Alcohol Consumption and Ultrasonographically Assessed Carotid Artery Wall Thickness and Distensibility

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Background. Although much has been written in recent years about the relation between alcohol and atherosclerotic disease, controversy exists as to whether and how alcohol exerts an effect on atherosclerosis in different sites.

Methods and Results. We tested the hypothesis that alcohol consumption is associated inversely with carotid atherosclerosis in a population sample of 45- to 64-year-old men and women who participated in the Atherosclerosis Risk in Communities (ARIC) Study and were free of cardiovascular disease at a baseline examination in 1987 to 1989. B-mode ultrasonography was used to determine carotid artery intimal-medial wall thickness and distensibility as indices of the degree of atherosclerosis. The level of alcohol consumption in the ARIC sample was generally low. Age-adjusted mean values of alcohol consumed (grams per week) were 72.0 for white and 74.3 for nonwhite men and 24.8 for white and 11.2 for nonwhite women. After adjustments for age, artery depth, education, body mass index, sport index, cigarette-years of smoking, low-density lipoprotein cholesterol, and diabetes mellitus, there was no significant cross-sectional association of reported current alcohol intake with either carotid artery wall thickness (among white and nonwhite men and nonwhite women) or distensibility (in any of the four sex-race groups). Among white women, the adjusted mean value of carotid artery wall thickness tended to be higher in light to moderate drinkers than in never or rare drinkers, but the difference across drinking status categories was of borderline statistical significance (P = .04) and may be of little biological importance.

Conclusions. The ARIC Study found no material cross-sectional association between current alcohol intake and carotid atherosclerosis but provides an opportunity in the future to study atherosclerosis progression and incident events in relation to alcohol consumption in a large population sample of men and women. (Circulation. 1993;88:2787-2793.)

Key Words • atherosclerosis • alcohol • ultrasonography

Carotid atherosclerosis is a major contributor to thrombotic stroke. It also is correlated positively with coronary atherosclerosis. Noninvasive ultrasonographic examination of carotid atherosclerosis may therefore provide useful information about risk of both cerebrovascular and coronary events. Epidemiological studies, with few exceptions, have indicated that moderate alcohol consumption is inversely associated with clinical manifestations of coronary heart disease (CHD). It also was reported that light to moderate alcohol consumption may have some protective effect on ischemic stroke.

The mechanism by which moderate alcohol intake might reduce CHD is not fully understood. It has been suggested that moderate alcohol consumption slows coronary atherosclerosis by beneficial effects on lipid metabolism, thrombogenesis, and fibrinolytic activity. If this were so, one also might expect an inverse association between moderate alcohol intake and carotid artery atherosclerosis. The Atherosclerosis Risk in Communities (ARIC) Study provided an opportunity to test this hypothesis using ultrasonographically determined carotid artery wall thickness and distensibility as indices of the degree of atherosclerosis. As distensibility may denote incipient atherosclerotic changes, we hypothesized alcohol consumption to be negatively correlated with the degree of carotid artery wall thickness but positively correlated with carotid distensibility.

Methods

Study Population

The ARIC Study is a prospective investigation of subclinical and clinical atherosclerotic diseases in four...
US communities: Forsyth County, NC; Jackson, Miss; the northwest suburbs of Minneapolis, Minn; and Washington County, Md. A population sample of 15,800 men and women aged 45 to 64 years was selected; in Jackson, blacks were sampled exclusively. A detailed description of the study design, sampling strategy, and examination procedures has been published previously. This article is based on baseline visit data collected from 1987 through 1989.

Risk Factor Measurements

Interviews in the home and clinic included assessment of socioeconomic factors, education level, smoking status, medical history, and medication use. A clinic examination included measurements of cardiovascular disease risk factors and a B-mode ultrasound examination of selected arterial sites. Subjects were asked to fast for 12 hours before clinic examination. Blood was drawn from the antecubital vein of seated participants using minimal trauma. Serum total cholesterol and triglycerides were measured by enzymatic methods, and high-density lipoprotein (HDL) cholesterol was measured after dextran-magnesium precipitation. Low-density lipoprotein cholesterol (LDL) was calculated using the Friedewald equation. Fasting serum glucose was assessed by hexokinase/glucose-6-phosphatase dehydrogenase method. Body mass index (kg/m²) was computed using measurements of weight (to the nearest pound, then converted to kilograms) and height (to the nearest centimeter). An index for physical activity in sports (sport index), ranging from 1 (low) to 5 (high), was derived using the Baecke physical activity questionnaire. Systolic and diastolic (fifth phase) blood pressures were measured three times in the right arm of seated participants using a random-zero sphygmomanometer; the mean of the last two measurements was used in the analysis.

Alcohol consumption was ascertained in a dietary interview conducted by trained interviewers. Subjects were asked whether they currently drank alcoholic beverages, and, if not, whether they ever drank. The cohort was classified by drinking status into three categories: never drinkers, former drinkers, and current drinkers. Former drinkers were asked to recall their usual alcohol intake per week before they quit but were not asked about alcohol type. Current drinkers were asked how often they usually drank wine, beer, or hard liquor, and the amount of alcohol consumed (in grams per week) was calculated assuming that 4 oz of wine equals 10.8 g, 12 oz of beer equals 13.2 g, and 1 oz of liquor equals 15.1 g of ethanol. Current drinkers were further subclassified into five groups: light drinkers (<10 g per week), light drinkers (11 to 70 g per week), lower moderate drinkers (71 to 140 g per week), upper moderate drinkers (141 to 280 g per week), and heavy drinkers (>280 g per week).

A variable for cigarette-years of smoking was calculated by multiplying the average number of cigarettes smoked per day by the number of years of smoking. Prevalent cardiovascular disease (CVD) was defined as angina or intermittent claudication by the Rose questionnaire; a self-reported, physician-diagnosed history of a heart attack or stroke; a prevalent Q wave on ECG; or a self-reported history of cardiovascular surgery or angioplasty. Prevalent diabetes mellitus was defined as an elevated serum glucose of ≥140 mg/dL (fasting), of ≥200 mg/dL (nonfasting), and/or a history of or treatment for diabetes.

Ultrasound Examination

Carotid artery atherosclerosis was determined by high-resolution B-mode ultrasonography. Trained technicians in each field center scanned the extracranial carotid arteries bilaterally. The arteries were divided into three segments: the distal 1.0-cm portion of the common carotid artery, the carotid bifurcation, and the proximal 1.0 cm of the internal carotid artery. Thus, including both sides, six artery segments were scanned. B-mode data were recorded onto high-resolution 3/4-in. cassettes and interpreted by trained readers according to a standardized protocol. Using magnified images, readers recorded for each segment up to 11 coordinate points from each echogenic interface that identified an arterial boundary. Due to technical difficulties, the scans of the 1273 participants examined during the first 6 months of the study have not yet been read. Because the ARIC sampling design provided for examinations of periodic probability subsamples, the unread group was a random subsample of the entire ARIC cohort.

The average intimal-medial thickness of the far wall of the six carotid artery segments was determined (ie, boundaries 4-5 in Fig 1). The rationale for this measure as an indicator of early atherosclerosis has been described. Poor visualization of carotid boundaries led to missing information in part of the six segments on some scans. Where possible, values for missing boundaries were imputed from the visualized boundaries. The first step of imputation was geometric interpolation of any of the 11 coordinate points that were missing, using a cubic splining technique. No splining was done when less than three coordinate points were available. If data were still insufficient for a segment, wall thickness was imputed from sex- and race-specific multivariate linear models, using as predictors the visualized boundaries, body mass index, and artery depth (Howard G, Evans GW, Espeland M, Sharrett AR, Chambless LE, Barnes R, Riley WA Jr, Heiss G. Atherosclerosis measured by ultrasound in population studies: implications of missing data. Manuscript in preparation.). Still, 1042 of the 14 436 scans read had no usable data. Of those remaining 13 394 scans, some imputation was required for 17% of the left common carotids, 18% of the right common carotids, 39% of the left bifurcations, 38% of the right bifurcations, 58% of the left internal carotids, and 57% of the right internal carotids. For the analyses, we took the overall average of the mean intimal-medial wall thickness (measured or imputed) of the six arterial segments.

Carotid artery distensibility was also measured using B-mode ultrasonography. A measure of arterial stiffness (as an artery becomes stiffer, the distensibility decreases), is defined as the percent volume increase occurring within an arterial segment during the cardiac cycle divided by the arterial pulse pressure. During 10 consecutive cardiac cycles, arterial wall echoes from the diagnostically opposite media-adventitia interfaces of the left common carotid artery 1 cm proximal to the bifurcation were electronically tracked to provide mean values of systolic and diastolic diameters. Simultaneously, systolic and dia-
Systolic blood pressure were measured automatically in the brachial artery. Carotid artery wall distensibility was then computed by the following formula:

\[
\text{Distensibility (\%/kPa)} = \frac{100 \times [D (S)^2 - D (D)^2]}{[D (D)^2 (MSBP - MDPB)]}
\]

where \( D (S) \) and \( D (D) \) are the mean systolic and mean diastolic carotid artery diameters, respectively, and MSBP and MDPB are the mean systolic and diastolic blood pressures, respectively. The unit of measurement is percent volume increase per kilopascal (kPa), where 1 kPa = 7.6 mm Hg. Because distensibility measurements were not instituted at the onset of the ARIC baseline examination and sometimes could not be obtained on obese subjects, only 4676 participants free of CVD had usable distensibility data. The average ethanol intake of those with distensibility data present was not significantly different \((P > .05)\) from those without distensibility data in nonwhites. White men with distensibility data had a slightly lower average alcohol intake (10 g/wk) than white men with distensibility missing \((P = .006)\). A similar difference of borderline statistical significance \((P = .05)\) was found among white women.

**Statistical Analysis**

To examine the association between alcohol consumption and early carotid atherosclerosis in asymptomatic subjects, we excluded from analysis ARIC participants with CVD at baseline \((n = 2234)\). (Analyses including participants with CVD were virtually identical.) Alcohol use was described by computing sex- and race-specific means and standard deviations or as percentage distributions. Alcohol stratum-specific adjusted average wall thickness and distensibility values were computed using sex- and race-specific ANCOVA. Covariates for adjustment included age, artery depth, cigarette-years of smoking, body mass index, sport index, and LDL cholesterol as continuous variables and diabetes mellitus and education level modeled as dummy variables. HDL cholesterol and blood pressure were not included because they may be mediators of alcohol effects. Associations were examined by using the SAS statistical package.35

**Results**

A total of 3630 white men, 4374 white women, 975 nonwhite men, and 1599 nonwhite women were free of CVD and had usable B-mode ultrasound data. As Fig 2 shows, the highest proportion of current drinkers was found among white men \((71\%)\); the next highest was among white women \((63\%)\) followed by nonwhite men \((50\%)\) and nonwhite women \((22\%)\). Age-adjusted mean values of ethanol consumed (grams per week) in the four race-sex groups were 72.0 for white and 74.3 for nonwhite men and 24.8 for white and 11.2 for nonwhite women.

The level of drinking among current drinkers in the ARIC Study was generally low (Fig 3). Among women drinkers, 80% of whites and 73% of nonwhites were in the category of rare to light drinking (up to 70 g of ethanol per week, that is, up to one drink per day). Less than 1% of white women \((n = 27)\) and 2% of nonwhite women \((n = 9)\) were heavy drinkers \(( \geq 280 \text{ g of ethanol per week})\). About 56% of white men were rare to light
drinkers, 35% were moderate drinkers, and about 9% were heavy drinkers. Nonwhite men had the greatest proportion of heavy drinkers (15%); still, about 44% of nonwhite men were rare to light drinkers and 41% of them were moderate drinkers.

Table 1 shows sex- and race-specific mean values of carotid artery wall thickness (in millimeters) by drinking categories. Adjustments were made for age, artery depth, education, body mass index, sport index, cigarette-years of smoking, LDL cholesterol, and diabetes mellitus (associations adjusted for age only showed a similar pattern). White men who were heavy drinkers had the greatest average intimal-medial wall thickness of 0.80 mm. No statistically significant difference was found in the adjusted mean values of carotid artery wall thickness across drinking status categories in white and nonwhite men and in nonwhite women. Among white women, the adjusted mean value of carotid wall thickness tended to be higher in light to moderate drinkers than in never or rare drinkers, but the difference in means across drinking status categories was of borderline statistical significance ($P = 0.04$).

Table 2 shows that among white men, the adjusted mean value of carotid artery wall distensibility tended to be lower in current drinkers than in never drinkers or former drinkers. However, no consistent pattern was discernable in other sex-race groups. The difference in adjusted mean values of carotid artery wall distensibility across drinking status categories was not statistically significant in any sex-race group.

The models shown in Tables 1 and 2 were repeated after excluding the LDL cholesterol variable, and results were virtually identical. The models were run again with the addition of systolic blood pressure; the impact was inconsistent across race-sex groups, and still none of the resulting associations between wall thickness and alcohol intake was statistically significant ($P > 0.05$).

**Discussion**

For the most part, moderate alcohol intake has been associated with lower incidence of CHD, and with lower risk of ischemic stroke. Few previous studies have examined the relation between alcohol consumption and carotid atherosclerosis. In a population-based cross-sectional study of 412 Finnish men aged 42 to 60 years, Salonen et al. found that usual alcohol consumption was inversely and significantly related to the severity of carotid atherosclerosis as assessed by high-resolution B-mode ultrasonography. Another report from the same study, however, showed that in a 2-year follow-up period, alcohol consumption was not significantly associated with the progression of carotid atherosclerosis as assessed by a change in intimal-medial thickening. In a cross-sectional study of 261 white men and women older than 50 years who had symptoms of cerebrovascular disease, Bogousslavsky et al. showed that light to moderate consumption of alcohol was inversely associated with extracranial carotid atherosclerosis as assessed by B-mode ultrasonography.

In the ARIC study of 45- to 64-year-old subjects free of clinical CVD, we found no significant cross-sectional association between current alcohol consumption and carotid atherosclerosis as determined by intimal-medial wall thickness or wall distensibility. The only finding that was at all statistically significant ($P = 0.04$) was a positive association between current alcohol consumption and carotid artery wall thickness in white women. However, the estimated increase in carotid artery intimal-medial thickness associated with moderate drinking may be of little biological importance. Excluding LDL cholesterol from the multivariate model did not affect these associations.

The advent of B-mode ultrasound measurements of carotid wall thickness and distensibility have made it possible to replace clinical manifestations of atherosclerosis with noninvasive end points and thereby better quantify the associations of various risk factors with atherosclerosis. The validity of the ultrasound method has been described by Pignoli et al. Previous reports by the ARIC Study and other investigators showed that carotid atherosclerosis measured by B-mode ultrasound methods is consistently related to major CVD risk factors such as age, cigarette smoking, LDL cholesterol, and, with some inconsistency, hypertension and HDL cholesterol. For example, compared with ARIC participants with wall thicknesses below the approximate 75th percentile, participants with thicknesses above the approximate 90th percentile were 3.1 times as likely to have ever smoked, 2.9 times as likely to have hypercholesterolemia, and 2.9 times more likely to have hypertension. This suggests that this method validly measures early atherosclerosis.

Because alcohol seems to be strongly associated with clinical CHD and ischemic stroke, a possible interpretation of our findings as well as those showing
TABLE 1. Sex- and Race-Specific Adjusted Values of Carotid Artery Wall Thickness (mm) by Drinking Status: The Atherosclerosis Risk in Communities Study, 1987 to 1989

<table>
<thead>
<tr>
<th></th>
<th>White</th>
<th>Nonwhite</th>
<th>White</th>
<th>Nonwhite</th>
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<tbody>
<tr>
<td></td>
<td>(n=3630)</td>
<td>(n=975)</td>
<td>(n=4374)</td>
<td>(n=1599)</td>
</tr>
<tr>
<td><strong>Men</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never drinker</td>
<td>0.77</td>
<td>0.01</td>
<td>0.77</td>
<td>0.01</td>
</tr>
<tr>
<td>Former drinker</td>
<td>0.78</td>
<td>0.01</td>
<td>0.78</td>
<td>0.01</td>
</tr>
<tr>
<td>Current drinker</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rare, ≤10 g/wk</td>
<td>0.77</td>
<td>0.01</td>
<td>0.77</td>
<td>0.02</td>
</tr>
<tr>
<td>Light, 11-70 g/wk</td>
<td>0.78</td>
<td>0.01</td>
<td>0.76</td>
<td>0.01</td>
</tr>
<tr>
<td>Lower moderate, 71-140 g/wk</td>
<td>0.77</td>
<td>0.01</td>
<td>0.76</td>
<td>0.02</td>
</tr>
<tr>
<td>Upper moderate, 141-280 g/wk</td>
<td>0.78</td>
<td>0.01</td>
<td>0.79</td>
<td>0.02</td>
</tr>
<tr>
<td>Heavy, &gt;280 g/wk</td>
<td>0.80</td>
<td>0.01</td>
<td>0.76</td>
<td>0.02</td>
</tr>
<tr>
<td><strong>P</strong></td>
<td>.33</td>
<td>.75</td>
<td>.04</td>
<td>.60</td>
</tr>
</tbody>
</table>

Values are adjusted for age, body mass index (kg/m²), artery depth, cigarette-years of smoking, low-density lipoprotein cholesterol, diabetes mellitus, education, and sport index.

*Value for overall test of difference in means across drinking status categories.


<table>
<thead>
<tr>
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<th>White</th>
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<th>White</th>
<th>Nonwhite</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>(n=1533)</td>
<td>(n=325)</td>
<td>(n=1980)</td>
<td>(n=540)</td>
</tr>
<tr>
<td><strong>Men</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never drinker</td>
<td>1.75</td>
<td>0.05</td>
<td>1.59</td>
<td>0.07</td>
</tr>
<tr>
<td>Former drinker</td>
<td>1.72</td>
<td>0.04</td>
<td>1.65</td>
<td>0.07</td>
</tr>
<tr>
<td>Current drinker</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rare, ≤10 g/wk</td>
<td>1.71</td>
<td>0.04</td>
<td>1.68</td>
<td>0.15</td>
</tr>
<tr>
<td>Light, 11-70 g/wk</td>
<td>1.69</td>
<td>0.03</td>
<td>1.59</td>
<td>0.08</td>
</tr>
<tr>
<td>Lower moderate, 71-140 g/wk</td>
<td>1.73</td>
<td>0.04</td>
<td>1.65</td>
<td>0.08</td>
</tr>
<tr>
<td>Upper moderate, 141-280 g/wk</td>
<td>1.69</td>
<td>0.05</td>
<td>1.37</td>
<td>0.12</td>
</tr>
<tr>
<td>Heavy, &gt;280 g/wk</td>
<td>1.65</td>
<td>0.06</td>
<td>1.50</td>
<td>0.11</td>
</tr>
<tr>
<td><strong>P</strong></td>
<td>.85</td>
<td>.46</td>
<td>.16</td>
<td>.19</td>
</tr>
</tbody>
</table>

Values are adjusted for age, body mass index (kg/m²), cigarette-years of smoking, low-density lipoprotein cholesterol, diabetes mellitus, education, and sport index.

*Value for overall test of difference in means across drinking status categories.

weak associations when coronary atherosclerosis was assessed by autopsy is that the main effect of alcohol is on the thrombotic process rather than on atherosclerosis. Indeed, alcohol increases fibrinolytic activity, suppresses platelet formation, and reduces platelet survival time. As was previously reported, alcohol consumption in the ARIC study was in fact negatively correlated with platelet count, fibrinogen, factor VII, factor VIII, von Willebrand factor, antithrombin III, and protein C.

Another possibility for our failure to find an association between alcohol and carotid atherosclerosis in ARIC is that alcohol has both negative and positive effects that counteract each other. For example, a positive association between alcohol consumption and blood pressure levels could be opposed by a positive association with HDL cholesterol. The impact of blood pressure on atherosclerosis may be primarily mediated through its effect on the sclerotic component of atherosclerosis. This effect may be stronger in large vessels (carotid arteries) than in medium-sized vessels (coronary arteries) and may offset the presumed favorable effects of alcohol on HDL and the thrombotic component of atherosclerosis.

Another possible conclusion from the literature and ARIC's findings is that alcohol intake is less strongly associated with atherosclerosis of the carotid arteries than with that of the coronary arteries. Differences in the roles of various risk factors in the pathogenesis of atherosclerosis of different vessels and of different sites in a single vessel such as the carotid artery have been reported. For example, lipids are important in...
coronary atherosclerosis, but Ford et al\textsuperscript{42} reported that only 4\% of the variation in the extent of carotid bifurcation atherosclerosis could be explained by serum lipids. Another study\textsuperscript{51} showed that LDL cholesterol levels were associated positively with common carotid intimal-medial wall thickening but that cigarette smoking had a stronger relation to carotid fibrous plaques than to wall thickening. Interestingly, this latter study did not show a significant cross-sectional association between hypertension and either manifestation of atherosclerosis of the common carotid artery. Further analyses from the same study showed that neither hypertension nor total HDL or HDL\textsubscript{2} cholesterol was a significant risk factor for progression of carotid atherosclerosis (intimal-medial thickening) over a 2-year period.\textsuperscript{37} If hypertension and HDL cholesterol are major mediators of alcohol effects on atherosclerosis, then the findings from this study raise the question of the importance of alcohol intake in the pathogenesis of carotid atherosclerosis.

In assessing the relation between alcohol consumption and carotid atherosclerosis, several methodological issues should be addressed. Low statistical power is an unlikely explanation for our null findings because the ARIC sample size was large, except perhaps for heavy drinkers. Questionnaire measurements of alcohol intake in epidemiological studies have been criticized because of underreporting, especially by heavy drinkers, resulting in misclassification and possibly recall bias. Although reported alcohol intake has not been validated in the ARIC Study, another population-based study\textsuperscript{52} showed a high correlation between reported frequency of drinking and the actual amount of alcohol consumed. Moreover, alcohol consumption (grams per week) in the ARIC Study was correlated with other risk factors in a manner consistent with other studies. For example, alcohol intake was positively and significantly correlated with cigarette smoking, total HDL cholesterol, and HDL\textsubscript{2} cholesterol and negatively correlated with LDL cholesterol (data not shown). This suggests that ARIC’s measurement of alcohol intake was reasonably valid.

The level of alcohol consumption in the ARIC Study was defined by usual intake of alcohol. The pattern of drinking, especially excess drinking (drunkenness), may also contribute to the role of alcohol in atherosclerosis. For example, in one study,\textsuperscript{53} blood pressure was related to both the frequency of drinking and a tendency to drink to excess, whereas serum cholesterol and hematocrit were related chiefly to frequency of drinking. The specific association of excess drinking with carotid atherosclerosis has not been assessed in the ARIC Study.

Conclusions

Although much has been written in recent years about the relation between alcohol and atherosclerosis, controversy still exists as to whether and how alcohol exerts an effect on atherosclerosis of different sites or its clinical manifestations. The ARIC Study found no material association but provides an opportunity in the future to study atherosclerosis progression and incident events in relation to alcohol consumption in a large population sample of men and women.

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Appendix

Forbush County Field Center: Cohort: Jeannette Bensen, MSc; Catherine Paton, Amy Haire; Delilah Posey. Surveillance: Catherine Burke, Deanna Horwitz, Carol Summers, Carmen Woody.

Jackson Field Center: Cohort: Royanne Barry, Faye Blackburn, Rajam Radhakrishman, Seshadri Raju, MD. Surveillance: David Conwill, MD, MPH; Connie Myers, Robert Watson, DVM, PLD; Nancy Wilson.

Minneapolis Field Center: Cohort: Marilyn Bowers, Bryna Lester, Gail Murton, Virginia Wyum. Surveillance: Richard Crow, MD; Janet Jeremiasion, RN; Nancy MacLennan, RN; Gina Tritel, RN.

Washington County Field Center: Carol Christman, Sonny Harrell, Joel Hill, Joan Nelling.

Central Hemostasis Laboratory: Valerie Stinson, Pam Pile, Hoang Pham, Teri Trevino.


References


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