# Factors Related to Premature Canities; A Cross-sectional Study in Lagos State, Nigeria

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***Authors' contributions***

*This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.*

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## ABSTRACT

**Background:** Canities, or graying or whitening hair, is a natural part of aging caused by reduced melanin production. Premature canities occur in humans or animals at a young age, with factors such as genetics, lifestyle, and environmental influences contributing. The study aims to evaluate the relationship between ABO Blood type, rhesus Factor, Genotype, Lifestyle, and Premature canities.
**Methods:** 259 respondents were involved and a cross-sectional descriptive study design was used to generate data. The respondents were selected using a multi-stage random sampling techniques and data collection was via descriptive questionnaire. Data obtained were analyzed using IBM SPSS version 25.
**Results:** A study of 259 participants found no significant association between premature canities and ABO blood type, rhesus, and genotype while lifestyle factors like smoking, and alcohol intake showed an association. Canities were found in various head regions, with no differences between

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sexes. Smoking and alcohol intake were more common in males. Depression was negatively associated with non-premature and premature canities. Nutrition intake was similar, but high carbohydrate and vegetable consumption was found to be statistically different.

**Conclusion:** No association between ABO blood type, rhesus factor, genotype with premature canities, and lifestyle (alcohol intake and smoking) showed possible association with premature canities.

## 1. INTRODUCTION

canities is referred to as graying or whitening of hair [1]. It is a natural part of the aging process, typically caused by a reduction in the production of melanin (the pigment solemnly responsible for the coloration of hair, skin, and eyes) [2]. As people age, their hair follicles produce less melanin, leading to a gradual loss of color and the appearance of gray or white hair [3]. However, humans or animals often exhibit canities at an early stage of life (1 – 30 years) which is termed premature canities [4]. The exact causes of premature canities are not always clear, several factors are said to contribute to and influence the occurrence [5]. These factors include genetic, lifestyle, and environmental factors.

Genetic factors resulting from an autosomal inheritance may be pathological (associated with various autoimmune disorders) or non-pathologic (such as smoking, stress, and psychological effects) contributing to the occurrence of premature canities [6]. Though genetic predisposition (autosomal inheritance) is a side influence of premature canities, on the other hand, several genes are involved in the regulation of hair pigmentation [7]. The most well-known is the melanocortin-1 receptor (MC1R) gene, which plays a role in determining hair color but variations in the MC1R gene have been linked to premature graying [8].

Blood group (ABO blood type), and rhesus factor recently have been of interest in the investigation of several diseases [9]. Several studies have been conducted to investigate the susceptibility of blood type in certain diseases. Edibamode et al., [10] reported that blood type B is more vulnerable to premature canities and blood type has a positive correlation with premature canities among the residents of Port Harcourt. Chaudhary and Mahotra, [11] have also stated that blood type O followed by B is more susceptible to premature canities. Despite the paucity of literature on the relationship between ABO blood type and canities, there are variations on the already facts among different populations. It could be attributed to environmental factors but the lifestyle of the population plays a vital role in influencing the occurrence of premature canities [12].

Ikeja is the capital city of Lagos state in southwestern Nigeria with over 861.3 thousand population. Is a place known for its exposure to industrialization and social embodiments [13]. The study explores the social life of the population to evaluate if there are associations with premature canities. Dietary consumption was also investigated. However, there is a dearth of literature on the relationship between blood type, genotype, and lifestyle with premature canities which has further motivated the interest of this study to evaluate the relationship between the blood type, rhesus factor, genotype, and lifestyle among individuals with premature canities.

## 2. MATERIALS AND METHODS

### 2.1 Study Design

A descriptive cross-sectional design was used to generate data on canities among the Ikeja, logos state population of Nigeria. A total of two hundred fifty-nine (259) respondents were involved in the study (168 males and 91 females). Premature canities were observed in the respondents within the age cut-off of 1-30 years while non-premature canities were observed within the age above 30 years. The respondents were sampled using a multi-stage random sampling to ensure that all respondents had an equal chance of being selected and the minimum sampling size was determined using the Cochran formula for a descriptive survey [14];

$$Sample \ size = \frac{Z_{1-\alpha/2}^2 \ p(1-p)}{d^2}$$

Where

$Z_{1-\alpha/2}$ = Standard normal variate (at 5% type 1 error) = 1.96

p = expected proportion of respondents (21%)

d = absolute error = 0.05

$$Sampleze = \frac{1.96^2 \times 0.21(1-021)}{0.05^2} = 254.82$$

For the study, the minimum sample size was rounded up to 259

## 2.2 Study Criteria

The study only recruited respondents with canities who had consented to be part of the study and are of the origin of Ikeja, of Lagos State, Nigeria. The study excludes respondents with no canities.

## 2.3 Method of Data Collection

For this study, data were collected using a descriptive questionnaire and a personal interview to gather information on the respondents. The questionnaire was subdivided into sections; A, B, C, and D. Section A; represents the socio-demographic data of the respondents comprising of age, sex, location, and nationality. Section B; represents the occurrence of the canities, areas, and age of the first observed canities. Section C: represents the blood group, genotype, and rhesus factors and section D represents the lifestyle which is further classified into smoking, alcoholism, depression, anemia, and nutrition. All respondents were issued a consent form to declare and participate in the study

following that the questionnaire was issued and retrieved.

## 2.4 Statistical Analysis

The data obtained for the study was subjected to statistical analysis using the International Business Machine of Statistical Package for Social Sciences (IBM SPSS version 25) and the results were presented as frequency and percentage. Chi-square was used as an inferential statistic for the study and a probability less than 0.05 was considered statistically significant (p<0.05). The confidence interval was denoted as 95%.

## 3. RESULTS

This study comprised two hundred and fifty-nine respondents (259); the males were 65% (n= 65) of the total population while the females were 35% (n= 91) (Fig. 3). All respondents in the study were observed to have canities but only 14.3% (n=37) had non-premature canities and 85.7% (222) of the total population had premature canities. 90% and 83.4% of the female and male population had premature canities and the association between canities based on sexes showed statistically insignificant (no sex differences) (Table .1).

Table 2 explores the association of canities based on blood group and it showed a majority of the premature canities were observed with blood group O, followed by blood groups A, B, and AB observed the least while among non-premature canities, blood type O was higher, followed by A, AB and the least was B. The association of the canities based on blood group showed that canities had no blood group differences (p>0.05). The genotype of the respondents was also explored to evaluate if there are associations between canities and genotype but the results from our study present that there are no genotype differences concerning canities (Table .3). In Table 4, the rhesus factors also showed no differences with premature canities (p>0.05).

### Table 1. Prevalence of canities based on gender

|  |  | Not premature canities | Premature canities | $X^2$ | p-value |
|---|---|---|---|---|---|
| sex | Female | 9 (10.0%) | 81 (90.0%) |  |  |
|  | Male | 28 (16.6%) | 141 (83.4%) | 2.07 | 0.15 |
| Total |  | 37 (14.3%) | 222 (85.7%) |  |  |

**Fig. 1. Image of premature canities of 50 years old Nigerian man whose onset commenced at age 15 - frontal view**



**Fig. 2. Image of premature canities of 50 years old Nigerian man whose onset commenced at age 15 - posterior view**
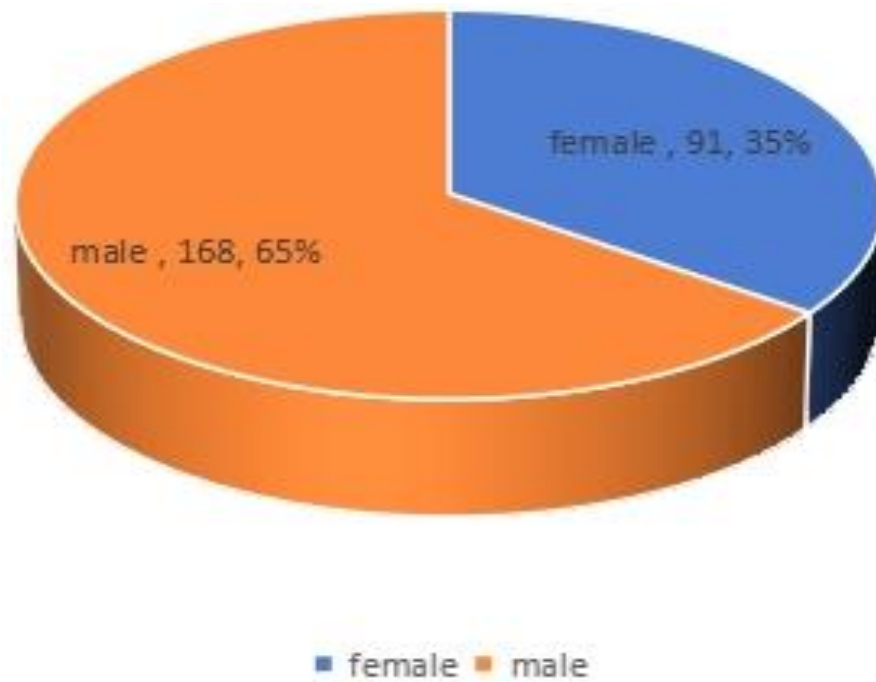
**Fig. 3. The sex of the population**

**Table 2. Distribution of canities based on blood group**

|            |    | non-premature | Premature canities | $X^2$ | p-value |
|------------|----|---------------|--------------------|-------|---------|
| Blood group | A  | 7 (13.5%)     | 45 (86.5%)         |       |         |
|            | AB | 3 (18.7%)     | 13 (81.3%)         |       |         |
|            | B  | 2 (9.1%)      | 20 (90.9%)         | 0.81  | 0.85    |
|            | O  | 25 (14.8%)    | 144 (85.2%)        |       |         |
| Total      |    | 37 (14.3%)    | 222 (85.7%)        |       |         |

**Table 3. Distribution of canities based on genotype**

|          |    | non-premature | premature canities | $X^2$ | p-value |
|----------|----|---------------|--------------------|-------|---------|
| Genotype | AA | 26 (16.6%)    | 131 (83.4%)        |       |         |
|          | AC | 0 (0.0%)      | 5 (100%)           | 2.184 | 0.34    |
|          | AS | 11 (11.3%)    | 86 (88.7%)         |       |         |
| Total    |    | 37 (14.3%)    | 222 (85.7%)        |       |         |

**Table 4. Distribution of canities based on rhesus factors**

|               |     | non-premature | premature canities | $X^2$ | p-value |
|---------------|-----|---------------|--------------------|-------|---------|
| Rhesus factor | RH- | 11 (16.7%)    | 55 (83.3%)         |       |         |
|               | RH+ | 26 (13.5%)    | 167 (86.5%)        | 0.41  | 0.52    |
| Total         |     | 37 (14.3%)    | 222 (85.7%)        |       |         |

Table 5 shows the distribution of the canities on the hairline among non-premature and premature canities between sexes. It showed that n=16 of the males and n=6 of the females of the total population of non-premature canities had canities on their hairline and the association displayed no differences (p>0.05) while premature canities were observed higher on the hairline among females compared to the males and the association shows that there is a related difference of premature canities of both males and females having canities on the hairline.

**Table 5.  The frequency distribution of canities in both sexes of the different regions of the scalp and face amongst residents of Ikeja**

| Hairline | | | Absent | Present | X² | p-value |
|---|---|---|---|---|---|---|
| non-premature | Female | | 3 (33.3%) | 6 (66.7%) | 0.27 | 0.71 |
| | Male | | 12 (42.9%) | 16 (57.1%) | | |
| premature canities | Female | | 27 (33.3%) | 54 (66.7%) | 41.92 | 0.00 |
| | Male | | 109 (77.3%) | 32 (22.7%) | | |
| Total | Female | | 30 (33.3%) | 60 (66.7%) | 35.36 | 0.00 |
| | Male | | 121 (71.6%) | 48 (28.4%) | | |
| **Parietal** | | | **Absent** | **Present** | **X2** | **p-value** |
| non-premature | Female | | 5 (55.6%) | 4 (44.4%) | 2.17 | 0.14 |
| | Male | | 8 (28.6%) | 20 (71.4%) | | |
| premature | Female | | 41(50.6%) | 40 (49.4%) | 0.03 | 0.87 |
| | Male | | 73 (51.4%) | 68 (48.6%) | | |
| Total | Female | | 46 (51.1%) | 44 (48.9%) | 0.24 | 0.63 |
| | Male | | 81 (47.9%) | 88 (52.1%) | | |
| **Temporal** | | | **Absent** | **Present** | **X2** | **p-value** |
| non-premature | Female | | 2 (22.2%) | 7 (77.8%) | 0.57 | 0.45 |
| | Male | | 10 (35.7%) | 18 (64.3%) | | |
| premature | Female | | 54 (66.7%) | 27 (33.3%) | 2.96 | 0.56 |
| | Males | | 85 (60.7%) | 55 (39.3%) | | |
| Total | Female | | 56 (62.2%) | 35 (37.8%) | 2.65 | 0.62 |
| | Male | | 95 (56.6%) | 73 (43.4%) | | |
| **Occipital** | | | **Absent** | **Present** | **X²** | **p-value** |
| Non premature | Sex | Female | 7 (77.8%) | 2 (22.2%) | 3.25 | 0.68 |
| | | Male | 12 (42.9%) | 16 (57.1%) | | |
| premature | Sex | Female | 72 (88.9%) | 9 (11.1) | 4.44 | 0.11 |
| | | Male | 109 (77.9%) | 31 (22.1%) | | |
| Total | Sex | Female | 79 (87.8%) | 11 (12.2%) | 8.66 | 0.013 |
| | | Male | 121(72.0%) | 47 (28.0%) | | |
| **Face** | | | **Absent** | **Present** | **X²** | **p-value** |
| non-premature | sex | Female | 6 (66.7%) | 3 (33.3%) | 22.80 | 0.00 |
| | | Male | 0 (0.0%) | 28 (100%) | | |
| premature | sex | Female | 73 (90.1%) | 8 (9.9 %) | 86.17 | 0.00 |
| | | Male | 36 (25.7%) | 104 (74.3%) | | |
| Total | sex | Female | 79 (87.8%) | 11 (12.2%) | 105.56 | 0.00 |
| | | Male | 36 (21.4%) | 132 (78.6%) | | |

The distribution of canities on the parietal region of the head it was observed that males had canities (non-premature and premature canities) on their parietal region compared to females. However, the association shows that there are no differences in sexes on premature canities and non-premature canities with canities on the parietal region of the head (p>0.05). Canities were also evaluated on the temporal region of the head, males were observed also to have a higher frequency of occurrence in both non-premature and premature canities compared to females, and the association showed no statistical differences in the occurrence of canities on the temporal region of the head in both non-premature and premature canities. The distribution of canities in both sexes among non-premature canities and premature canities on the occipital region of the head, the findings state that among sexes there are no differences in the distribution of canities (non-premature and premature canities) on the occipital region of the head though the distribution of canities showed significant sexual differences on the face among non-premature canities and premature canities.

The study further explores the association of canities with various lifestyles and Table 6 shows the association of canities among the respondents who smoke, the findings reviewed that non-premature canities among males and

females showed no statistical differences while premature canities showed sexual differences in the distribution of smoke where the finding show that majority of the smokers are males.

Alcoholism is a lifestyle practice among the population, the findings from this study showed that there are no sexual differences in the intake of alcohol in the non-premature canities while the respondents with premature canities display a significant association (p<0.05) on intake of alcohol between the sexes where it reports that majority of the respondents that drink alcohol are males (Table 7).

Table 8 shows the distribution of those who felt depression due to the occurrence of canities. The findings from our study have shown a negative association (p>0.05) among non-premature canities and premature canities. Anemia is the deficiency of healthy red blood cells, Table 9 shows the distribution of those with anemia on canities and it presents that a minority of the population with canities either non-premature or premature canities had anemia and the association showed no sexual differences among the non-premature canities while there were sexual differences observed among the premature canities where the higher frequency was observed in females.

**Table 6. Distribution of canities on lifestyle (Smoking)**

| | | | Don't smoke | Smoke | $X^2$ | p-value |
|---|---|---|---|---|---|---|
| Non-premature | Sex | Female | 9(100%) | 0 (0.0%) | 2.77 | 0.96 |
| | | Male | 21 (75%) | 7 (25.0%) | | |
| Premature | Sex | Female | 73 (89%) | 9 (11%) | 19.37 | 0.00 |
| | | Male | 86 (61.4%) | 54 (38.6%) | | |
| Total | Sex | Female | 82 (90.1%) | 9 (9.9%) | 20.89 | 0.00 |
| | | Male | 107 (63.7%) | 61 (36.3%) | | |

**Table 7. Distribution of canities on lifestyle (Alcohol)**

| | | | Don't take alcohol | Alcohol intake | $X^2$ | p-value |
|---|---|---|---|---|---|---|
| Non-premature | Sex | Female | 6 (66.7%) | 3 (33.3%) | 0.76 | 0.38 |
| | | Males | 14 (50.0%) | 14 (50.0%) | | |
| Premature | Sex | Female | 58 (70.7%) | 24 (29.3%) | 18.66 | 0.00 |
| | | Male | 57 (43.7%) | 83 (56.3%) | | |
| Total | Sex | Female | 64 (70.3%) | 27 (29.7%) | 18.38 | 0.00 |
| | | Male | 71 (42.3% | 97 (57.7%) | | |

**Table 8. Distribution of those who experience depression on the canities**

| | | | Don't experience depressed | Experience depressed | $X^2$ | p-value |
|---|---|---|---|---|---|---|
| Non-premature | Sex | Female | 7 (77.8%) | 2 (22.2%) | 0.31 | 0.57 |
| | | Male | 24 (85.7%) | 4 (14.3%) | | |
| Premature | Sex | Female | 63 (76.8%) | 19 (23.2%) | 0.21 | 0.27 |
| | | Male | 98 (70.0%) | 42 (30.0%) | | |
| Total | Sex | Female | 70 (76.9%) | 21 (23.1%) | 0.57 | 0.45 |
| | | Male | 122 (72.6%) | 46 (27.4%) | | |

**Table 9. Distribution of anemia on canities**

| | | | No anemia | Anemia | $X^2$ | p-value |
|---|---|---|---|---|---|---|
| Non-premature | Sex | Female | 9 (100%) | 0 (0.0%) | 0.32 | 0.56 |
| | | Male | 27 (96.4%) | 1 (3.6%) | | |
| Premature | Sex | Female | 64 (78.0%) | 18 (22.0%) | 29.90 | 0.00 |
| | | Male | 139 (99.3%) | 1 (0.7%) | | |
| Total | Sex | Female | 73 (80.2%) | 18 (19.8%) | 28.63 | 0.00 |
| | | Male | 166 (98.8%) | 2 (1.2%) | | |

**Table 10. Distribution of nutrition on canities**

| Less carbohydrate | | not less carbohydrate | Less carbohydrate | $X^2$ | p-value |
|---|---|---|---|---|---|
| Non-premature | Female | 9 (100%) | 0 (0.0%) | 1.049 | 0.31 |
| | Male | 25 (89.3%) | 3 (10.7%) | | |
| Premature | Female | 68 (62.9%) | 14 (37.1%) | 2.94 | 0.08 |
| | Male | 127 (90.7%) | 13 (9.3%) | | |
| Total | Female | 77 (84.6%) | 14 (15.4%) | 1.98 | 0.16 |
| | Male | 152 (90.5%) | 16 (9.5%) | | |
| **More carbohydrate** | | **Not more carbohydrate** | **More carbohydrate** | **$X^2$** | **p-value** |
| Non-premature | Female | 4 (44.4%) | 5 (55.6%) | 0.78 | 0.37 |
| | Male | 8 (28.6%) | 20 (71.4%) | | |
| Premature | Female | 38 (46.3%) | 44 (53.7%) | 6.62 | 0.04 |
| | Male | 41 (29.3%) | 99 (70.7%) | | |
| Total | Female | 42 (46.2%) | 49 (53.8%) | 7.51 | 0.02 |
| | Male | 49 (29.2%) | 119 (70.8%) | | |
| **More vegetable** | | **Not more vegetable** | **More vegetable** | **$X^2$** | **p-value** |
| Non-premature | Female | 7 (77.8%) | 2 (22.2%) | 0.085 | 0.77 |
| | Male | 23 (82.1%) | 5 (17.9%) | | |
| Premature | Female | 58 (70.7%) | 24 (29.3%) | 5.78 | 0.02 |
| | Male | 118 (84.3%) | 22 (15.7%) | | |
| Total | Female | 65 (71.4%) | 26 (28.6%) | 5.67 | 0.17 |
| | Male | 141 (83.9%) | 27 (16.1%) | | |
| **Less protein** | | **Not less protein** | **Less protein** | **$X^2$** | **p-value** |
| Non-premature | Female | 9 (100%) | 0 (0.0%) | 1.05 | 0.31 |
| | Male | 25 (89.3%) | 3 (10.7%) | | |
| Premature | Female | 69 (84.1%) | 13 (15.9%) | 2.74 | 0.09 |
| | Male | 128 (91.4%) | 12 (8.6%) | | |
| Total | Female | 78 (85.7%) | 13 (14.3%) | 1.76 | 0.19 |
| | Male | 153 (91.1%) | 15 (8.9%) | | |
| **More Protein** | | **Not more protein** | **More Protein** | **$X^2$** | **p-value** |
| Non-premature | Female | 6 (66.7%) | 3 (33.3%) | 0.017 | 0.89 |
| | Male | 18 (64.3%) | 10 (35.7%) | | |
| Premature | Female | 64 (78.0%) | 18 (22.0%) | 0.79 | 0.38 |
| | Male | 116 (82.9%) | 24 (17.1%) | | |
| Total | Female | 70 (76.9%) | 21 (23.1%) | 0.28 | 0.59 |
| | Male | 134 (78.8%) | 34 (21.2%) | | |
| **Less vegetable** | | **Not less vegetable** | **Less vegetable** | **$X^2$** | **p-value** |
| Non-premature | Female | 9 (100%) | 0 (0.0%) | 1.44 | 0.23 |
| | Male | 24 (85.7%) | 4 (14.3%) | | |
| Premature | Female | 72 (87.8%) | 10 (12.2%) | 0.73 | 0.39 |
| | Male | 117 (83.6%) | 23 (16.4%) | | |
| Total | Female | 81 (89.0%) | 10 (11.0%) | 1.24 | 0.26 |
| | Male | 141 (83.9%) | 27 (16.1%) | | |

The study explored the nutrition intake concerning canities and it was observed that there were no sexual differences in the intake of less carbohydrate, high carbohydrate, high vegetable, less vegetable, high protein, and less protein consumption, among the non-premature canities population while statistical differences were observed in respondents with high consumption of carbohydrate and vegetable (Table 10).

## 4. DISCUSSION

The present study evaluates the relationship between the ABO blood type, genotype, rhesus factor, and lifestyle of the Ikeja population with premature canities where non-premature canities were used as a control over the premature canities. The findings of the study present that two hundred and fifty-nine respondents had canities with 14.3% being non-premature canities while 85.7% were premature canities. Males were observed to be prone to canities more often than females though the association with sex has shown that there are no gender differences which implies that the prevalence of canities is not sexual based but rather males were observed more with both premature and non-premature canities. The finding is consistent with the previous study by Panhard et al., [15] who in their report on the African population have stated no sexual differences.

ABO blood type and rhesus factors have been studied to evaluate association and linkage to several diseases [16]. The present study explores the association of ABO blood type and rhesus factor in the population with canities. The findings show that blood type O predominates the population with non-premature canities and premature canities. However, the association has indicated no relationship with canities (premature and non-premature canities). It is also necessary to know that while blood type is not directly linked to canities, other health factors associated with blood may indirectly influence hair health. Furthermore, this has motivated the interest to further investigate the blood by evaluating the protein outside the red blood cells (rhesus factor) which in our study has shown that there are still no statistical differences between rhesus factor and canities. The findings imply that growing premature canities or not is not dependent on any blood type or rhesus factor. The findings agree with Farshchian and Hamidi, [17] that canities are not dependent on blood type or rhesus factor though our finding was contradicted by Edibamode et al., among residents in Port Harcourt, Nigeria that ABO blood type is statistically dependent on canities. However, they reported that the rhesus factor on the other hand has no association with canities.

Genetic factors play a crucial role in determining the fate of every individual regarding the occurrence of premature canities due to mutation of the melanocortin-1 receptor (MC1R) gene [18]. However, it is a point of interest that the present study evaluated the association of genotype with canities. Our findings present that the AA genotype predominated in the population and no possible association was observed ($p>0.05$) between genotype and canities. The findings suggest that genotype has no possible link to canities in a populational study but individual genetic variation leading to autosomal inheritance could be genetic dependent.

Premature canities have been said to adversely affect the appearance, self-esteem, and socio-cultural acceptance of the affected individual [19]. To this effect, it has necessitated exploring the different areas where canities are observed. The hairline, parietal, temporal, occipital, and the face were examined. The findings of this study present that males are highly observed with canities (premature and non-premature canities) in the hairline, parietal, temporal occipital, and face. The association of non-premature canities shows no related sexual differences in the different areas of the head except for the face while the association with premature canities was observed with no sexual differences in the areas except for the hairline, and face. The findings are inconsistent with previous studies [20-22].

Stress and lifestyle factors such as alcoholism and smoking are other factors said to influence the occurrence of premature canities [23] because high levels of stress and an unhealthy lifestyle can contribute to premature aging which directly will influence premature canities [24]. The study sees the association between alcohol intake and smoking in premature canities and the findings present that males are observed in alcohol intake compared to females which is also applicable to smoking. However, the association shows statistically insignificant among the non-premature canities while among the premature canities, it was revealed that it was statistically significant. These findings suggest that alcohol intake accelerates aging and various adverse effects on health by generating reactive oxygen species (ROS) leading to oxidative stress, alcohol being a diuretic, could lead to dehydration, and chronic dehydration can contribute to dry skin thereby influencing aging. Smoking also generates ROS leading to oxidative stress and smoking has been linked to the breakdown of collagen and elastin in the skin, leading to premature wrinkles and sagging directly influencing premature aging. These findings agree with previous studies [25-28].

The study investigated the nutrition intake concerning canities and discovered that there were no sexual differences in the intake of less carbohydrate, high carbohydrate, high vegetable, less vegetable, high protein, and less protein consumption among the non-premature canities population, while statistical differences were observed in respondents with high carbohydrate and vegetable consumption. The findings suggest that there is a need to improve diet consumption to build a healthy body system rather than staying unhealthy, directly and indirectly accelerating aging which is capable of influencing the occurrence of premature canities [29].

The above-discussed work shows some similarities and differences in the relationship between the ABO blood type, rhesus factor, genotype, lifestyle, and premature canities using non-premature canities as a control. The differences could be attributed to the geographical areas used.

## 5. CONCLUSION

There are no possible associations between ABO blood type, rhesus factor, genotype, and premature and non-premature canities. Alcohol intake and smoking as a lifestyle have shown an association with premature canities thereby accelerating aging and the regions of premature and non-premature canities are statistically insignificant with sexes. Nutritional status is shown as statistically significant with canities on more carbohydrate and more vegetable intake.

## CONSENT

As per international standards, written consent was issued to every respondent to declare their consent to participate in the study underlining the importance and nature of the study. The signed consent forms were retrieved and documented by the authors.

## ETHICAL APPROVAL

The study was approved by the ethical committee of the University of Port Harcourt, Nigeria.

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## COMPETING INTERESTS

Authors have declared that no competing interests exist.

## REFERENCES

1. Saxena S, Gautam RK, Gupta A, Chitkara A. Evaluation of systemic oxidative stress in patients with premature canities and correlation of severity of hair graying with the degree of redox imbalance. International Journal of Trichology. 2020; 12(1):16-23.
2. Solano F. Photoprotection and skin pigmentation: Melanin-related molecules and some other new agents obtained from natural sources. Molecules. 2020;25(7): 1537.
3. Asz-Sigall D, Ortega-Springall MF, Smith-Pliego M, Rodríguez-Lobato E, Martinez-Velasco MA, Arenas R, Vincenzi C, Tosti A. White hair in alopecia areata: clinical forms and proposed physiopathologic mechanisms. Journal of the American Academy of Dermatology. 2023;89(4): 758-63.
4. Saxena S, Gautam RK, Gupta A, Chitkara A. Evaluation of systemic oxidative stress in patients with premature canities and correlation of severity of hair graying with the degree of redox imbalance. International Journal of Trichology. 2020; 12(1):16-23.
5. Anastassakis K, Anastassakis K. The effects of aging on the hair follicle. Androgenetic Alopecia From A to Z: Basic Science, Diagnosis, Etiology, and Related Disorders. 2022:83(1)-94.
6. Pisetsky DS. Pathogenesis of autoimmune disease. Nature Reviews Nephrology. 2023;19(8):509-24.
7. Wolff H. Diseases of hair. Braun-Falco s Dermatology. 2020;1-33.
8. O'Sullivan JD, Nicu C, Picard M, Chéret J, Bedogni B, Tobin DJ, Paus R. The biology of human hair greying. Biological Reviews. 2021;96(1):107-28.
9. Abegaz SB. Human ABO blood groups and their associations with different diseases. BioMed research international. 2021;2021: 1-9.
10. Edibamode EI, Adamma SA, Obbtavwe ABO blood group linkage to early grey hair appearance in south-southerners: a cross-sectional study of port harcourt residents,

journal of Anatomical Science. 2017; 8(1):228-135

11.   Chaudhary S, Mahotra N. Early canities among undergraduate medical students of a medical college: A descriptive cross-sectional study. JNMA: Journal of the Nepal Medical Association. 2023;61 (259):220.

12.   Ungvari A, Kiss T, Gulej R, Tarantini S, Csik B, Yabluchanskiy A, Mukli P, Csiszar A, Harris ML, Ungvari Z. Irradiation-induced hair graying in mice: an experimental model to evaluate the effectiveness of interventions targeting oxidative stress, DNA damage prevention, and cellular senescence. Gero Science. 2024;1-8.

13.   Monyei CG, Adewumi AO, Obolo MO, Sajou B. Nigeria's energy poverty: Insights and implications for smart policies and framework towards a smart Nigeria electricity network. Renewable and Sustainable Energy Reviews. 2018;81:158 2-601.

14.   Asiwe N, Irozulike FC, Filima PL, Wedeh MS, Yirate BN. Pattern of earlobe attachment among igbo ethnic group of Nigeria. Asian Journal of Advanced Research and Reports. 2023;17(11):344-9.

15.   Panhard S, Lozano I, Loussouarn G. Greying of the human hair: A worldwide survey, revisiting the '50'rule of thumb. British Journal of Dermatology. 2012;167(4):865-73.

16.   Asiwe Jerome Ndudi, Umoren Elizabeth Bassey, Kolawole Tolunigba Abisola, Etim Kingsley Bassey, Agbeluyi Adedolapo Adeola, Asiwe Nicholas and Igbokwe Vincent, Does ABO blood type implicate susceptibility to respiratory abnormalities? A controlled cross-sectional study among indigenous university students in Rivers State, Nigeria, Journal of Complementary and Alternative Medical Research.2023; 23(1):35-45.

17.   Farshchian M, Hamidi A. The epidemiology of premature canities and the associated affecting factors in the students of Hamadan Medical University. Avicenna Journal of Clinical Medicine. 1997;4(1): 0.

18.   O'Sullivan JD, Nicu C, Picard M, Chéret J, Bedogni B, Tobin DJ, Paus R. The biology of human hair greying. Biological Reviews. 2021;96(1):107-28.

19.   Pandhi D, Khanna D. Premature graying of hair. Indian journal of dermatology, venereology and leprology. 2013;79: 641.

20.   Singal A, Daulatabad D, Grover C. Graying severity score: A useful tool for evaluation of premature canities. Indian Dermatology Online Journal. 2016;7(3):164-7.

21.   Sonthalia S, Priya A, Tobin DJ. Demographic characteristics and association of serum Vitamin B12, ferritin and thyroid function with premature canities in Indian patients from an urban skin clinic of North India: A retrospective analysis of 71 cases. Indian Journal of Dermatology. 2017;62(3): 304-8.

22.   Yadav D, Chander R, Mendiratta V, Debnath E, Bisherwal K, Das S. A study of micronutrient levels in premature canities in children. Indian Journal of Paediatric Dermatology. 2022;23(4): 297-301.

23.   Mahendiratta S, Sarma P, Kaur H, Kaur S, Kaur H, Bansal S, Prasad D, Prajapat M, Upadhay S, Kumar S, Kumar H. Premature graying of hair: Risk factors, co-morbid conditions, pharmacotherapy and reversal—A systematic review and meta-analysis. Dermatologic Therapy. 2020;33(6):e13990.

24.   Kaur K, Kaur R, Bala I. Therapeutics of premature hair graying: A long journey ahead. Journal of Cosmetic Dermatology. 2019;18(5):1206-14.

25.   Liguori I, Russo G, Curcio F, Bulli G, Aran L, Della-Morte D, Gargiulo G, Testa G, Cacciatore F, Bonaduce D, Abete P. Oxidative stress, aging, and diseases. Clinical Interventions in Aging. 2018;757-72.

26.   Thomas DR, Cote TR, Lawhorne L, Levenson SA, Rubenstein LZ, Smith DA, Stefanacci RG, Tangalos EG, Morley JE, Council D. Understanding clinical dehydration and its treatment. Journal of the American Medical Directors Association. 2008;9(5):292-301.

27.   Hooper L, Bunn D, Jimoh FO, Fairweather-Tait SJ. Water-loss dehydration and aging. Mechanisms of Ageing and Development. 2014;136:50-8.

28.   Raitio A. Smoking and skin: Comparison of the appearance, physical qualities, morphology, collagen synthesis and extracellular matrix turnover of skin in smokers and non-smokers. University of Oulu; 2005.

29. Kandola K, Bowman A, Birch-Machin MA. Oxidative stress–a key emerging impact factor in health, ageing, lifestyle and aesthetics. International Journal of Cosmetic Science. 2015;37: 1-8.

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