groups and health care providers with lower rates of recommended medication use. These measures alone may not be sufficient, and investment in other, innovative approaches to promoting evidence-based prescribing practices is warranted. The correlation between use of these therapies and beneficial outcomes in these and other subpopulations also needs further assessment.

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1. DeFrances CJ, Cullen KA, Kozak LJ. National Hospital Discharge Survey: 2005 annual summary with detailed diagnosis and procedure data [Table 12]. *Vital Health Stat 13.* December 2007;(165):1-209.
2. Lloyd-Jones D, Adams R, Carnethon M, et al; American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Heart disease and stroke statistics–2009 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. *Circulation.* 2009;119 (3):480-486.
3. Packer M, Fowler MB, Roecker EB, et al; Carvedilol Prospective Randomized Cumulative Survival (COPERNICUS) Study Group. Effect of carvedilol on the morbidity of patients with severe chronic heart failure: results of the carvedilol prospective randomized cumulative survival (COPERNICUS) study. *Circulation.* 2002;106(17):2194-2199.
4. Jhund PS, Macintyre K, Simpson CR, et al. Long-term trends in first hospitalization for heart failure and subsequent survival between 1986 and 2003: a population study of 5.1 million people. *Circulation.* 2009;119(4):515-523.
5. Putnam W, Burge FI, Lawson B, et al. Evidence-based cardiovascular care in the community: a population-based cross-sectional study. *BMC Fam Pract.* April 1, 2004;5:6.
6. Gheorghiade M, Gattis WA, O'Connor CM. Treatment gaps in the pharmacologic management of heart failure. *Rev Cardiovasc Med.* 2002;3(suppl 3): S11-S19.

Coffee Intake and Glucose Homeostasis: Is There a Role for Body Iron?

Since the original report by van Dam et al,[1] coffee drinking has been associated with a decreased risk of type 2 diabetes mellitus in a number of epidemiological studies. However, body iron has for over a century been known to cause diabetes if in overt excess, manifested as the "bronzed diabetes"–hereditary hemochromatosis. We hypothesize in line with Mascitelli et al[2] in their letter to the editor regarding a study by Pereira et al[3] that the protective effect that coffee shows toward type 2 diabetes mellitus is perhaps, at least partially, explained by the iron absorption inhibitory effect of coffee. If this were so, subjects who consume much coffee should have both lower body iron stores and better glucose homeostasis compared with people who drink less or no coffee.

Methods. We looked at the association of coffee consumption with body iron and glucose homeostasis in 2682 men, aged 42 to 60 years, in the Kuopio Ischaemic Heart Disease Risk Factor Study (KIHD) in eastern Finland. The KIHD study has been approved by the joint research ethics committee of the University of Kuopio and Kuopio University Hospital. Dietary intake of foodstuffs was estimated by a 4-day food record,[4] body iron was assessed as serum ferritin concentration, and glucose homeostasis was studied by the updated homeostasis model assessment (HOMA2) insulin resistance (IR) and pancreatic β-cell function (%β). The steady-state nonlinear HOMA2 models the interplay between hepatic glucose output, body glucose uptake, and insulin secretion and produces computational IR and %β parameters.[5] The model sets the normal IR to 1.0 and the normal %β to 100. Ferritin, glucose, and insulin measurements were carried out in fasting state samples, as previously described.[6]

Results. The mean (SD) values for the study subjects was 53.1 (5.1) years for age; 566 (297) mL/d for coffee intake; 168 (152) µg/L for serum ferritin concentration; 1.51 (0.89) for IR; and 112% (39%) for %β.

In an unadjusted linear regression model, IR decreased 2.1% per 100-mL increase in coffee intake and %β decreased 0.8%. Adjustment for ferritin level attenuated the association of coffee intake with IR from −0.021 (*P* < .001) to −0.009 (*P* = .03) (difference, −54%) and the association of coffee intake with %β from −0.843 (*P* = .001) to −0.706 (*P* = .008) (difference, −16%).

In the multivariate-adjusted linear regression models, serum ferritin adjustment weakened the age- and body mass index (BMI)-adjusted association of coffee intake with IR markedly (**Table**, model 2). Multivariate analyses of coffee intake and %β were very resistant to adjustments. Furthermore, serum ferritin concentra-

Table. Multivariate-Adjusted Linear Regression of Coffee Intake With HOMA2 IR and %β

Model	HOMA2 IR, Regression Coefficient (95% CI)	P Value	HOMA2 %β, Regression Coefficient (95% CI)	P Value
Model 1				
Coffee intake (per 100 mL/d)	−0.010 (−0.020 to <0.000)	.04	−0.579 (−1.073 to −0.081)	.02
Age, y	0.005 (−0.001 to 0.010)	.11	−0.045 (−0.333 to 0.244)	.76
BMI	0.140 (0.132 to 0.148)	<.001	3.42 (2.30 to 3.83)	<.001
Model 2				
Coffee intake (per 100 mL/d)	−0.004 (−0.014 to 0.006)	.43	−0.580 (−1.079 to −0.081)	.02
Age, y	0.008 (0.002 to 0.013)	.007	−0.045 (−0.336 to 0.245)	.76
BMI	0.130 (0.122 to 0.138)	<.001	3.42 (2.99 to 3.85)	<.001
Serum ferritin concentration (per 10 µg/L)	0.010 (0.008 to 0.012)	<.001	−0.002 (−0.103 to 0.099)	.94

Abbreviations: %β, pancreatic β-cell function; BMI, body mass index; IR, insulin resistance; HOMA2, updated homeostasis model assessment.

tion was not associated with %β in these models, probably because adding BMI to our statisical model made the association between iron and B-cell function disappear (regression coefficient, −0.002 [P=.96] with BMI [Table, model 2] vs 0.200 [P<.001] without BMI [model not shown]). Further adjustment for other dietary factors that relate to iron balance, such as intakes of red meats, vegetables, and milk, and for total energy intake did not confound the observed associations significantly (data not shown).

Comment. These results suggest that coffee consumption may be associated with both body iron stores and glucose homeostasis as measured by HOMA2 IR and HOMA2 %β. Furthermore, the results suggest that the association of coffee intake with IR may be partially explained by a decrease in body iron level.

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1. van Dam RM, Feskens EJ. Coffee consumption and risk of type 2 diabetes mellitus. *Lancet.* 2002;360(9344):1477-1478.
2. Mascitelli L, Pezzetta F, Sullivan JL. Inhibition of iron absorption by coffee and the reduced risk of type 2 diabetes mellitus [letter]. *Arch Intern Med.* 2007; 167(2):204-205.
3. Pereira MA, Parker ED, Folsom AR. Coffee consumption and risk of type 2 diabetes mellitus: an 11-year prospective study of 28 812 postmenopausal women. *Arch Intern Med.* 2006;166(12):1311-1316.
4. Wallace TM, Levy JC, Matthews DR. Use and abuse of HOMA modeling. *Diabetes Care.* 2004;27(6):1487-1495.
5. Voutilainen S, Rissanen TH, Virtanen J, Lakka TA, Salonen JT; Kuopio Ischemic Heart Disease Risk Factor Study. Low dietary folate intake is associated with an excess incidence of acute coronary events: the Kuopio Ischemic Heart Disease Risk Factor Study. *Circulation.* 2001;103(22):2674-2680.
6. Laaksonen DE, Niskanen L, Punnonen K, et al. Sex hormones, inflammation and the metabolic syndrome: a population-based study. *Eur J Endocrinol.* 2003; 149(6):601-608.

Predictors of Flight Diversions and Deaths for In-flight Medical Emergencies in Commercial Aviation

In-flight medical emergencies are clinically challenging and may result in aircraft diversions, which are costly and inconvenient, or in-flight deaths. We provide an updated description of in-flight medical emergencies and systematically examine predictors of medical flight diversions and in-flight deaths.

Methods. This is a retrospective cohort study of consecutive in-flight emergencies of a large commercial airline based in Hong Kong. Between December 2003 and November 2008, all passengers with in-flight medical problems for whom emergency medical advice was sought by satellite telephone were studied. For incidence-per-flown-mile calculations, only paid trips were included. Variables associated with diversion and deaths were identified using univariate and multivariate logistic regression analyses. Ethical approval for the study was granted by the local institutional review board.

Results. There were 4068 medical emergencies, with 46 diversions and 30 deaths in the 5-year period, giving an incidence of 11.63 medical emergencies, 0.13 diversions, and 0.09 deaths per billion revenue passenger kilometers. Cardiac events accounted for 6.1% of all emergencies but resulted in 23.9% of the diversions and 66.7% of all in-flight deaths. An automated external defibrillator (AED) was used 23 times (0.6% of all events), but only 1 shock was delivered in the 5-year period. The AED was deployed in the management of 63.3% of cases that led to in-flight death.

Increasing age (**Figure**), altered mental status, and AED use were significant risk factors for diversions and