Implications of a New Definition of Vitamin D Deficiency in a Multiracial US Adolescent Population: The National Health and Nutrition Examination Survey III

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Implications of a New Definition of Vitamin D Deficiency in a Multiracial US Adolescent Population: The National Health and Nutrition Examination Survey III

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What's Known on This Subject
Vitamin D is necessary for bone mineralization and the prevention of rickets. In adults, vitamin D deficiency has been linked to many chronic diseases. The prevalence of deficiency is estimated to range from 15% to 80% depending on the population and how deficiency is defined.

What This Study Adds
Using a large nationally representative data set, we estimated the prevalence of vitamin D deficiency in adolescents. In addition, we estimated the risk of deficiency by race/ethnicity, gender, age, and weight after adjustment for sociodemographic and regional factors.

ABSTRACT

OBJECTIVE. In children, vitamin D deficiency can interfere with bone mineralization, leading to rickets. In adults, it is linked to cardiovascular disease, insulin resistance, and hypertension. Accurate estimates of the prevalence of vitamin D deficiency are complicated by the lack of consensus as to optimal vitamin D status. Currently, individuals with serum 25-hydroxyvitamin D levels of <11 ng/mL are classified as vitamin D deficient. Experts collectively have proposed that minimum levels be at least 20 ng/mL. Our objectives were to (1) determine the national prevalence of vitamin D deficiency in adolescents by using both the current and recommended cutoffs and (2) examine the implications of the new recommendation after adjustment for various factors.

METHODS. Data were obtained from National Health and Nutrition Examination Survey III, a cross-sectional survey administered to a nationally representative sample of noninstitutionalized civilians aged 2 months and older. Analyses were restricted to 2955 participants aged 12 to 19 with serum 25-hydroxyvitamin D levels. Relationships between serum 25-hydroxyvitamin D levels and sociodemographic variables were evaluated by using logistic regression.

RESULTS. Changing the definition of vitamin D deficiency from <11 to <20 ng/mL increased the prevalence from 2% to 14%. After adjustment for all covariates, non-Hispanic black adolescents had 20 times the risk of serum 25-hydroxyvitamin D <20 ng/mL compared with non-Hispanic white adolescents. The risk of deficiency was more than double for females compared with males. An inverse relationship between weight and serum 25-hydroxyvitamin D levels was found. Overweight adolescents had increased risk of deficiency compared with normal-weight adolescents.

CONCLUSIONS. There was a disproportionate burden of vitamin D deficiency in the non-Hispanic black adolescent population. Routine supplementation and monitoring of serum levels should be considered. Females and overweight adolescents are at increased risk. The consequences of chronic vitamin D deficiency in adolescents should be prospectively investigated.

VITAMIN D IS known to be necessary for bone metabolism. However, there are few dietary sources of vitamin D available to meet the daily recommended requirement.1 In addition, many individual and environmental factors interfere with the ability to have sufficient sun exposure to produce vitamin D endogenously.2 Over the past 4 decades, increasing scientific evidence has linked vitamin D deficiency to many chronic diseases including hypertension, immune dysfunction, cancer, diabetes, and cardiovascular disease.3,4 In adolescents, these relationships have only begun to be explored. It is reasonable to infer that because many of the potential consequences of vitamin D deficiency develop over time, prevention should begin in childhood.
The prevalence of vitamin D deficiency has been reported to range from 15% to 80%. Generally, the prevalence varies in different populations but is also a function of how deficiency is defined. Accurate estimates are complicated by the lack of consensus as to optimal vitamin D status as determined by plasma 25-hydroxyvitamin D (25(OH)D) concentration. Currently, individuals with serum levels of <11 ng/mL are classified as being vitamin D deficient. However, there is evidence that biochemical and skeletal sequelae of vitamin D deficiency may actually manifest at a higher cutoff of 30 to 35 ng/mL.

Because the sequelae of vitamin D deficiency occur at a higher level than the current standards, many experts have proposed that minimum acceptable serum levels be increased to at least 20 ng/mL. The purpose of this study is to (1) determine the national prevalence of vitamin D deficiency in adolescents by using both current and recommended cutoffs and (2) identify sociodemographic and geographic risk factors that are associated with vitamin D deficiency as defined by the new recommendation.

**METHODS**

**Survey and Sample**

The data used were from the Third National Health and Nutrition Examination Survey (NHANES III), 1988–1994, which is a periodic survey conducted by the National Center for Health Statistics of the Centers for Disease Control and Prevention (CDC). NHANES III contains data on 33,994 individuals and was administered to a representative sample of noninstitutionalized civilians aged 2 months and older who resided in the United States. The NHANES III data set oversampled persons who were >60 years of age, black, or Mexican American and those who had low income.

Laboratory data from an initial cohort of 3441 participants aged 12 to 19 years were analyzed. Participants with missing serum levels of 25(OH)D \( (n = 486) \) or missing data for the additional variables were excluded. A final sample of 2629 with complete information was available for multiple logistic regression analysis. The protocol was approved by the Weill Cornell Medical College institutional review board.

**Study Variables**

All variables included in the analysis were based on the NHANES III survey design and are described briefly below.

Age was calculated by using the birth date obtained from the screener questionnaire. Race/ethnicity was based on self-reported race and ethnicity. The 4 categories were non-Hispanic white, non-Hispanic black, Mexican American, and other.

Weight was divided into 3 categories: normal, at risk for overweight, and overweight. To place the subject in the appropriate category, BMI was calculated by using weight in kilograms divided by the square of height in meters. Then, per CDC guidelines, BMI percentiles were determined by using age- and gender-specific growth charts. These percentiles were used to characterize a child’s nutritional status as follows: BMI < 85th percentile was normal, BMI between the 85th and 95th percentiles was at risk for overweight, and BMI ≥ 95th percentile was overweight.

Vitamin D was based on serum 25(OH)D levels that were determined by using the INCSTAR 25(OH)D assay (INCSTAR Corp, Stillwater, MI). A detailed description of this technique is found in the NHANES laboratory procedure manual. Because 25(OH)D is the predominant circulating form of vitamin D, it is the primary indicator of an individual’s vitamin D status. Per current standards, vitamin D deficiency was defined as a serum 25(OH)D level of <11 ng/mL (27.5 nmol/L). For comparison, a serum 25(OH)D level of <20 ng/mL (50 nmol/L) was used, as recommended by the 13th Workshop Consensus for Vitamin D Nutritional Guidelines, held in British Columbia, Canada (April 2007).

Education level was defined by using the highest level of education attained by the family reference person designated during the screener interview as the person ≥17 years of age who owned or rented the dwelling unit. If no one owned or rented the unit, the family reference person was the first family member mentioned by the respondent who was 17 years of age. Education was categorized as 0 to 8 years, 9 to 11 years, high school graduate, and college (>13 years).

Income level was defined as low, middle, and high on the basis of the acceptable eligibility cut points of the poverty income ratio (PIR) for the US Department of Agriculture (USDA) food assistance programs. PIR was computed as a ratio of 2 components. The numerator was the midpoint of the observed family income category in the family questionnaire. The denominator was the poverty threshold, which varies by the calendar year in which the family was interviewed and the size of the family. Poverty threshold values (in dollars) are produced annually by the Census Bureau and are adjusted for inflation between calendar years. The PIR allows income data to be comparable across the years of the survey. Low, middle, and high income categories were defined as a PIR of 0 to 1.850, 1.851 to 3.50, and ≥3.501, respectively. The upper limit of the low-income category was defined as 185% of the federal poverty guidelines as the USDA.

Urban classification was based on the 1993 USDA rural-urban codes developed by Butler and Beale. These codes describe metropolitan and nonmetropolitan counties by degree of urbanization and nearness to metropolitan areas. The USDA codes were recoded into 2 categories: urban and rural. Urban refers to counties and neighboring counties of metropolitan areas with a population of ≥1 million.

Regional classification was defined by the Census Bureau and refers to the 4 geographic regions of the United States. The regions are defined as Northeast, Midwest, South, and West. As a limitation of the NHANES III study design, not all states were included in the sample. Therefore, regional estimates may not be entirely representative for a given region.
**Statistical Methods**

Statistical analysis was performed by using SAS 9.1 software (SAS Institute, Inc, Cary, NC). To make valid inferences accounting for the NHANES III complex survey design, sampling weights were incorporated to produce population estimates.\(^\text{24}\) SAS proc survey procedures, which included weight, cluster, and strata statements, were used to address this issue.\(^\text{25-26}\) Mean serum levels of 25(OH)D were computed and compared between groups by using the 2-tailed \(t\) test or analysis of variance where appropriate. The prevalence of vitamin D deficiency was determined by using two definitions: the current standard of <11 ng/mL and the new recommendation of <20. Analysis was performed by using simple and multiple logistic regression models using the binary outcome of vitamin D deficiency as defined by the new recommendation. Multiple logistic regression models were created manually by adding each variable significantly associated with vitamin D deficiency in the univariate analysis to the model individually and assessing its effect. All \(P\) values were 2-sided, with statistical significance evaluated at the .05 level. BMI percentiles were calculated using a publicly available SAS program provided by the CDC.\(^\text{27}\)

**RESULTS**

**Participant Demographics and Mean Serum 25(OH)D Levels**

There were 2955 adolescents aged 12 to 19 in the sample, with a mean age of 15.4 years. The proportion of boys to girls was equal. Approximately 15% of the sample was non-Hispanic black, 8% was Mexican American, and 66% was non-Hispanic white. Approximately 10% of the sample was overweight (Table 1). The mean serum 25(OH)D level for the entire sample was 32.1 ng/mL. Mean serum 25(OH)D levels decreased with increasing weight and differed by gender, race/ethnicity, region, income, and education.

**Prevalence of Vitamin D Deficiency**

Changing the definition of vitamin D deficiency from <11 to <20 ng/mL resulted in an increase in the overall prevalence from 2% to 14%. The difference was most pronounced for non-Hispanic black adolescents, in whom the prevalence increased from 11% to 50%. As BMI percentile increased, the prevalence of vitamin D deficiency increased (Fig 1). Using simple linear regression (data not shown), a 1% increase in BMI percentile resulted in a 5% decrease in serum 25(OH)D level (\(P < .001\)).

**Racial and Gender Differences in Mean Serum 25(OH)D Levels**

Mean serum 25(OH)D levels according to race/ethnicity were lowest in non-Hispanic black adolescents after adjusting for all other covariates. After additional stratification according to gender, means were lower for girls than boys in every race/ethnic category. Non-Hispanic black girls had the lowest mean serum 25(OH)D levels after adjustment for all covariates (Fig 2).

**Risk Factors Associated With Vitamin D Deficiency**

Race/ethnicity, age, weight, region of the United States, whether the area was urban or rural, income, and education were independent predictors of vitamin D deficiency regardless of the definition. Specifically, non-Hispanic black adolescents had a 20.4 times increased odds of serum 25(OH)D levels being <20 ng/mL than non-Hispanic white adolescents (95% confidence interval [CI]: 12.5–33.3). Mexican American adolescents were also at increased risk of vitamin D deficiency (odds ratio [OR]: 4.6 [95% CI: 2.8–7.4]). Adolescents with low income status had 3 times the odds of deficiency compared with those of high income status (95% CI: 1.85–

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**TABLE 1** Characteristics of Study Sample and Distribution of Serum 25(OH)D (N = 2955)

<table>
<thead>
<tr>
<th>Sample Size (% of Total Sample)</th>
<th>Mean Serum 25(OH)D, ng/mL (SE)</th>
<th>(P^b)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall</td>
<td>2955 (100)</td>
<td>32.1 (0.69)</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>1577 (49.3)</td>
<td>29.8 (0.63)</td>
</tr>
<tr>
<td>Male</td>
<td>1378 (40.7)</td>
<td>34.2 (0.91)</td>
</tr>
<tr>
<td>Race/ethnicity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-Hispanic white</td>
<td>760 (66.3)</td>
<td>36.1 (0.86)</td>
</tr>
<tr>
<td>Non-Hispanic black</td>
<td>1040 (15.2)</td>
<td>21.0 (0.55)</td>
</tr>
<tr>
<td>Mexican American</td>
<td>1005 (8.3)</td>
<td>27.2 (0.48)</td>
</tr>
<tr>
<td>Other</td>
<td>150 (10.2)</td>
<td>26.4 (1.03)</td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td></td>
</tr>
<tr>
<td>12 y</td>
<td>394 (12.4)</td>
<td>34.0 (1.08)</td>
</tr>
<tr>
<td>13 y</td>
<td>386 (12.4)</td>
<td>30.2 (0.10)</td>
</tr>
<tr>
<td>14 y</td>
<td>383 (14.3)</td>
<td>32.5 (1.15)</td>
</tr>
<tr>
<td>15 y</td>
<td>353 (11.3)</td>
<td>31.9 (1.11)</td>
</tr>
<tr>
<td>16 y</td>
<td>392 (12.9)</td>
<td>33.0 (1.42)</td>
</tr>
<tr>
<td>17 y</td>
<td>377 (13.1)</td>
<td>31.7 (1.28)</td>
</tr>
<tr>
<td>18 y</td>
<td>341 (11.6)</td>
<td>30.6 (1.11)</td>
</tr>
<tr>
<td>19 y</td>
<td>329 (11.9)</td>
<td>32.3 (1.15)</td>
</tr>
<tr>
<td>Weight</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>2069 (74.0)</td>
<td>33.0 (0.72)</td>
</tr>
<tr>
<td>At risk for overweight</td>
<td>454 (14.8)</td>
<td>31.2 (1.30)</td>
</tr>
<tr>
<td>Overweight</td>
<td>395 (10.4)</td>
<td>27.5 (0.70)</td>
</tr>
<tr>
<td>Missing</td>
<td>37 (1.2)</td>
<td>30.2 (0.96)</td>
</tr>
<tr>
<td>Region</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Northeast</td>
<td>327 (18.7)</td>
<td>33.5 (1.47)</td>
</tr>
<tr>
<td>Midwest</td>
<td>543 (23.3)</td>
<td>35.5 (1.60)</td>
</tr>
<tr>
<td>South</td>
<td>1347 (36.0)</td>
<td>29.3 (0.92)</td>
</tr>
<tr>
<td>West</td>
<td>738 (22.1)</td>
<td>31.7 (1.67)</td>
</tr>
<tr>
<td>Metropolitan area</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Urban</td>
<td>1391 (48.0)</td>
<td>30.6 (0.99)</td>
</tr>
<tr>
<td>Rural</td>
<td>1564 (52.0)</td>
<td>33.4 (0.97)</td>
</tr>
<tr>
<td>Income index</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low</td>
<td>1682 (40.6)</td>
<td>28.9 (0.65)</td>
</tr>
<tr>
<td>Medium</td>
<td>690 (31.2)</td>
<td>34.1 (0.92)</td>
</tr>
<tr>
<td>High</td>
<td>297 (22.3)</td>
<td>35.3 (1.79)</td>
</tr>
<tr>
<td>Missing</td>
<td>286 (7.9)</td>
<td>26.9 (1.07)</td>
</tr>
<tr>
<td>Education</td>
<td></td>
<td></td>
</tr>
<tr>
<td>≤8th grade</td>
<td>678 (11.6)</td>
<td>29.1 (0.92)</td>
</tr>
<tr>
<td>9th–11th grade</td>
<td>557 (11.4)</td>
<td>28.7 (1.02)</td>
</tr>
<tr>
<td>High school graduate</td>
<td>941 (33.9)</td>
<td>32.0 (0.89)</td>
</tr>
<tr>
<td>College</td>
<td>768 (40.3)</td>
<td>34.1 (0.92)</td>
</tr>
<tr>
<td>Missing</td>
<td>1 (0.09)</td>
<td>26.4 (1.03)</td>
</tr>
</tbody>
</table>

\(a\) Weighted proportion.
\(b\) Missing data were excluded for \(P\)-value computation in relevant univariate analysis.
4.85). Girls had more than twice the odds of deficiency compared with boys (95% CI: 1.68–3.07). Overweight adolescents had a 75% increase in odds of deficiency compared with normal-weight adolescents (95% CI: 1.28–2.38) (Table 2).

In the multiple logistic regression model, race/ethnicity, gender, age, weight, and education remained statistically significant predictors of vitamin D deficiency after adjustment for all covariates. The risk of vitamin D deficiency was more pronounced for girls and overweight adolescents after full adjustment. Girls were more likely to be vitamin D deficient compared with boys (OR: 2.73 [95% CI: 1.88–3.95]). Overweight adolescents had almost twice the odds of deficiency compared with normal-weight adolescents (OR: 1.97 [95% CI: 1.26–3.08]).

DISCUSSION

The major strength of this study is that data from a large, nationally representative sample were analyzed to arrive at our conclusions. Serum vitamin D data had not yet been released in the more recent NHANES data sets, ne-
cessitating the use of data from NHANES III. We identified only 1 study, which was conducted by Looker et al., which used NHANES III data to evaluate the relationship between season and serum vitamin D levels. This study did not specifically quantify the national prevalence of vitamin D deficiency in the adolescent population, and it did not control for any potential confounders.

On the basis of current data from smaller studies, together with increasing trends of insufficient dietary vitamin D intake, we can speculate that the national prevalence of vitamin D deficiency is probably much higher than that reported here. Approximately 50% to 60% of children receive the recommended USDA intake of 200 IU of vitamin D daily via diet and/or supplementation. There is evidence that the recommended daily intake would need to be doubled to achieve a minimum serum 25(OH)D level of 20 ng/mL. Using a cutoff of 20 ng/mL, we estimated that the national prevalence of vitamin D deficiency was 14%, which differs from that in other published studies. The study performed by Gordon et al., found that 42% of adolescents in their sample had serum 25(OH)D levels of <20 ng/mL. Using the same criteria, Harkness and Cromer found that the prevalence of deficiency was 54% in adolescent girls. Both cross-sectional studies were performed in urban settings and may have a higher prevalence of deficiency than reported here because minority groups were overrepresented. If we had chosen a cutoff of 30 ng/mL to define vitamin D deficiency, the level above, which has been suggested as optimal, an alarming 48% of the adolescent population would be deficient. This underscores the public-health significance of this issue.

The most significant finding in this study was the substantial difference in the prevalence of vitamin D deficiency according to race/ethnic group, which has also been reported in other studies. However, our model controls for various sociodemographic, economic, and geographic factors to better characterize the relationship between vitamin D deficiency and race/ethnicity. Because our study had a cross-sectional design, association, not causality, can be demonstrated. Our results confirm that there is a disproportionate burden of vitamin D deficiency in the non-Hispanic black adolescent population. We included the “other” race/ethnic category in our analysis to estimate the risk of vitamin D deficiency in individuals of heterogeneous ancestry or from nontraditional categories. This population represents 10% of the overall sample with a 21% prevalence of vitamin D deficiency.

We found an inverse relationship between BMI percentile and serum 25(OH)D, which has been reported in adults. In the pediatric population, BMI alone is insufficient for comparing individuals and can be misleading. BMI is expected to increase with age as part of normal growth. The BMI percentile, which is based on age and gender, is more useful in standardizing the interpretation of BMI in children. For example, a 12-year-old boy with a BMI of 24 is considered at risk for overweight, whereas a 16-year-old boy with the same BMI would be considered normal weight. Because vitamin D is fat soluble, its deficiency may be magnified in obesity because of its sequestration in body fat, decreasing its bioavailability. This group may require oral vitamin D doses, which approach the upper limit of 2000 IU/day. Longitudinal studies are needed to establish dosing parameters that consider the influence of BMI. In addition, obesity is a known risk factor for many chronic diseases that have also been linked to vitamin D deficiency; therefore, our results are of clinical importance. The attributable risk of superimposed chronic vitamin D deficiency in overweight adolescents with regard to the development of cardiovascular disease, metabolic syndrome, and impaired glucose tolerance should be prospectively explored.

Although literature demonstrates that intake of vitamin D decreases with increasing age, we found only a marginal increase in risk of vitamin D deficiency with increasing age. It is possible that we were unable to demonstrate a stronger association between age and vitamin D deficiency in this study because it was limited to a specific age group.

Several studies have investigated vitamin D deficiency in inner-city adolescent populations, but to our knowledge none have assessed the influence of an urban environment compared with a rural environment. Adolescents living in an urban environment may be predisposed to vitamin D deficiency, because the dense concentration of buildings in these areas may physically interfere with sunlight exposure. However, this variable was of marginal significance after adjustment for other covariates in the model (OR: 1.58 [95% CI: 0.99–2.52]; P = .057).

Both season and latitude are known to affect cutaneous production of vitamin D. Generally, vitamin D deficiency is more prevalent in the winter than in summer and in northern latitudes compared with southern. In NHANES III, data were collected in the summer in the northern United States and in the winter in the southern United States. Consequently, the distinct contributions of season and latitude cannot be assessed. However, regional differences do exist. Looker et al. found that the regional prevalence was lowest in the north and highest in the south regardless of the cutoff used to define vitamin D deficiency. Our findings are consistent with these results (data not shown). When we compared serum 25(OH)D levels in the north to those in the south, no statistically significant differences were found after controlling for all covariates (P = .12).

The univariate analysis initially demonstrated a positive relationship between income level and mean serum vitamin D levels. After adjustment for all variables in the logistic regression model, however, income was not an independent predictor. Approximately 7% of the sample did not report their income or income category. Consequently, nonresponse bias may have influenced our results. We found an association between race/ethnicity and the reporting of income (P < .001). Missing income information was highest for those within the other and Mexican American categories (12.5% and 12.4%, respectively) and lowest for those in the non-Hispanic white category (6.3%). Therefore, income was not missing at random. Complex imputation techniques can be
used to address this issue but were not performed because they can introduce more bias. In addition, we created an alternative model that included subjects who did not report income and found no significant difference in results.

Because serum parathyroid hormone levels were not measured in the NHANES III, the biochemical impact of vitamin D deficiency could not be assessed. Vitamin D deficiency can cause hypocalcemia and hyperparathyroidism. Hyperparathyroidism stimulates calcium conservation and increased excretion of phosphate. Although serum calcium and phosphorus levels were available, without parathyroid hormone their relationship to vitamin D cannot be accurately interpreted.

CONCLUSIONS
On the basis of our results, a substantial proportion of adolescents, particularly non-Hispanic black adolescents, may be at risk. Because numerous factors contribute to deficiency, routine supplementation should be considered along with monitoring of serum levels to document the attainment of adequate vitamin D. These measures can inform future dosing and dietary guidelines. Of particular concern is the inverse relationship between weight and serum 25(OH)D. Because vitamin D is stored in body fat, simply increasing the dosage of vitamin D may not be effective in overweight adolescents. As the prevalence of childhood obesity increases, vitamin D deficiency may increase as well. In this group, appropriate nutrition could solve both problems. Our finding that girls are at increased risk of deficiency may have additional ramifications for those who become pregnant during adolescence, which was not explored here. The prenatal nutritional status of the mother directly affects the nutritional status of the fetus and neonate. Prenatal vitamin D deficiency may increase maternal risk of preeclampsia and gestational diabetes and may be associated with reduced bone mineralization in the offspring.

In 2003, the American Academy of Pediatrics established vitamin D supplementation guidelines to ensure that children and adolescents who do not get regular sunlight exposure or ingest at least 500 mL of fortified milk daily receive 200 IU of vitamin D daily. Additional research as to physicians’ knowledge and practices with regard to vitamin D deficiency is needed to determine if this policy has been implemented effectively.

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Dr Saintonge had full access to all of the data in the study, takes responsibility for the integrity of the data and the accuracy of the data analysis, and was responsible for the whole content including conception and design, acquisition of the data, statistical analysis, and interpretation of the data, and drafting and revision of the manuscript; Dr Bang was responsible for part of the content including analysis and interpretation of the data, critical revision of the manuscript, and supervision of the first author, Dr Saintonge; Dr Gerber was responsible for part of the content including conception and design, analysis and interpretation of the data, critical revision of the manuscript, and supervision of the first author, Dr Saintonge.


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