Lack of Improvement in Outpatient Management of Congestive Heart Failure in the United States

Congestive heart failure (CHF) accounts for 3% of admissions to US hospitals, and the diagnosis carries a mortality rate of 20% at 1 year and 80% at 8 years.1,2 Numerous advances in the chronic medical management of CHF, including angiotensin antagonists, β-blockers, and aldosterone antagonists, have significantly reduced mortality in clinical trial populations with varying degrees of CHF severity.3,4 Practices proven in clinical trials, however, do not always readily translate to community practice.5,6 Previous studies in outpatient populations in the late 1990s through the early 2000s observed suboptimal adoption of evidence-based therapy for CHF. Using nationally representative data, we evaluated whether patterns of medication use have improved.

Methods. We used data from the National Disease and Therapeutic Index (NDTI) physician survey produced by IMS Health (Plymouth Meeting, Pennsylvania) to characterize contemporary trends in the outpatient use of recommended medications for CHF from January 1994 through March 2009. Estimates for 2009 are made using data from January through March 2009. The NDTI is an ongoing physician survey that provides nationally representative diagnostic and medication use information on patients treated by office-based, private practice physicians in the continental United States.

We used descriptive analyses to determine the proportion of visits where the use of selected medication classes was reported. For the NDTI estimates, 95% confidence intervals (CIs) were calculated using tables of relative standard errors that accounted for the complex, multistage NDTI sampling design.

Results. The number of patient visits for CHF declined gradually over the 15-year study period, from 10.9 million nonhospital visits in 1994 to 8.5 in 2000 and to 5.7 million visits in 2008. Physician-reported degree of CHF severity for patient visits did not change appreciably over time. Angiotensin-converting enzyme inhibitor (ACEI) or angiotensin II receptor blocker (ARB) use gradually increased from 34% (95% CI, 32%-36%) in 1994 to 45% (95% CI, 43%-46%) in 2002 (Figure). However, after 2002, there was a steady decline in ACEI or ARB use, decreasing to 32% (95% CI, 27%-33%) in 2009. Because ARB use remained steady after 1998, fluctuating between 4% and 9%, the trend in ACEI and ARB use was entirely due to the rise and fall in ACEI use for CHF. We observed a gradual increase in β-blocker use for outpatient CHF visits from 11% (95% CI, 9%-12%) in 1998 to a peak of 44% (95% CI, 42%-46%) in 2006. After 2006, there was a decline in β-blocker use to 37% (95% CI, 31%-40%) in 2009.

There was a slow increase in aldosterone antagonist use in CHF from 1% in 1998 to 11% in 2003, maintaining a fluctuating plateau through 2009 (range, 8%-12%). A stable proportion of patients with CHF was reported to be receiving treatment with digoxin from 1994 to 1997 (range, 39%-43%), with a substantial decline after 1997 to 32% in 1999, to 20% in 2004, and to 10% in 2009. The use of diuretics declined slowly over 15 years from 69% in 1994 to 56% in 2009.

Comment. Initial adoption of evidence-based therapies for CHF through the 1990s and mid-2000s was modest. What we observe after the mid-2000s is more troubling. Some therapies that previously were increasing slowly have reached a plateau. Other recommended therapies have declined. The persistence of this trend could lead to a regression in the beneficial outcomes achieved through the increasing use of these therapies. The current framework used to promulgate evidence-based therapy for CHF does not appear to be sufficient to facilitate appropriate levels of therapy. Our results suggest that further improvements in the adoption process are needed, perhaps through targeting at-risk patient subgroups.
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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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 al2 in their letter to the editor regarding a study by Pereira et al3 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 the multivariate-adjusted 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 −0.843 (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-