

sessions. For each strategy, the associated compliance was estimated using survival curves at 12, 24, and 52 weeks and tested statistically using the log rank test. A Cox proportional hazards model was used to estimate the relative risk of compliance for each interval. Median time of follow up was 24 weeks for ST1 and 20 weeks for ST2. Clinical baselines characteristics were similar in both study groups. By univariate analysis, the absence of previous smoking ($p < 0.05$), and the strategy evaluated ($p < 0.001$) were the only predictors of compliance. The compliance curves were significantly different between both strategies (log rank test < 0.001). The only independent predictor of compliance was the strategy used, with the ST2 showing a significantly better compliance than ST1. Relative risk at 12 weeks RR = 2.3 [1.8, 2.9]; at 24 weeks, RR = 2.95 [2.3, 3.7] and at 52 weeks, RR = 4.25 [3.2, 5.6].

Conclusion: This study shows that the strategy with a combination of multiple interventions to increase patient commitment and involvement in a variety of activities, significantly improved the compliance to a rehabilitation program. This is of utmost importance since at least three months in a program is the minimum time required to achieve a clinical impact.

1081-74 Anti-inflammatory Effect Associated With Triglyceride Reduction

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Dislipidemia, inflammation and impaired fibrinolysis increase the risk for coronary events and death. Lipid-lowering has been found to reduce this risk. In a randomized, double-blind, cross-over study of hypertriglyceridemic men ($n = 12$, 39-64 yrs; 6 with stable ischemic heart disease), we investigated whether lipid-lowering influenced C-reactive protein (CRP) and baseline and post-exercise fibrinolysis. A morning fasting blood sample was taken pre- and post-maximal exercise (known to stimulate fibrinolysis) before and after 2-months' treatment with gemfibrozil (G: 600 mg \times 2/d) or placebo (P), for measurements of lipids, CRP, tissue-type plasminogen activator (t-PA, $\mu\text{g/L}$), plasmin-antiplasmin (PAP, $\mu\text{g/L}$) and plasminogen activator inhibitor-1 (PAI-1).

As expected, during P, patients with IHD, vs those without, had higher CRP (median 4.5 vs 1.1 mg/L, $p = 0.006$) and PAI-1 (median 86 vs 44 $\mu\text{g/L}$, $p = 0.006$). Moreover, CRP and PAI-1 were strongly related ($r = 0.84$, $p = 0.0006$). A significant, similar increase of t-PA antigen was observed in all study phases after exercise (post-) compared with before (pre-) ($p < 0.0003$). After G, baseline triglycerides (TG, mg/dL) and CRP were both significantly reduced, and PAP concentrations were increased:

	TG	CRP	PAP	t-PA pre	t-PA post
Placebo	485 \pm 82	2.4	164	12	17
Gemfibrozil	286 \pm 51	1.4	245	12	18
p	0.008	0.04	0.003	0.4	NT

[†] mean \pm SEM, other values are medians. Units are in text. NT = not tested.

Our data suggest that TG reduction in hypertriglyceridemic men has both anti-inflammatory and pro-fibrinolytic effects. This may explain, at least in part, the clinical benefits of lipid-lowering.

1081-75 Grape Juice Inhibits Human ex Vivo Platelet Aggregation While Orange and Grapefruit Juices do Not

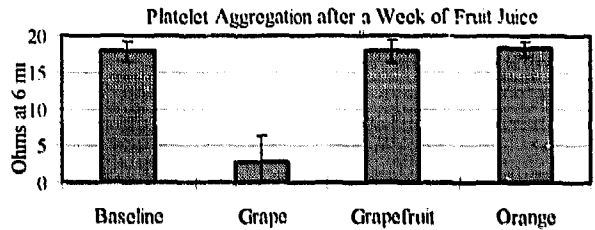
J.G. Keovil, H. Osman, N. Maalej, J.D. Folts. *Cardiology Section, University of Wisconsin, Madison WI, USA*

Background: Platelets contribute to the progression of atherosclerosis and precipitate coronary thrombosis leading to myocardial infarction (MI). Red wine and purple grape juice (GJ) contain flavonoids (Fs) with antioxidant and anti-platelet properties thought to reduce death from coronary artery disease (CAD). GJ prevents experimental thrombosis in monkey carotid and dog coronary arteries (Folts model). Orange juice (OJ) and grapefruit juice (GFJ) contain a different class of Fs than GJ (flavones vs. flavonols).

Methods: Ten healthy human subjects drank 5-7.5 ml/kg/day of GJ, OJ or GFJ daily for 7-10 days each in a three arm crossover design. Whole blood platelet aggregation (PA) studies were done at baseline and after each juice had been consumed.

Results: Drinking grape juice for one week inhibited the PA response to 1 $\mu\text{g/ml}$ of collagen by 84% \pm 20% ($p = 0.003$). OJ and GFJ had no effect on PA. Platelet inhibition was still at full effect two days after stopping GJ consumption, but was gone in seven days.

Conclusion: Drinking GJ daily for one week potently inhibited PA while OJ and GFJ had no effect. It appears that the flavonols in GJ, taken orally are more effective PA inhibitors than the flavones in OJ and GFJ. The GJ platelet inhibitory effect was stronger than a published standard for aspirin



suggesting that GJ, taken daily, may be as effective as aspirin in decreasing risk of MI.

1081-76 Health's Impact on Drinking Behavior: The Atherosclerosis Risk in Communities (ARIC) Study

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Background: It has been suggested that the putative protective effect of moderate alcohol consumption on cardiovascular disease is because of self-selection for drinking by those who are healthier.

Methods: To test how a change in health affects drinking behavior, we studied 174 black women, 252 black men, 2065 white women, and 1991 white men from the ARIC study who were drinkers at visit 1, who had no history of disease (myocardial infarction, stroke, hypertension, diabetes, cancer, or lung disease) at visit 1, and who reported drinker status and disease status approximately 3 years later at visit 2. The ethnic/gender-specific, age- and income-adjusted odds of stopping drinking were modeled by logistic regression analyses for those who developed one or more of the diseases by visit 2 in comparison to those who remained disease free.

Results: In three ethnic/gender strata, the development of disease was strongly associated with stopping drinking. The odds ratios (95% CI) were as follows: Black men, OR = 2.9 (1.46-5.69); white men, OR = 1.6 (1.08-2.37); white women OR = 1.8 (1.25-2.69); and black women, OR = 0.6 (0.21-1.68).

Conclusion: Change in awareness of disease status is associated with a change in drinking behavior and should be considered when evaluating the cross-sectional association of alcohol and disease outcomes.

1082 Cost Analysis of Coronary Revascularization

Monday, March 30, 1998, 3:00 p.m.-5:00 p.m.

Georgia World Congress Center, West Exhibit Hall Level

Presentation Hour: 4:00 p.m.-5:00 p.m.

1082-89 Total Hospital Cost for Coronary Revascularization: Hospital (UB92) and Professional (RBRVS) Components

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Total hospital cost of coronary revascularization include hospital and professional (Prof) components. Hospital cost is often determined from the hospital charges (UB92), reduced to cost by departmental cost to charge ratios. Prof costs are generally either not included or estimated from Medicare reimbursement for specific procedures, which will not cover all services provided during an episode of care (EOC). At Emory University there is centralized billing, permitting collection of all CPT codes for an EOC. CPT codes (and modifiers) may be related to Prof resource use by their relative value units (RVUs). RVUs include work and practice components which may be added to form total RVUs. RVUs for all services during an EOC are then summed and multiplied by the Medicare conversion factor to calculate Prof cost. Total cost of coronary revascularization was determined in 3,132 patients undergoing PTCA and 2,078 CABG in 1994 & 1995, in 1995 \$:

	CABG 94	CABG 95	PTCA 94	PTCA 95
Hospital Cost [†]	\$20,069	\$16,333	\$9,046	\$6,908
Work RVUs	47	42	20	17
Total RVUs	109	100	52	46
Prof Cost	\$4,358	\$3,918	\$2,071	\$1,713
Total Cost	\$24,355	\$20,116	\$11,072	\$8,601

All measures of resource use declined from 1994 to 1995, $p < 0.0001$. Ability to predict total cost from the Emory clinical database was modest unless length of stay (LOS) was included in the model: for CABG $r^2 = 0.16$