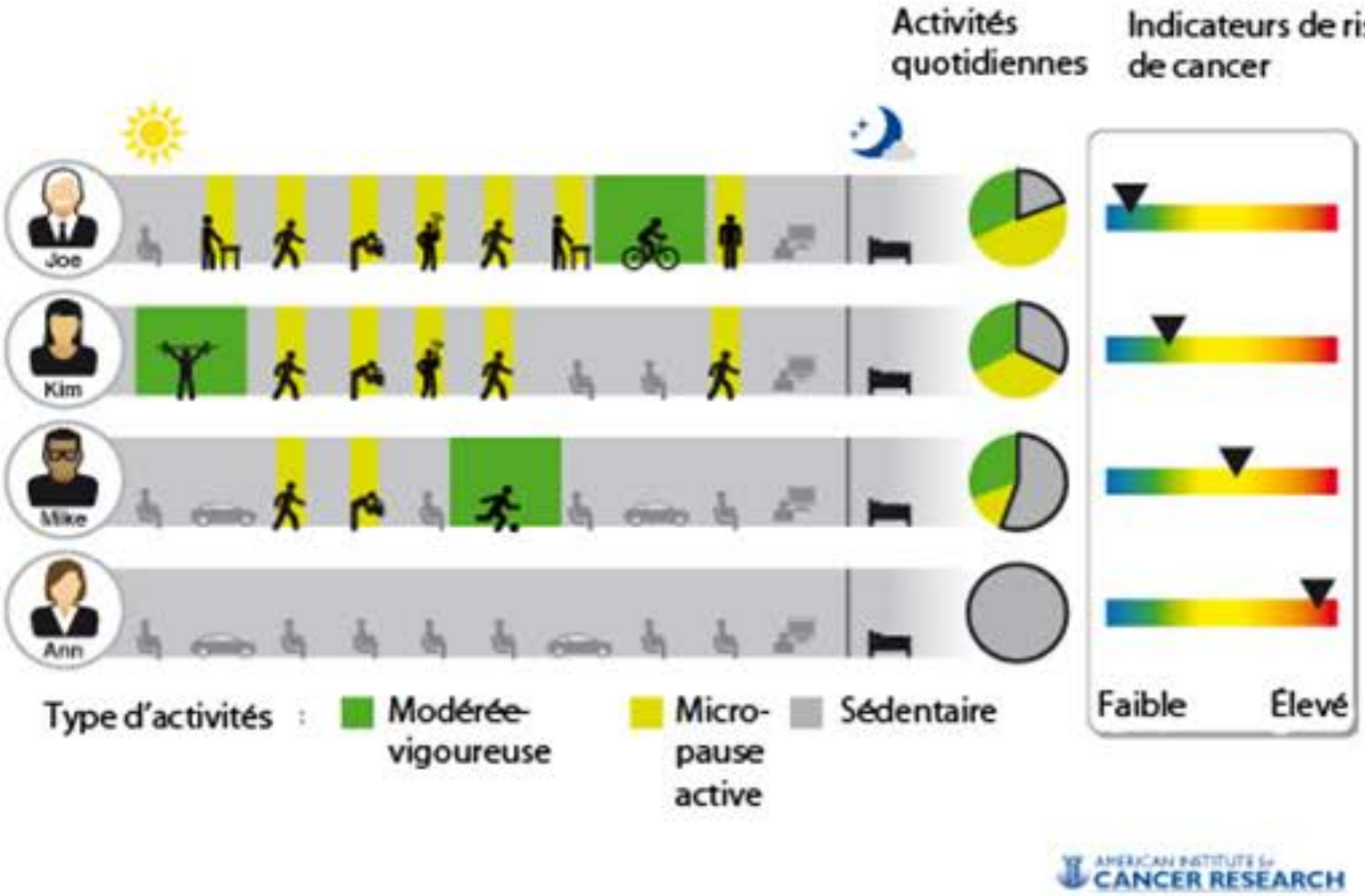


Figure 1 **Multivariable-Adjusted HRs for Total CVD for Joint Association Between Sedentary Time and Physical Activity**

The multivariable model was stratified by age and includes race; education; income; marital status; smoking; family history of myocardial infarction; depression; alcohol intake; hours of sleep; intake of total calories, saturated fat, and fiber; and body mass index. The p for interaction is 0.94.
CVD = cardiovascular disease; HR = hazard ratio; MET = metabolic equivalent task.


Activités quotidiennes
Indicateurs de risque de cancer
Joe
Kim
Mike
Ann
Type d'activités :
Modérée-vigoureuse
Micro-pause active
Sédentaire
Faible
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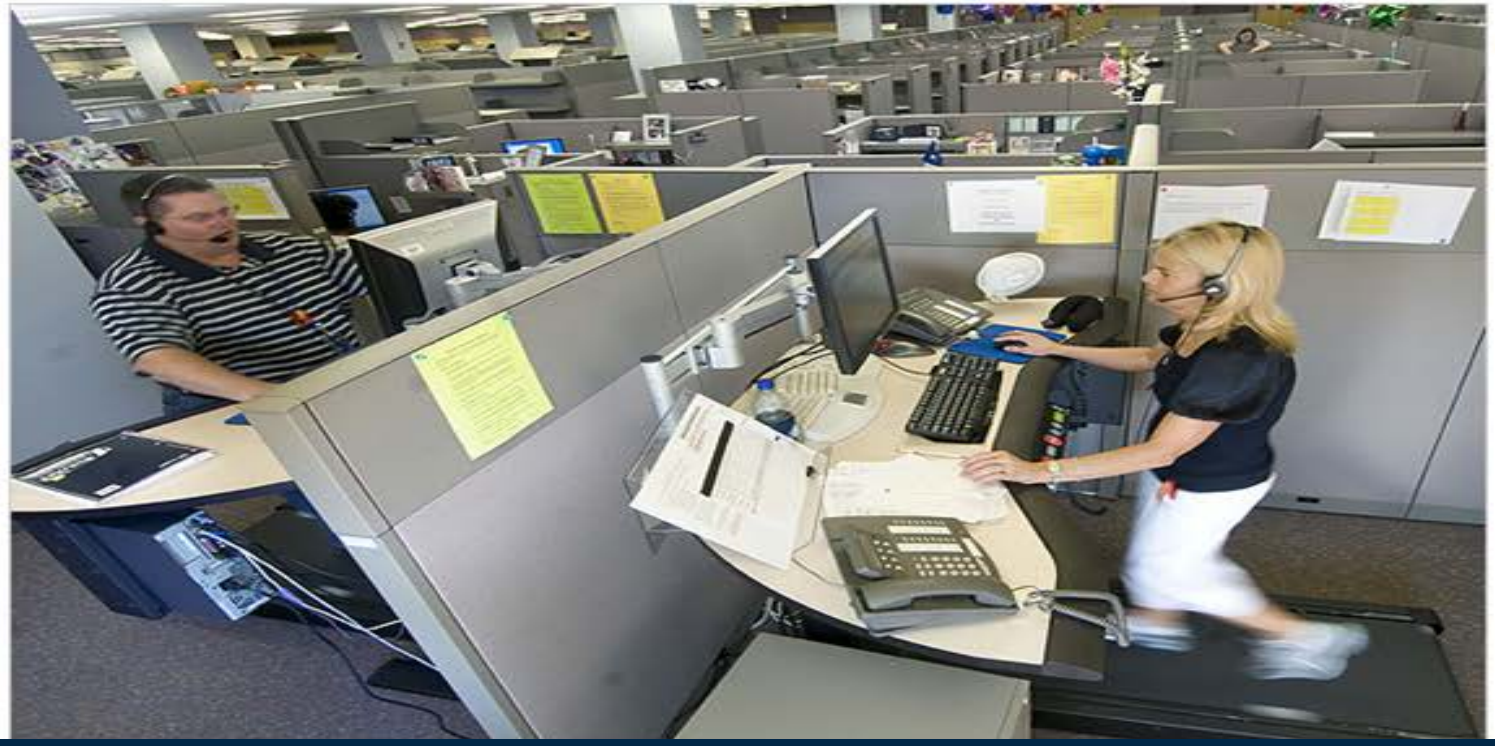

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- En moyenne, les Canadiens ont regardé 28 heures de télévision par semaine, nombre plus élevé que la moyenne de 26,5 heures en 2009. Cette augmentation est attribuable à l'utilisation d'un nouvel outil de mesure, plus précis.

Television Viewing and Risk of Type 2 Diabetes, Cardiovascular Disease, and All-Cause Mortality

A Meta-analysis

Anders Grøntved, MPH, MSc
Frank B. Hu, MD, PhD

Context Prolonged television (TV) viewing is the most prevalent and pervasive sedentary behavior in industrialized countries and has been associated with morbidity and mortality. However, a systematic and quantitative assessment of published studies is not available.

Objective To perform a meta-analysis of all prospective cohort studies to determine the association between TV viewing and risk of type 2 diabetes, fatal or nonfatal cardiovascular disease, and all-cause mortality.

Data Sources and Study Selection Relevant studies were identified by searches of the MEDLINE database from 1970 to March 2011 and the EMBASE database from 1974 to March 2011 without restrictions and by reviewing reference lists from retrieved articles. Cohort studies that reported relative risk estimates with 95% confidence intervals (CIs) for the associations of interest were included.

Data Extraction Data were extracted independently by each author and summary estimates of association were obtained using a random-effects model.

Data Synthesis Of the 8 studies included, 4 reported results on type 2 diabetes (175 938 individuals; 6428 incident cases during 1.1 million person-years of follow-up), 4 reported on fatal or nonfatal cardiovascular disease (34 253 individuals; 1052 incident cases), and 3 reported on all-cause mortality (26 509 individuals; 1879 deaths during 202 353 person-years of follow-up). The pooled relative risks per 2 hours of TV viewing per day were 1.20 (95% CI, 1.14-1.27) for type 2 diabetes, 1.15 (95% CI, 1.06-1.23) for fatal or nonfatal cardiovascular disease, and 1.13 (95% CI, 1.07-1.18) for all-cause mortality. While the associations between time spent viewing TV and risk of type 2 diabetes and cardiovascular disease were linear, the risk of all-cause mortality appeared to increase with TV viewing duration of greater than 3 hours per day. The estimated absolute risk differences per every 2 hours of TV viewing per day were 176 cases of type 2 diabetes per 100 000 individuals per year, 38 cases of fatal cardiovascular disease per 100 000 individuals per year, and 104 deaths for all-cause mortality per 100 000 individuals per year.

Conclusion Prolonged TV viewing was associated with increased risk of type 2 diabetes, cardiovascular disease, and all-cause mortality.

JAMA. 2011;305(23):2448-2455 www.jama.com

Author Affiliations: Institute of Sport Science and Clinical Biomechanics, Department of Exercise Epidemiology, Center of Research in Childhood Health, University of Southern Denmark, Odense (Mr Grøntved); and Departments of Nutrition (Mr Grøntved and Dr Hu) and Epidemiology (Dr Hu), Harvard School of Public Health, Channing Laboratory, Harvard Medical School and Brigham and Women's Hospital (Dr Hu), Boston, Massachusetts.

Corresponding Author: Frank B. Hu, MD, PhD, Harvard School of Public Health, 655 Huntington Ave, Boston, MA 02115 (frank.hu@channing.harvard.edu).

Clinical Review Section Editor: Mary McGrae McDermott, MD, Contributing Editor. We encourage authors to submit papers for consideration as a Clinical Review. Please contact Mary McGrae McDermott, MD, at mdm608@northwestern.edu.

CME available online at www.jamaarchivescme.com and questions on p 2476.

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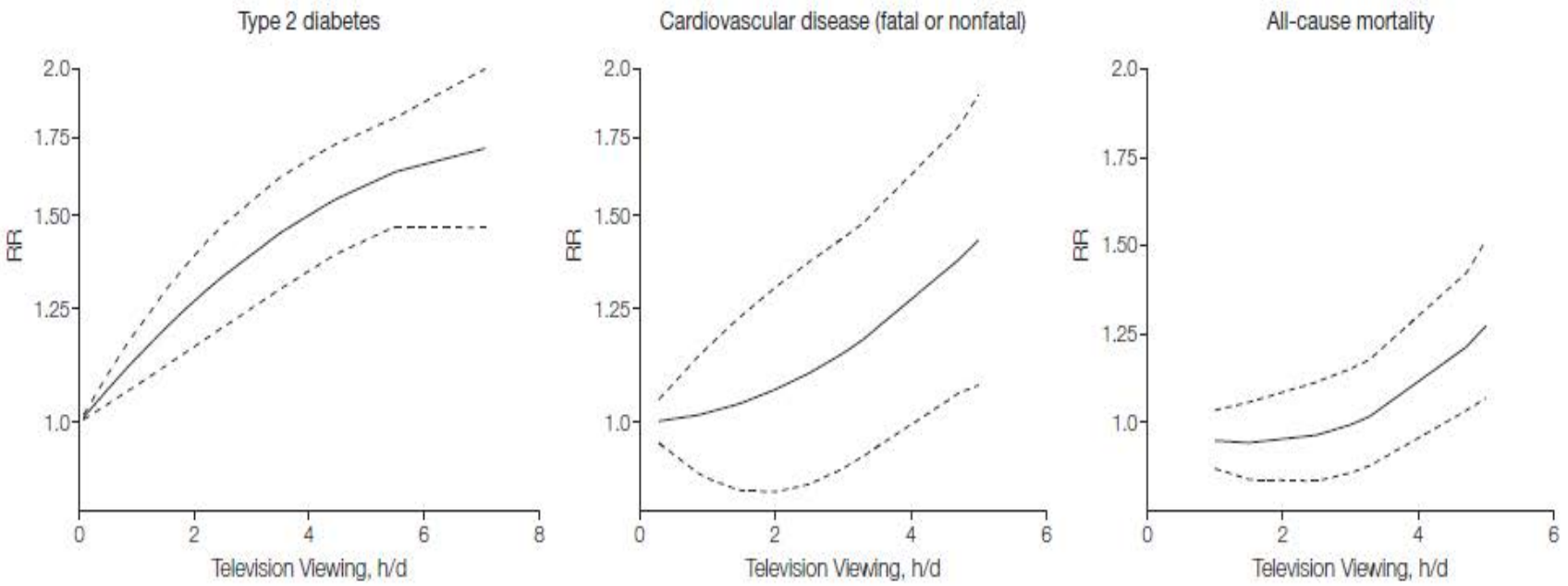
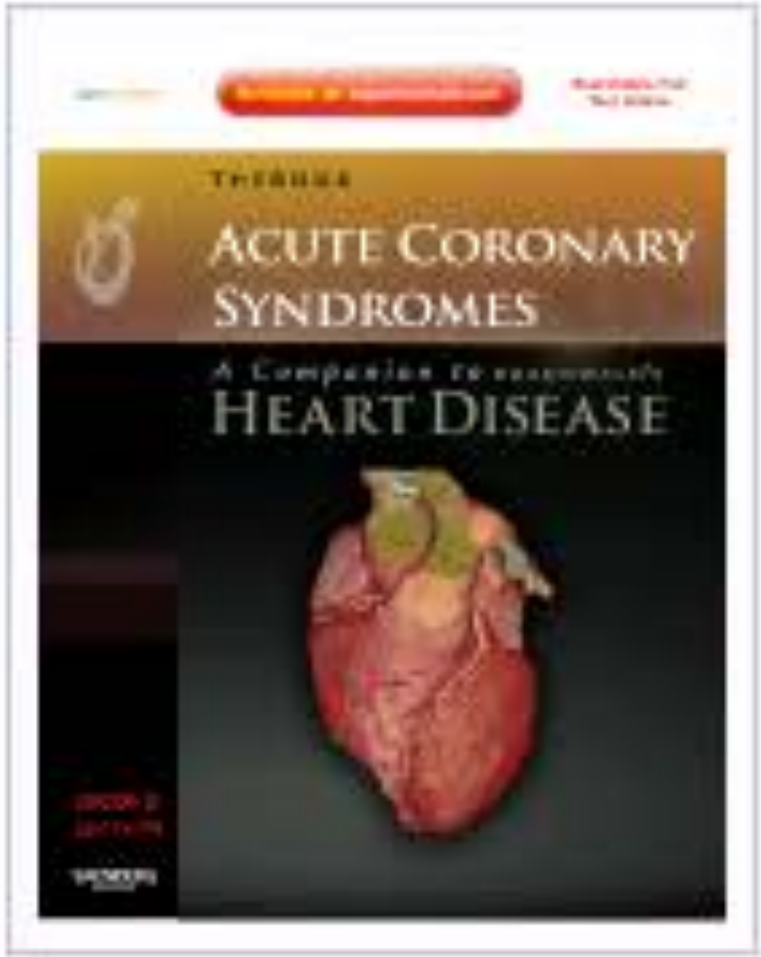
Figure 3. Dose-Response Relationship Between Television Viewing and Risk of Type 2 Diabetes, Cardiovascular Disease, All-Cause Mortality



Dotted lines represent the 95% confidence intervals for the fitted trend. The dose-response relationship plot between television (TV) viewing (hours per day) and risk of type 2 diabetes (4 studies), cardiovascular disease (4 studies), and all-cause mortality (3 studies) was estimated with random-effects meta-regression,[10] which allowed for a nonlinear response by including a quadratic term of TV viewing time. The test for a nonlinear relationship was only significant for all-cause mortality ($P = .007$). In subsequent piecewise regression, the best model fit was obtained at an inflection point of 3 hours of TV viewing per day ($P = .01$ for difference in slopes).


ACUTE CORONARY SYNDROMES
HEART DISEASE

CHAPTER 32

Exercise Training After an Acute Coronary Syndrome

Juneau Nigan

CLINICAL BENEFITS

Numerous publications have documented the efficacy of exercise-based cardiac rehabilitation (CR) after myocardial infarction (MI) or coronary artery bypass surgery. Because most studies published were underpowered to detect significant mortality reductions, many meta-analyses were performed to address this issue. Three recent meta-analyses[1-3] have confirmed earlier reviews published in the late eighties.[4,5]

In their publication, Clark and colleagues[1] reviewed 63 randomized trials including a total of 21,295 patients. For the

COST-EFFECTIVENESS OF EXERCISE-BASED CARDIAC REHABILITATION

A review of 15 studies on the economic impact of exercise-based CR was published by Papadakis and colleagues in 2005.[6] The authors concluded that the range of cost per life-year gained was between $2193 and $28,193 and from $668 to $16,118 per quality-adjusted life-year gained.

Ades and colleagues[7] studied the cost effectiveness of CR after myocardial infarction. Their results show a cost of $2130 per life-year saved in the late 1980s and $4950 per life-year saved in 1995. They concluded

TABLE 32–1	Exercise Prescription for Subjects with Coronary Heart Disease

Aerobic Training

Intensity:*

- Heart rate: 65%-85% of maximal HR** or 40%-60% of the HR reserve (HR reserve = (maximal HR – resting HR) + resting HR)
- Gas exchange measurements: 40%-60% of maximal VO_2.
- Perceived exertion: Borg scale 12-14.

Frequency: 3-5 sessions/week.

Duration: 20-45 minutes/session.

Resistance Training

Intensity: 30%-40% of 1-RM for upper body exercises. 40%-60% of 1-RM for lower body exercises.

Repetitions: 10-15 per set.

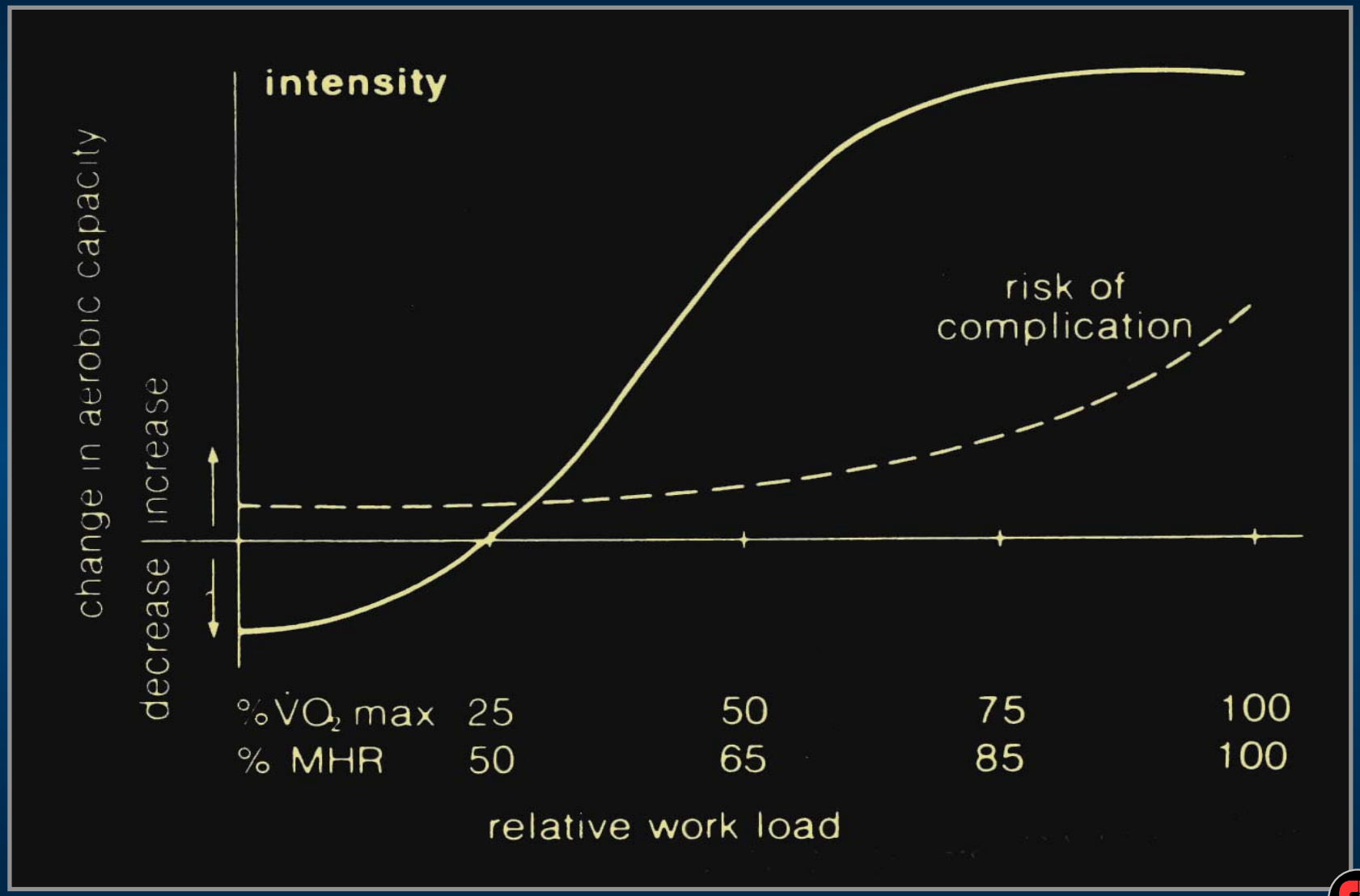
Number of sets: 8-10 sets of different exercises.

Frequency: 2-3 sessions/week.

*As measured during a symptom-limited exercise test.

**See text if exercise-induced ischemia is present during the exercise test.

HR, heart rate; 1-RM, maximum weight that can be lifted to complete one repetition.

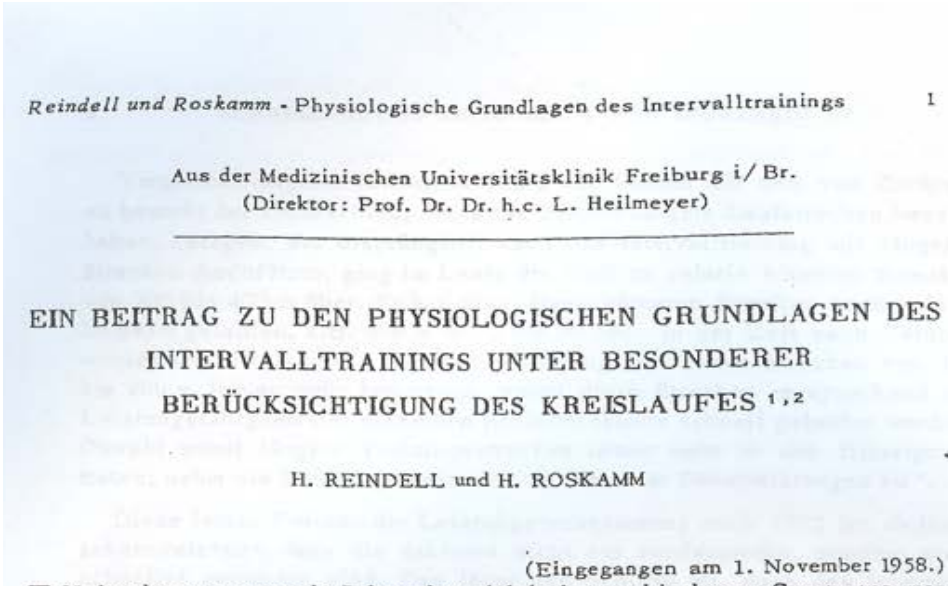

intensity
change in aerobic capacity
increase
decrease
risk of
complication
%VO2 max 25 50 75 100
% MHR 50 65 85 100
relative work load

ECHELLE DE BORG

Perception de la fatigue

	6
TRES TRES FACILE	7
	8
TRES FACILE	9
	10
ASSEZ FACILE	11
	12
UN PEU DIFFICILE	13
	14
DIFFICILE	15
	16
TRES DIFFICILE	17
	18
TRES TRES DIFFICILE	19
	20

EXERCICE INTERMITTENT : HISTORIQUE



Reindell und Roskamm - Physiologische Grundlagen des Intervalltrainings 1

Aus der Medizinischen Universitätsklinik Freiburg i/ Br.
(Direktor: Prof. Dr. Dr. h.c. L. Heilmeyer)

EIN BEITRAG ZU DEN PHYSIOLOGISCHEN GRUNDLAGEN DES INTERVALLTRAININGS UNTER BESONDERER BERÜCKSICHTIGUNG DES KREISLAUFES [1,2]

H. REINDELL und H. ROSKAMM

(Eingegangen am 1. November 1958.)

Reindell (1950's)

Emile Zatopek (1952)

Astrand et coll.

Intermittent muscular work.

Acta Physiol Scand 1960 ; 48 : 448-453

Circulation
JOURNAL OF THE AMERICAN HEART ASSOCIATION

American Heart Association.
Learn and Live.

Core Components of Cardiac Rehabilitation/Secondary Prevention Programs: 2007 Update: A Scientific Statement From the American Heart Association Exercise, Cardiac Rehabilitation, and Prevention Committee, the Council on Clinical Cardiology; the Councils on Cardiovascular Nursing, Epidemiology and Prevention, and Nutrition, Physical Activity, and Metabolism; and the American Association of Cardiovascular and Pulmonary Rehabilitation

Gary J. Balady, Mark A. Williams, Philip A. Ades, Vera Bittner, Patricia Comoss, JoAnne M. Foody, Barry Franklin, Bonnie Sanderson and Douglas Southard

Circulation 2007;115;2675-2682; originally published online May 18, 2007;

- Develop an individualized exercise prescription for aerobic and resistance training that is based on evaluation findings, risk stratification, comorbidities (eg, peripheral arterial disease and musculoskeletal conditions), and patient and program goals. The exercise regimen should be reviewed by the program medical director or referring physician, modified if necessary, and approved. Exercise prescription should specify frequency (F), intensity (I), duration (D), modalities (M), and progression (P).
  - For aerobic exercise: F=3-5 days/wk; I=50-80% of exercise capacity; D=20-60 minutes; and M=walking, treadmill, cycling, rowing, stair climbing, arm/leg ergometry, and others using continuous or interval training as appropriate.
  - For resistance exercise: F=2-3 days/wk; I=10-15 repetitions per set to moderate fatigue; D=1-3 sets of 8-10 different upper and lower body exercises; and M=calisthenics, elastic bands, cuff/hand weights, dumbbells, free weights, wall pulleys, or weight machines.
- Include warm-up, cool-down, and flexibility exercises in each exercise session.
- Provide progressive updates to the exercise prescription and modify further if clinical status changes.
- Supplement the formal exercise regimen with activity guidelines as outlined in the Physical Activity Counseling section of this table.

Accepted Manuscript

Provocative Issues in Heart Disease Prevention

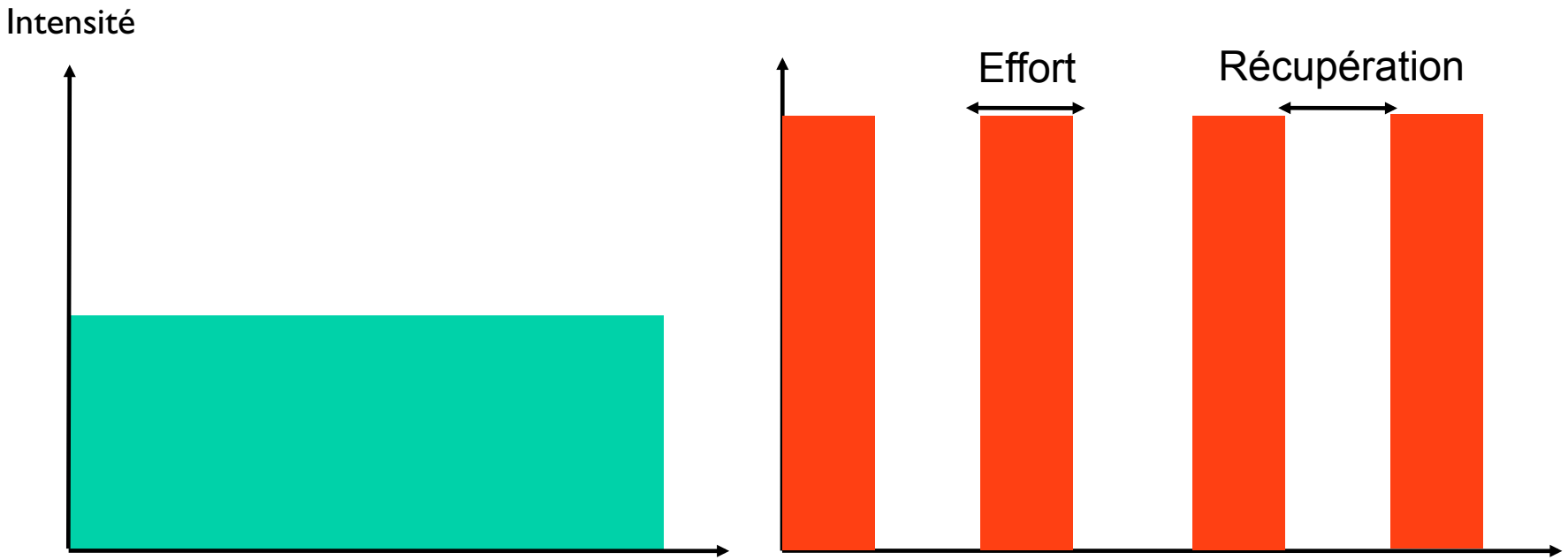
Martin Juneau, M.D Douglas Hayami, M.D Mathieu Gayda, Ph.D Sebastien Lacroix, PhD Anil Nigam, M.D

PII: S0828-282X(14)01398-1

DOI: 10.1016/j.cjca.2014.09.014

Reference: CJCA 1404

To appear in: *Canadian Journal of Cardiology*



Continu

Intermittent

Caractéristiques d'un exercice intermittent

Intensité de l'exercice (%VAM, %VO_2max, %record)

Durée de l'exercice (minutes, secondes)

Durée de la récupération (minutes, secondes)

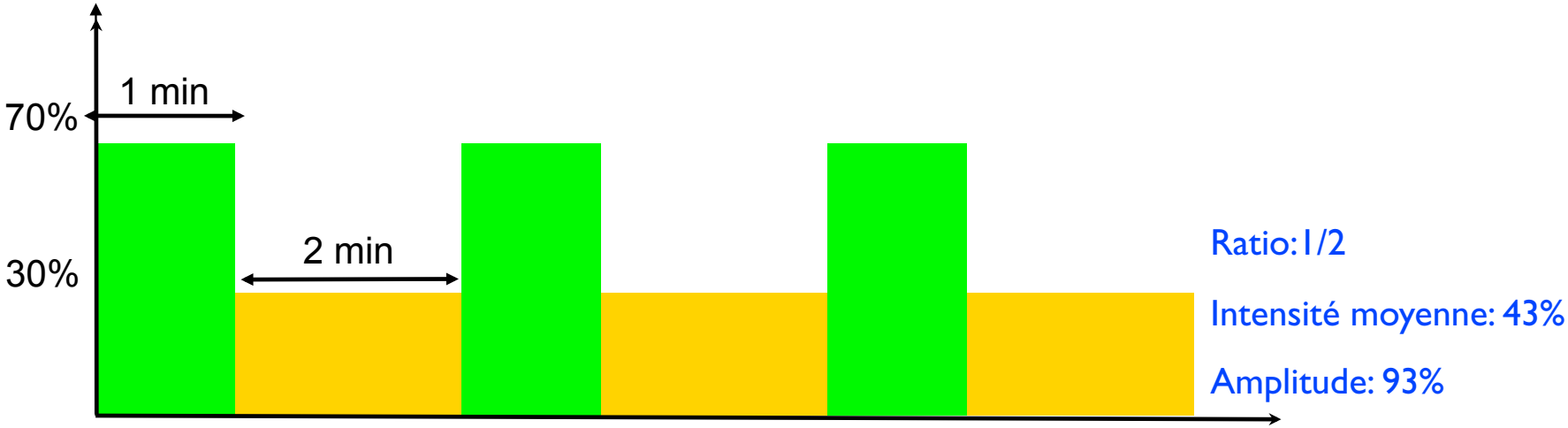
Type de la récupération (active, passive)

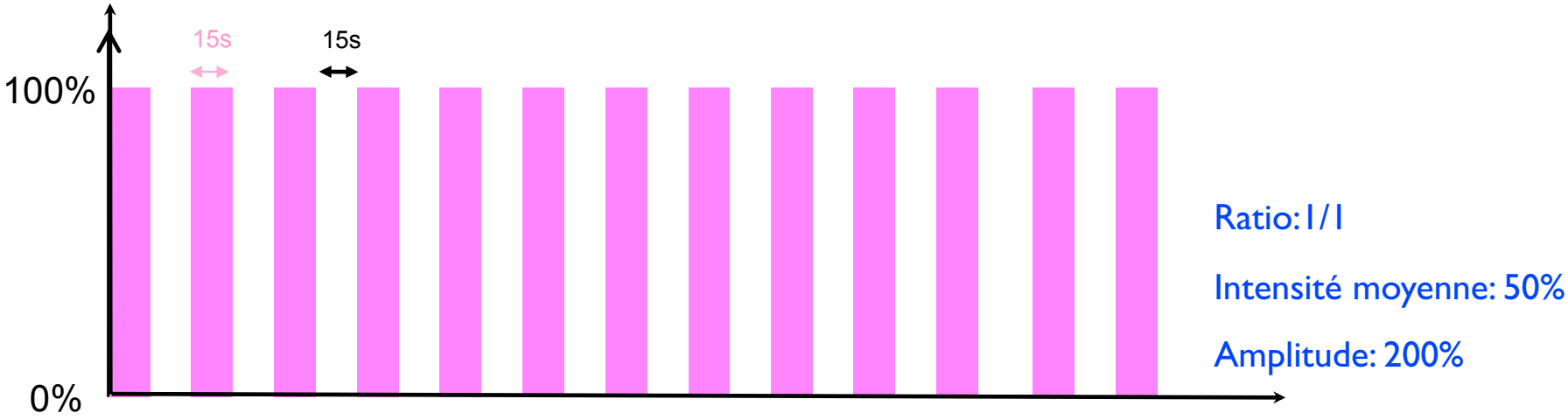
Nombre de répétitions de l'exercice (n)

La manipulation des paramètres d'une séance d'entraînement intermittent a un effet majeur sur la performance et/ou sur la réponse physiologique

Intérêts de l'exercice intermittent à haute intensité

- - Adaptations centrales et périphériques
- - S'habituer à faire des efforts fractionnés
- - Meilleure récupération entre les efforts.
- - Maintenir un haut niveau de VO_2 pendant une longue durée.
- - Travailler à des vitesses que l'on ne peut pas maintenir en continu





Saltin et al. 1960

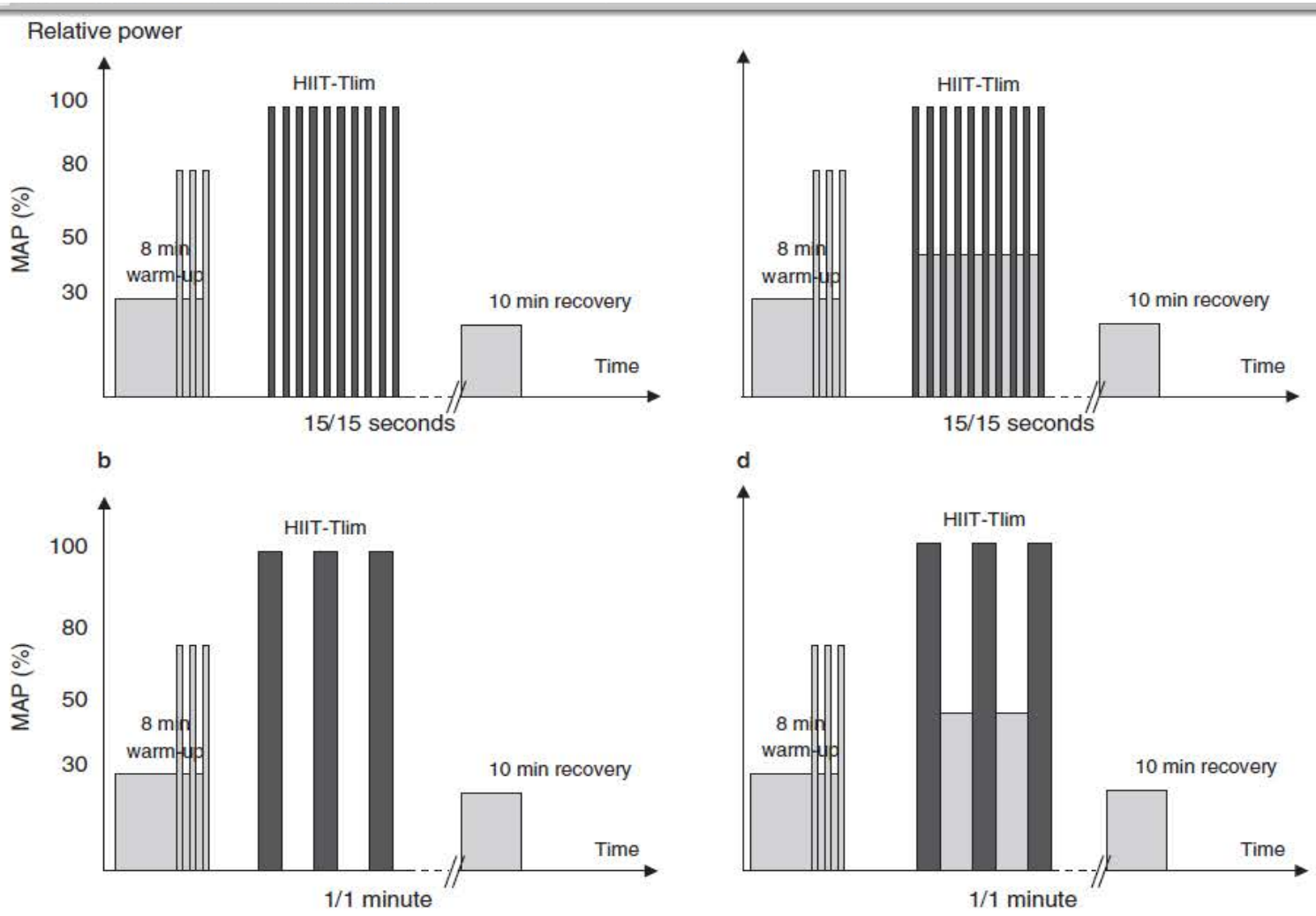


Fig. 1. Different training protocols. Mode (**a**) and (**b**) ratio 1 : 1, mean intensity 50%, amplitude 200%; mode (**c**) and (**d**) ratio 1 : 1, mean intensity 75%, amplitude 66%. **HIIT** = high-intensity interval training; **MAP** = maximal aerobic power; **Tlim** = time to exhaustion.

2. General Principles of Interval Training Prescription

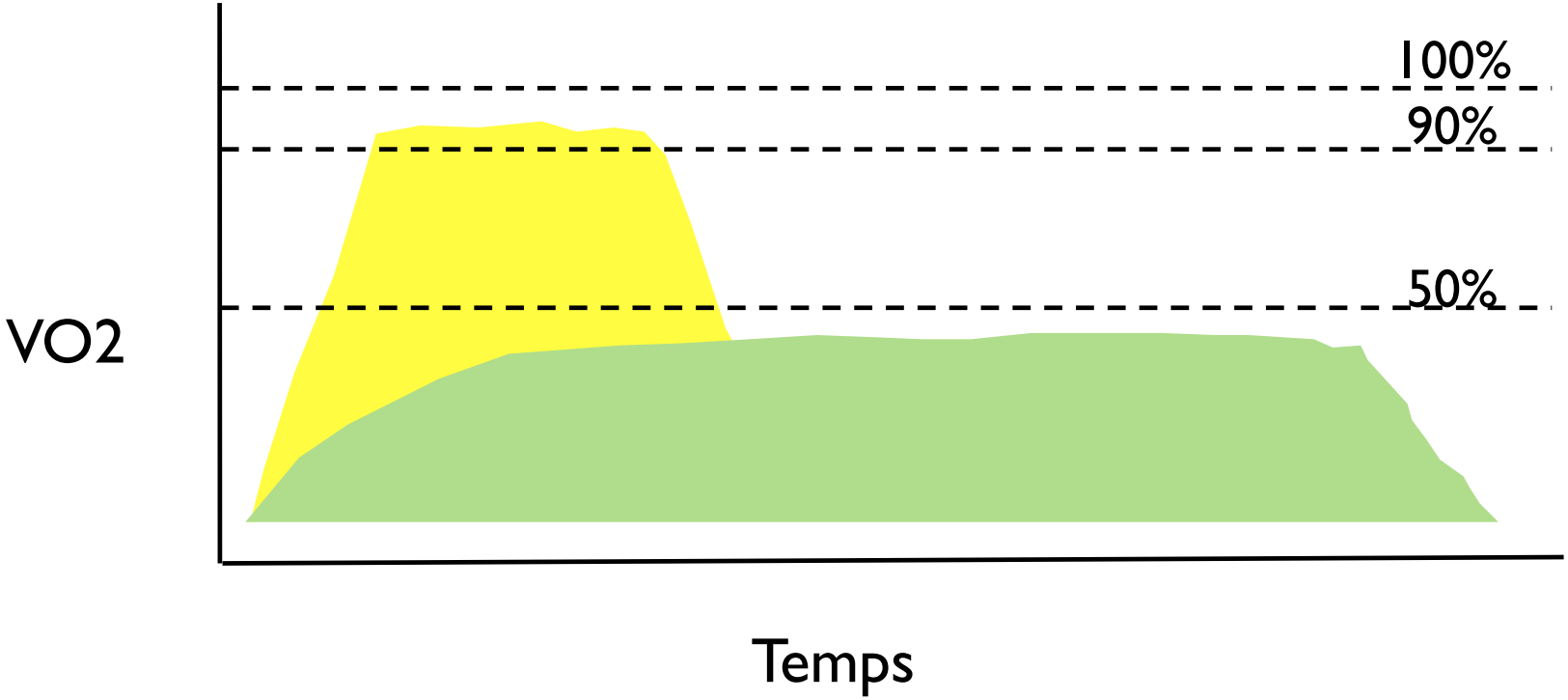
In healthy trained subjects, the improvement in $\dot{V}O_{2peak}$ with exercise training appears to correlate with the time spent at a high level of oxygen uptake ($\dot{V}O_2$).[40] It is thus usual to measure this parameter to determine the acute physiological requirements of different interval-training protocols.[37,40-44] In training for sports, three categories of interval training inducing different physiological responses are usually described: long intervals (3–15 minutes, intensity 85–90% maximal oxygen uptake [$\dot{V}O_{2max}$]), moderate intervals (1–3 minutes,

Table I. Examples of calculations using Saltin's parameters for two different interval-training protocols (A, B)

Saltin's parameters	Mode A	Mode B
Duration of exercise phase	15 sec	1 min
Exercise intensity (PPO)	120% of PPO	100% of PPO
Duration of recovery phase	15 sec	30 sec
Type and intensity of recovery	Passive (0%)	Active (50% of PPO)
Ratio	1/1	2/1
Mean intensity	60%	83%
Amplitude	200%	60%

PPO = peak power output.

Pour ↑ VO_2max, il faut stimuler VO_2max, donc s'entraîner à des intensités proches du VO2 max



Étude de la réponse aiguë à l'exercice intermittent à haute intensité chez le patient coronarien

Thèse de Doctorat effectuée en cotutelle

Institut de Cardiologie de Montréal /
Département de Kinésiologie
Faculté des études supérieures
Université de Montréal

et

Inserm U858
École doctorale Biologie Santé et Biotechnologies
Université de Toulouse

par

Thibaut Guiraud

Thèse présentée à la Faculté des études supérieures de l'Université de Montréal en vue de l'obtention du grade de de Philosophia Doctor (Ph.D) en sciences de l'activité physique

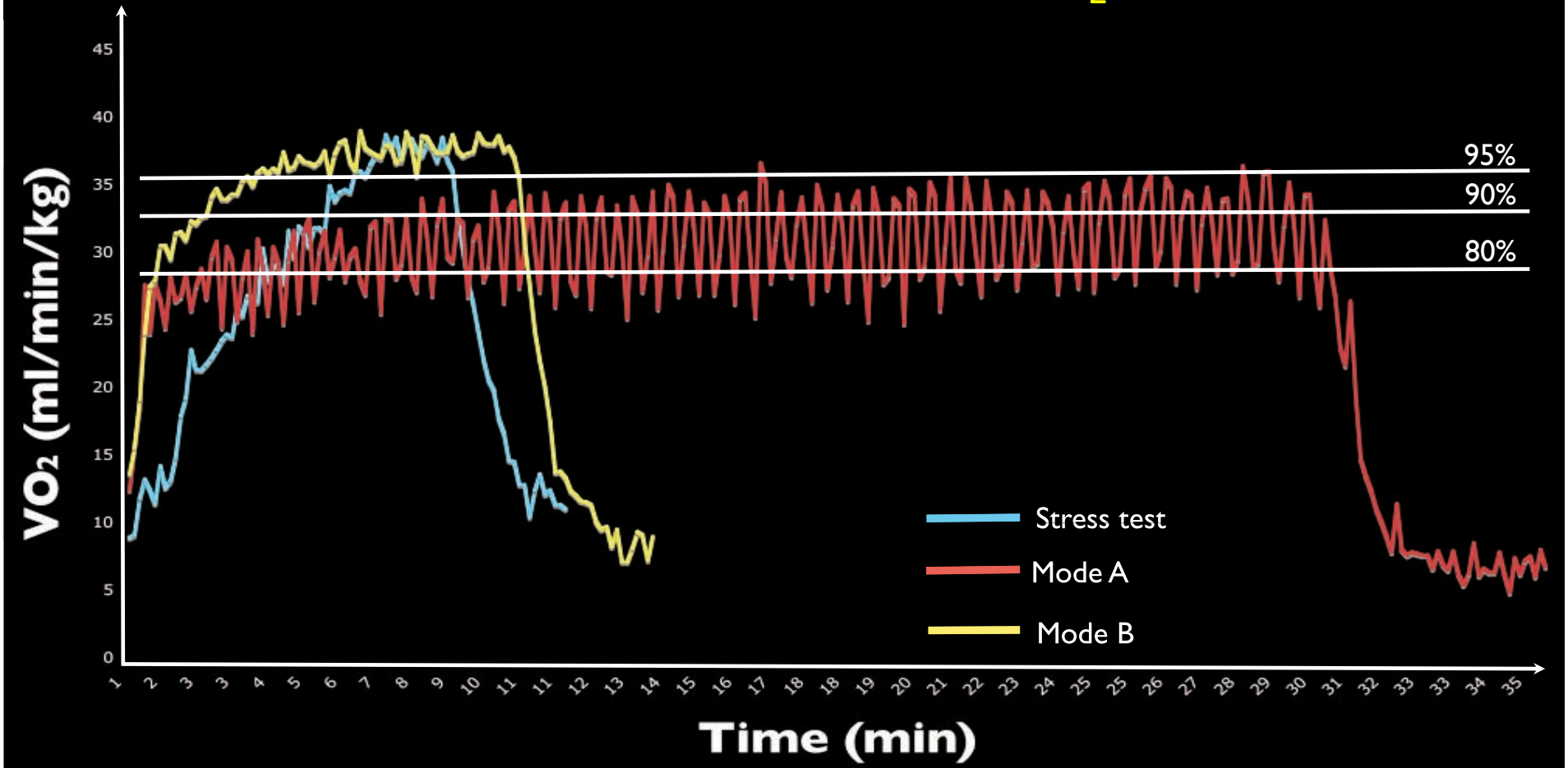
et à

l'école doctorale Biologie Santé Biotechnologies de l'Université de Toulouse en vue de l'obtention du grade de Docteur en sciences.

INSTITUT DE CARDIOLOGIE DE MONTRÉAL

Université de Montréal

Methods: time spent near VO_2max



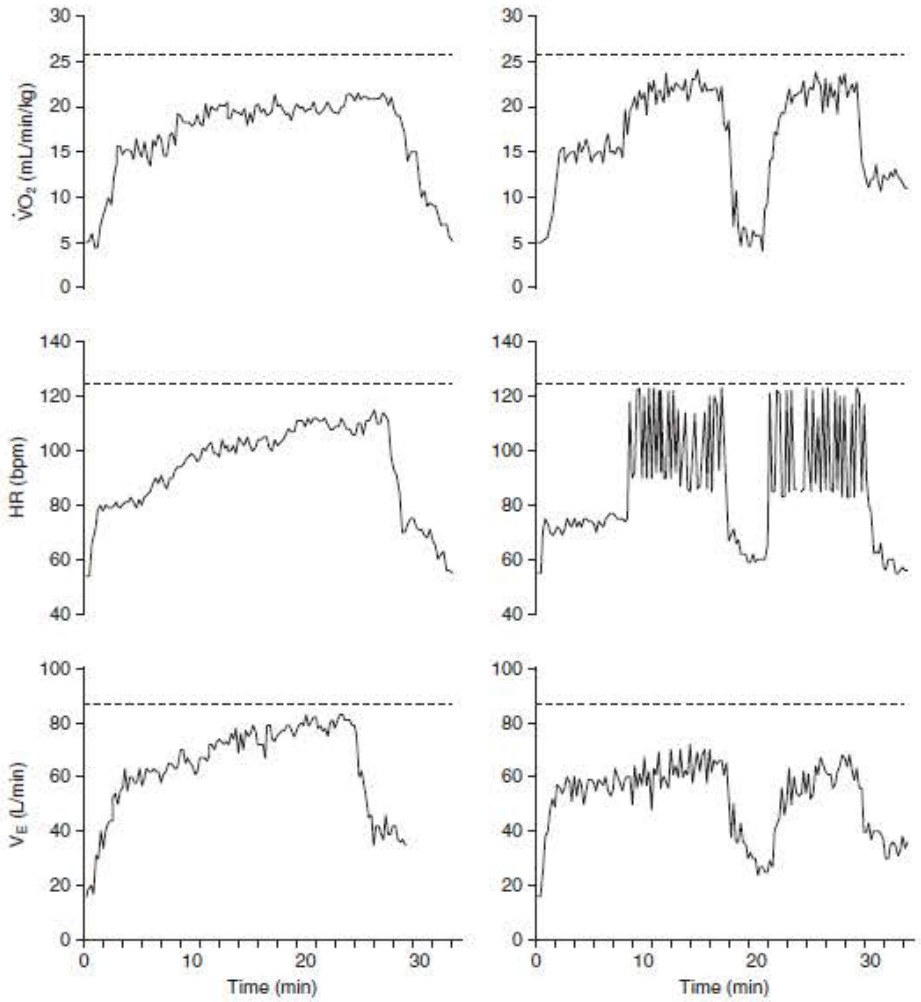
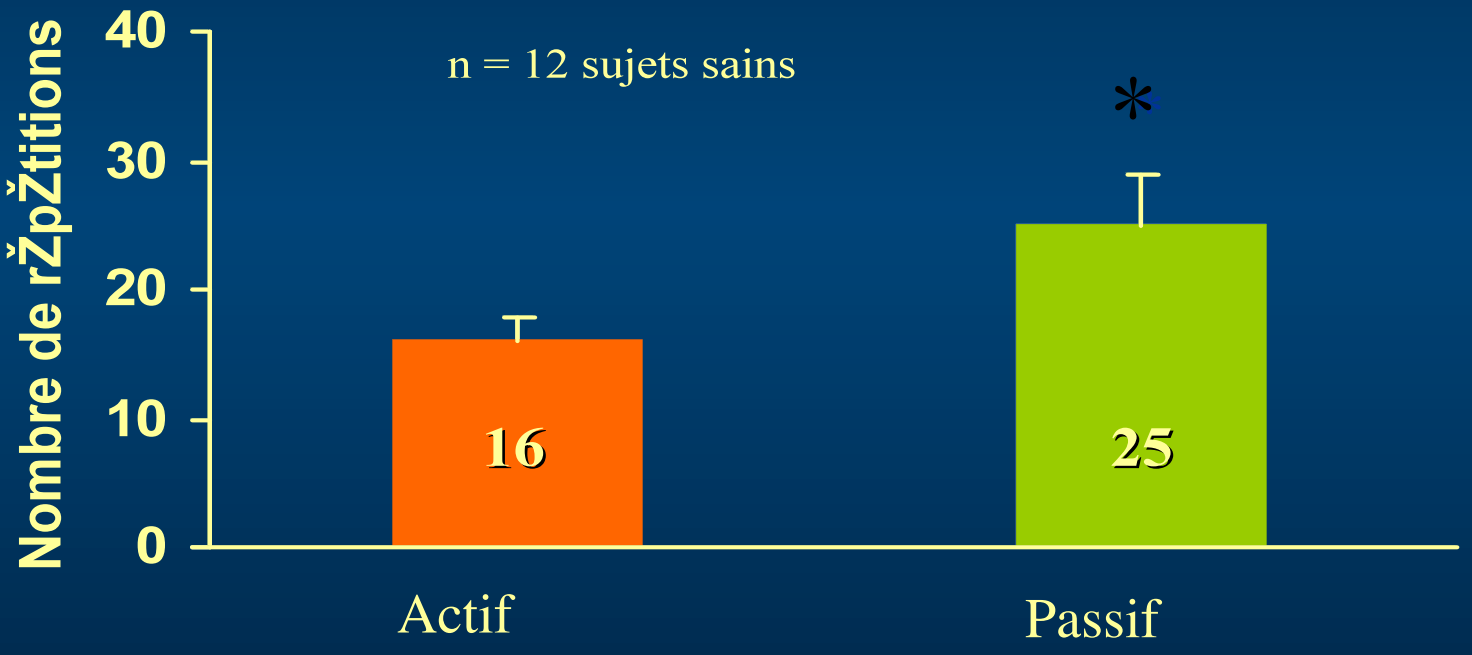


Fig. 2. Oxygen consumption, heart rate and ventilation in a patient during continuous exercise (left panel) and interval training (right panel). The continuous exercise consisted of maintaining an intensity of 70% of MAP for 28.7 minutes, and high-intensity interval training after a 10-minute warm-up at 50% of MAP consisted of two series of 10 minutes each comprising 15-second periods of exercise at 100% of MAP interspersed with 15-second phases of passive recovery. The two series were separated by 4 minutes of rest and ended with a 5-minute recovery period (ratio 1 : 1, mean intensity 50%, amplitude 200%). The dotted line shows the maximal values for this coronary artery disease patient for the three criteria. **bpm** = beats per minute; **HR** = heart rate; **MAP** = maximal aerobic power; $\mathbf{V_E}$ = ventilation; $\mathbf{\dot{V}O_2}$ = oxygen consumption.

Effet du type de récupération sur la performance intermittente

15 secondes à 120% de la VMA; récupération = 15 secondes à 0 ou à 50%



Dupont et coll.
Emprunté À Laurent Bosquet
Eur J Appl Physiol 2003 ; 89 : 548-554

MJ

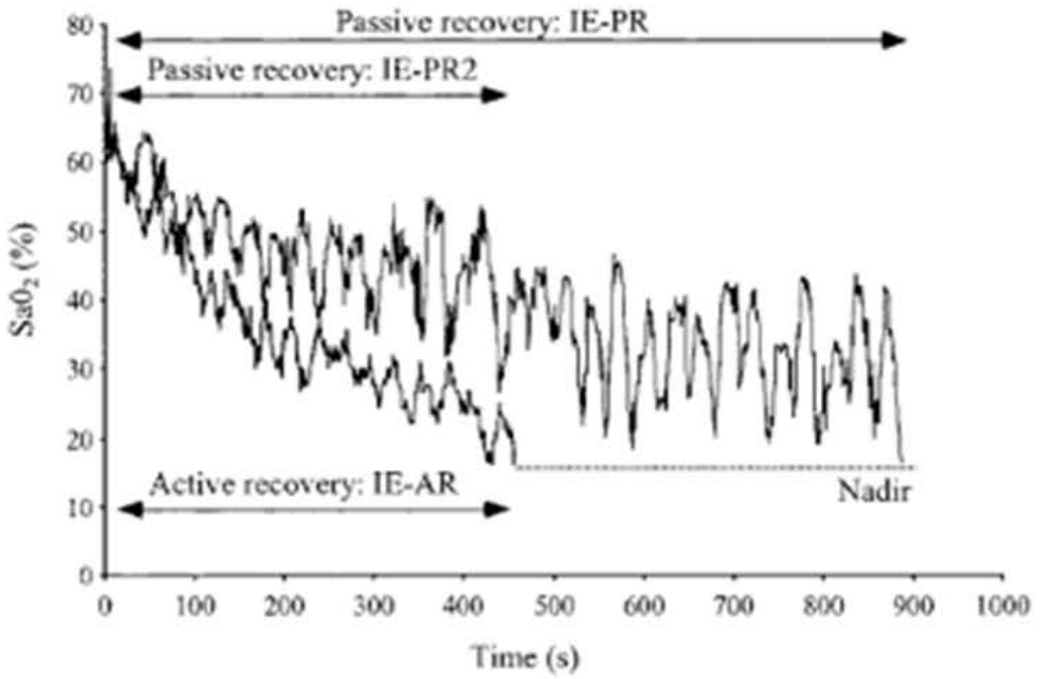
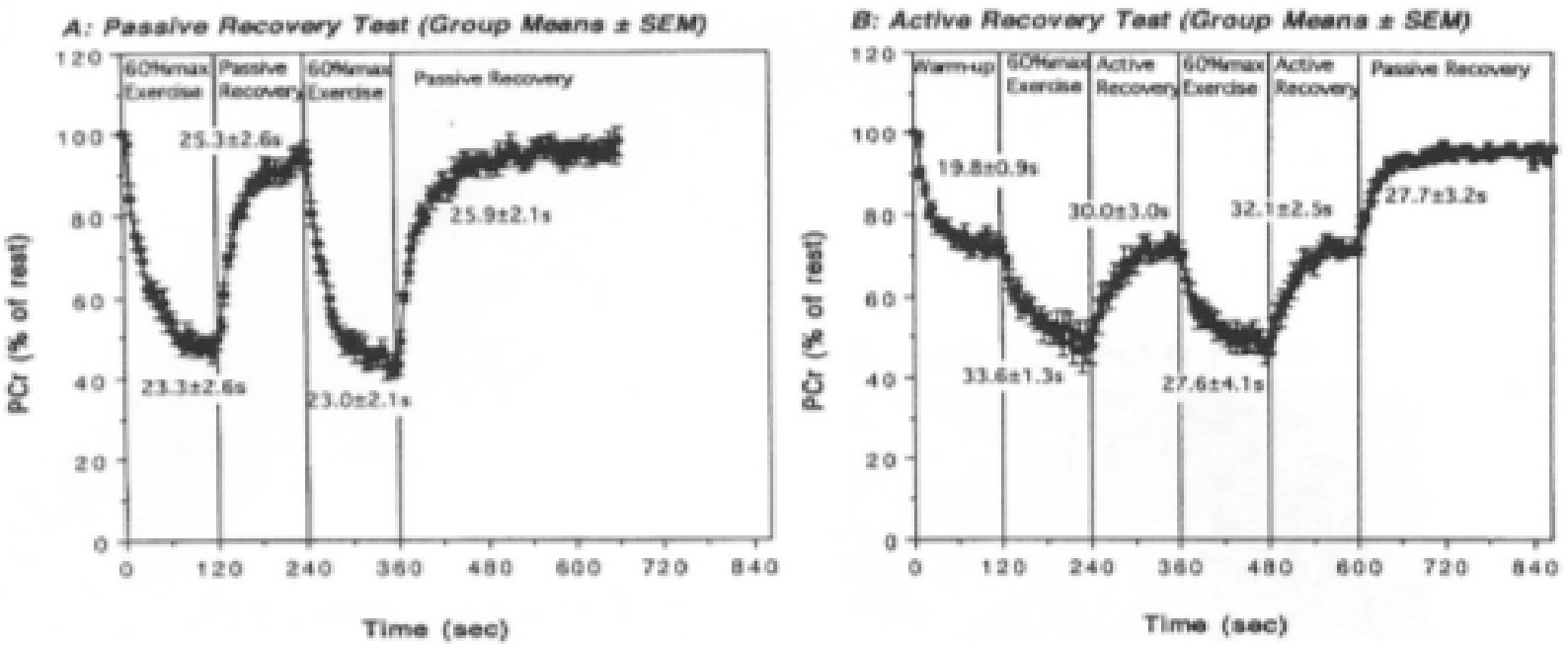


Figure 9. Variations de HbO2 au cours de deux entrainements intermittents type 15/15 (Tlim) avec récupération active (40%) et passive (Dupont et al. 2004)

Variations de la PCr (actif Vs passif)



IN PATIENTS WITH CAD

High Intensity Interval exercise (HIIE)

VS

Moderate Intensity Continuous exercise (MICE)

- **Equivalent energy expenditure**
- **Lower mean ventilation**
- **Lower perceived exertion**
- **More efficient and preferred**

Guiraud T et al. *Med Sci Sports Exer*. 2011.

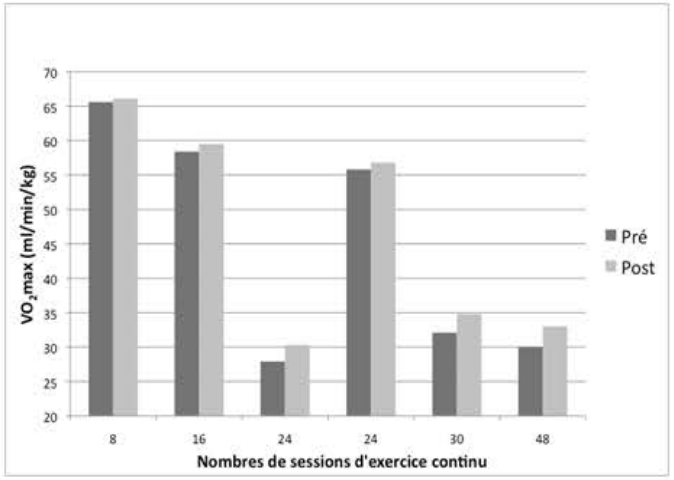
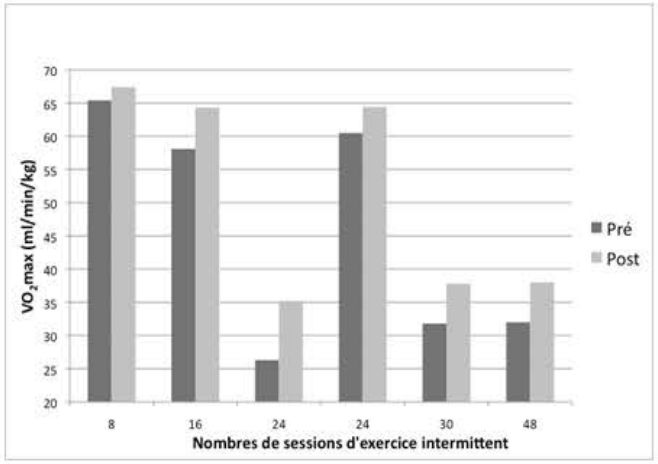


Figure 12. Études comparant les modifications de VO_2max après une période d'entraînement continu (Daussin et al. 2007; Helgerud et al. 2001; Helgerud et al. 2007; Laursen et al. 2005; Rognmo et al. 2004; Warburton et al. 2005)



Obese patients: HIIE and Mediterranean Diet

- Superior effects on body composition (fat mass and WC)
- Blood pressure
- Cholesterol and triglycerides
- Exercise capacity and resting heart rate

Gremeaux et al. *Am J Phys Med Rehabil*. 2012.
Landaeta-Diaz et al. *Eur J Prev Cardiol*. 2012

Eur J Appl Physiol
DOI 10.1007/s00421-009-1287-z

ORIGINAL ARTICLE

Optimization of high intensity interval exercise in coronary heart disease

Thibaut Guiraud · Martin Juneau · Anil Nigam · Mathieu Gayda · Philippe Meyer · Said Mekary · François Paillard · Laurent Bosquet

Accepted: 27 October 2009

Acute Responses to High-Intensity Intermittent Exercise in CHD Patients

THIBAUT GUIRAUD[1,2], ANIL NIGAM[1], MARTIN JUNEAU[1], PHILIPPE MEYER[1], MATHIEU GAYDA[1], and LAURENT BOSQUET[2,3]

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CLINICAL SCIENCES

REVIEW ARTICLE

Sports Med 2012; 42 (7): 587-605
0112-1642/12/0007-0587/$49.95/0

High-Intensity Interval Training in Cardiac Rehabilitation

Thibaut Guiraud,[1,2,3] *Anil Nigam*,[1] *Vincent Gremeaux*,[1,4,5] *Philippe Meyer*,[1,6] *Martin Juneau*[1,7] and *Laurent Bosquet*[7,8]

1 Montreal Heart Institute, Cardiovascular Prevention Centre (centre ÉPIC), Université de Montréal, Montréal, Québec, Canada

Exercise above the ischemic threshold and serum markers of myocardial injury

Martin Juneau MD[1,2,3], Nathalie Roy MD[4], Anil Nigam MD[1,2,3], Jean-Claude Tardif MD[1,2,3], Lucie Larivée RN[2]

M Juneau, N Roy, A Nigam, J-C Tardif, L Larivée. Exercise above the ischemic threshold and serum markers of myocardial injury. Can J Cardiol 2009;25(10):e338-e341.

BACKGROUND: Current guidelines for exercise training in coronary patients state that in the presence of exercise-induced ischemia, the heart rate during exercise should be at least 10 beats/min below the heart rate associated with an ST segment depression of 1 mm or greater. For patients with a relatively low ischemic threshold, this recommendation does not allow for a sufficient training stimulus.

OBJECTIVE: To document the effects of a single session of exercise above the ischemic threshold on biochemical markers of myocardial injury in stable coronary patients with exercise-induced ischemia. Because creatine kinase (CK) and its MB isoenzyme (CK-MB) can both increase after exercise because of skeletal muscle injury, troponin T was also measured.

METHODS: Twenty-one patients with documented coronary artery disease underwent two 20 min exercise sessions. The intensity of the first exercise training session was fixed at a heart rate below the ischemic threshold (ie, approximately 10 beats/min lower than the heart rate associated with the appearance of an ST segment depression of 1 mm or greater). The intensity of the second exercise session was fixed at a heart rate above the ischemic threshold.

L'exercice au-dessus du seuil ischémique et des marqueurs sériques de lésion myocardique

HISTORIQUE : Selon les lignes directrices actuelles relativement à l'entraînement à l'exercice chez les patients atteints d'une maladie cardiovasculaire, en présence d'ischémie induite par l'exercice, le rythme cardiaque pendant l'exercice devrait se situer au moins à 10 battements/min sous celui associé à une dépression du segment ST de 1 mm ou plus. Dans le cas des patients chez qui le seuil ischémique est relativement faible, cette recommandation n'assure pas un stimulus d'entraînement suffisant.

OBJECTIF : Documenter les effets d'une seule séance d'exercice au-dessus du seuil ischémique sur les marqueurs biochimiques de lésion myocardique chez des patients atteints d'une coronaropathie stable souffrant d'ischémie induite par l'exercice. Puisque la créatine kinase (CK) et ses isoenzymes MB (MB-CK) peuvent augmenter après l'exercice en raison d'une lésion du muscle squelettique, on a également mesuré la troponine T.

MÉTHODOLOGIE : Vingt et un patients atteints d'une coronaropathie documentée ont effectué deux séances d'exercice de 20 minutes. L'intensité de la première séance d'entraînement à l'exercice était établie

TABLE 4
Serum markers before and after exercise

	Below ischemic threshold	Above ischemic threshold
Before training		
CK, U/L	145±41	110±27
CK-MB, U/L	6±1	8±1
Troponin T, ng/mL	0.009±0.009	0.014±0.011
6 h post-training		
CK, U/L	151±36	127±32
CK-MB, U/L	9±2	8±1
Troponin T, ng/mL	0.015±0.008	0.014±0.012
24 h post-training		
CK, U/L	132±27	118±32
CK-MB, U/L	9±3	9±1
Troponin T, ng/mL	0.011±0.008	0.011±0.007

Data presented as mean ± SD. Normal ranges: creatine kinase (CK) 24 U/L to 195 U/L; CK-MB isoenzyme 0 U/L to 30 U/L; troponin T <0.1 ng/mL. No significant differences noted for all comparisons

From the Cardiac Rehabilitation and Prevention Center (Centre ÉPIC) (PM, TG, MG, MJ, AN), Montreal Heart Institute, University of Montreal, Montreal, Canada; and Department of Kinesiology (LB), University of Montreal, Montreal, Canada.

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Disclosures:

Funding Support provided by the ÉPIC Foundation and Montreal Heart Institute Foundation.

0894-9115/09/8810-0001/0
American Journal of Physical Medicine & Rehabilitation

High-Intensity Aerobic Interval Training in a Patient with Stable Angina Pectoris

ABSTRACT

Meyer P, Guiraud T, Gayda M, Juneau M, Bosquet L, Nigam A: High-intensity aerobic interval training in a patient with stable angina pectoris. Am J Phys Med Rehabil 2009;88:000–000.

Recently, high-intensity aerobic interval training was shown to be more effective than continuous moderate-intensity exercise for improving maximal aerobic capacity and endurance in patients with coronary heart disease. However, patients with exercise-induced ischemia were not included in those studies. We present the acute cardiopulmonary responses of a 67-yr-old man with stable angina pectoris during a 34-min session of high-intensity aerobic interval training. Exercise was well tolerated with neither significant arrhythmia nor elevation of cardiac

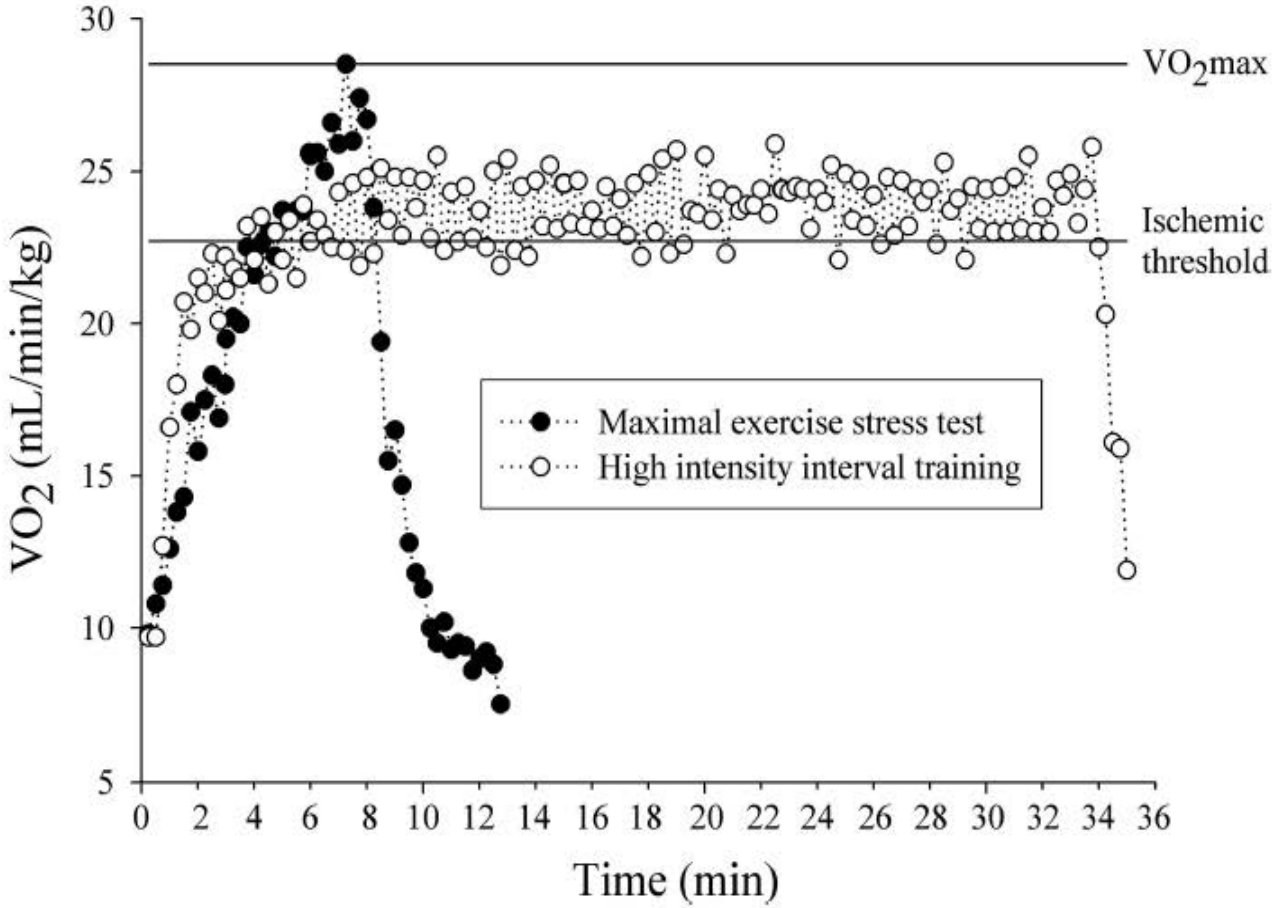

FIGURE 1 *Oxygen uptake (averaged every 15 secs) during maximal exercise stress test and high-intensity interval training.*

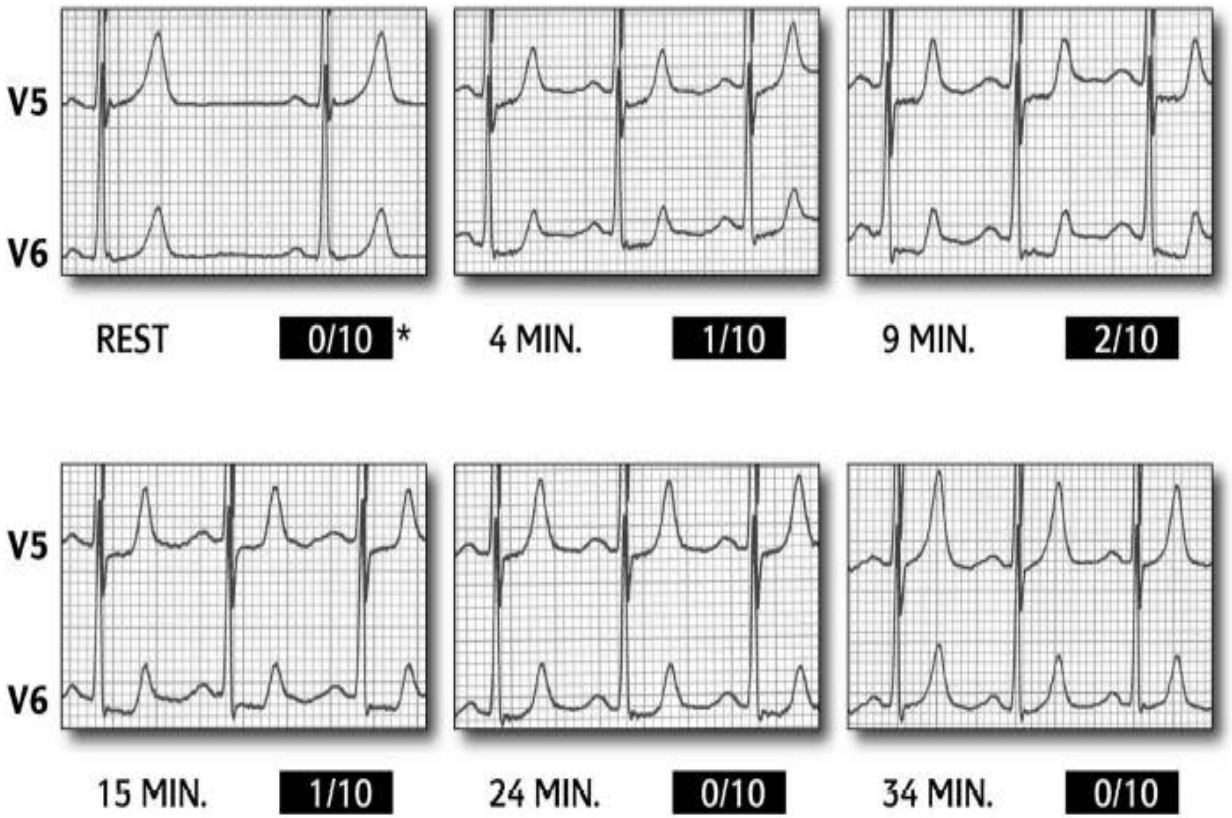

FIGURE 2 *ECGs (leads V5 and V6) and perceived angina during a 34-min session of high-intensity interval training. *Rating scale of perceived angina (0–10).*

Canadian Journal of Cardiology ■ (2013) 1–3

Brief Rapid Report

Comparison of Carbohydrate and Lipid Oxidation During Continuous and Intermittent Exercise in Patients With Chronic Heart Failure

Mathieu Gayda, PhD,[a,b,c] Eve Normandin, MSc,[a,b] Philippe Meyer, MD,[a,b,d] Martin Juneau, MD,[a,b,c] and Anil Nigam, MD[a,b,c]

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High intensity interval training versus moderate continuous training in patients post acute coronary syndrome

Douglas Hayami[1,2,3], M Gayda[2,3], J-F Larouche[2,3], G Lapierre[2,3], C Henri[1,2], E Thorin[1,2], J-C Tardif[1,2], M Juneau[1,2,3], A Nigam[1,2,3]

[1]Institut de Cardiologie de Montréal, [2]Université de Montréal

[3]Centre de médecine préventive et d'activité physique de l'Institut de cardiologie de Montréal (EPIC)

Background

- Acute coronary syndrome (ACS) is a significant risk factor for morbidity and mortality
- Exercise training in patients post ACS is associated with a significant reduction in mortality and future cardiovascular events.
  - Moderate intensity continuous exercise training (MICET) is currently recommended.
  - High intensity interval training (HIIT) has been explored as an alternative to MICET.
- The acute effects of this modality in coronary heart disease (CHD) patients has been reported by our group.
- The chronic effects are poorly described with conflicting results.
- We conducted a randomized clinical trial to compare the chronic effect MICET vs HIIT in patients post ACS

Methods

Randomized unblinded single center clinical trial

Recruitment: Chart review of patients discharged from MHI CCU

screened

Inclusion criteria
- >18 years, S/P ACS (UA, NSTEMI, STEMI), LVEF ≥ 40%
- Complete revascularization
- Stable dose medication
- Able to perform maximal cardiopulmonary exercise test

Exclusion criteria
- Presence of symptoms suggestive of ischemia, HF, arrythmia
- Recent CABG
- Significant valvular disease
- Poorly controlled BP
- Resting ECG abormalities: LBBB, atrial arrythmias, paced rhythm
- Significant systemic disease

46 patients randomized

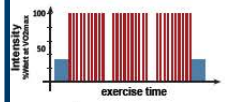
Anthropometric, clinical, biochemical

VO2max

Training: supervised individual or small group

36 sessions, 12 week, 2-3 session/week: isocaloric HIIT or MICET

HIIT n = 26
- 5min warm up/cool down 30%
- 3 x 20 x 15sec, 15sec passive recovery
- 4 min passive recovery between sets



17 HIIT

MICET n = 20
- 5min warm up/cool down 30%
- 1 x 37min

19 MICET

Anthropometric, clinical, biochemical

VO2max

echocardiogram

9 Dropouts
- HIIT
  - 1 unstable angina unrelated to exercise training
  - 1 new atrial fibrillation unrelated to exercise training
  - 1 enrolled in 2 studies
  - 1 MSK
  - 1 protracted pneumonia
  - 3 non-adherent: time conflict, no longer willing to participate
- MICET
  - 1 COPD, unable to achieve minimum exercise capacity

Statistical analysis
- analysis based on 37 individuals who completed 12 week training period
- Sample size calculation: predicted: 25% increase in V02 max in the HIIT group and 8% in the MICET group, 4 ± 5.5 mg/kg/min. 30 patients per group: 80% power, difference using a two-sided alpha = 0.05.
- T-test comparisons between training modality and training effect for normally distributed variables, Mann-Whitney and Wilcoxon test for respective analysis between and within group differences non-normally distributed continuous variables. 2-sided $p < 0.05$ considered statistically significant.
- data: means ± standard deviation

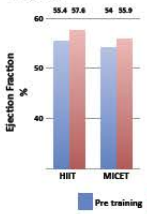
Results

Baseline characteristics	entire cohort	HIIT n = 18	MICET n = 19	p
Age years (range)	61 (35-74)	62 (42-74)	60 (35-74)	ns
Male %	78	72	84	
Anthropometric				
Mass kg	82.6 ± 16	79.2 ± 15.1	85.8 ± 16.6	ns
LBM kg	59 ± 12	55.6 ± 10.5	62 ± 12.9	ns
BMI kg/m2	28.5 (4.7)	28.1 ± 4.8	28.9 ± 4.7	ns
Waist Circumference cm	100.4	97.8 ± 8.8	98.8 ± 10.4	ns
SBP mmHg	127.6 ± 15.5	123.4 ± 16	131.5 ± 14	ns
DBP mmHg	71.8 ± 9.4	69.3 ± 8.7	74 ± 9.7	ns
Resting HR min-1	67.5 ± 10.5	67.6 ± 10.8	67.4 ± 10.4	ns
Risk factors				
Dyslipidemia %	66	47.1	84.2	
Hypertension %	50	47.1	52.6	
Diabetes %	11	5.9	15.8	
Current smoker	22.2	5.9	36.8	
Medications				
Aspirin %	95	88.9	100	
Beta blockers %	83	76.5	89.5	
Statin %	89	94.1	89.5	
ACE-I or ARA %	86	88.2	89	
Time intervals				
Time from ACS to randomization weeks	9.4 (3.4 - 35.4)	8.7 (3.4 - 19)	10.1 (3.4 - 35.4)	ns
Training duration weeks (range)	18.17 (12.7-38.7)	17.9 (12.7-29.3)	18.5 (12.7-38.7)	ns
Time from ACS to first echo	2.7 ± 5.7	3.3 ± 4.4	2.0 ± 6.9	ns
Time from study end to f/u echo	5.2 ± 11.4	4.1 ± 12.2	6.2 ± 11	ns

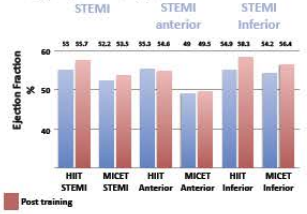
Training effect	HIIT pre	HIIT post	MICET pre	MICET post
Anthropometric				
BMI kg/m2	28.1 ± 4.8	28.3 ± 5.3	28.9 ± 4.7	28.9 ± 4.4
mass kg	79.2 ± 15.1	79.2 ± 16	85.8 ± 16.6	85.7 ± 16.4
lean body mass kg	55.6 ± 10.5	56.1 ±10.6	62 ± 12.9	61.7 ± 12
Waist circumference cm	97.8 ± 8.83	98.8 ± 10.4	102.7 ± 12.6	101.4 ± 12
Cardiopulmonary				
peak power W	115.8 ± 44	132.8 ± 46.7*	132 ± 4 ± 42	153 ± 50*
RER at VO2peak	1.19 ± 0.8	1.17 ± 0.07	1.16 ± 0.1	1.15 ± 0.07
Echocardiographic				
Peak E velocity cm/s	7.7 ± 1.7	8.0 ± 1.6**	7.3 ± 1.6	8.1 ± 1.0
E/A	1.0 ± 0.30	1.16 ± 0.39*	1.08 ± 0.55	1.2 ± 0.35

*p < 0.05
**p=0.056

Ejection fraction by group

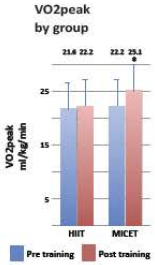


Ejection fraction by group and ACS type

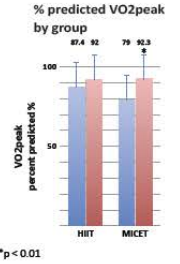


Training effect: VO2peak

VO2peak by group

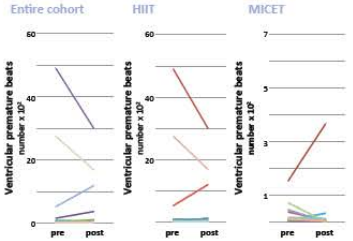


% predicted VO2peak by group

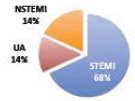


*p < 0.01

Ventricular premature beats pre vs post training

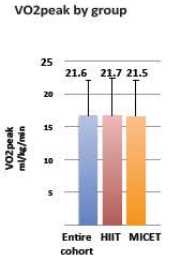


ACS Type

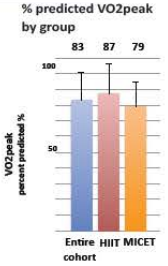


STEMI infarct territory

VO2peak by group



% predicted VO2peak by group



Limitations

- Small number of randomized patients: did not achieve pre-specified sample size
- Adherence to prespecified training duration was mediocre: mean training duration > 12 weeks, wide range
- Baseline fitness level between groups was non-significantly different.

Conclusions

- HIIT in stable, low risk patients post ACS, EF ≥ 40% is safe with no evidence of increased arrythmia or adverse effect on ejection fraction.
- No significant increase in VO2max was observed in the HIIT group, while 13% increase in VO2max was observed in the MICET group.
- Further studies are required to investigate the chronic effectiveness of HIIT and different HIIT protocols in post ACS patients.v

Centre de médecine Préventive et d'activité physique de l'Institut de Cardiologie de Montréal EPIC

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Université de Montréal

Contact: Dr. Anil Nigam (anil.nigam@icm-mhi.org)

MJ

BMJ Case Rep. 2013 Feb 13;2013. pii: bcr2012007668. doi: 10.1136/bcr-2012-007668.

The effect of high-intensity aerobic interval training on postinfarction left ventricular remodelling.

Godfrey R[1], Theologou T, Dellegrottaglie S, Binukrishnan S, Wright J, Whyte G, Ellison G.

Author information

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Abstract

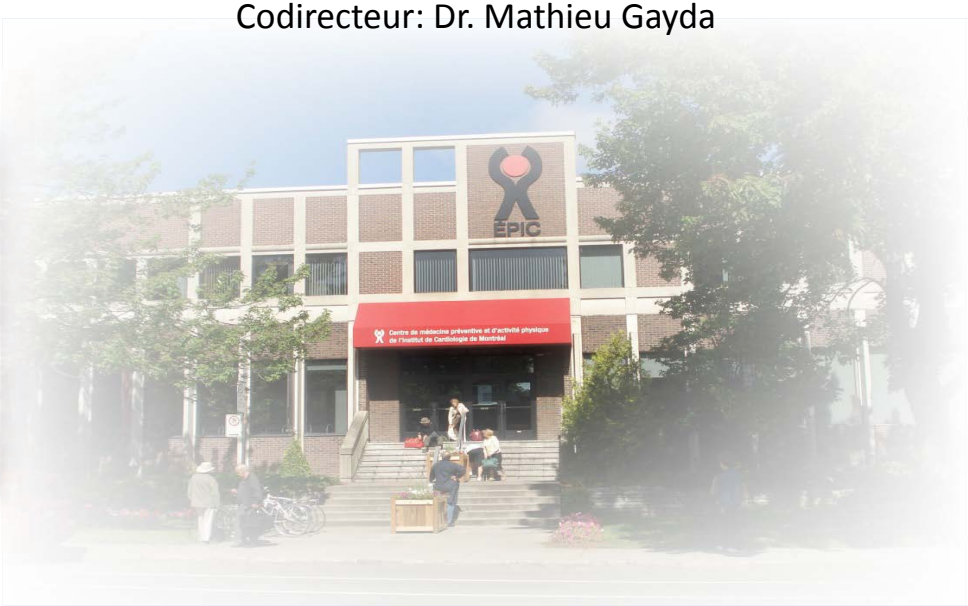
This is the third in a series of case studies on an individual with normal coronaries who sustained an idiopathic acute myocardial infarction . Bilateral pulmonary emboli almost 2 years post-myocardial infarction (MI) revealed coagulopathy as the cause. The original MI resulted in 16% myocardial scar tissue. An increasing number of patients are surviving MI, hence the burden for healthcare often shifts to heart failure. Accumulating evidence suggests high-intensity aerobic interval exercise (AHIT) is efficacious in improving cardiac function in health and disease. However, its impact on MI scar has never been assessed. Accordingly, the 50-year-old subject of this case study undertook 60 weeks of regular AHIT. Successive cardiac MRI results demonstrate, for the first time, a decrease in MI scar with exercise and, alongside mounting evidence of high efficacy and low risk, suggests AHIT may be increasingly important in future prevention and reversing of disease and or amelioration of symptoms.

Comparaison des réponses physiologiques lors d'un exercice incrémental maximal sur vélo immergé et sur terrain sec : Aspects biomécaniques, cardiopulmonaires et hémodynamiques

Mauricio Garzon Camelo

Directeur Département de Kinésiologie :
Professeur Jean-Marc Lavoie.

Directeur de recherche: Dr. Martin Juneau
Codirecteur: Dr. Mathieu Gayda



ICM-01-01-2012-08

October 30th, 2012
12:02 AM ET

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Comments (2 comments)

Easier to sweat while wet, study finds

During spinning class, I often find myself wishing I was in a pool. For one, water would make the sweat dripping down my back less noticeable. Two, it has to be easier to sneak a break when the instructor can't see your legs below the surface.

MJ

Plan de présentation

- ✓ Introduction
- ✓ Objectifs de recherche
- ✓ Méthodologie
- ✓ Résultats
- ✓ Conclusion

Introduction

L'immersion peut influencer les réponses cardiopulmonaires et hémodynamiques au cours de l'exercice par rapport à l'exercice sur terraine sec

Augmentation: Retour veineux, débit cardiaque, volume d'éjection systolique

Diminution: Fréquence cardiaque, $VO_{2,}$ résistance vasculaire périphérique

ICM-01-01-2012-08

Alberton et al. *J Sports Med Phys Fitness 2009;49:142-151*
Chu & Rhodes. *Sports Medicine 2001; 31*(1), 33-46.

Chu & Rhodes *Sports Med 2001;31(1)*
Alberton et al. *J Sports Med Phys Fitness 2009;49:142-151*
Gayda et al. *Am J Phys Med Rehabil 2010;89:722-730*

ÉPIC

INSTITUT DE CARDIOLOGIE DE MONTRÉAL

AFFILIÉ À Université de Montréal

ICM-01-01-2012-08

VO_2 et puissance (Watts) vs. cadence de pédalage (RPM) sur (IE)

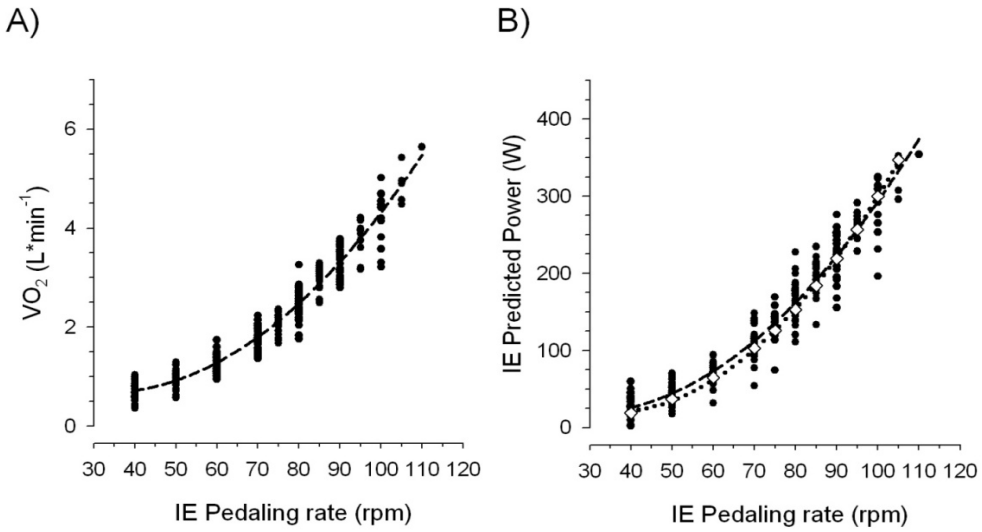


Fig. 3

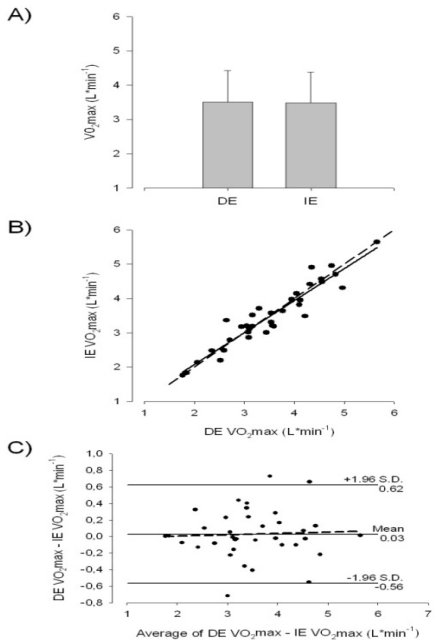
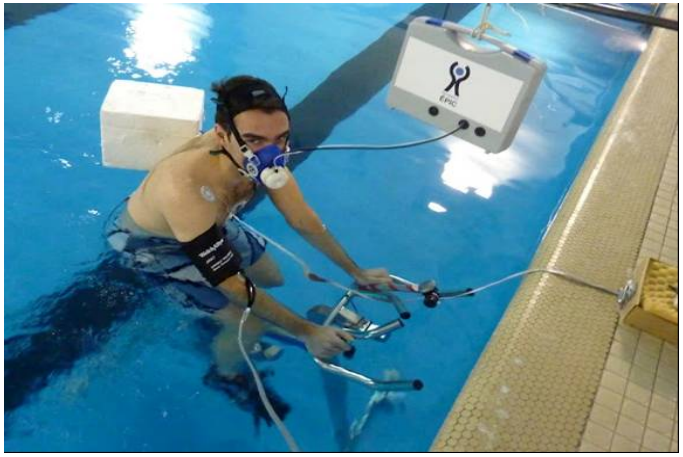


Fig. 4

ICM-01-01-2012-08

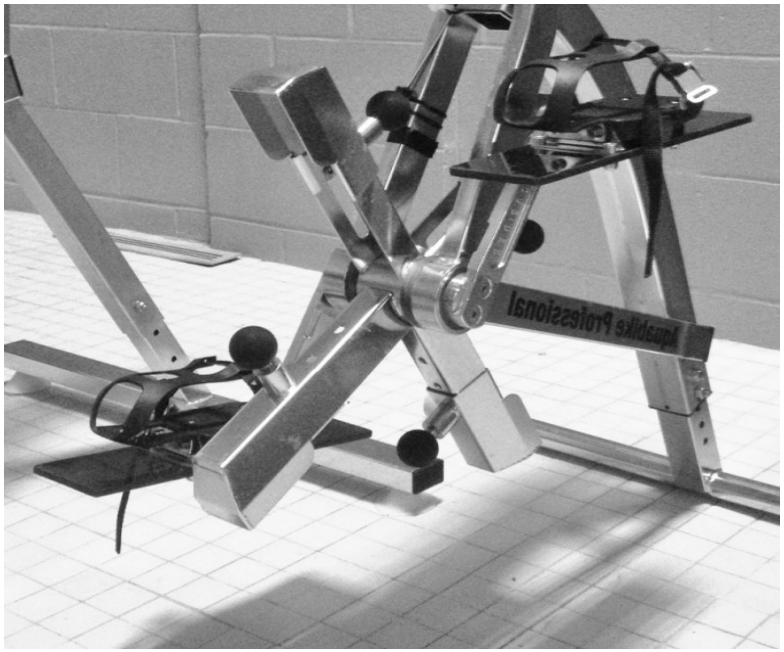
2ème étude

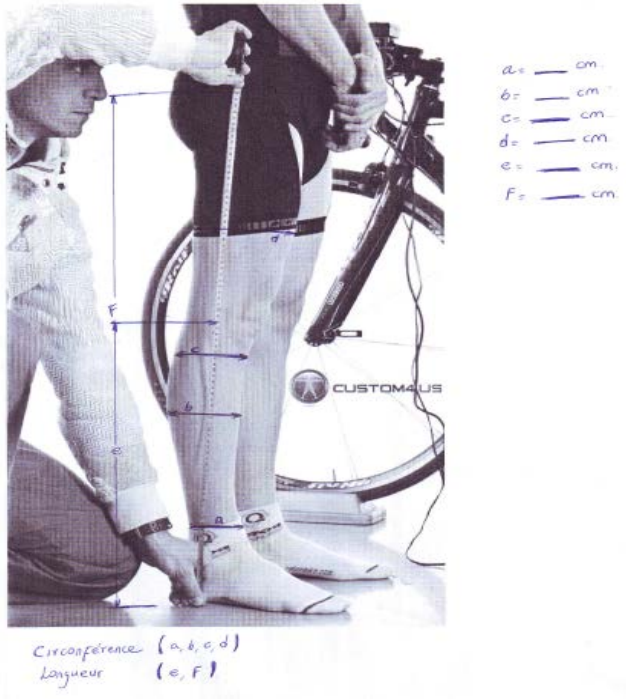
Comparer les réponses cardiopulmonaires et hémodynamiques lors d'un exercice incrémental réalisé sur ergocycle sur terrain sec (VS) vs ergocycle aquatique (VA) en immersion au niveau de la poitrine à une puissance externe (P_{ext}) équivalente



Calcul biomécanique

La pale, le levier, la pédale et la jambe





Équations obtenues

IE P_{ext} (Watts) = $0.0004(rpm)^{2.993}$
(r^2 = 0.99, SEE = 7.6 W, p<0.0001)

rpm	w
40	25
50	49
60	84
70	133
75	164
80	199
85	238
90	283
95	332
100	387

IE (rpm) = $13.91 \times DE\ P_{ext}(Watts)^{0.329}$
(r^2 = 0.99, SEE = 1.5 W, p < 0.0001).

w	rpm
25	40
50	50
75	58
100	63
125	68
150	72
175	76
200	79
225	83
250	86
275	88
300	91
385	99

ICM-01-01-2012-08

2eme étude

Contents lists available at ScienceDirect

Journal of Science and Medicine in Sport

journal homepage: www.elsevier.com/locate/jsams

Original research

Cardiovascular and hemodynamic responses on dryland vs. immersed cycling

Mauricio Garzon[a,b,d], Martin Juneau[a,b,c], Olivier Dupuy[a,f], Anil Nigam[a,b,c], Laurent Bosquet[f], Alain Comtois[e], Mathieu Gayda[a,b,c,*]

Comparer les réponses cardiopulmonaires et hémodynamiques lors d'un exercice incrémental réalisé sur VS vs VA en immersion au niveau de la poitrine à une puissance externe (P_{ext}) équivalente

3eme étude

Journal of Strength and Conditioning Research

IMMERSIBLE ERGOCYCLE PRESCRIPTION AS A FUNCTION OF RELATIVE EXERCISE INTENSITY

--Manuscript Draft--

Mauricio Garzon, Mathieu Gayda, Anil Nigam, Alain-Steve Comtois, Martin Juneau

Étudier la relation entre les différentes expressions de l'intensité relative de l'exercice (FC (%FCmax, %FCR) et VO_2 (%VO_2max, %VO_2R)) pour obtenir la méthode la plus appropriée afin de prescrire l'exercice sur VA

CCC VANCOUVER OCTOBRE 2014

- **RANDOMIZED CONTROLLED TRIAL OF HIGH INTENSITY INTERVAL TRAINING VS MODERATE INTENSITY CONTINUOUS EXERCISE TRAINING IN PATIENTS POST ACUTE CORONARY Highlighted Poster Dr Douglas Hayami 27 octobre 10h30 à 11h30**

- COMPARAISON OF HEART RATE RECOVERY AND PARASYMPATHETIC REACTIVATION PARAMETERS AFTER A MAXIMAL EXERCICE, A MODERATE-INTENSITY CONTINUOUS EXERCISE AND HIGH-INTENSITY INTERVAL EXERCISE IN YOUNG AND OLD HEALTY SUBJECTS AND STABLE CORONARY PATIENTS. Highlighted Poster Dr Mathieu Gayda 27 octobre de 10h30 à 11h30

- **CEREBRAL OXYGENATION/PERFUSION, CARDIAC HEMODYNAMICS DURING EXERCISE AND COGNITIVE FUNCTIONS IN OBESE PATIENTS. Gabriel Lapierre 27 octobre 10h00 à 16h30 (présent au poster de 15h30 à 16h30)**

- CEREBRAL OXYGENATION/PERFUSION, CARDIOPULMONARY AND HEMODYNAMIC RESPONSES DURING MAXIMAL INCREMENTAL EXERCISE IN HEART TRANSPLANT RECIPIENTS VS. HEALTHY CONTROL SUBJECTS. Audrey Desjardins **27 octobre de 10h00 à 16h30 (présent au poster de 15h30 à 16h30)**

- **VALIDATION OF A MEDITERRANEAN FOOD FREQUENCY QUESTIONNAIRE FOR THE POPULATION OF QUEBEC. Jennifer Cantin 27 octobre de 10h00 à 16h30 (présent au poster de 15h30 à 16h30)**

- HIGH INTENSITY AEROBIC INTERVAL EXERCISE IN HEALTHY YOUNG AND OLDER ADULTS: HEMODYNAMIC AND SKELETAL MUSCLE SUBSTRATE USE. **Jean-François Larouche 27 octobre de 10h00 à 16h30 (présent au poster de 15h30 à 16h30)**

- **STATIN THERAPY DECREASES VO2PEAK WITHOUT AFFECTING EXERCISE ENDURANCE, ENDOTHELIAL FUNCTION OR ARTERIAL STIFFNESS AMONG SUBJECTS WITH PRIMARY UNTREATED HYPERCHOLESTEROLEMIA. Dr Philippe Sosner 27 octobre de 10h00 à 16h30 (présent au poster de 15h30 à 16h30)**

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EuroPRevent 2014

» Douglas Hayami

High intensity interval training and lifestyle intervention in patients with abdominal obesity: effect on submaximal and maximal BP

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High intensity interval training and lifestyle intervention in patients with abdominal obe...

Abstract: P154

High intensity interval training and lifestyle intervention in patients with abdominal obesity: effect on submaximal and maximal BP

Authors:

D Hayami[1], M Gayda[1], G Marquis-Gravel[2], V Guilbeault[3], E Latour[3], M Juneau[1], A Nigam[1], [1]University of Montreal, Montreal Heart Institute, Cardiovascular Prevention Centre (Centre EPIC) - Montreal - Canada, [2]University of Montreal - Montreal - Canada, [3]Montreal Heart Institute, Cardiovascular Prevention Centre (Centre EPIC) - Montreal - Canada,

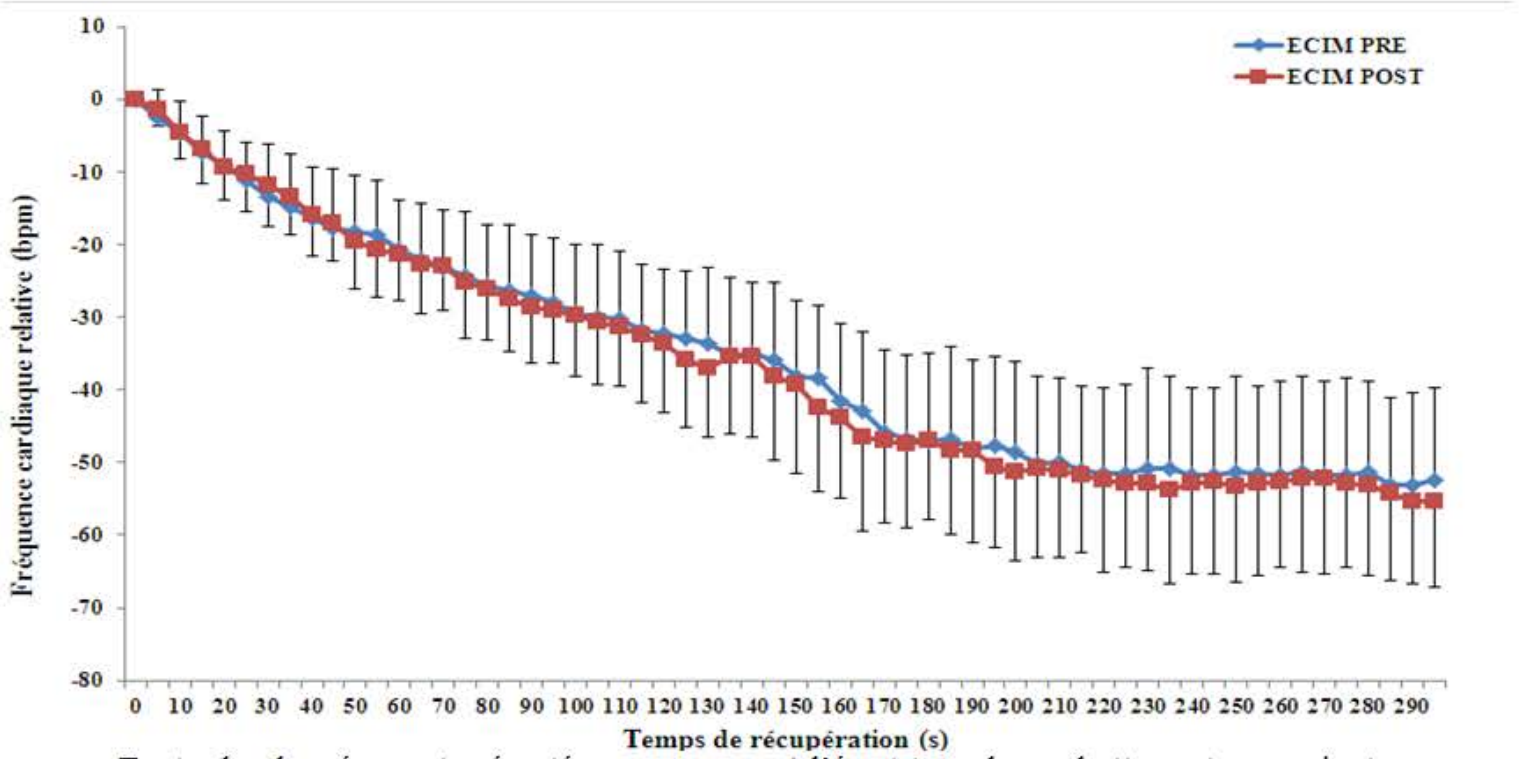
Effet d'un entrainement intermittent de haute intensité vs. un entrainement continu à intensité modérée sur la fréquence cardiaque de récupération, la variabilité de la fréquence cardiaque et les arythmies chez des patients post-syndrome coronarien aigu.

Rénia Amoussou (B.Sc) [1-3], Doug Hayami (M.D) [1-3], Anil Nigam (M.D) [1-3],

Martin Juneau (M.D)[1-3], Mathieu Gayda (Ph.D) [1-3]

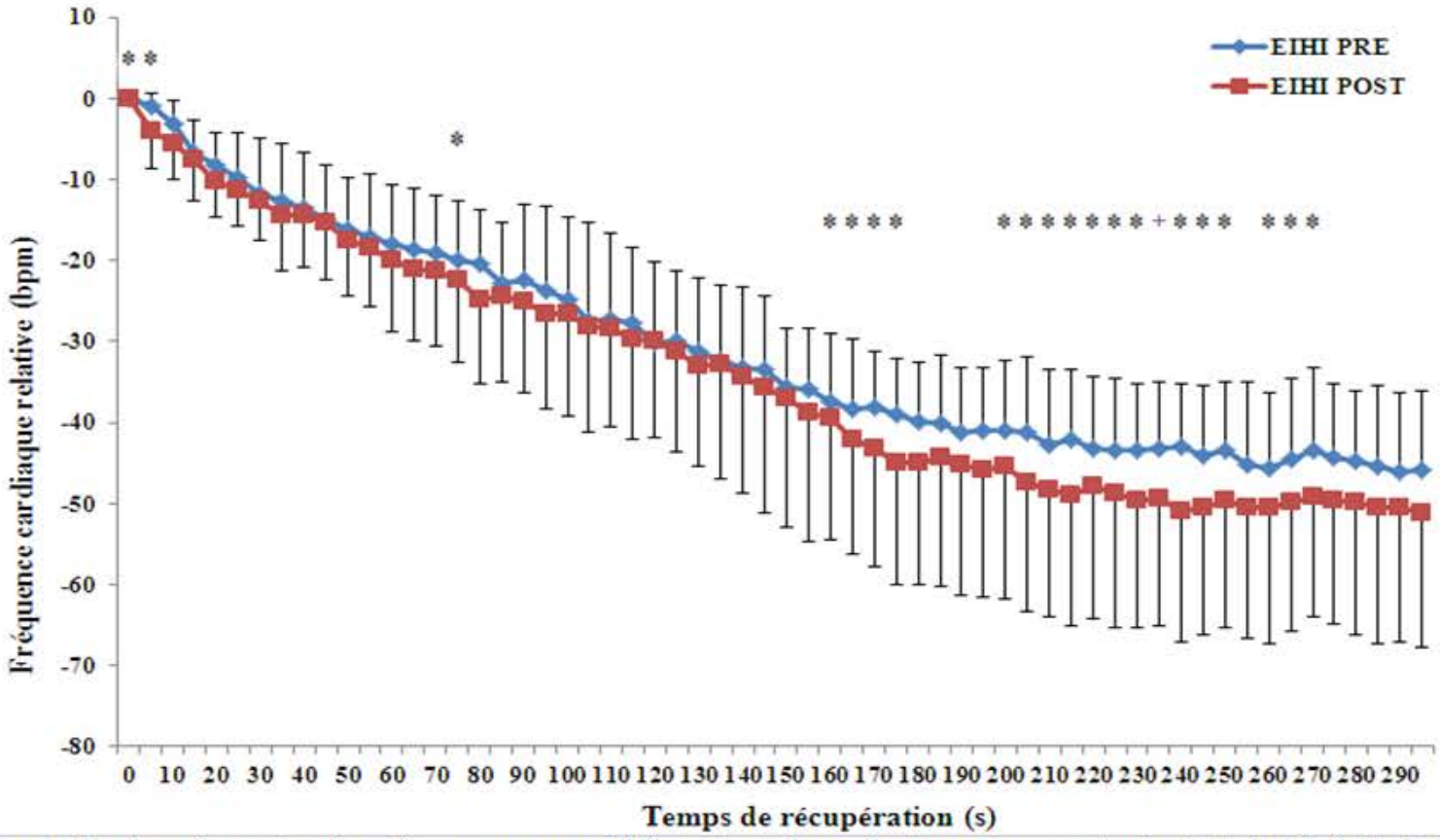
1) Centre de médecine préventive et d'activité physique, Institut de Cardiologie de Montréal (Centre ÉPIC), Montréal, Québec, Canada.
2) Centre de recherche, Institut de Cardiologie et Université de Montréal, Montréal, Québec, Canada.
3) Faculté de médecine, Université de Montréal, Montréal, Québec, Canada.

MJ



Toutes les données sont présentées en moyenne ± l'écart-type. bpm : battements par minute.

Figure 1. Cinétiques des fréquences cardiaques de récupération avant et après un entrainement continu à intensité modéré (ECIM) de 3 mois chez des patients PSCA.



Toutes les données sont présentées en moyenne ± l'écart-type. bpm : battements par minute. *=P<0,05, †=P<0,01.

Journal of the American College of Cardiology
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FOCUS ISSUE: CARDIOMETABOLIC RISK

Simvastatin Impairs Exercise Training Adaptations

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Douglas J. Oberlin, MSc,† Scott P. Naples, MSc,† Justin Fletcher, MSc,†
Grace M. Meers, BSc,§ Meghan Ruebel, MA,‖ M. Harold Laughlin, PhD,¶
Kevin C. Dellsperger, MD, PhD,§ Paul J. Fadel, PhD,# John P. Thyfault, PhD†‡**
Durham, North Carolina; Columbia, Missouri; and Little Rock, Arkansas

Objectives This study sought to determine if simvastatin impairs exercise training adaptations.

Background Statins are commonly prescribed in combination with therapeutic lifestyle changes, including exercise, to reduce cardiovascular disease risk in patients with metabolic syndrome. Statin use has been linked to skeletal muscle myopathy and impaired mitochondrial function, but it is unclear whether statin use alters adaptations to exercise training.

Methods This study examined the effects of simvastatin on changes in cardiorespiratory fitness and skeletal muscle mitochondrial content in response to aerobic exercise training. Sedentary overweight or obese adults with at least 2 metabolic syndrome risk factors (defined according to National Cholesterol Education Panel Adult Treatment Panel III criteria) were randomized to 12 weeks of aerobic exercise training or to exercise in combination with simvastatin (40 mg/day). The primary outcomes were cardiorespiratory fitness and skeletal muscle (vastus lateralis) mitochondrial content (citrate synthase enzyme activity).

Results Thirty-seven participants (exercise plus statins: $n = 18$; exercise only: $n = 19$) completed the study. Cardiorespiratory fitness increased by 10% ($p < 0.05$) in response to exercise training alone, but was blunted by the addition of simvastatin resulting in only a 1.5% increase ($p < 0.005$ for group by time interaction). Similarly, skeletal muscle citrate synthase activity increased by 13% in the exercise-only group ($p < 0.05$), but decreased by 4.5% in the simvastatin-plus-exercise group ($p < 0.05$ for group-by-time interaction).

Conclusions Simvastatin attenuates increases in cardiorespiratory fitness and skeletal muscle mitochondrial content when combined with exercise training in overweight or obese patients at risk of the metabolic syndrome. (Exercise, Statins, and the Metabolic Syndrome; NCT01700530) (J Am Coll Cardiol 2013;62:709–14) © 2013 by the American College of Cardiology Foundation

JACC Vol. 62, No. 8, 2013
August 20, 2013:709–14

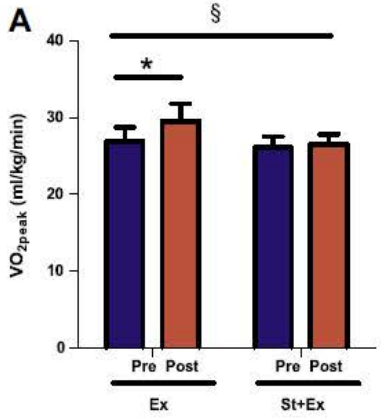

Figure 1 Cardiorespiratory Fitness

(A) Peak oxygen consumption (Vo_2peak) before (Pre) and after (Post) 12 weeks of supervised aerobic exercise training (Ex) or combination exercise-plus-statin therapy (St+Ex). **(B)** Vo_2peak presented as within-group change (Δ) from baseline. Data are expressed as mean ± SE. $^*p < 0.005$ for within-group change from baseline. §$p < 0.005$ for between-group difference in change from baseline.

JACC Vol. 62, No. 8, 2013
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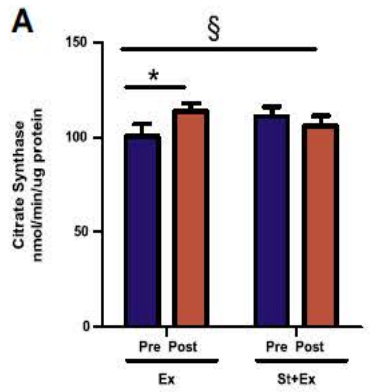

Figure 2 **Citrate Synthase Activity, a Marker of Skeletal Muscle Mitochondrial Content**

(A) Citrate synthase activity before and after 12 weeks of supervised aerobic exercise training or combination exercise-plus-statin therapy. **(B)** Citrate synthase activity presented as within-group change from baseline. Data are expressed as mean ± SE. $^{*}p < 0.05$ for within-group change from baseline. §$p < 0.05$ for between-group difference in change from baseline. Abbreviations as in Figure 1.

12-weeks of lipid-lowering therapy decrease exercise tolerance without affecting endothelial function or arterial stiffness among subjects with primary untreated hypercholesterolemia

Philippe Sosner (MD) [1-5], Mathieu Gayda (PhD) [1-3], Gary Mitchell (MD) [6], Julie Lalongé [1,2], Sébastien Lacroix (PhD) [1-3]
Martin Juneau (MD) [1-3], Jean-Claude Tardif (MD) [2,3], Anil Nigam (MD) [1-3].

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Background

Conduit vessel non-compliance and endothelial dysfunction may be manifestations of a similar process which lead to atherosclerosis.[1] Statin therapy may have beneficial effects on these parameters. We sought to study the effect of statin therapy on endothelial function, aortic stiffness, and their potential impact on VO_2 peak and submaximal exercise endurance.

Methods

In this double-blind, placebo-controlled trial, 20 patients with primary untreated hypercholesterolemia and free of other risk factors or cardiovascular disease *(Table 1)* were randomized 1:1 to pravastatin 40 mg daily or placebo for 12 weeks.

	Placebo (n = 10)		Statin (n = 10)		ANOVA
	Pre	Post	Pre	Post	*P* value
Age (years)	55 ± 12		57 ± 7		0.58
Sex	5 male; 5 female		7 male; 3 female		0.65
Weight (kg)	75.8 ± 16.2	75.6 ± 15.2	74.9 ± 15.7	75.0 ± 16.4	a: 0.92 b: 0.86 c: 0.71
BMI (kg/m²)	26.9 ± 3.1	26.9 ± 2.9	26.5 ± 4.4	26.5 ± 4.7	a: 0.83 b: 0.97 c: 0.83
Heart rate (bpm)	66.6 ± 3.9	70.4 ± 6.3	69.1 ± 11.7	69.0 ± 9.7	a: 0.88 b: 0.13 c: 0.11
SBP (mm Hg)	128.5 ± 15.3	125.5 ± 16.74	123.7 ± 9.1	121.5 ± 14.5	a: 0.42 b: 0.45 c: 0.91
DBP (mm Hg)	78.5 ± 5.8	76.5 ± 6.7	78.0 ± 4.8	75.0 ± 7.1	a: 0.66 b: 0.14 c: 0.76
HbA1c (%)	5.43 ± 0.29	5.90 ± 0.42	5.07 ± 0.76	5.73 ± 0.25	a: 0.38 b: 0.13 c: 0.71
Fasting glucose (mmol/L)	5.17 ± 0.54	5.17 ± 0.49	4.90 ± 0.43	5.18 ± 0.55	a: 0.68 b: 0.09 c: 0.09
Total cholesterol (mmol/L)	6.79 ± 0.69	6.18 ± 0.94	6.65 ± 0.49	5.23 ± 0.79	a: 0.11 b: <0.0001 c: 0.01
HDL-cholesterol (mmol/L)	1.41 ± 0.37	1.41 ± 0.28	1.38 ± 0.37	1.39 ± 0.37	a: 0.88 b: 0.93 c: 0.93
LDL-cholesterol (mmol/L)	4.77 ± 0.47	4.15 ± 0.72	4.43 ± 0.33	3.03 ± 0.73	a: 0.01 b: <0.0001 c: 0.008
Triglycerides (mmol/L)	1.38 ± 0.60	1.39 ± 0.73	1.98 ± 1.28	1.79 ± 0.90	a: 0.22 b: 0.33 c: 0.50

Table 1. General characteristics.

Values are mean ± SD. Subject characteristics before (Pre) and after 12 weeks (Post) of statin or placebo treatment. a, condition effect; b, time effect; c, interaction effect (condition x time). *BMI, body mass index; bpm, beats per minute; SBP, systolic blood pressure; DBP, diastolic blood pressure; HbA1c, hemoglobin A1c; HDL, high-density lipoprotein; LDL, low-density lipoprotein.*

We non-invasively assessed:

- endothelial function using ultrasound-guided brachial artery flow mediated dilatation (FMD) method;[2]
- aortic stiffness using tonometry-derived carotid-femoral pulse wave velocity (cfPWV);[3]
- VO_2 peak on a maximal exercise test;
- endurance time on a submaximal exercise test.

Results

After 12 weeks, In pre/post comparisons in statin group, we observed a decrease in LDL-cholesterol (4.49 ± 0.34 vs. 3.03 ± 0.73 mmol/L, P = 0.01, g = 0.79) *(Table 1)*, no changes in FMD (7.0 ± 3.7 vs. 10.1 ± 4.7 %, P = 0.12, g = -0.34), cfPWV (7.69 ± 1.87 vs. 8.28 ± 2.17 m/s, P = 0.17, g = -0.14) and submaximal exercise duration (1326 ± 649 vs. 1230 ± 862 sec, P = 0.67, g = 0.06) *(Table 2)*, but a decrease in VO_2 peak (29.77 ± 6.07 vs. 25.59 ± 4.60 mL/min/kg, P = 0.03, g = 0.36) that did not changed in placebo group (27.85 ± 8.83 vs. 28.93 ± 9.62 mL/min/kg, P = 0.65, g = -0.06) *(Table 3) (Figure 1)*.

	Placebo		Statin		ANOVA
	Pre	Post	Pre	Post	*P* value
Arterial stiffness	(n = 10)	(n = 6)	(n = 10)	(n = 6)	
Central SBP (mm Hg)	107.0 ± 18.6	103.9 ± 14.4	102.3 ± 16.9	101.7 ± 10.0	a: 0.67 b: 0.65 c: 0.76
Central DBP (mm Hg)	59.8 ± 8.0	61.0 ± 11.8	60.5 ± 7.7	60.2 ± 6.7	a: 0.98 b: 0.88 c: 0.79
Augmentation index (%)	14.1 ± 10.2	18.5 ± 18.6	10.4 ± 3.3	16.4 ± 7.8	a: 0.44 b: 0.36 c: 0.89
cfPWV (m/s)	9.00 ± 2.33	8.65 ± 2.12	7.69 ± 1.87	8.28 ± 2.17	a: 0.65 b: 0.29 c: 0.76
Endothelial function	(n = 10)	(n = 8)	(n = 10)	(n = 8)	
Baseline brachial artery diameter (mm)	3.65 ± 0.84	3.79 ± 0.71	3.66 ± 0.52	3.71 ± 0.73	a: 0.92 b: 0.31 c: 0.40
NTG-mediated dilatation (%)	11.2 ± 5.9	12.0 ± 3.9	10.89 ± 3.2	12.3 ± 4.5	a: 0.61 b: 0.36 c: 0.51
FMD (%)	7.5 ± 4.1	9.2 ± 2.9	7.0 ± 3.7	10.1 ± 4.7	a: 0.88 b: 0.053 c: 0.64

Table 2. Vascular characteristics.

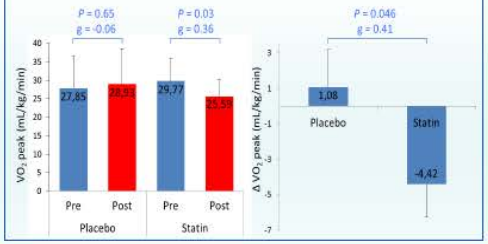
Values are mean ± SD. Subject characteristics before (Pre) and after 12 weeks (Post) of statin or placebo treatment. a, condition effect; b, time effect; c, interaction effect (condition x time). *SBP, systolic blood pressure; DBP, diastolic blood pressure; cfPWV, carotid-femoral pulse wave velocity; NTG, nitroglycerin; FMD, Flow mediated dilatation.*

	Placebo		Statin		ANOVA
	Pre	Post	Pre	Post	*P* value
Sub-maximal exercise test	(n = 10)	(n = 9)	(n = 10)	(n = 9)	
Total exercise time (sec)	1536 ± 893	1562 ± 948	1326 ± 649	1230 ± 862	a: 0.36 b: 0.72 c: 0.86
Maximal exercise test	(n = 10)	(n = 10)	(n = 10)	(n = 9)	
Maximum HR (bpm)	162.9 ± 14.6	165.8 ± 19.6	158.2 ± 14.9	152.3 ± 17.1	a: 0.22 b: 0.63 c: 0.08
% Maximum predicted HR	96.8 ± 6.8	98.4 ± 9.4	95.0 ± 8.4	91.4 ± 9.1	a: 0.22 b: 0.59 c: 0.09
% Heart rate reserve	94.8 ± 11.2	97.2 ± 16.0	90.9 ± 13.4	86.4 ± 14.2	a: 0.23 b: 0.63 c: 0.14
RER max	1.15 ± 0.09	1.12 ± 0.06	1.11 ± 0.06	1.10 ± 0.06	a: 0.23 b: 0.54 c: 0.47
VO_2 peak (L/min)	2.17 ± 0.94	2.25 ± 0.96	2.23 ± 0.65	1.95 ± 0.43	a: 0.81 b: 0.26 c: 0.09
VO_2 peak (mL/min/kg)	27.85 ± 8.83	28.93 ± 9.62	29.77 ± 6.07	25.59 ± 4.60	a: 0.86 b: 0.25 c: 0.07

Table 3. Exercise tests results.

Values are mean ± SD. Subject characteristics before (Pre) and after 12 weeks (Post) of statin or placebo treatment. a, condition effect; b, time effect; c, interaction effect (condition x time). HR, heart rate; bpm, beats per minute; VO_2, oxygen uptake; RER, respiratory exchange ratio.

Figure1. VO_2 peak before (Pre) and after 12 weeks (Post) of placebo or statin treatment (left) and changes in peak (right). Data are mean and SD (left) or standard error (right). VO_2, oxygen uptake. Hedge's (g) effect size: small (0.2 < g ≤ 0.5), moderate (0.5 < g ≤ 0.8), or large (g > 0.8).



Conclusion

In patients with previously untreated hypercholesterolemia, 12-weeks of statin therapy had deleterious effects on VO_2 peak without affecting vascular function. One explanation could be deleterious effect of statin on mitochondrial function.[4]

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MJ

ORIGINAL RESEARCH | Annals of Internal Medicine

The Association Between Midlife Cardiorespiratory Fitness Levels and Later-Life Dementia

A Cohort Study

Laura F. DeFina, MD; Benjamin L. Willis, MD, MPH; Nina B. Radford, MD; Ang Gao, MS; David Leonard, PhD; William L. Haskell, PhD; Myron F. Weiner, MD; and Jarett D. Berry, MD, MS

Table 2. Cox Proportional Hazards Model for Incident Alzheimer Disease and Related Types of Dementia

Effect, by Model Adjustment	Hazard Ratio (95% CI)	*P* Value
Adjustment 1 (n = 19 458)*		
Quintile 1 (reference)	1.00	–
Quintile 2	0.87 (0.75–1.01)	0.069
Quintile 3	0.78 (0.67–0.91)	0.001
Quintile 4	0.70 (0.60–0.81)	<0.001
Quintile 5	0.64 (0.54–0.76)	<0.001

J Rehabil Med

SHORT COMMUNICATION

EFFECT OF INTERVAL TRAINING ON COGNITIVE FUNCTIONING AND CEREBRAL OXYGENATION IN OBESE PATIENTS: A PILOT STUDY

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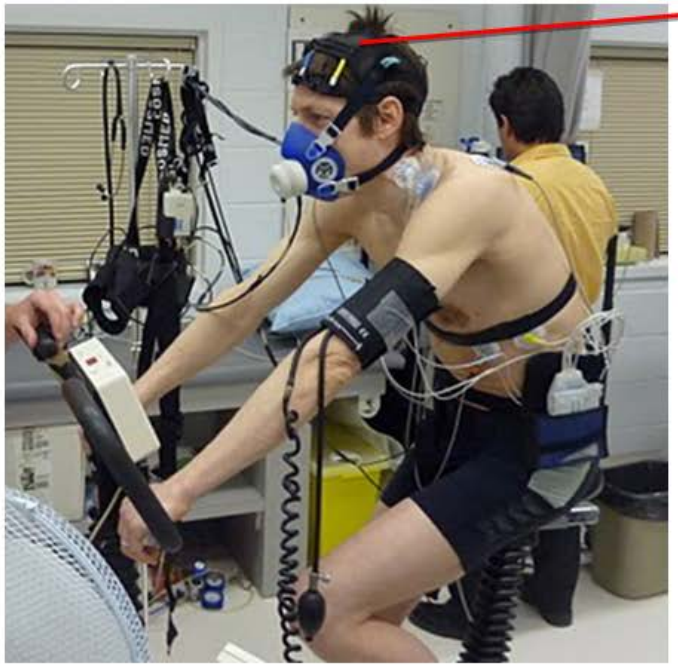
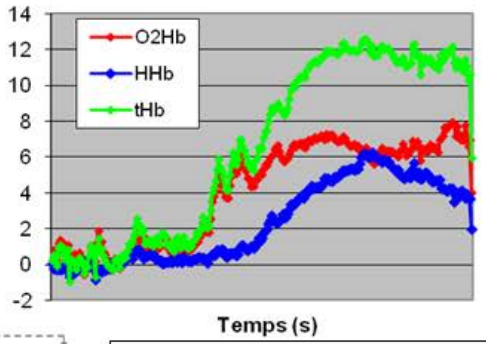
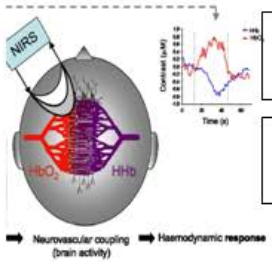
Epreuve d'effort triangulaire



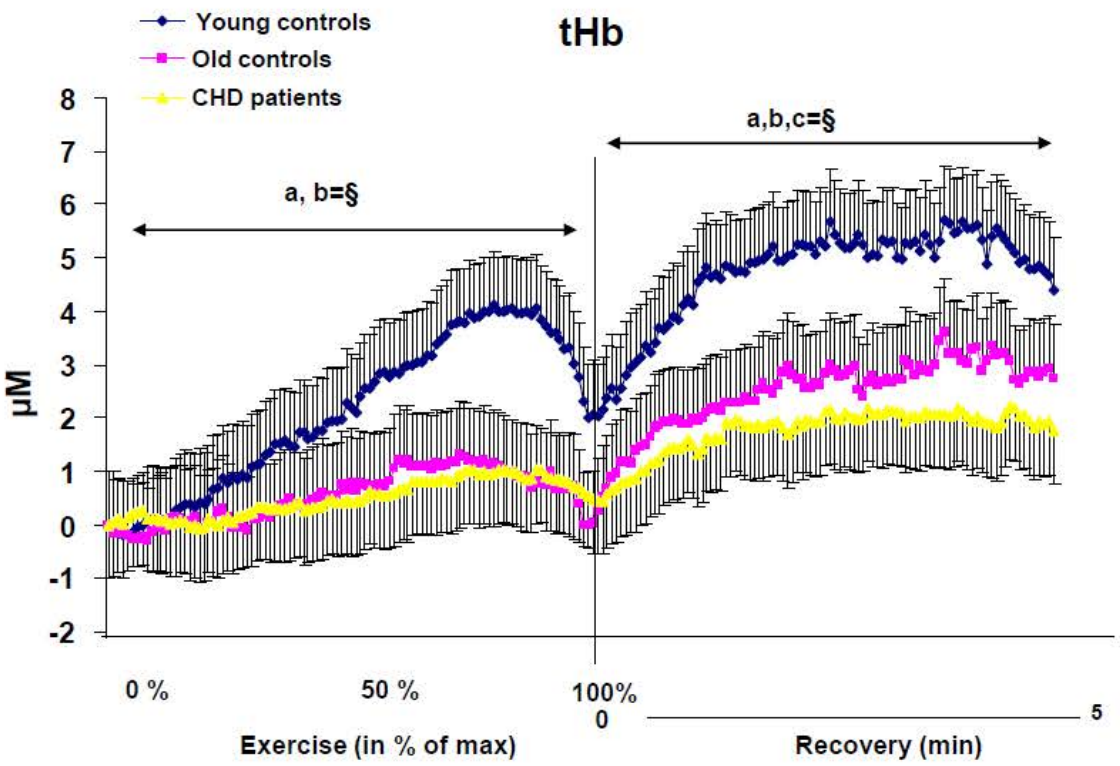




Activation :
↑ O2Hb et ↓ /stabilité HHb

Désactivation :
↓ O2Hb et ↑ HHb

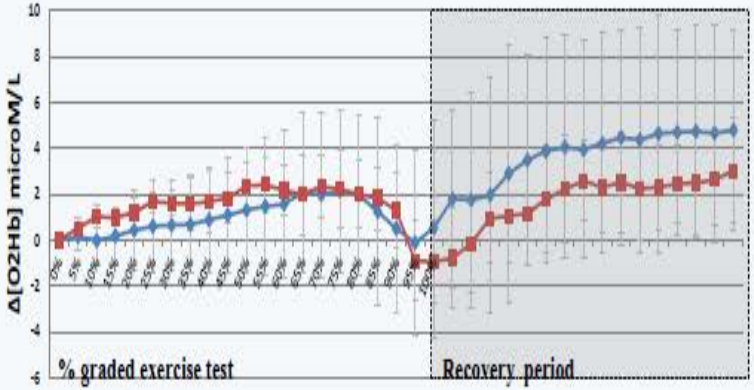
Oxygénation cérébrale: NIRS
Oxymon, Artinis, Netherlands



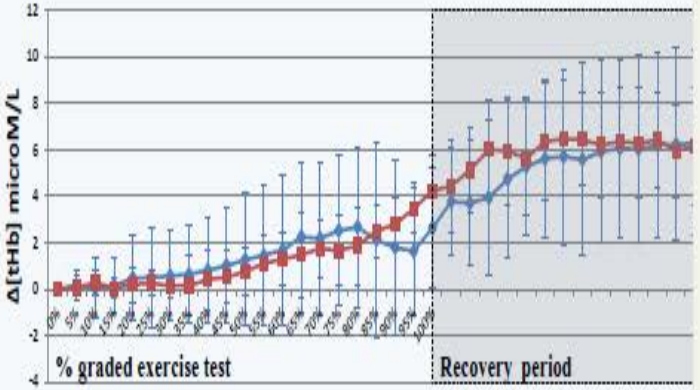
tHb: total hemoglobin
Group effect: a= young vs. old, b=young vs. CHD, c=CHD vs. old, §=p<0.0001

e 2- Changes in cerebral oxygenation during maximal exercise testing

O2Hb



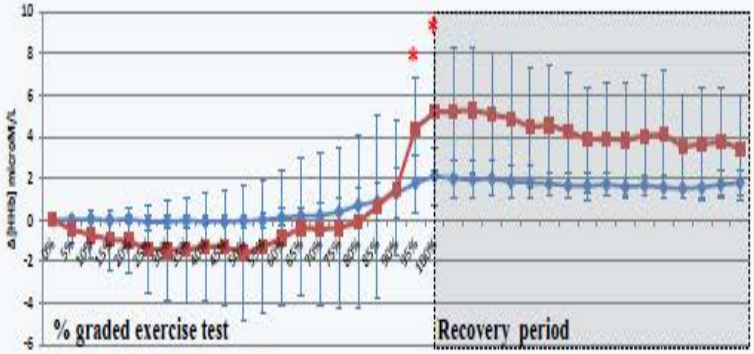
tHb



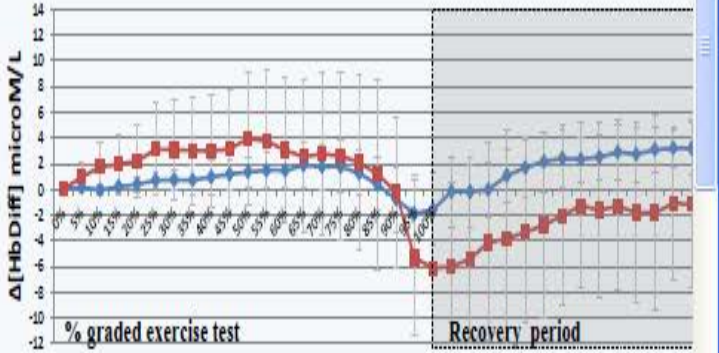
before

after

HHb



HbDiff



nt changes with p-value p<0,05

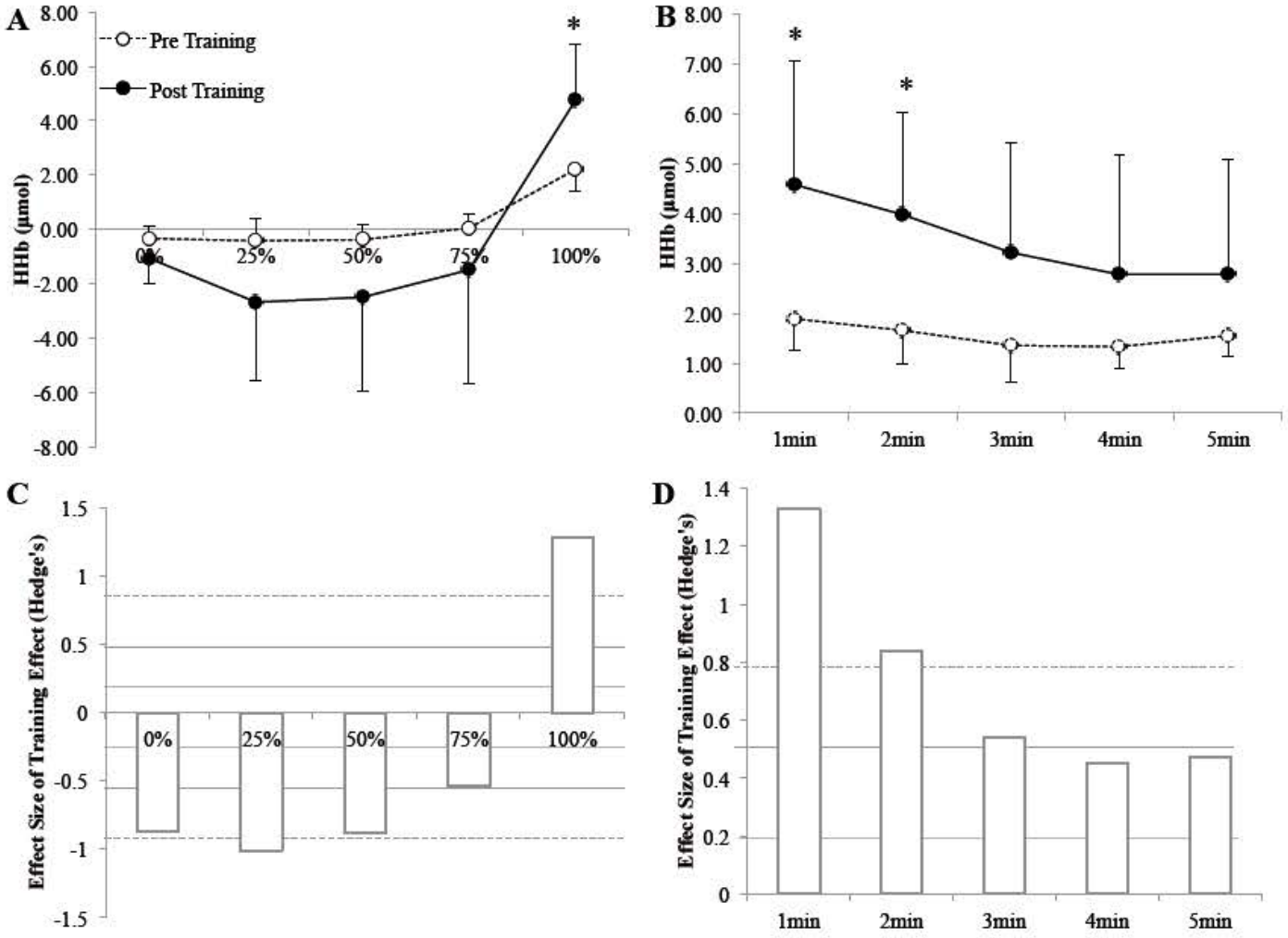
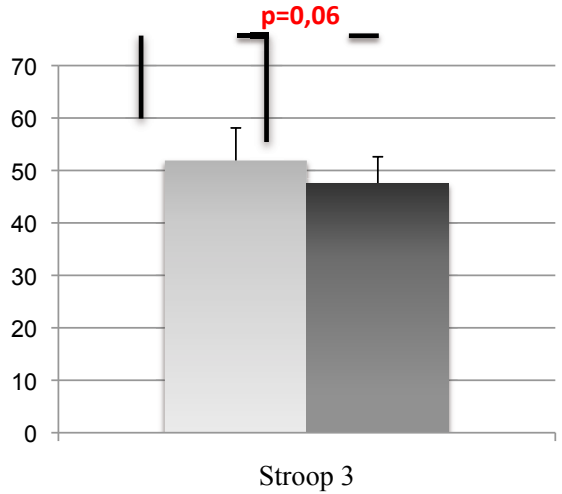
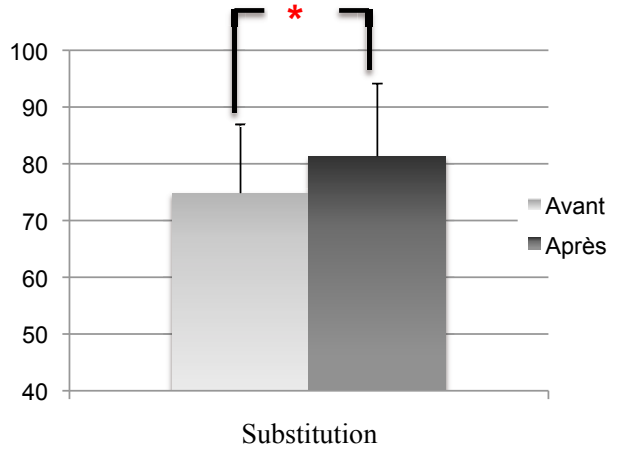


Fig. 1. Effect of high-intensity interval training (HIIT) on deoxyhaemoglobin (Δ[HHb]) signal] during (A) maximal exercise and (B) during recovery. Magnitude of difference during (C) exercise and (D) recovery. Dotted lines represent strength of magnitude effect according the Cohen's scale. $*p < 0.05$.

Résultats - Cognitif

- Exécutif





- Corrélations

	R	P- value
Stroop – Tâche d'inhibition		
Perte de poids	**0,87**	**<0,05**
Diminution du tour de taille	**0,79**	**<0,05**
Gain en PMA	**-0,85**	**<0,05**
Empan de chiffre - Endroit		
Gain en PMA	**0,92**	**<0,05**

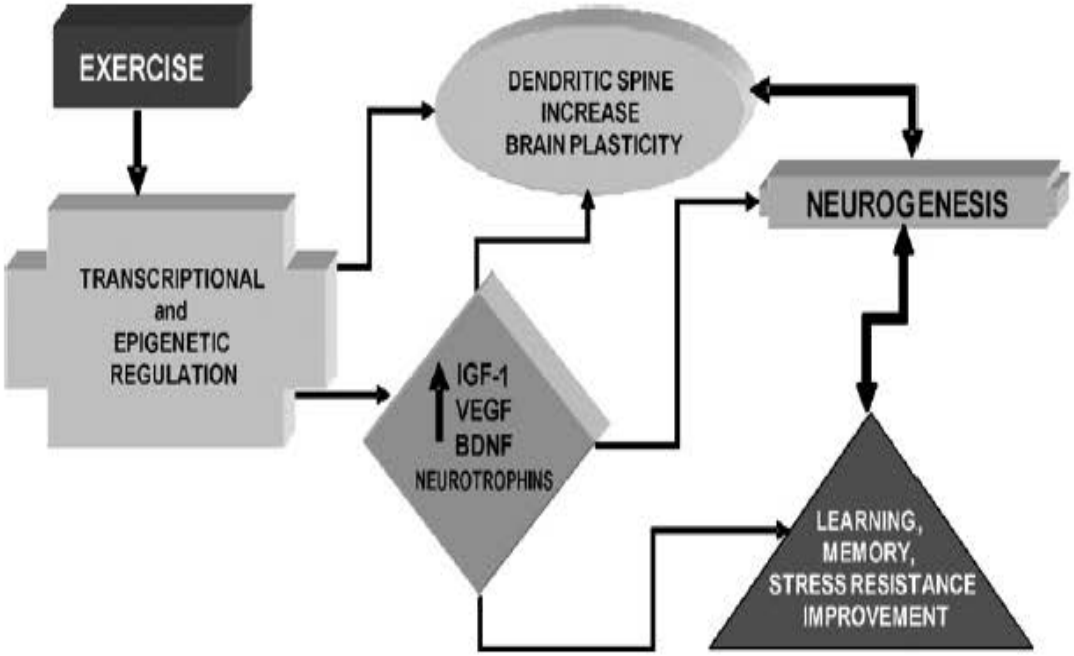


Fig. 1. A model for the effect of exercise on molecular, neuroplastic and cognitive parameters. Physical activity has positive neurophysiological effects mediated by changes in the transcriptional profiles of growth and neurotrophic factors such as VEGF, IGF-1 and BDNF that lead to improved efficiency of the capillary system and neuroplastic mechanisms in the brain. Although the precise molecular pathways that sustain these processes are unknown, recent data have described the epigenetic impact of physical exercise in peripheral tissues and in the brain. In mice such changes have been correlated with cognitive enhancement and increased stress resistance.

Run for your life … at a comfortable speed and not too far

James H O'Keefe,[1,2] Carl J Lavie[3,4]

During the Greco-Persian War in 490 BCE, Phidippides, a 40-year-old herald messenger (professional running-courier) ran the 26 miles from a battlefield near Marathon, Greece, into Athens carrying momentous news of Greek victory. Upon arriving at the Acropolis, he proclaimed: 'Joy, we have won!' and then immediately collapsed and died.[1] Fast-forward about 2500 years to an era when the baby-boomer's came of age and long-distance running boomed. The prevailing logic held that aerobic exercise is clearly good for one's health and that, if some is good, more must be better. In 1975, Dr Thomas

may not kill you, it may erase many of the health advantages of regular moderate exercise.

Indeed, regular vigorous exercise is probably the single best step a person can take to ensure robust CV health. In a study of 416 000 adults followed for a mean of 8 years, 40–50 min per day of vigorous exercise reduced risk of death by about 40% (figure 1).[7] In that study, at about 45 min, a point of diminishing returns is reached whereby longer exercise efforts do not appear to translate into lower death risk. Light to moderate physical activity reduced death rates too, albeit not as

morbidity and mortality (figure 2).[8] However, fitness levels above 12 metabolic equivalents do not seem to translate into additional gains in CV health and longevity. Thus, if one is training to be able to run at speeds above 7.5 miles per hour, this is being done for some reason other than further improvements in life expectancy.

CV DAMAGE FROM EXCESSIVE EXERCISE

High-intensity exercise sessions lasting beyond 1–2 h cause acute volume overload of the atria and right ventricle (RV), which can bring about overstretching and micro-tears in the myocardium, as evidenced by a transient rise in cardiac biomarkers, including troponin and B-natriuretic peptide and a fall in the RV ejection fraction.[9] Although within 1 week, these transitory abnormalities

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Clinical Research

Transient Myocardial Tissue and Function Changes During a Marathon in Less Fit Marathon Runners

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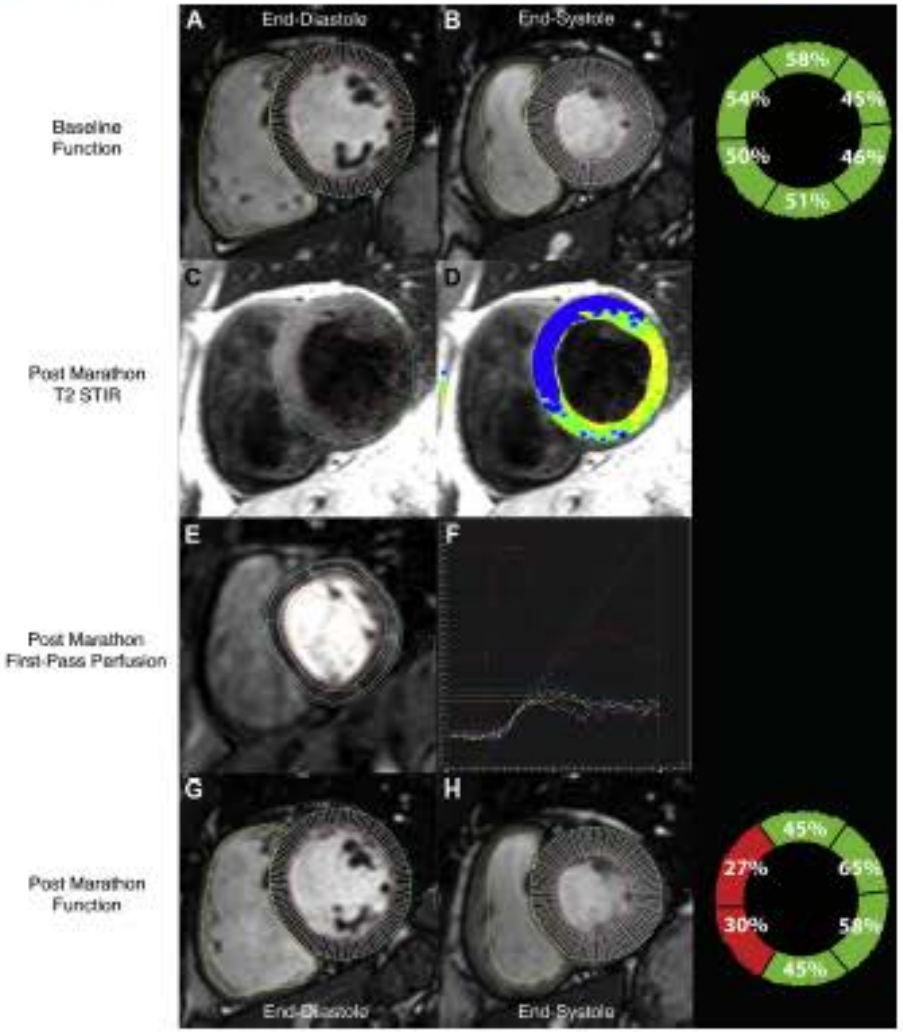

Figure 1. Example of changes observed in 1 marathon runner (single midventricular slice for clarity). Baseline function at end-diastole (**A**) and end-systole (**B**) and resulting systolic wall thickening (**to the right**) derived from fractional shortening averaged for each LV segment from 100 automatically generated radial chords. Post-marathon function at identical slice location (**G** and **H**). Edema identified as T2 STIR Ratio > 2 providing a bright signal (**C**) illustrated in **blue** by overlaid colour-coded map (**D**). First-pass segmental perfusion (**E**) with corresponding contrast curves for all 6 segments of the index slice normalized to LV blood pool contrast kinetics (**F**). Septal segments decreased function, increased edema, and decreased perfusion after the marathon. LV, left ventricle.

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doi:10.1093/eurheartj/ehr397

CLINICAL RESEARCH

Exercise-induced right ventricular dysfunction and structural remodelling in endurance athletes

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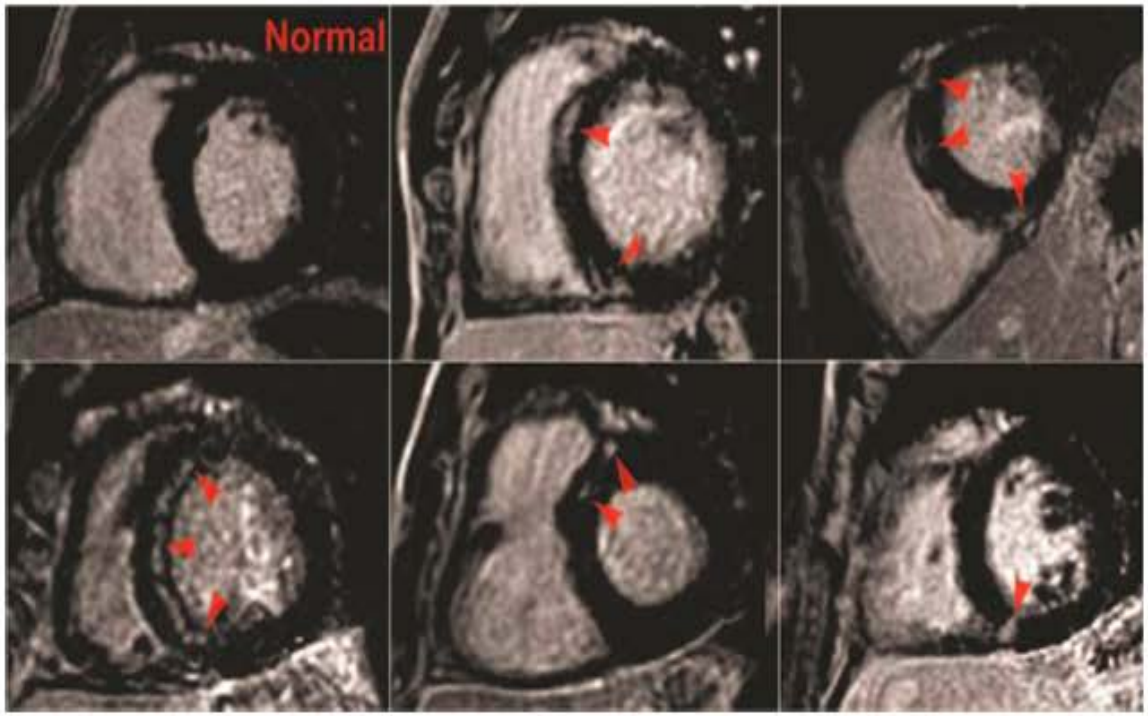

Figure 3 Delayed gadolinium enhancement in five athletes. Images of five athletes in whom focal delayed gadolinium enhancement (DGE) was identified in the interventricular septum (indicated with arrows) when compared with an athlete with a normal study (top left).

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European Heart Journal
doi:10.1093/eurheartj/eht188

CLINICAL RESEARCH

Risk of arrhythmias in 52 755 long-distance cross-country skiers: a cohort study

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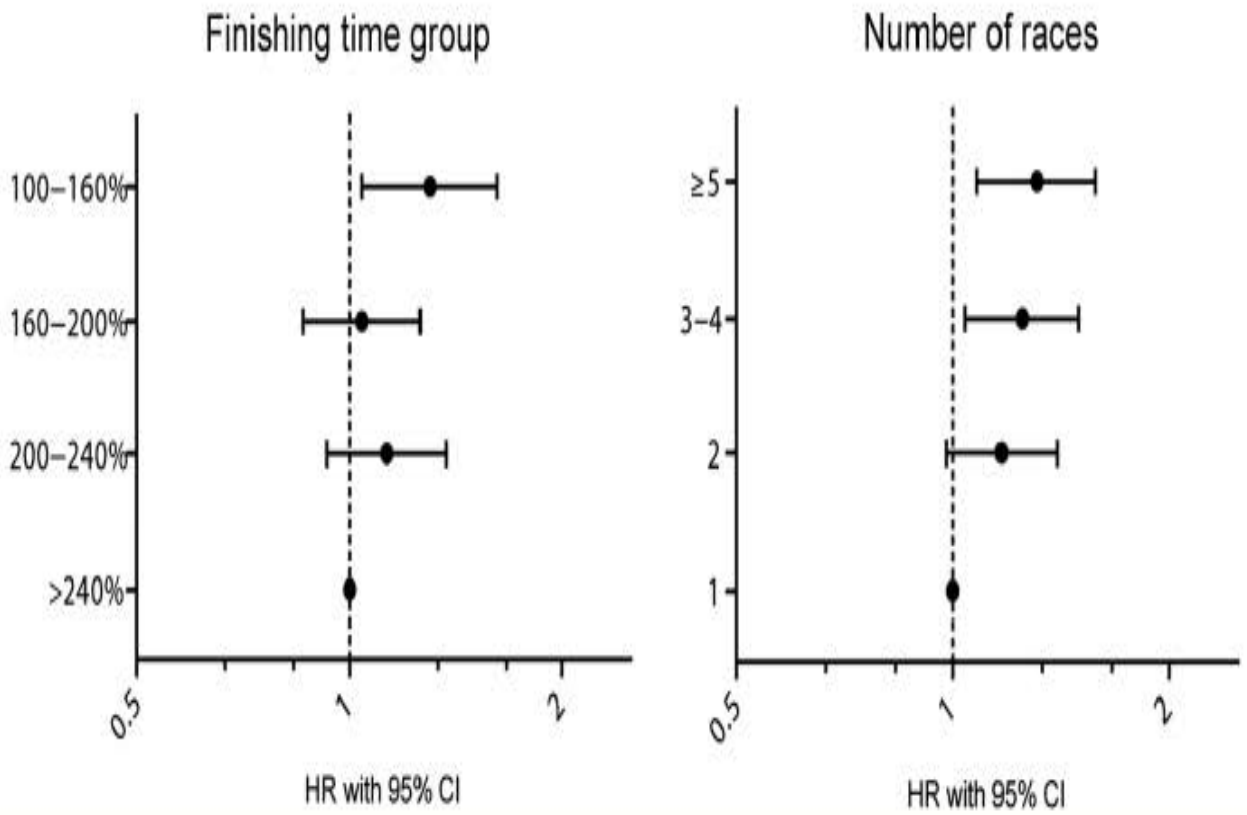


Figure 2 Hazard ratios of any arrhythmia with 95% confidence intervals (log scale) by finishing time group in per cent of winning time and number of previous races. Model adjusted for age, occupation, and education level.

Arrhythmia/Electrophysiology

Cardiac Arrhythmogenic Remodeling in a Rat Model of Long-Term Intensive Exercise Training

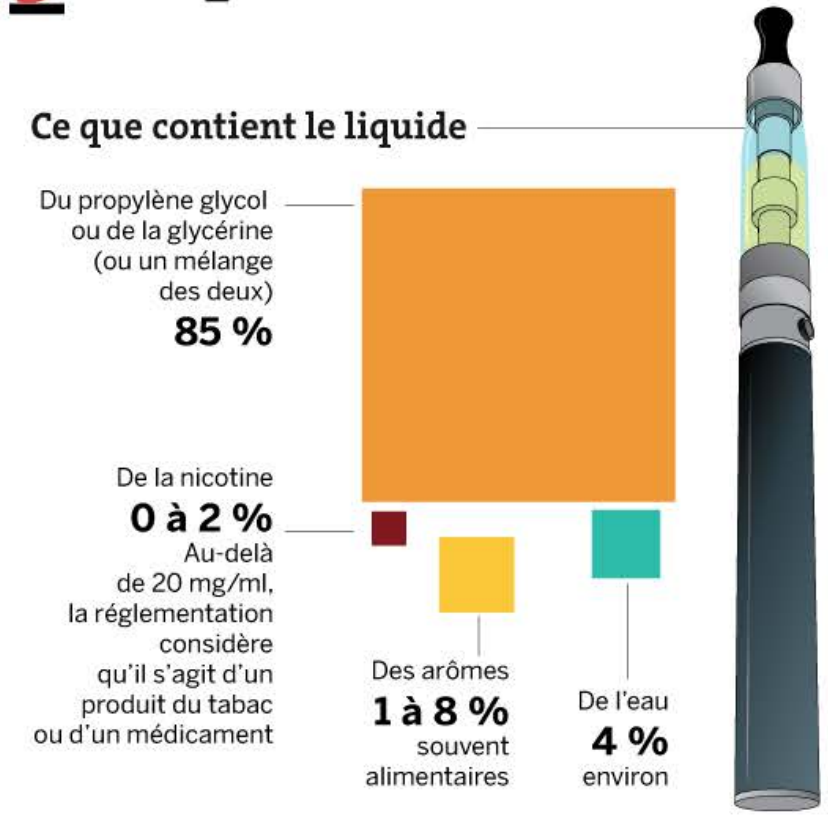
Begoña Benito, MD*; Gemma Gay-Jordi, PhD*; Anna Serrano-Mollar, PhD; Eduard Guasch, MD; Yanfen Shi, MD; Jean-Claude Tardif, MD; Josep Brugada, MD, PhD; Stanley Nattel, MD†; Lluis Mont, MD, PhD†

Background—Recent clinical studies suggest that endurance sports may promote cardiac arrhythmias. The aim of this study was to use an animal model to evaluate whether sustained intensive exercise training induces potentially adverse myocardial remodeling and thus creates a potential substrate for arrhythmias.

Methods and Results—Male Wistar rats were conditioned to run vigorously for 4, 8, and 16 weeks; time-matched sedentary rats served as controls. Serial echocardiograms and in vivo electrophysiological studies at 16 weeks were obtained in both groups. After euthanasia, ventricular collagen deposition was quantified by histological and biochemical studies, and messenger RNA and protein expression of transforming growth factor-β1, fibronectin-1, matrix metalloproteinase-2, tissue inhibitor of metalloproteinase-1, procollagen-I, and procollagen-III was evaluated in all 4 cardiac chambers. At 16 weeks, exercise rats developed eccentric hypertrophy and diastolic dysfunction, together with atrial dilation. In addition, collagen deposition in the right ventricle and messenger RNA and protein expression

Extrait de l'étude Etincel
de l'Observatoire des drogues et toxicomanies

3 Un produit « sain » ?



Contrairement à la fumée de cigarette, la vapeur de l'e-cigarette ne contient pas :

De monoxyde de carbone (gaz asphyxiant)

De particules fines solides (qui provoquent maladies respiratoires, cardiaques et vasculaires)

EN NANOGRAMMES

	D'une cigarette	D'une e-cigarette
Phénanthrène	350	48
Anthracène	130	7
Pyrène	130	36
Benzopérylène	60	Non détectable
Chrysène	50	Non détectable
Benzanthracène	45	Non détectable
Benzo(a) pyrène	35	Non détectable
Benzofluoranthène	30	Non détectable
Méthyl phénanthrène	30	5
Benzo(e) pyrène	16	Non détectable
Indeno pyrène	12	Non détectable

Cancérogène probable

Cancérogène possible

Non classable en cancérogène

Health risks of e-cigarette vapour

- e-cigarette vapours contain some toxic substances. The levels of the toxicants are **9-450 times lower** than in cigarette smoke and, comparable with trace amounts found in a nicotine inhalator (Formaldehyde, Acetaldehyde, Cd, Ni, Pb).[1]

Table 4 Comparison of toxins levels between conventional and electronic cigarettes

Toxic compound	Conventional cigarette (µg in mainstream smoke) [35]	Electronic cigarette (µg per 15 puffs)	Average ratio (conventional vs electronic cigarette)
Formaldehyde	1.6–52	0.20–5.61	9
Acetaldehyde	52–140	0.11–1.36	450
Acrolein	2.4–62	0.07–4.19	15
Toluene	8.3–70	0.02–0.63	120
NNN	0.005–0.19	0.00008–0.00043	380
NNK	0.012–0.11	0.00011–0.00283	40

NNK, N′-nitrosonornicotine (NNN) and 4-(methylnitrosoamino)-1-(3-pyridyl)-1-butanone; NNN, N′-nitrosonornicotine.

1. Goniewicz ML, Knysak J, Gawron M, Kosmider L, Sobczak A, Kurek J, Prokopowicz A, Jablonska-Czapla M, Rosik-Dulewska C, Havel C, Jacob P 3rd, Benowitz N. Levels of selected carcinogens and toxicants in vapour from electronic cigarettes. Tob Control. 2013 Mar 6. [Epub ahead of print]

L'e-cigarette et les jeunes - 15 mai 2014

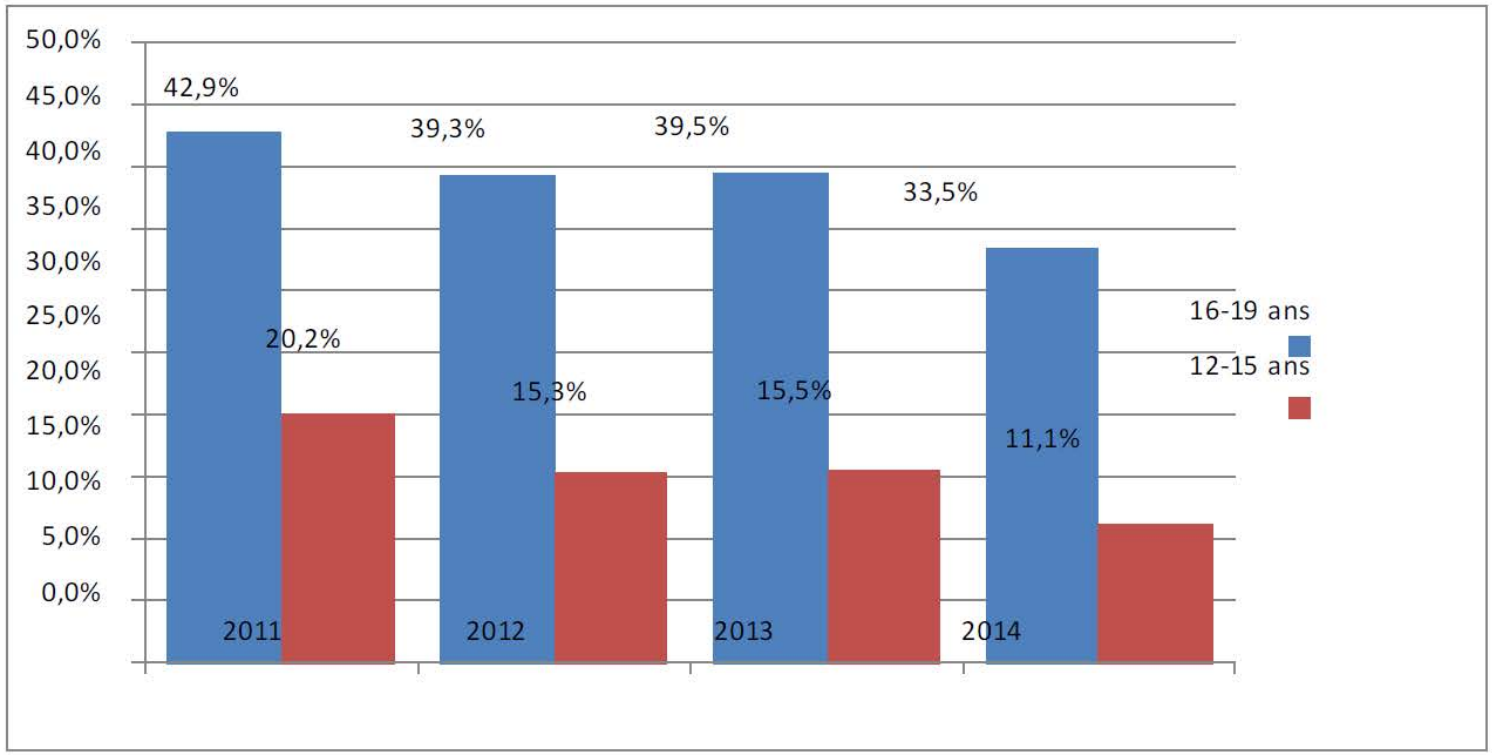
Contrairement à ce qui était redouté, l'e-cigarette apparaît en 2014 chez les jeunes Parisiens davantage comme un produit de « ringardisation » du tabac que comme un produit d'entrée en tabagisme.

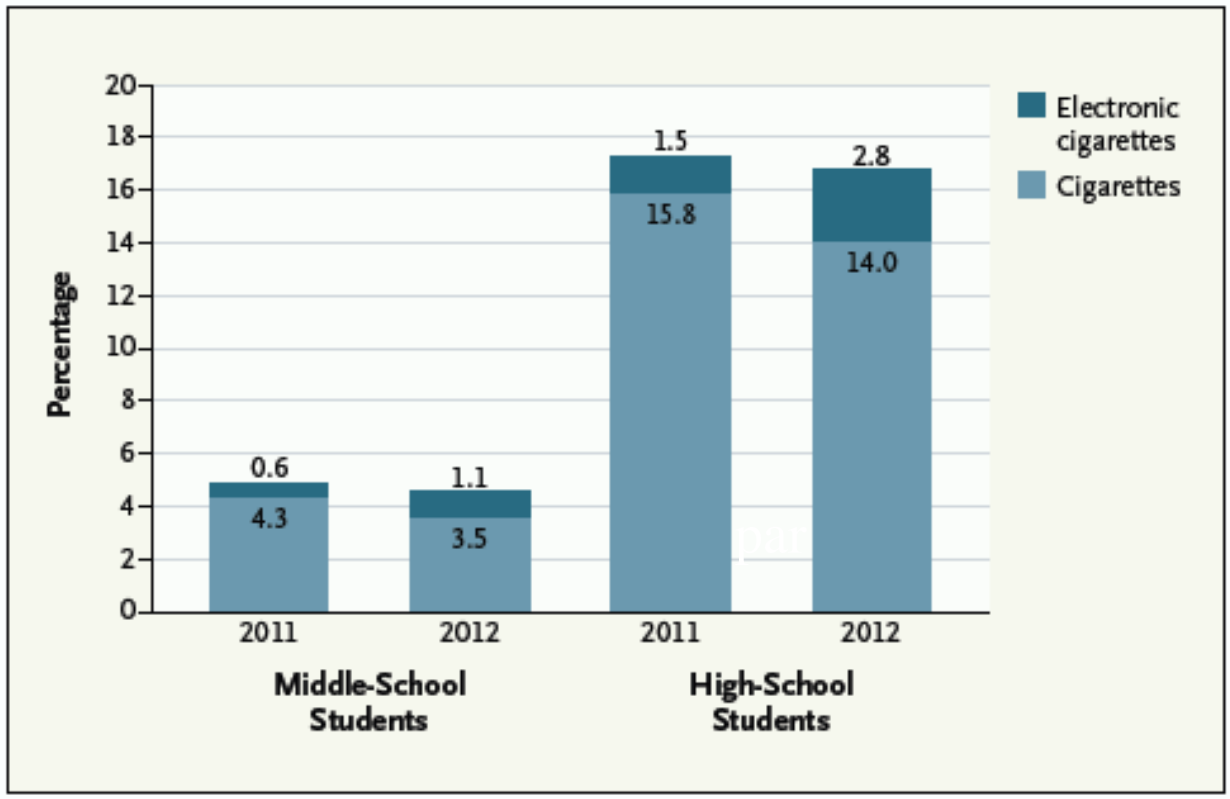
Avant propos

L'e-cigarette ou cigarette électronique a débarqué en masse depuis moins de deux ans en France.

La cigarette électronique apparaît de plus en plus comme un produit de sortie du tabac à l'échelon individuel et à l'échelon collectif comme le montrent les données françaises et anglaises[1] qui sont les deux pays européens où l'e-cigarette est bien disponible (alors qu'en Belgique par exemple où la vente d'e-liquide nicotiné est interdite, les rares vapoteurs belges viennent s'approvisionner en France et la Belgique voit son taux de fumeurs et ses ventes de cigarettes augmenter comme le dénonce l'ABDV)[2].

✓ Bonne nouvelle : la consommation de tabac quotidienne ou occasionnelle baisse chez les collégiens et lycéens parisiens





Use of Cigarettes and Electronic Cigarettes by U.S. Students in 2011 and 2012.
Data are from the Centers for Disease Control and Prevention.

Amy L. Fairchild, Ph.D., M.P.H., Ronald Bayer, Ph.D., and James Colgrove, Ph.D., M.P.H. *The Renormalization of Smoking? E-Cigarettes and the Tobacco « Endgame ».* The New England Journal of Medicine. Perspective, January 23, 2014. p. 294.

- On voit clairement d'après ces données CDC que la consommation de cigarettes de tabac baisse de 2011 à 2012 chez les jeunes américains pendant que la consommation de cigarettes électroniques augmente.
- On voit également très bien que la cigarette électronique ne constitue pas un tremplin vers le tabac puisque la consommation de cigarettes de tabac baisse à mesure que la consommation de cigarettes électroniques augmente.

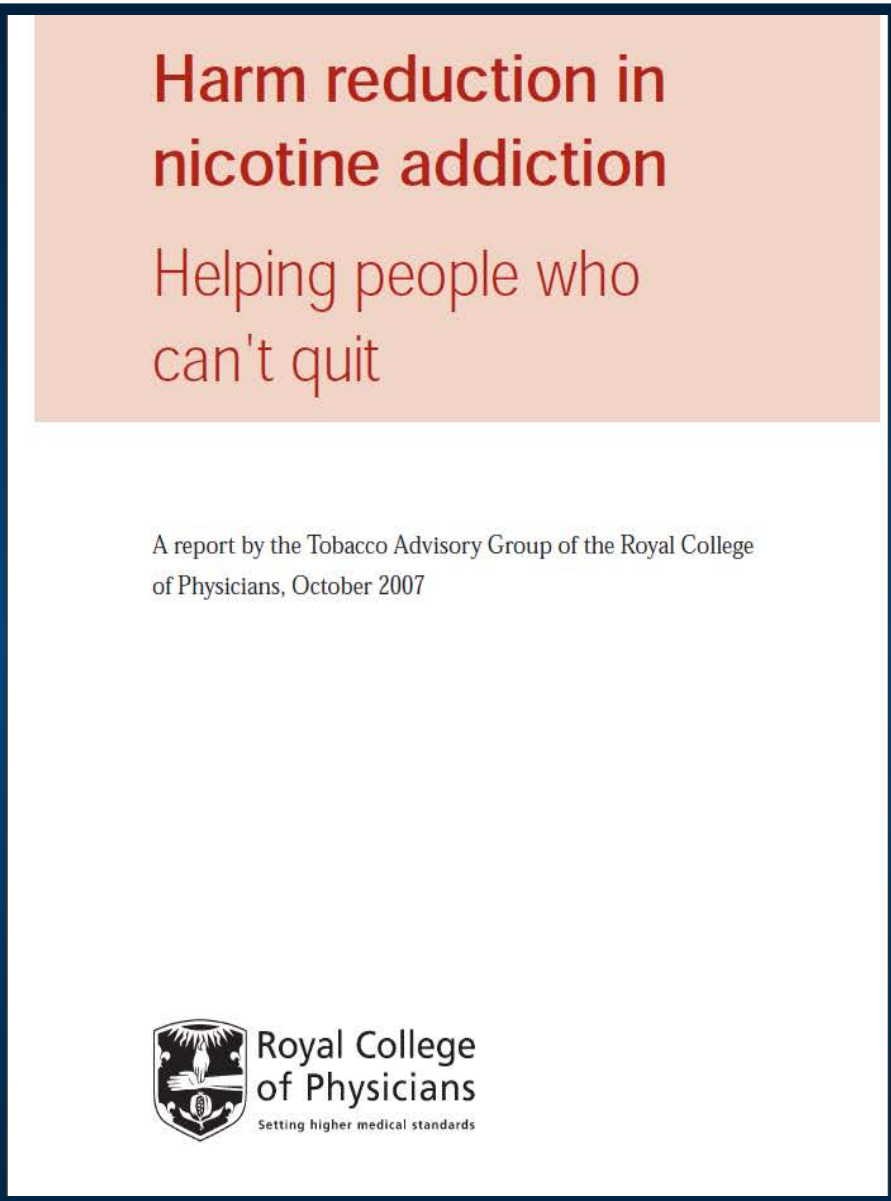
MJ

Here are the numbers from the three phases of the YSS specifically related to the current smoking status of teenagers in Quebec and Canada:

Québec	Grades 6-9	Grades 10-12
2008-09	7.6%	16.6%
2010-11	4.3%	8.7%
2012-13	4.4%	7.0%

Canada	Grades 6-9	Grades 10-12
2008-09	3.5%	13.2%
2010-11	2.2%	10.0%
2012-13	1.9%	7.8%

Again, these numbers suggest that electronic cigarettes are not renormalizing tobacco use amongst kids.


Harm reduction in nicotine addiction
Helping people who can't quit
A report by the Tobacco Advisory Group of the Royal College of Physicians, October 2007
Royal College of Physicians
Setting higher medical standards

MJ

RRPs Opportunity

PHILIP MORRIS INTERNATIONAL

- Commercialization on track for 2014:
  - Platform 1 in city test phase
  - E-vapor products in the EU:
    - Acquisition of UK-based Nicocigs Limited

Heated Tobacco Products | **Nicotine Containing Products**

Platform 1

Platform 2

Platform 3

Platform 4 Generation 1

Ready for Commercialization

Product Development Ongoing

Ready for Commercialization

Note: Reduced-Risk Products ("RRPs") is the term we use to refer to products that have the potential to reduce individual risk and population harm. The products depicted are subject to ongoing development and therefore the descriptions are illustrative and do not necessarily represent the latest stages of product development

RECOMMANDATIONS

- HOMOLOGATION PAR SANTÉ CANADA
- NE **PAS** RENDRE LA CE **MOINS** ACCESSIBLE QUE LA CIGARETTE DE TABAC
- PERMETTRE CERTAINES SAVEURS
- PERMETTRE DE VAPOTER DANS LES POINTS DE VENTE (BOUTIQUES SPÉCIALISÉES)
- NE PAS MÉDICALISER (LE TABAC N'EST PAS PRESCRIT PAR UN MD)
- PAS DE PUB "LIFESTYLE"
- PAS DE CE DANS ENDROITS PUBLICS
- PAS DE VENTE AUX MINEURS

MERCI