

Occurrence and Evaluation of White Spot Lesions in Orthodontic Patients: A Pilot Study.

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ABSTRACT

Orthodontic treatment may cause an increase in the rate of enamel decalcification on tooth surfaces, producing White Spot Lesions (WSL). Orthodontic patients are at a higher risk for decalcification because orthodontic appliances retain food debris which leads to increased plaque formation. Dental plaque, an oral biofilm formed by factors including genetics, diet, hygiene, and environment, contains acid producing bacterial strains with a predominance of *Mutans Streptococcus* (MS). MS and others metabolize oral carbohydrates during ingestion, the byproducts of which acidify the biofilm to begin a process of enamel decalcification and formation of WSL.

This study tests if patients in orthodontic treatment at Temple University can be used as subjects for further longitudinal study of WSL risk factors. Twenty patients between the ages of ten to eighteen after three months or greater of treatment were enrolled to determine if duration of treatment, hygiene, sense of coherence, obesity, diet frequencies, age and gender correlated with development of WSL. Of these, age is positively correlated with the number of untreated decayed surfaces. WSL and plaque levels may negatively correlate with increased brushing frequency and duration, while flossing frequency demonstrated a statistically significant negative correlation. This population may be suitable for further study because of its high incidence of WSL (75%), however difficulty in enrollment and patient attrition necessitates that future studies be modified.

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CHAPTER 1

INTRODUCTION

White spot lesions (WSL) are localized chalky roughened areas of enamel demineralization in the earliest stages of caries (Ogaard et al. 1998) which form due to a specific and complex composition of oral bacterial acid production (Torakovic 2012; Ogaard 1998). There is normally a balance between demineralization and remineralization as bacterial plaque acids dissipate and salivary minerals penetrate the enamel surface. If the demineralization process is favored, layers of enamel calcium and phosphate hydroxyapatite crystals begin to break down (Khalaf et al. 2014), forming WSL which may progress to cavitated lesions in need of restoration.

WSL are a particular concern during orthodontic treatment since fixed orthodontic appliances create plaque retention areas that are difficult to clean and quickly raise bacterial populations, and without adequate hygiene cause an increased acid challenge to enamel (Khalaf et al. 2014; Artun et al. 1986). Further the irregular shapes of orthodontic appliances also limit the self-cleansing ability of saliva, lips, tongue and cheeks (Heymann et al. 2013). The highest incidence of WSL occurs on maxillary lateral incisors and canines, followed closely by the mandibular premolars (Artun et al. 1986). Small tooth surface areas between the orthodontic bracket and gum tissue which are hard to clean leave these teeth at high risk. Bends in orthodontic wires and elastic hooks are often placed close to these teeth during treatment which further complicate adequate patient hygiene (Artun et al. 1986). Although these observations appear intuitive, there is no consensus as to exactly why white spots develop in some patients but not in others with similar hygiene habits. Recent development has shown a genetic component to

caries, ranging from enamel crystal quality to variation in salivary molecules such as CD14, a co-receptor that detects bacterial lipopolysaccharide in immunological response to oral bacteria (Viera et al. 2014). When studies without a genetic component are conducted it must be kept in mind that a piece of the picture may be missing from the results.

Although promising preventive methods of WSL are currently being developed, there is currently no standard of care. Most research correlates proper oral hygiene, diet and the use of fluoride with a reduction of WSL (Khaelf et al. 2014; Featherstone 2004; Kidd 2011). Even with this information available the prevalence of WSL can reach as high as 92% (Khaelef et al. 2014). Treatment is attempting to re-mineralize, or mechanically reducing WSL once already formed (Yap 2014). This process is time consuming, and would be unnecessary if white spot lesions could be prevented.

CHAPTER 2

REVIEW OF THE LITERATURE

2.1 Identification of White Spot Lesions

White spots lesions (WSL) can range from being visually obvious, to clinically hidden until the tooth is dried. This creates difficulty assessing formation and progression. One method for detection is the International Caries Detection and Assessment System (ICDAS). ICDAS has gained acceptance as a practical way for clinicians to quickly and routinely evaluate a carious lesion. It has the benefit of reliably detecting caries before they have become cavitated without the use of an explorer, which can potentially create iatrogenic cavitations (Lussi 1991, Dodds 1993). The ICDAS system uses a series of codes ranging from zero, which is sound enamel, to six, which is a distinct cavitation involving at least half of the tooth, to grade lesions based on their visual appearance. Codes 1 and 2 represent the initial (visible only after the tooth is dried) and late white spot lesions (clinically visible on a wet tooth) respectively, while code 3 is when cavitation begins.

2.2 Progression of White Spot Lesions

The caries process begins with the dynamic stability stage which is when normal fluctuations in pH lead to teeth undergoing demineralization and remineralization in a ratio that leads to net remineralization. When acidification increases, it leads to what is termed the acidogenic stage, where the oral cavity undergoes a moderate frequent

exposure to acid that may shift the tooth from net remineralization to demineralization. The final stage is the aciduric stage, which is characterized by severe prolonged acidification. Bacteria that thrive in acidic conditions dominate and further lower the pH, leading to increased mineral loss and the initiation of new lesions as well as progressions of prior lesions (Takahashi et al. 2011). Torlakovic et al. in 2012 used the Human Oral Microbe Identification Microarray (HOMIM) to detect about 300 bacterial species in a longitudinal study of changes in composition of the oral biome during development of carious lesions around orthodontic bands. They observed that 25 bacterial types, including *Streptococcus mutans* (*S. Mutans*), were significantly associated with initial WSLs, whereas 14 other bacteria were significantly associated with sound enamel, indicating that there is greater complexity to the progression of enamel lesions than earlier thought. Regardless of these findings, *S. Mutans* has been shown to play a central role in the initiation of caries (Tanzer et al. 2001). *Streptococcus mutans* has the ability to create water insoluble glucan used for the initial attachment of bacteria to teeth. It is acidogenic in the presence of sucrose, thrives at a low pH and has been shown in a multitude of studies to be a primary agent in the initiation of caries (Baelstrom et al. 2014; Jung et al. 2014).

2.3 Salivary Buffering

Saliva serves as the oral cavities natural buffering mechanism. In a healthy mouth changes in the pH are neutralized, keeping the acidity below the critical demineralizing level. The amount of saliva and its buffering capacity correlate with a decrease in caries experienced (Al-Alimi et al 2014; Ericsson et al. 1959). The buffering capacity of saliva

has three major components: bicarbonate, phosphate and protein buffering systems (Lenander-Lumikari et al. 2000). Each system has a different pH range of buffering capacity and together protects the oral cavity from large changes in pH, with the bicarbonate buffering system playing the largest role. When a patient has low buffering capability as well as low salivary flow, they are at an increased risk of a pH shift that can lead to net demineralization and WSL (Lenander-Lumikari et al. 2000; Lagerlöf et al. 1994). The most common test for salivary flow is to have the patient chew and measure the saliva produced. Buffering capacity is measured after collecting saliva and exposing it to different levels of acidity. It can be used to assess a patient's demineralization risk. (Lenander-Lumikari et al. 2000). Variations in salivary buffering capacity play a key role in enamel protection as acidic challenge to oral pH fluctuates with ingestion of sugar or fermentable carbohydrates.

2.5 Dietary Role in Formation of WSL

Sweetened beverages like soda and juice contain high concentrations of sugars, which when frequently ingested increases the daily acidic challenge to the enamel. (Marshall et al. 2005). High levels of daily sugar intake have been linked with overweight patients (Vasanti et al. 2006).

One method of evaluating a patient's weight is by using the body mass index (BMI). BMI is calculated using the patient's weight in kilograms and dividing it by the square of their height in meters. The CDC publishes height and weight anthropometric measurements that percentiles the American child and adolescent population from which BMI is calculated. The categories of BMI percentiles are underweight (less than the fifth

percentile), healthy weight (fifth percentile to less than 85th percentile), overweight (85th to less than the 95th percentile), and finally obese (greater than or equal to the ninety-fifth percentile). Because of the increased prevalence of obesity in the United States, estimated to be from 15% to 33% in adults and from 6% to 19% in children between 1980 to 2007 (Ogden et al., 2007), understanding comorbidity with oral health is an important consideration. Comorbid conditions include the risk of type 2 diabetes in children, adolescents and adults (Fagot-Campagna 2000; Oguma et al. 2005) as well as liver and cardiovascular disease (Eckel 2002). Obese subjects are at increased risk for the development of periodontal disease (Genco et al. 2005) and dental caries (Burt et al. 2006), but the mechanisms for these associations remain uncertain. Yuan et al. (2012) have recently reviewed the problems associated with obesity that affect oral health such as increased sugar intake and more frequent sugar exposure, and strongly advocate an emphasis of these topics and greater use of physical evaluations in clinical dental education. Although studies of children 6 to 9 years old (Werner et al. 2010) and age 8 and under (D'Mello et al. 2011) did not find a significant relationship between BMI and caries (Costacurta et al. 2011) demonstrated that correlations were significant in 6 to 12 year olds using percent body fat mass, as determined by dual energy X-ray Absorptiometry (DXA), instead of BMI values. It was further suggested that determination of childhood obesity by BMI may lead to misclassifications that could explain the conflicting data on associations between obesity and dental caries. Furthermore, the selection in their study of a slightly older population with more permanent dentition may reflect a higher susceptibility to caries due to the quality of enamel in newly erupted teeth. As yet, there are no reports of potential effects of obesity

on the presence and development of WSLs in adolescents undergoing orthodontia with fixed appliances. Accordingly, there is a need to investigate whether obese adolescents might be at higher risk for WSLs during treatment.

2.6 Oral Hygiene and WSL

Oral hygiene includes brushing and flossing, and is a patient's ability to keep their mouth clean. It varies based on a patient's brushing technique, duration, frequency as well as the use of floss. Orthodontic brackets and wires serve as obstacles which retain plaque and are difficult to clean around. Patients with inadequate oral hygiene are at an increased risk of developing WSLs, because the prolonged retention of plaque maintains the acidic challenge to the tooth (Bishara et al. 2008). The best prevention of WSL is by implementing a good oral hygiene regimen (Khalef et al. 2014; Yap et al. 2014; Bishara et al. 2008) including adequate brushing frequency, as well as brushing duration. Satisfactory oral hygiene disrupts the plaque found on teeth, and prevents the demineralization process from occurring (Bishara et al. 2008). Patients with poor oral hygiene are almost nine times more likely to experience WSL (Khalef et al. 2014). Changing a patient's oral hygiene habits is difficult. The best method is to evaluate their technique and advise on their timing and frequency (Clarkson et al. 2009).

2.7 Patient Compliance

Patient compliance is crucial in orthodontic treatment. A patient must have good oral hygiene, few, if any, missed appointments, and a willingness to follow instructions. One method of predicting a patient's willingness to partake in treatment is to measure their sense of coherence (SOC). SOC uses a series of questions on a scale from one to seven asking the patient about their outlook on life. A patient's SOC characterizes their ability to see environmental challenges as meaningful and manageable, and in doing so, take responsibility for their own care (Eriksson et al. 2005). It measures how people manage environmental stress to predict compliance. A weak sense of coherence increases the likelihood of poor oral health and is tied strongly to a tooth brushing frequency of less than once a day (Savolainen et al. 2005; Bernabe et al. 2010). Psychosocial models for oral health related behaviors usually parallel similar models used for overall systemic health. One that has been useful to oral health is the Salutogenetic theory of Antonovsky (Antonovsky 1987). This theory hypothesizes that people with a greater sense of coherence (SOC) have better overall health and respond to stressors in the environment more successfully. Sense of Coherence is the confidence one has that stimuli in the environment are structured, predictable and explicable. SOC increases in people who believe they have the resources to cope with environmental stimuli and that coping with the environment is a worthy investment. Antonovsky developed the first SOC test in 1987 which has 45 questions answered on a likert scale from 1 - 7. The SOC scale is a reliable, valid and cross culturally applicable instrument for measuring how people manage environmental stress and health (Eriksson et al. 2005). A weak sense of

coherence increases the likelihood of poor oral health and a tooth brushing frequency of less than once a day (Savolainen et al. 2005).

CHAPTER 3

AIMS OF THE INVESTIGATION

The primary aim will determine if the graduate orthodontic patient population at Temple University is viable for clinical trials investigating white spot lesions

The secondary aims are to study the caries severity of white spot lesions (ICDAS codes 1 and 2) with patient characteristics including: age, gender, diet, BMI, hygiene and social outlook.

CHAPTER 4

MATERIALS & METHODS

4.1 The Sample

The subjects were twenty English speaking patients ten to eighteen years old in braces for at least three months. Patients with a previous history of poor compliance or cognitive impairment were excluded. Recruitment was during clinic hours, where consents were obtained. Each patient that participated in the study had their consent forms placed in a patient specific folder that was locked in the orthodontic clinic. The patients were then de-identified by assigning them three digit numbers. The patients were assigned randomly to one of the two treatment groups.

Two credential protected electronic folders were created for the study. One folder held an excel sheet consisting of the patient's name, relevant data, and de-identification number. The second folder held the patient's number and all data collected on that patient.

When the patient arrived they were asked to not brush their teeth and their wires were removed. Each patient was informed that they would have data collection once every two months for a total of four, as shown in Table 1. The data collected from the initial time point T0 was used for this thesis.

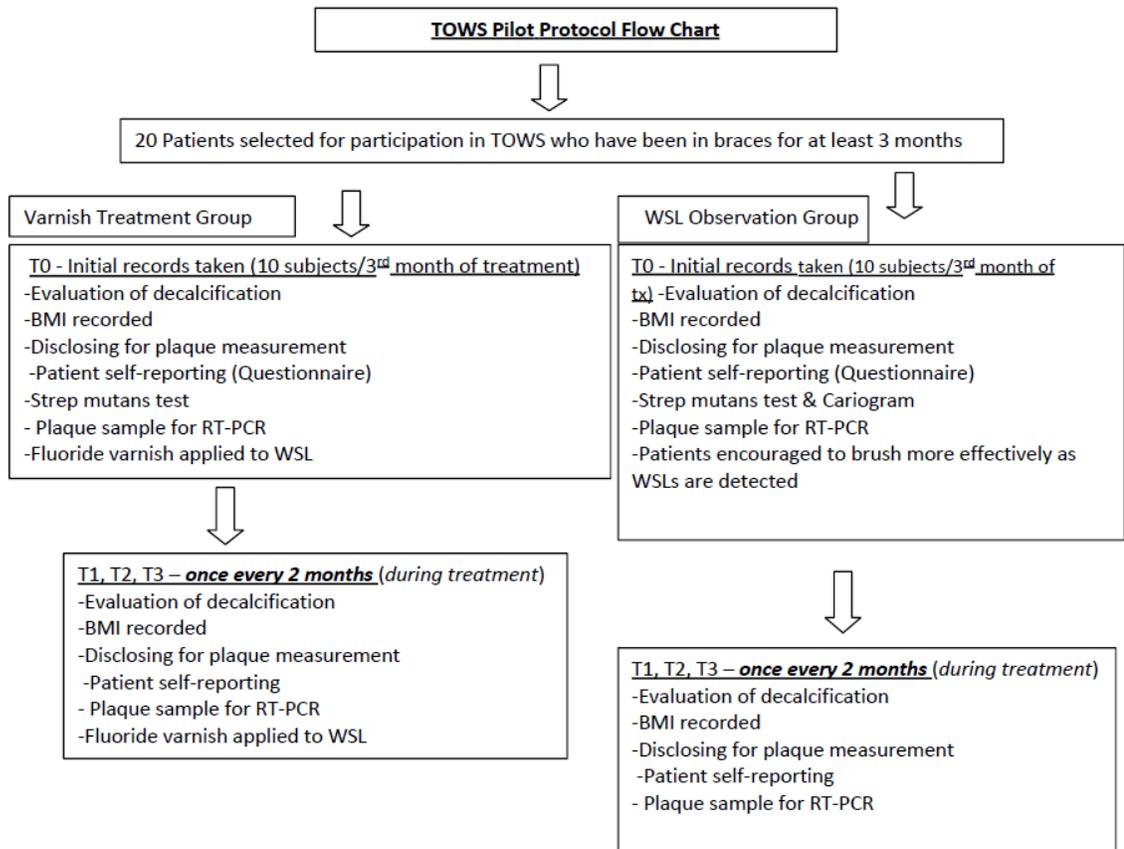


Figure 1: TOWS data collection flow chart

4.2 Bacterial Testing

Plaque samples were collected with a sterile tooth pick from the buccal enamel surface of all non-molar teeth. The tooth pick was then smeared along the edges of a tube in order to suspend the sample in 1X Phosphate Buffered Saline. Dr. Horton isolated DNA from samples using standard extraction protocol for isolation of bacterial genomic DNA (Nadkami et al. 2009).

Patients had their buccal plaque scraped along their brackets with a toothpick that was then smeared on a Strip Mutans™ plastic strip. The patient was then given a paraffin

pellet to chew for one minute before swallowing the stimulated saliva. Two thirds the strip (Strip mutans™) was inserted into the mouth and rotated on the tongue 10 times. The Saliva and plaque strips were then placed into a culture vial containing selective culture broth with a bacitracin disk. The vial was stored temporarily in a refrigerator at 8⁰ Celsius until it was transported to the laboratory where it was incubated at 35⁰ Celius for forty-eight hours before being read. Strip mutans™ results were scored blindly by Dr. Sciote and Dr. Horton, on a scale of 0 to 3, with 0 being no bacterial species, 2 being moderate density, and 3 being an excess of species (Calibrated by Figure 1). The strips were each scored for the highest density area. Each score represents a different colony forming unit (CFU) density as seen in figure 2. The strips were photographed as shown in figure 3, and stored in a credential protected database that included no patient identifying data.

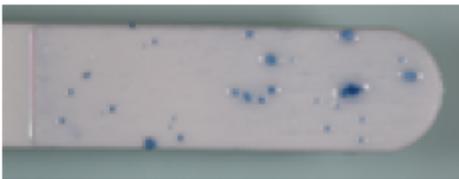
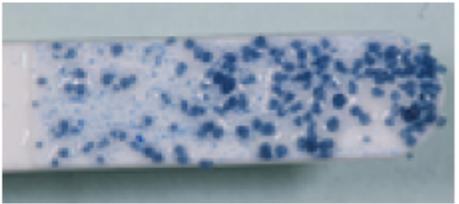
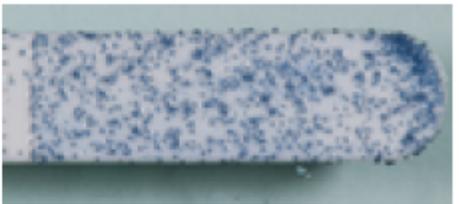
<p align="center">Figure 2</p> <p align="center"><i>S mutans</i> Density in Stripmutans Solution</p>		
0	Clear	
1	Mild Density	
2	Moderate Density	
3	Severe Density	

Figure 2 Representative quantity categories of oral *S mutans* assayed using Stripmutans™ diagnostic strips.

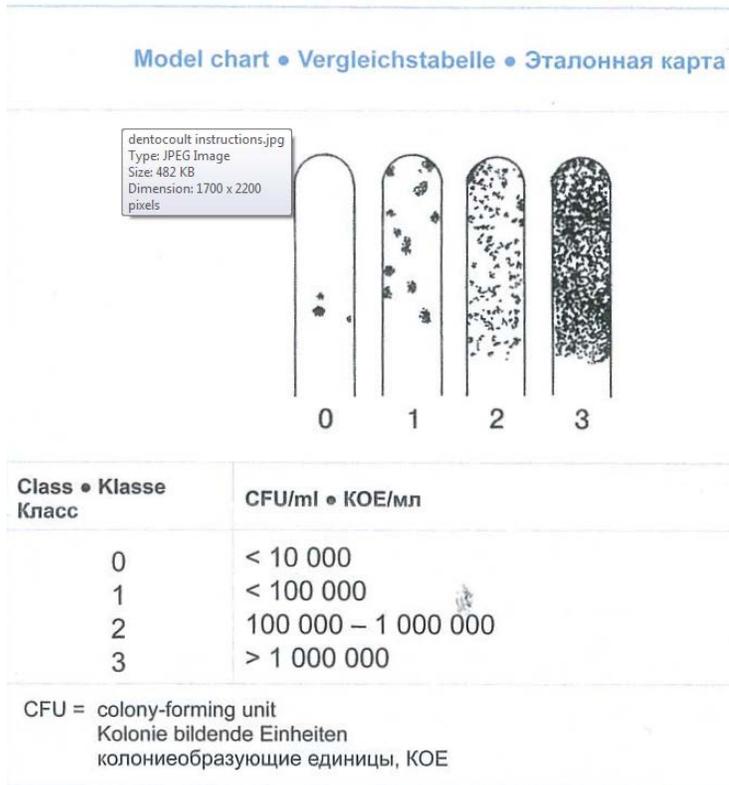


Figure 3: Dentocult SM Strip Mutans CFU Instruction manual reading chart



Figure 4: Temple orthodontic clinic severe density *S. Mutans* (Patient #999).

4.3 Plaque Scoring

In order to stain the patient's plaque they were given a disclosing tablet and asked to chew for 30 seconds before rinsing with eight ounces of water. After staining, the teeth were examined for the density of plaque at the cervical third of the buccal, lingual, mesial, and distal tooth surfaces in the established method of Silness and Loe (Silness 1964; Loe 1967). A zero indicated no plaque present; 1 indicated a film of plaque present on the tooth; 2 represented moderate accumulation of soft deposits in the gingival pocket or on the tooth that could be seen by the naked eye; 3 represented an abundance of soft matter within the pocket or on the tooth. Four areas on each tooth were assigned a score from 0 to 3. Scores for each tooth were then totaled and divided by the four surfaces scored. To determine a total plaque index (PI) for an individual, the scores for each tooth were totaled and divided by the number of teeth examined. Four ratings were then assigned: 0 = excellent, 0.1-0.9 = good, 1.0-1.9 = fair, 2.0-3.0 = poor. For this experiment the patient had incisors, canines and premolars of both jaws totaled and then divided by the number of present teeth.



Figure 5: Disclosing Method Used For Plaque Scores

Surface	Score
Buccal	3
Lingual	2
Mesial	1
Distal	2

$$\text{Plaque Index} = (3+2+1+2) / 4 = 2.0$$

Figure 6: Plaque index calculation

Patients then brushed thoroughly until all surface plaque was removed, and no disclosing coloration could be seen on the surface of the teeth.

4.4 ICDAS White Spot Detection

The surfaces of the patient's teeth were visually examined using the ICDAS method (Ismail et al. 2007).

The following grading criterion was used:

Code Description

0 Sound

1 First Visual Change in Enamel (seen only after prolonged air drying or restricted to within the confines of a pit or fissure)

2 Distinct Visual Change in Enamel

3 Localized Enamel Breakdown (without clinical visual signs of dentinal involvement)

- 4 Underlying Dark Shadow from Dentin
- 5 Distinct Cavity with Visible Dentin
- 6 Extensive Distinct Cavity with Visible Dentin

The WSL caries severity was calculated as the sum of all ICDAS codes 1 and 2.

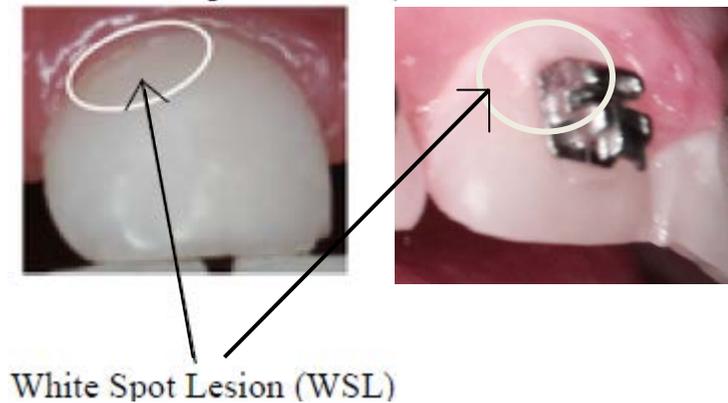


Figure 7: ICDAS CODES 1 and 2 respectively

4.5 Patient Self-Reporting

Three questionnaires were given to the patient: Oral Hygiene Frequency Questionnaire (OHFQ), Harvard Youth/Adolescent Food Frequency Questionnaire (HYA FFQ), and the thirteen question Sense of Coherence (SOC) questionnaire. The oral hygiene frequency questionnaire (Figure 3) was developed for this study by Dr. Sciote as a means of quickly elucidating a patients oral hygiene habits. Each answer was used independently as a separate hygiene factor.

	0	1	2	3	4	5+
1. How many times a day do you brush your teeth?						
2. How many times a day do you floss your teeth?						
3. How long do you brush your teeth? (minutes)						
4. How many times a day do you eat sweets or drink a sweetened drink?						

Figure 8: Oral Hygiene Frequency Questionnaire (OHFQ)

HYA FFQ (Figure 4) has been optimized for simplicity and ease of use. The questions have been carefully worded to gain maximum useful patient information (Rockett et al 1997). Instead of scoring using Harvard’s proprietary algorithm each answer was used as a separate risk factor and evaluated against the patients plaque scores, prevalence of WSL, and quantities of plaque and salivary *S. Mutans* to create a risk profile similar to the methods of Khalef et al. in 2014.

DRINKS			
1. Diet soda/pop (1 can or individual bottle) A Never/less than 1 per month B 1-3 bottles per month C 1 bottle per week D 2-4 bottles per week E 5-6 bottles per week F 1 bottle per day G 2 bottles per day H 3 or more bottles per day	2. Soda/pop—not diet (1 can or individual bottle) A Never/less than 1 per month B 1-3 bottles per month C 1 bottle per week D 2-4 bottles per week E 5-6 bottles per week F 1 bottle per day G 2 bottles per day H 3 or more bottles per day	3. What is the usual serving size of the soda/pop you drink (any type)? A <12 oz. B 12 oz. (e.g., can) C 16-20 (individual bottle) D 21+oz. (e.g., Big Gulp) E Don't know or don't drink	4. Sugared iced-tea, fruit drinks, punch, lemonade, Sunny D, Kool-Aid or other non-carbonated fruit drink—NOT JUICE (1 glass, can or individual bottle) A Never/less than 1 per month B 1-3 bottles per month C 1 bottle per week D 2-4 bottles per week E 5-6 bottles per week F 1 bottle per day G 2 bottles per day H 3 or more bottles per day
5. Sport drinks (e.g., Powerade or Gatorade) or sugar-sweetened vitamin water (individual bottle) A Never/less than 1 per month B 1-3 bottles per month C 1 bottle per week D 2-4 bottles per week E 5-6 bottles per week F 1 bottle per day G 2 bottles per day H 3 or more bottles per day	6. Sugar-free or low calorie energy drinks—Red Bull Sugarfree, Lo-carb Monster Energy (individual can/bottle) A Never/less than 1 per month B 1-3 bottles per month C 1 bottle per week D 2-4 bottles per week E 5-6 bottles per week F 1 bottle per day G 2 bottles per day H 3 or more bottles per day	7. Regular energy drinks—Red Bull, Rock Star (individual can/bottle) A Never/less than 1 per month B 1-3 bottles per month C 1 bottle per week D 2-4 bottles per week E 5-6 bottles per week F 1 bottle per day G 2 bottles per day H 3 or more bottles per day	8. Smoothies (e.g., medium Jamba Juice or Orange Julius) A Never/less than 1 per month B 1-3 glasses per month C 1 glass per week D 2-4 glasses per week E 5-6 glasses per week F 1 glass per day G 2 glasses per day H 3 or more glasses per day

Figure 9: Harvard Youth/Adolescent Food Frequency Questionnaire (HYA FFQ)

Sense of coherence was scored using the established method of first reverse coding answers #1,2,3,7,10 (1 scored as 7, 2 scored as 6 etc.) and then summing to get a total SOC score between 13 and 91 (Naaldenberg et al. 2011; Savolainen et al. 2005).

4.6 Body Mass Index

The height in centimeters and weight in kilograms was measured using a SECA stadiometer and scale on loan from Temple University's Center of Obesity Research and Education. The BMI was calculated by dividing the patient's weight in kilograms by the square of their height in meters. CDC tables were then used to compare a patient's BMI with what is expected at their age to find their BMI percentile.

RESULTS

5.1 DESCRIPTIVE STATISTICS

Data from 20 patients including: age at data collection, sex, BMI percentile, ICDAS scores, plaque scores, *S. Mutans* saliva and plaque levels, sense of coherence, brushing frequency, and drink intake data were tabulated into a data matrix and compared using spearman correlation coefficients. The subjects consisted of 14 females and 6 males with a mean age at data collection of 14 years, with the youngest patient being 11, the oldest being 18 with a standard deviation of 2.25 years. The patients mean BMI percentile was 55.62 with a range of 1.10 to 97.3 and a standard deviation of 37.28. The percentiles were distributed with 3 obese, 5 overweight, 3 normal, and two underweight patients. This means that forty percent of our sample population consisted of patients overweight or obese. Fifteen of the twenty subjects had at least one WSL giving our patient population an incidence of 75%.

GENDER	AGE	BMI%	ICDS	TX DURATION
F	12	96.2	0	620
F	12	14.9	0	600
F	14	1.1	0	244
F	14	22.2	0	1058
F	15	16.6	1	405
F	11	2	1	82
F	14	76.4	2	150
F	17	91.4	2	241
F	17	66.4	3	318
F	11	94.2	3	350
F	12	61	6	205
F	18	95	10	122
F	15	31.8	12	816
F	18	29.7	17	314
M	12	97.3	0	118
M	16	95.1	3	90
M	14	11.8	6	659
M	14	91.6	7	734
M	14	27.6	14	363
M	17	90.1	17	561

Table 1: Gender, Age, BMI Percentile, and Treatment Duration Sorted by Caries

Severity (DS 1 + 2)

Underweight	Less than the 5th percentile
Healthy weight	5th percentile to less than the 85th percentile
Overweight	85th to less than the 95th percentile
Obese	Equal to or greater than the 95th percentile

Table 2: BMI Classifications

5.2 Duration of braces with Number of Untreated Decayed Surfaces Including ICDAS Codes 1 and 2

Duration of braces (days after bonding) showed little if any correlation with caries severity (Number of untreated decayed surfaces including ICDAS scores 1 and 2) $R=.17$. A P-value of 0.49 showed this was not statistically significant.

5.3 Patient Age with Number of Untreated Decayed Surfaces Including ICDAS Codes 1 and 2

Patient age (at T0) showed moderate correlation with caries severity (Number of untreated decayed surfaces including ICDAS scores 1 and 2) $R=.53$ with a statistically significant P-value of 0.02.

5.4 Gender with Number of Untreated Decayed Surfaces Including ICDAS Codes 1 and 2

In this sample of 20 patients 14 were females with a mean Number of Untreated Decayed Surfaces Including ICDAS Codes 1 and 2 of 4.07. Six were males with a mean Number of Untreated Decayed Surfaces Including ICDAS Codes 1 and 2 of 7.83. The Kruskal-Wallis test showed that this data had a P value of 0.16 which was not significant in correlating gender with prevalence of white spot lesions.

5.5 BMI Percentile with Number of Untreated Decayed Surfaces Including ICDAS Codes 1 and 2

BMI percentile showed little if any correlation with caries severity (Sum of ICDAS scores 1 and 2) $R=.24$. A P-value of 0.30 showed this was not statistically significant.

Pt ID	Age T0	Sex	Saliva <i>S. Mutans</i>	Plaque <i>S. Mutans</i>	SOC	ICDS
111	15	F	2.5	2.625	77	1
135	14	M	2	2.125	53	7
218	15	F	0	0.375	71	12
302	12	F	0	1	53	6
319	12	F	0	0	55	0
444	12	F	3	2.375	64	0
470	14	M	1	0.375	66	6
448	11	F	1	1.5	51	1
541	16	M	2	2.25	62	3
641	18	F	0.5	1.125	60	10
645	14	F	1	2.125	56	2
662	17	M	3	2.875	60	17
670	17	F	1	1.25	30	2
678	17	F	2	1.5	80	3
706	18	F	2	1.75	65	17
711	11	F	2	2.25	59	3
726	14	F	3	1.25	77	0
730	12	M	1.5	1.625	64	0
989	14	M	3	2.875	58	14
999	14	F	3	3	51	0

Table 3: Saliva and plaque *S. Mutans* with SOC and WSL.

5.6 Plaque *S. Mutans* with Number of Untreated Decayed Surfaces Including ICDAS Codes 1 and 2

Plaque *S. Mutans* quantities showed little if any correlation with caries severity lesions (Sum of ICDAS scores 1 and 2) $R=.0019$ A P-value of 0.99 showed this was not statistically significant.

5.7 Saliva *S. Mutans* with Number of Untreated Decayed Surfaces Including ICDAS Codes 1 and 2

Saliva *S. Mutans* quantities showed little if any correlation with caries severity (Sum of ICDAS scores 1 and 2) $R = -.096$ A P-value of 0.69 showed this was not statistically significant.

5.8 Plaque Scores with Number of Untreated Decayed Surfaces Including ICDAS Codes 1 and 2

Plaque scores showed a low correlation with caries severity (Sum of ICDAS scores 1 and 2) $R = .42$ A P-value of 0.07 showed this was not statistically significant.

5.9 SOC Scores with Number of Untreated Decayed Surfaces Including ICDAS Codes 1 and 2

SOC scores showed a little if any correlation with caries severity (Sum of ICDAS scores 1 and 2) $R = .08$ A P-value of 0.72 showed this was not statistically significant.

5.10 SOC Scores with Plaque Scores

SOC scores showed little if any correlation with a patients plaque scores. $R = .08$ A P-value of 0.73 showed this was not statistically significant.

5.11 Harvard Youth Adolescent Food Frequency Questionnaire

We assessed the frequency of the different survey answers with the number of untreated decayed surfaces including ICDAS Codes 1 and 2, as well as plaque scores.

DRINKS

1. Diet soda/pop (1 can or individual bottle)
 A Never/less than 1 per month
 B 1-3 bottles per month
 C 1 bottle per week
 D 2-4 bottles per week
 E 5-6 bottles per week
 F 1 bottle per day
 G 2 bottles per day
 H 3 or more bottles per day

2. Soda/pop—not diet (1 can or individual bottle)
 A Never/less than 1 per month
 B 1-3 bottles per month
 C 1 bottle per week
 D 2-4 bottles per week
 E 5-6 bottles per week
 F 1 bottle per day
 G 2 bottles per day
 H 3 or more bottles per day

3. What is the usual serving size of the soda/pop you drink (any type)?
 A <12 oz.
 B 12 oz. (e.g., can)
 C 16-20 (individual bottle)
 D 21+oz. (e.g., Big Gulp)
 E Don't know or don't drink

4. Sugared iced-tea, fruit drinks, punch, lemonade, Sunny D, Kool-Aid or other non-carbonated fruit drink—NOT JUICE (1 glass, can or individual bottle)
 A Never/less than 1 per month
 B 1-3 bottles per month
 C 1 bottle per week
 D 2-4 bottles per week
 E 5-6 bottles per week
 F 1 bottle per day
 G 2 bottles per day
 H 3 or more bottles per day

5. Sport drinks (e.g., Powerade or Gatorade) or sugar-sweetened vitamin water (individual bottle)
 A Never/less than 1 per month
 B 1-3 bottles per month
 C 1 bottle per week
 D 2-4 bottles per week
 E 5-6 bottles per week
 F 1 bottle per day
 G 2 bottles per day
 H 3 or more bottles per day

6. Sugar-free or low calorie energy drinks—Red Bull Sugarfree, Lo-carb Monster Energy (individual can/bottle)
 A Never/less than 1 per month
 B 1-3 bottles per month
 C 1 bottle per week
 D 2-4 bottles per week
 E 5-6 bottles per week
 F 1 bottle per day
 G 2 bottles per day
 H 3 or more bottles per day

7. Regular energy drinks—Red Bull, Rock Star (individual can/bottle)
 A Never/less than 1 per month
 B 1-3 bottles per month
 C 1 bottle per week
 D 2-4 bottles per week
 E 5-6 bottles per week
 F 1 bottle per day
 G 2 bottles per day
 H 3 or more bottles per day

8. Smoothies (e.g., medium Jamba Juica or Orange Julius)
 A Never/less than 1 per month
 B 1-3 glasses per month
 C 1 glass per week
 D 2-4 glasses per week
 E 5-6 glasses per week
 F 1 glass per day
 G 2 glasses per day
 H 3 or more glasses per day

Figure 9: Harvard Youth/Adolescent Food Frequency Questionnaire Repeated Figure 7.

5.11A HYA1 with WSL and Plaque Scores

We assessed white spot lesions and plaque scores for question one: “frequency of diet soda/pop intake”, and determined the mean scores between subject answers as shown in Table 4.

Table 4: Frequency of diet soda/pop intake (1 can or individual bottle) and the corresponding plaque scores and caries severity (DS 1 and 2) of white spot lesions.

HYAFFQ Question 1				
	1a (n=12)	1b (n=5)	1d (n=2)	1g (n=1)
White Spot Score	4.17	7.20	8.50	1.00
Plaque Score	1.58	1.65	0.53	1.00

For the white spot and plaque scores, differences were not significant and the P-values were 0.58 and 0.53, respectively.

Table 5: Box-and-Whisker plot for the frequency of diet soda/pop intake and number of untreated decayed surfaces including ICDAS codes 1 and 2.

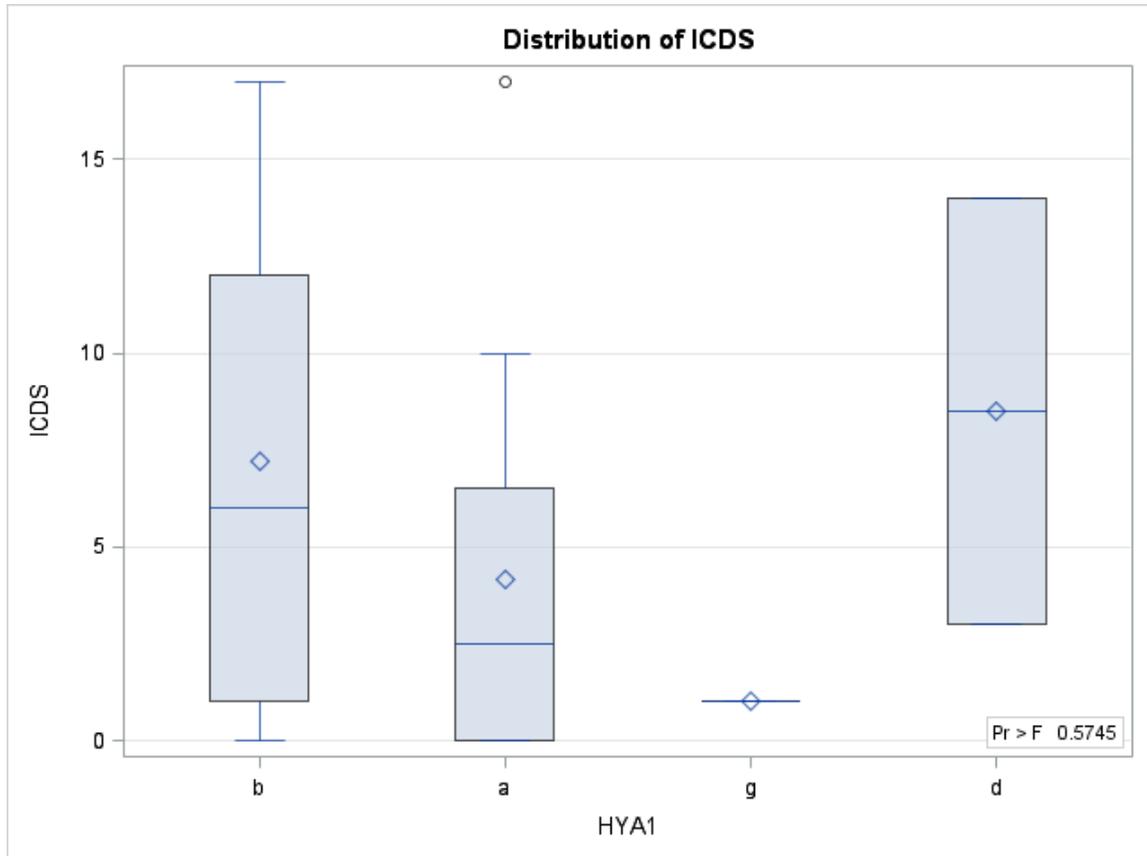
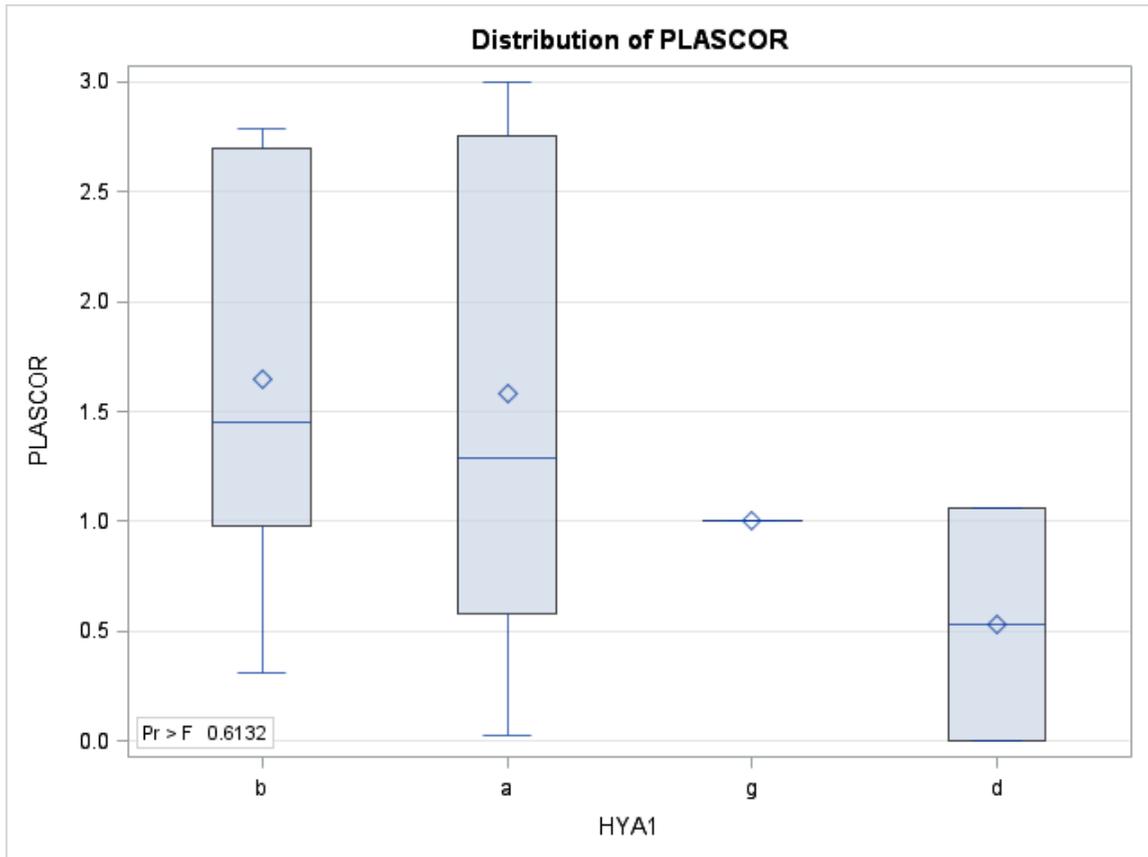


Table 6: Box-and-Whisker plot for the frequency of diet soda/pop intake and plaque scores.



5.11B HYA2 with WSL and Plaque Scores

We assessed white spot lesions and plaque scores for Question 2 “Soda/Pop non-diet” and determined the mean scores between subject answers in Table 7.

Table 7: Frequency of soda/pop (not diet; 1 can or individual bottle) intake and the corresponding plaque scores and caries severity (DS 1 and 2) of white spot lesions.

HYAFFQ Question 2								
	2a (n=6)	2b (n=3)	2c (n=3)	2d (n=2)	2e (n=1)	2f (n=2)	2g (n=1)	2h (n=2)
White Spot Score	4.67	2.33	3.00	12.00	0.00	8.50	2.00	8.50
Plaque Score	1.18	1.49	1.25	3.00	1.23	1.78	0.16	1.52

For the white spot and plaque scores, differences were not significant and the P-values were 0.51 and 0.46, respectively.

Table 8: Box-and-Whisker plot for the frequency of soda/pop intake (not diet) and white spot scores.

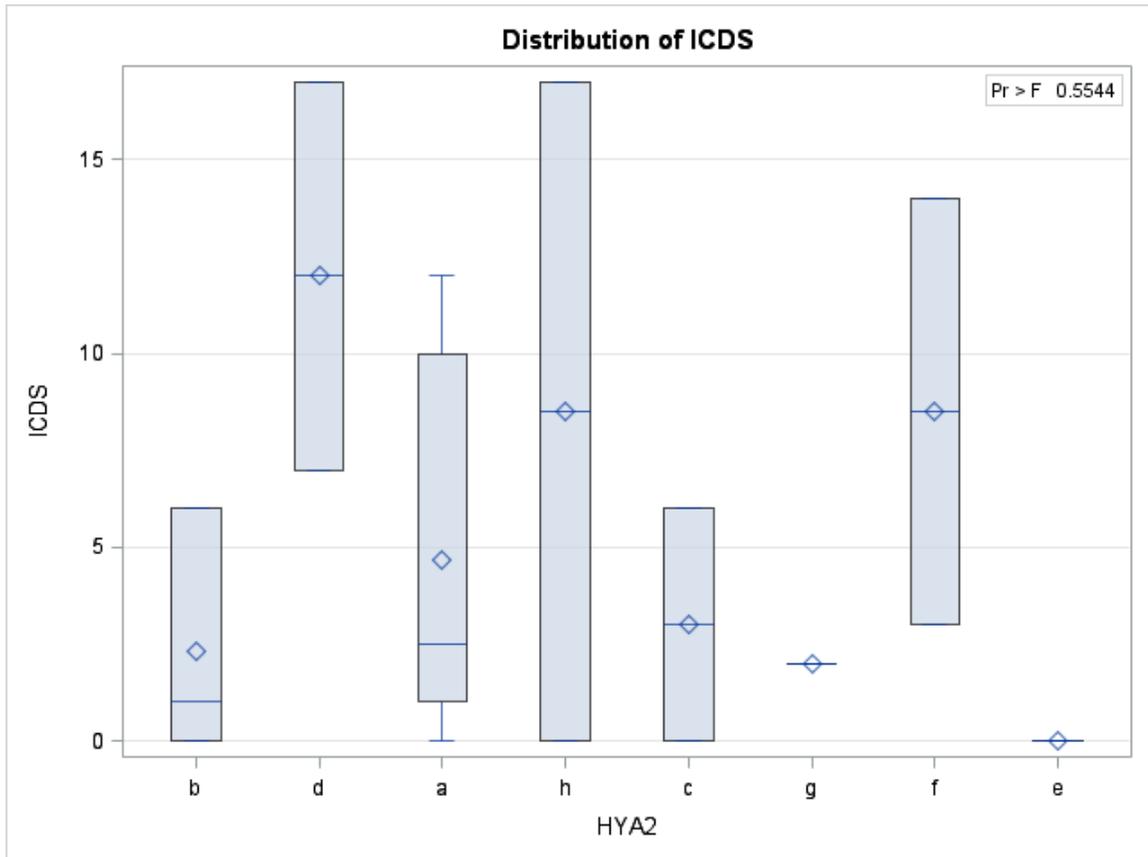
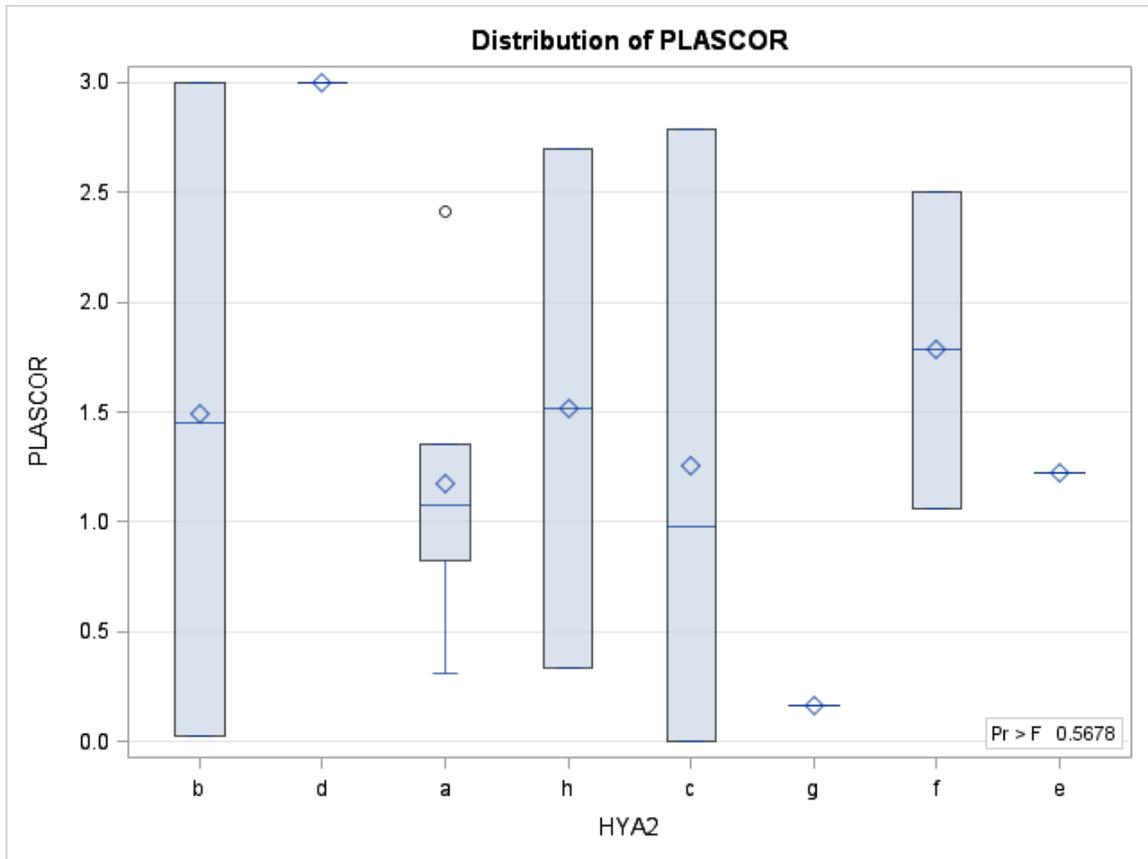


Table 9: Box-and-Whisker plot for the frequency of soda/pop intake (not diet) and plaque scores.



5.11C HYA3 with WSL and Plaque Scores

We assessed white spot lesions and plaque scores for Question 3 “What is the usual serving size of the soda/pop that you drink?” and determined the mean scores between subject answers in in Table 10.

Table 10: Average serving size of soda/pop and the corresponding plaque scores and caries severity (DS 1 and 2) of white spot lesions.

HYAFFQ Question 3					
	2b (n=3)	2c (n=3)	2d (n=2)	2e (n=1)	2f (n=2)
White Spot Score	1.00	6.89	4.75	5.50	0.00
Plaque Score	1.45	1.72	1.03	1.38	1.38

For the white spot and plaque scores, differences were not significant and the P-values were 0.62 and 0.56, respectively.

Table 11: Box-and-Whisker plot for the average soda/pop size and white spot scores.

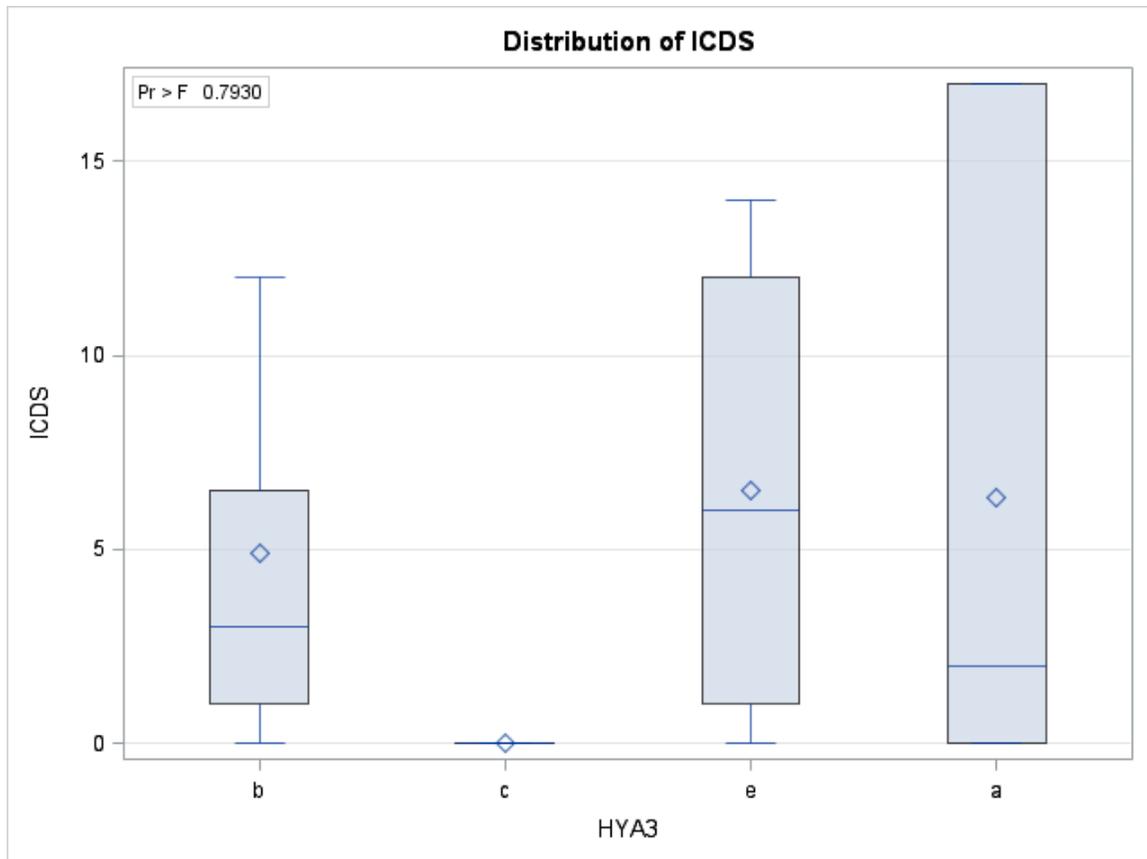
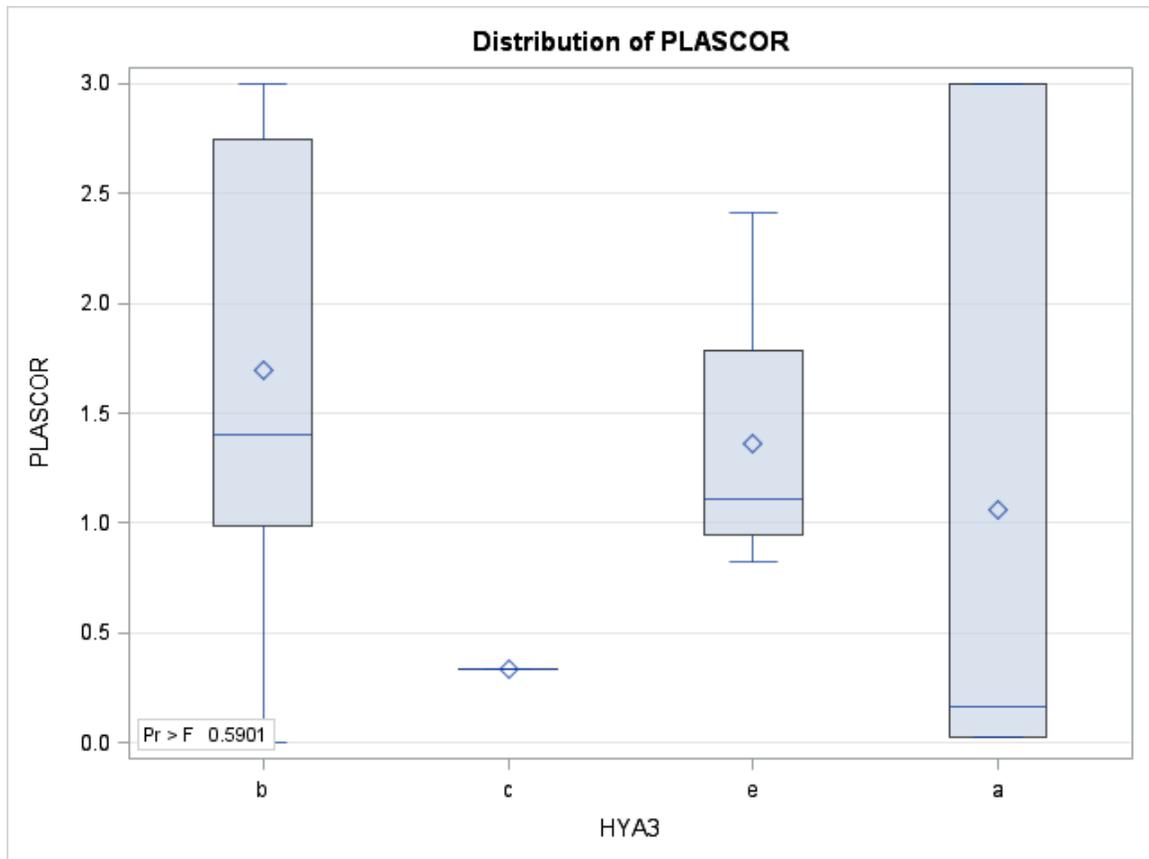


Table 12: Box-and-Whisker plot for the average serving size for the soda/pop and white spot scores.



5.11D HYA4 with WSL and Plaque Scores

We assessed white spot lesions and plaque scores for Question 4: Non-carbonated, non-juice sugary drinks, and determined the mean scores between subject answers in Table 13.

Table 13: Frequency of intake of sugared drinks or other non-carbonated fruit drinks (not juice) and the corresponding plaque scores and Caries Severity (DS 1 and 2) of white spot lesions.

HYAFFQ Question 4							
	4b (n=6)	4c (n=3)	4d (n=5)	4e (n=2)	4f (n=2)	4g (n=1)	4h (n=1)
White Spot Score	8.67	0.67	3.40	10.00	0.50	0.00	12.00
Plaque Score	2.12	0.61	1.27	2.75	0.67	1.23	0.31

For the white spot and plaque scores, differences were not significant and the P-values were 0.06 and 0.17, respectively.

Table 14: Box-and-Whisker plot for the frequency of intake of sugared drinks or other non-carbonated fruit drinks (not juice) and white spot scores.

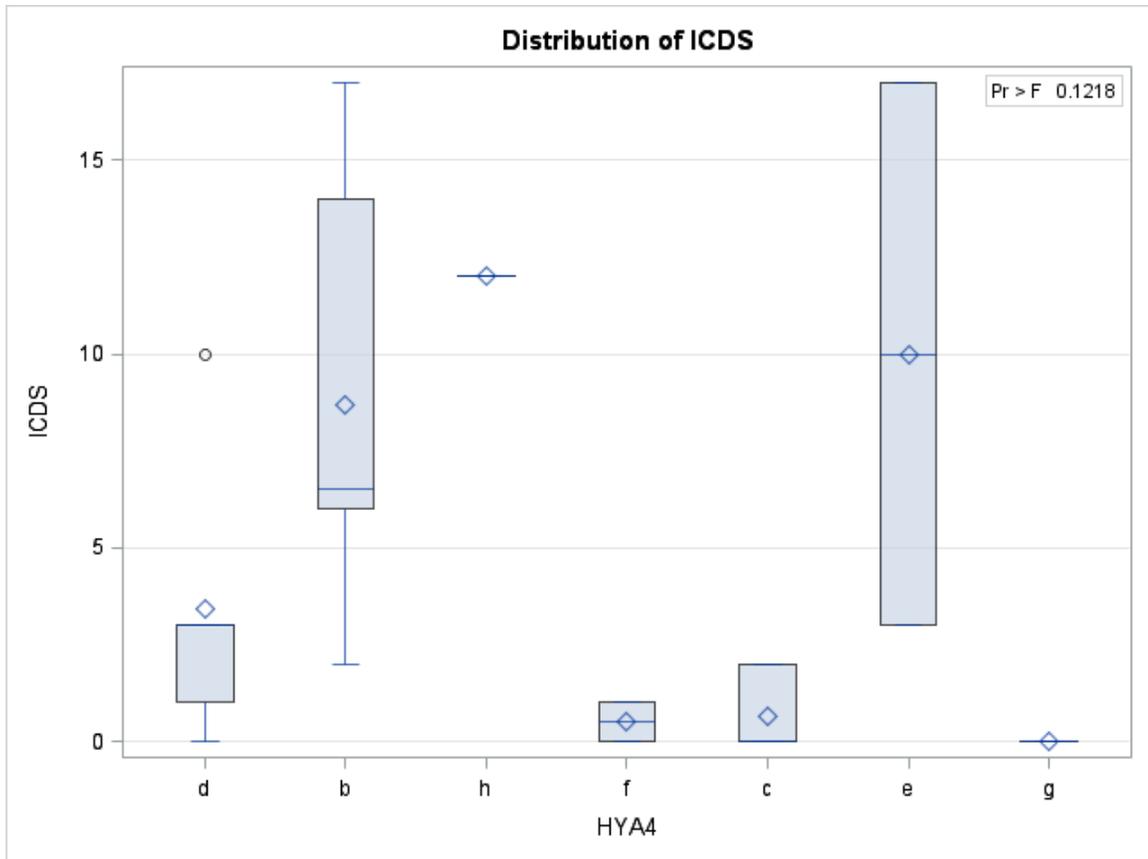
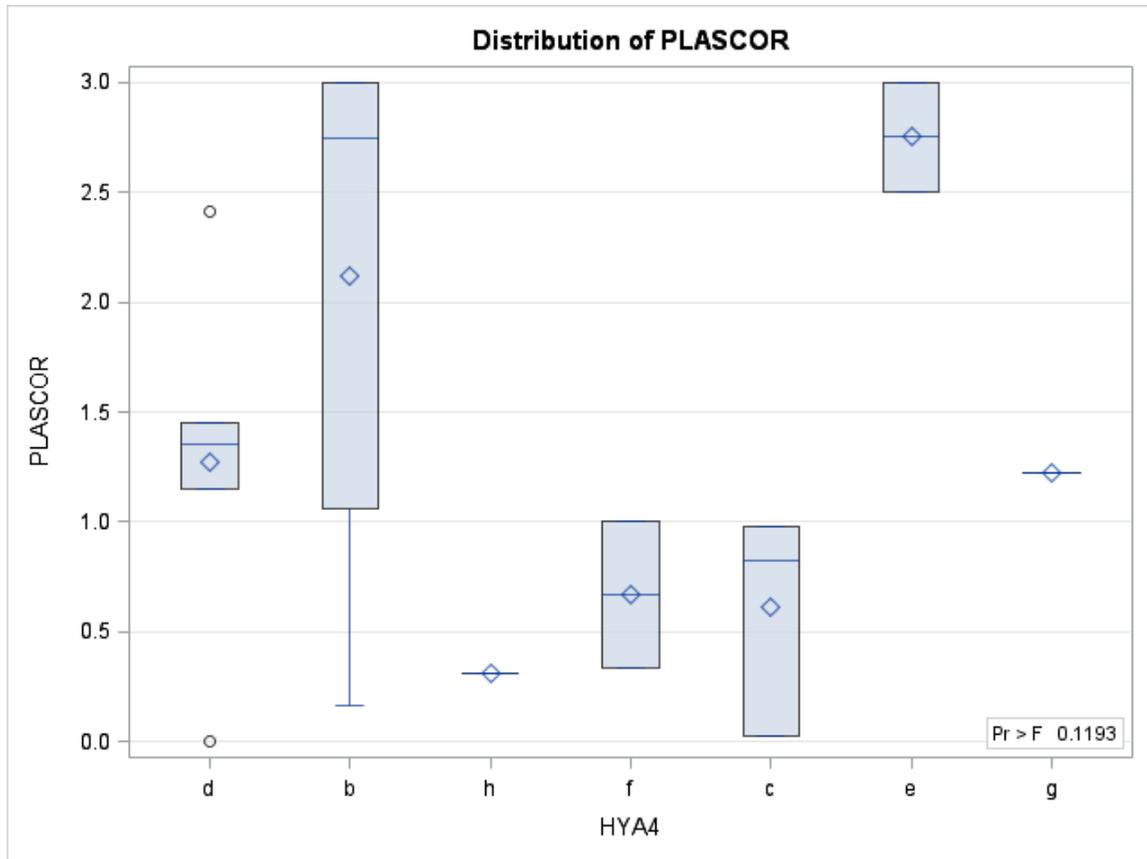


Table 15: Box-and-Whisker plot for the frequency of intake of sugared drinks or other non-carbonated fruit drinks (not juice) and plaque scores.



5.11E HYA5 with WSL and Plaque Scores

We assessed white spot lesions (DS 1 + 2) and plaque scores for Question 5 “Frequency of intake of sports drinks or sugar-sweetened vitamin water” and determined the mean scores between subject answers in Table 16.

Table 16: Frequency of intake of sports drinks or sugar-sweetened vitamin water and the corresponding plaque scores and Caries Severity (DS 1 and 2) of white spot lesions.

HYAFFQ Question 5					
	5a (n=12)	5b (n=2)	5c (n=2)	5d (n=3)	5f (n=1)
White Spot Score	5.67	5.00	7.00	3.33	2.00
Plaque Score	1.69	2.18	1.02	0.80	0.16

For the white spot and plaque scores, differences were not significant and the P-values were 0.83 and 0.36, respectively.

Table 17: Box-and-Whisker plot for the frequency of intake of sports drinks or sugar-sweetened vitamin water and white spot scores.

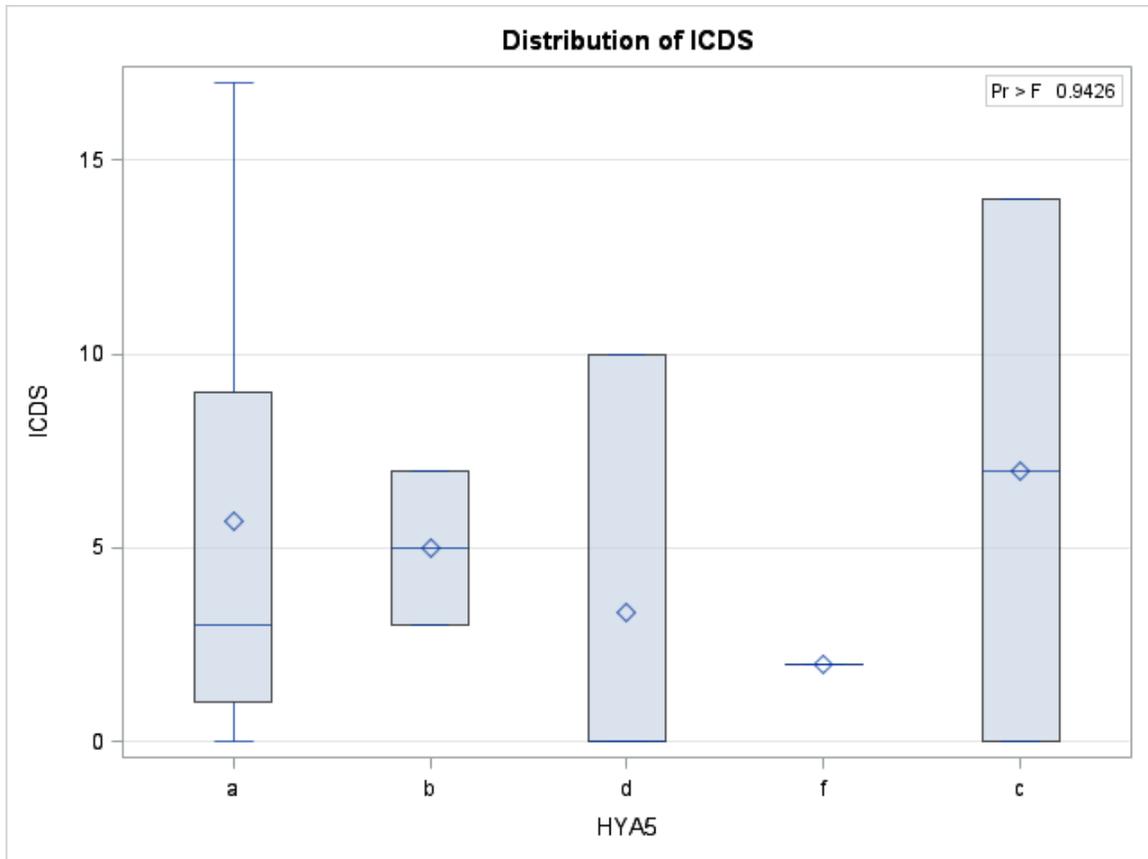
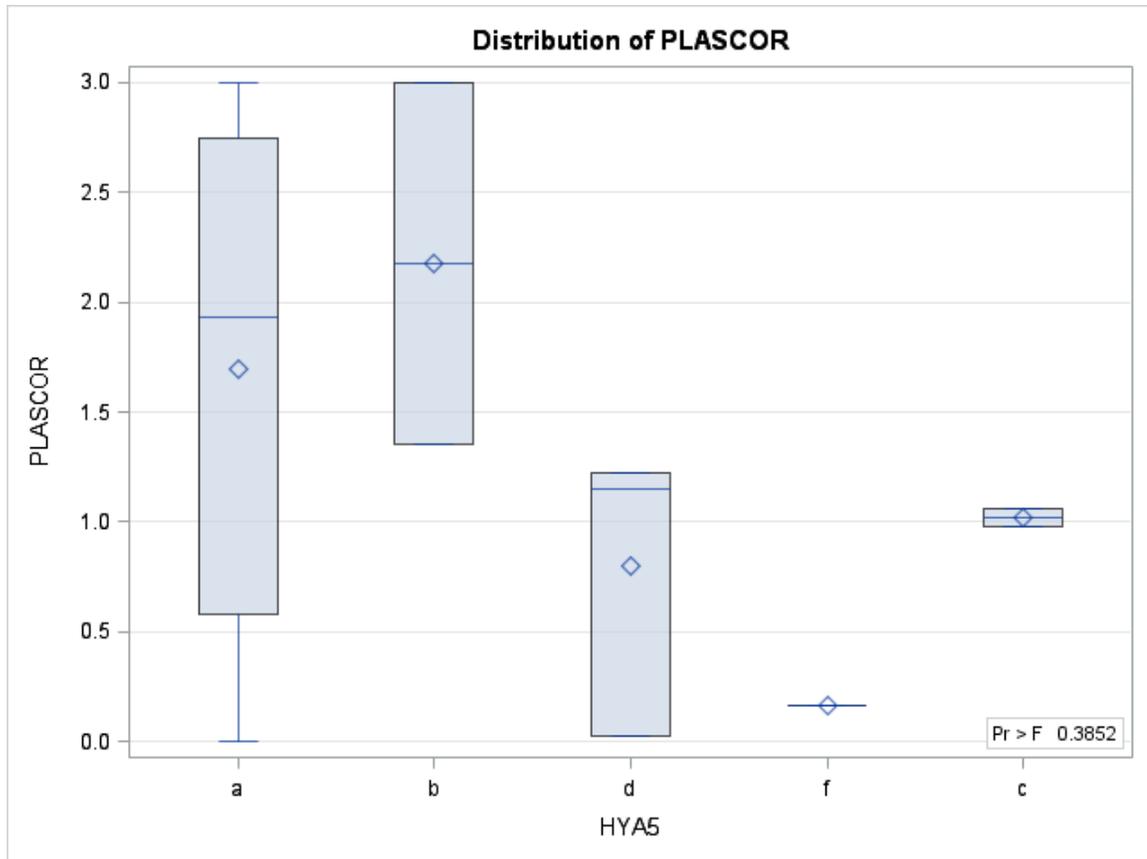


Table 18: Box-and-Whisker plot for the frequency of intake of sports drinks or sugar-sweetened vitamin water and plaque scores.



5.11F HYA6 with WSL and Plaque Scores

We assessed white spot lesions and plaque scores for Question 6 “Frequency of intake of sugar-free or low calorie energy drinks” and determined the mean scores between subject answers in Table 19.

Table 19: Frequency of intake of sugar-free or low calorie energy drinks and the corresponding plaque scores and Caries Severity (DS 1 and 2) of white spot lesions.

HYAFFQ Question 6		
	6a (n=19)	6b (n=1)
White Spot Score	4.74	14.00
Plaque Score	1.49	1.06

For the white spot and plaque scores, differences were not significant and the P-values were 0.19 and 0.79, respectively.

Table 20: Box-and-Whisker plot for the frequency of intake of sugar-free or low calorie energy drinks and white spot scores.

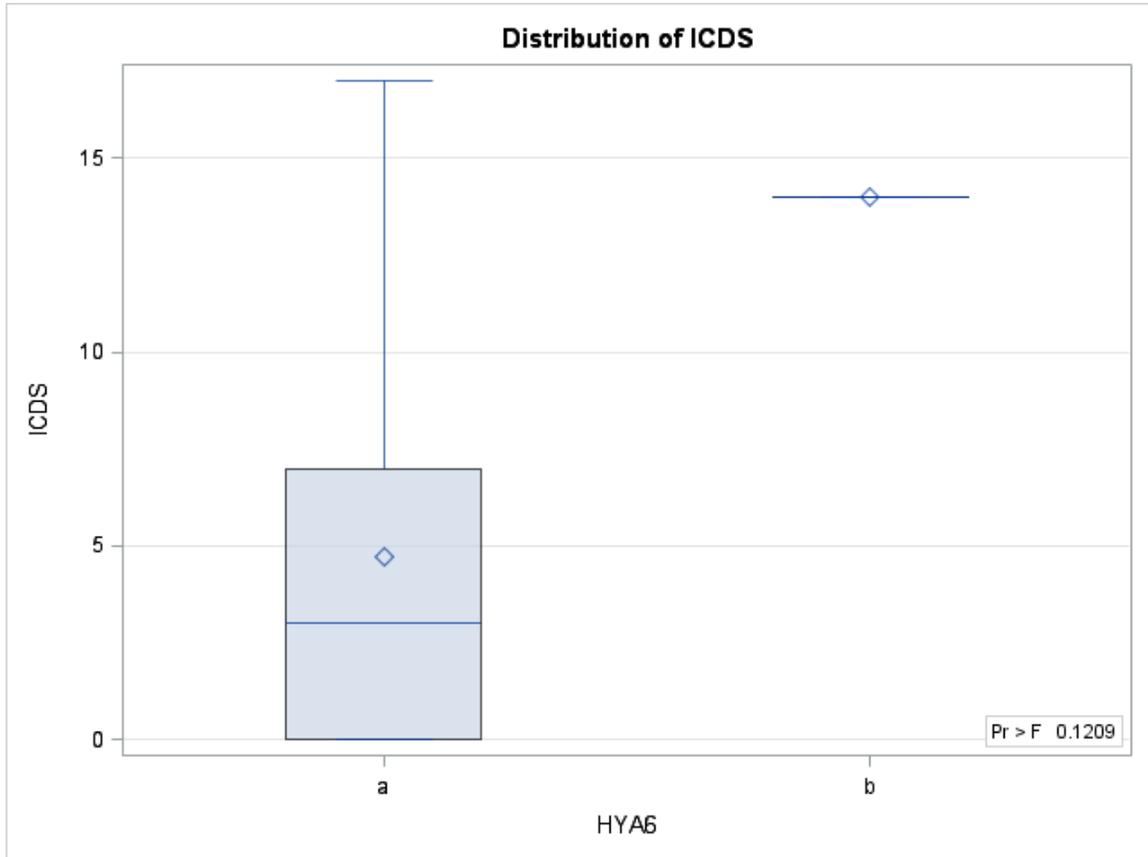
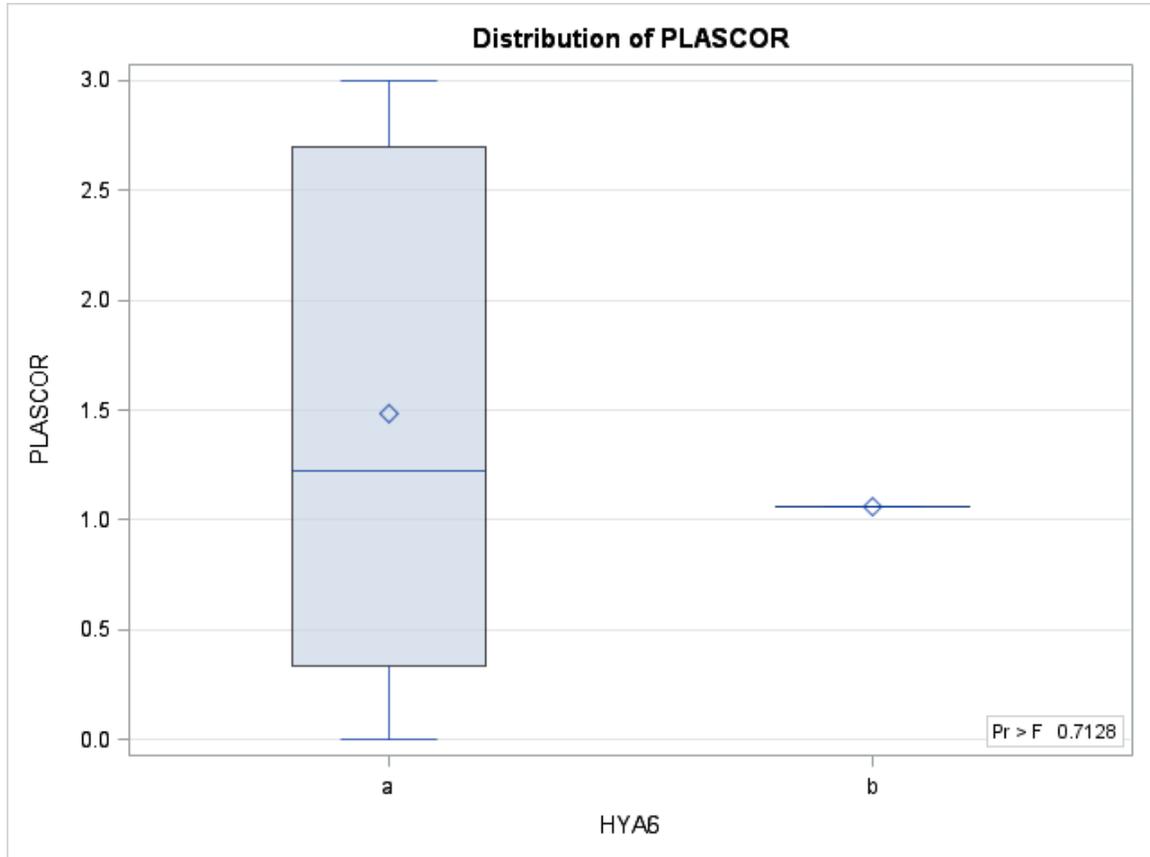


Table 21: Box-and-Whisker plot for the frequency of intake of sugar-free or low calorie energy drinks and plaque scores.



5.11G HYA7 with WSL and Plaque Scores

We assessed white spot lesions and plaque scores for Question 7 “Frequency of intake of regular energy drinks” and determined the mean scores between subject answers in Table 22.

Table 22: Frequency of intake of regular energy drinks and the corresponding plaque scores and caries severity (DS 1 and 2) of white spot lesions.

HYAFFQ Question 7			
	7a (n=17)	7b (n=2)	7c (n=1)
White Spot Score	4.18	9.50	14.00
Plaque Score	1.47	1.58	1.06

For the white spot and plaque scores, differences were not significant and the P-values were 0.26 and 0.96, respectively.

Table 23: Box-and-Whisker plot for the frequency of intake of regular energy drinks and white spot scores.

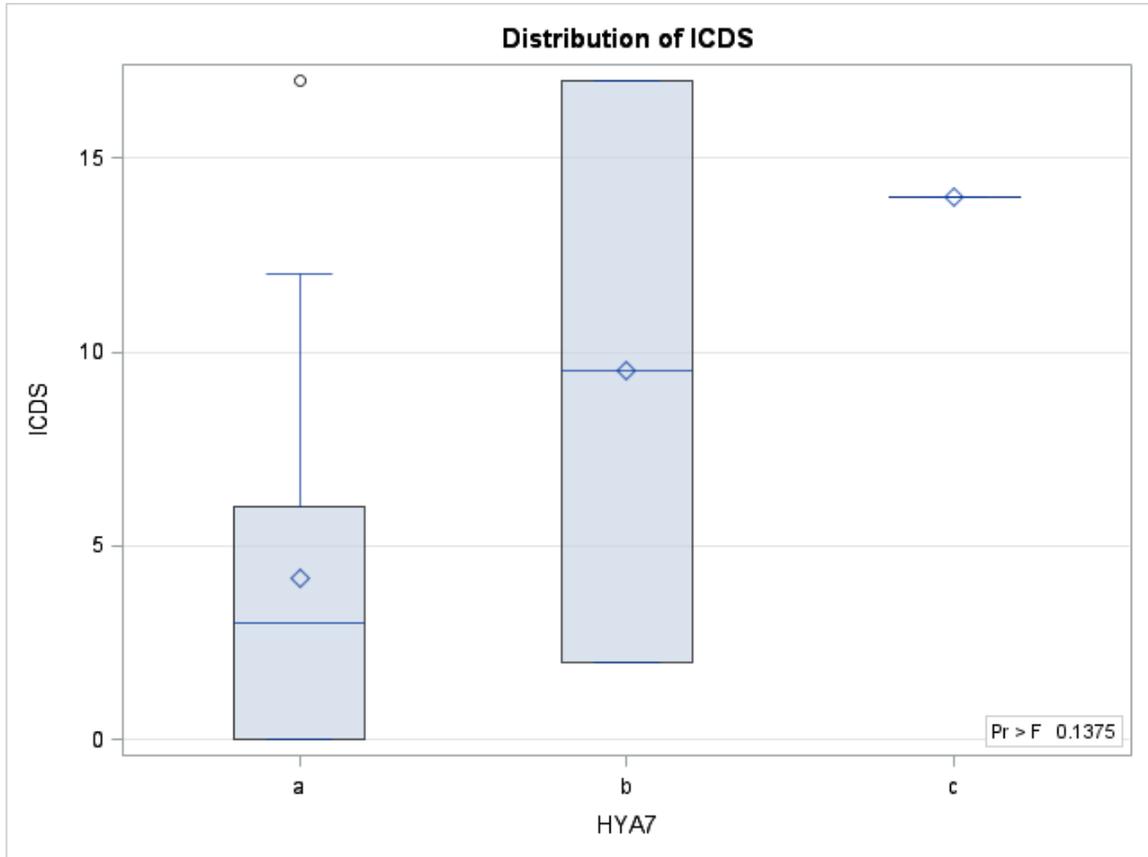
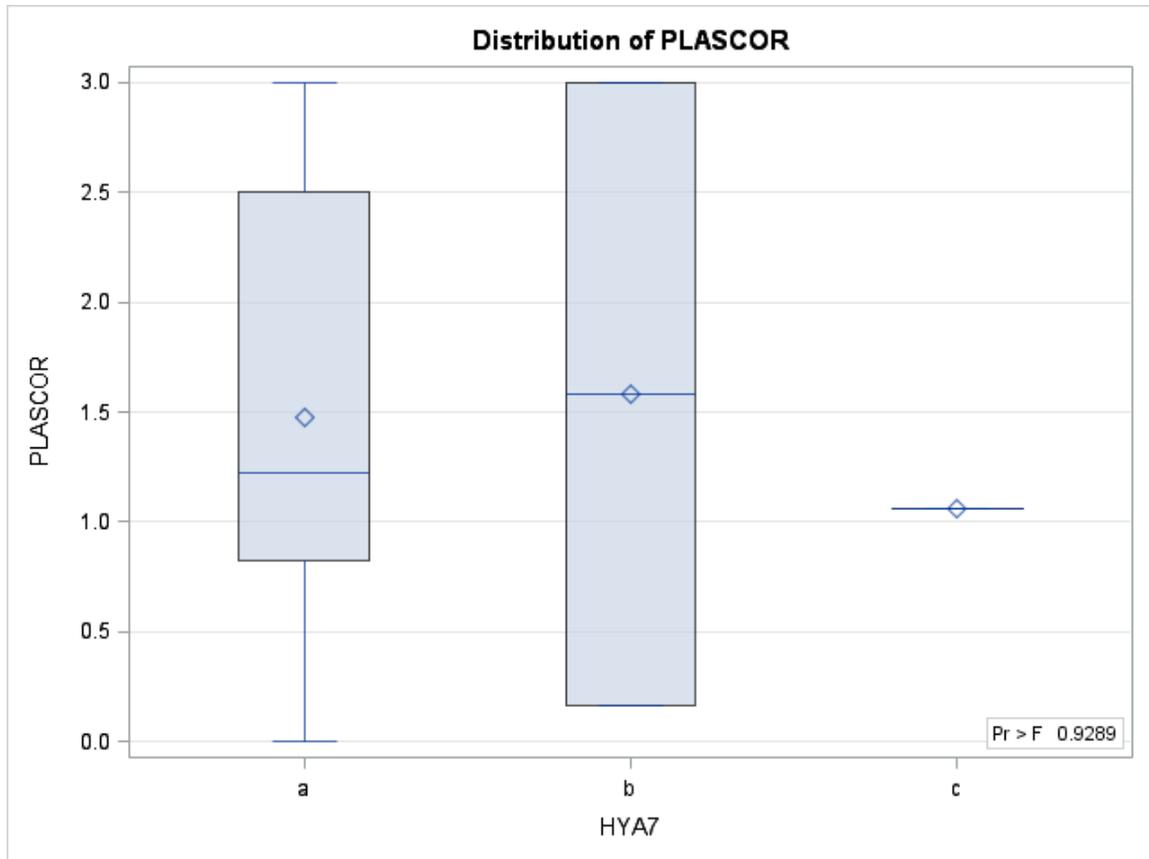


Table 24: Box-and-Whisker plot for the frequency of intake of regular energy drinks and plaque scores.



5.11H HYA8 with WSL and Plaque Scores

We assessed white spot lesions and plaque scores for Question 8 “Frequency of intake of smoothies” and determined the mean scores between subject answers in Table 25.

Table 25: Frequency of intake of smoothies and the corresponding plaque scores and caries severity (DS 1 and 2) of white spot lesions.

HYAFFQ Question 8					
	8a (n=6)	8b (n=7)	8c (n=3)	8d (n=2)	8e (n=2)
White Spot Score	7.17	3.57	1.33	6.00	10.00
Plaque Score	1.71	1.44	1.09	0.66	2.18

For the white spot and plaque scores, differences were not significant and the P-values were 0.36 and 0.60, respectively.

Table 26: Box-and-Whisker plot for the frequency of intake of smoothies and white spot scores.

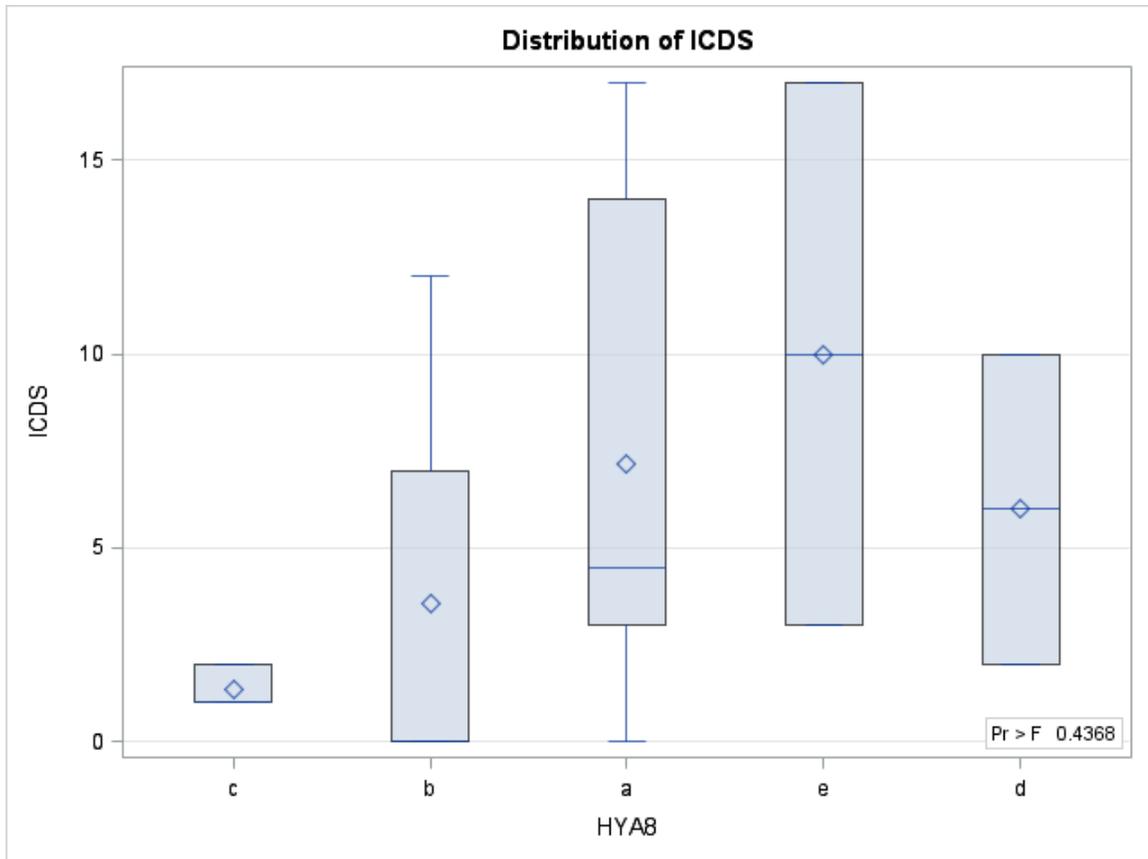
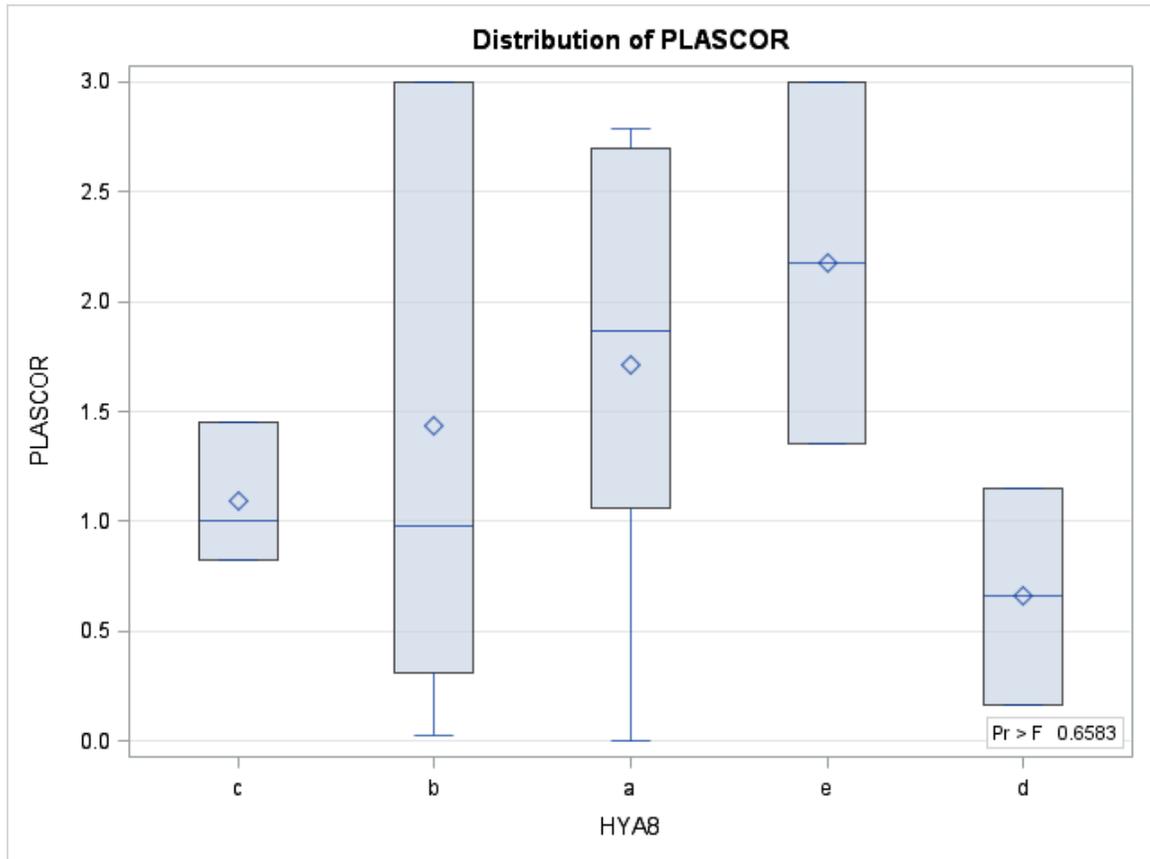


Table 27: Box-and-Whisker plot for the frequency of intake of smoothies and plaque scores.



5.12 Oral Hygiene Frequency Questionnaire

We assessed the frequency of the different survey answers with white spot lesions and plaque scores.

	0 A	1 B	2 C	3 D	4 E	5+ F
1. How many times a day do you brush your teeth?						
2. How many times a day do you floss your teeth?						
3. How long do you brush your teeth? (minutes)						
4. How many times a day do you eat sweets or drink a sweetened drink?						

5.12A OHF1 with WSL and Plaque Scores

We assessed white spot lesions and plaque scores for Question 1 “How many times a day do you brush your teeth?” and determined the mean scores between subject answers in Table 28.

Table 28: Tooth brushing per day and the corresponding plaque scores and caries severity (DS 1 and 2) of white spot lesions.

OHF Q1		
	1b (n=3)	1c (n=17)
White Spot Score	3	1.47
Plaque Score	1.69	1.41

For the white spot and plaque scores, differences were not significant. The P-values were 0.49 and 0.93, respectively.

Table 29: Box-and-Whisker plot for the frequency of diet soda/pop intake and white spot scores.

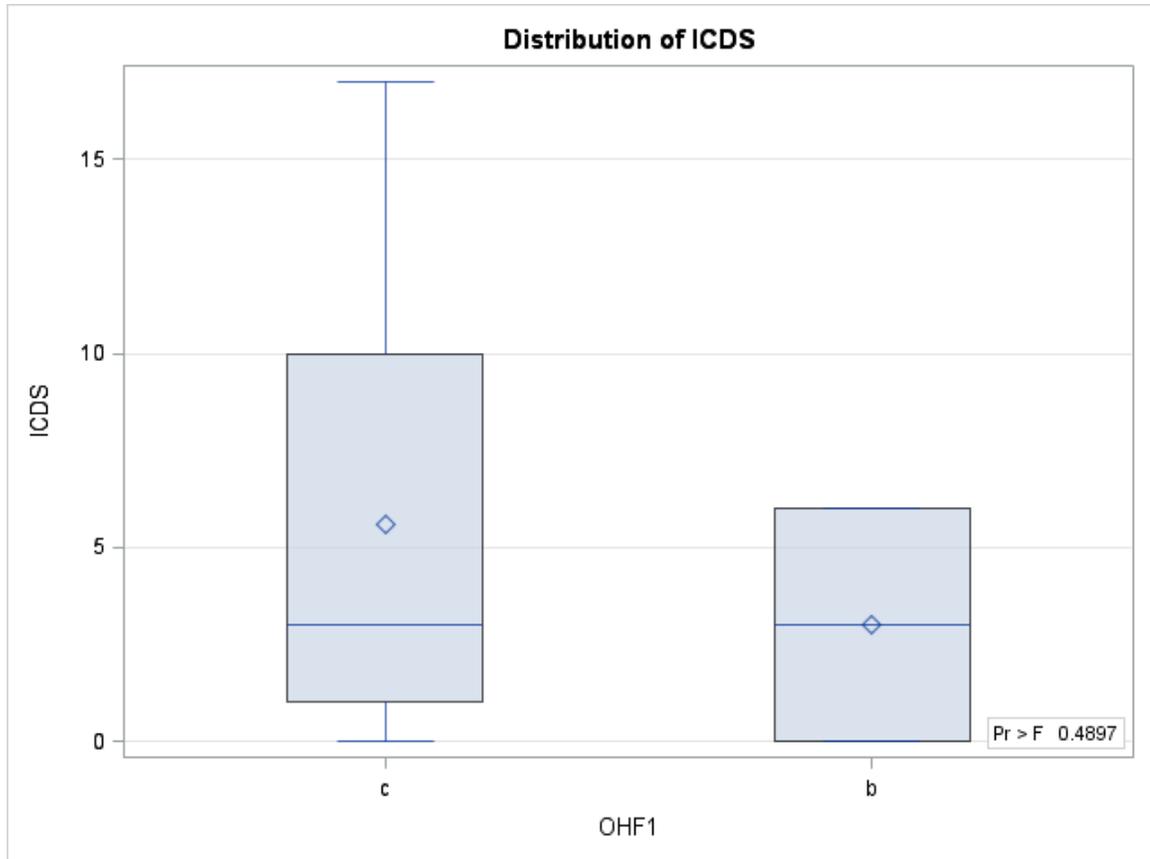
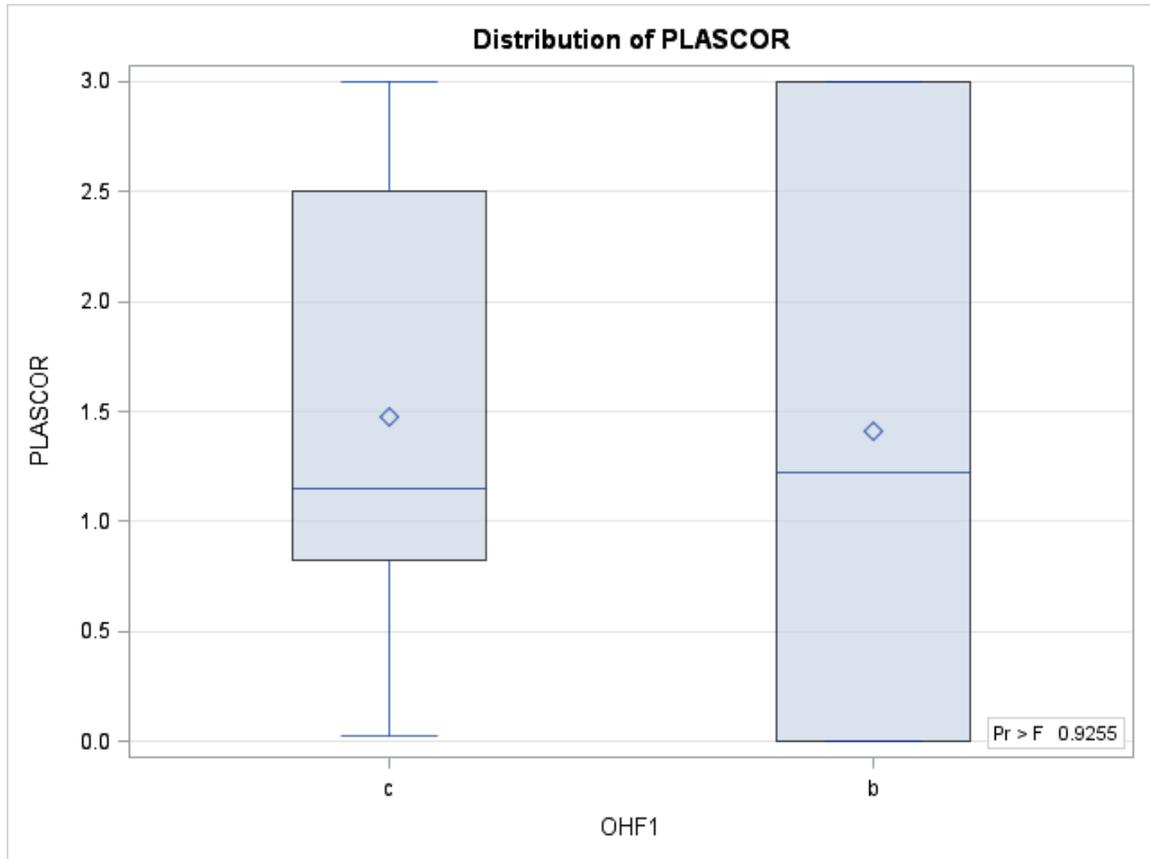


Table 30: Box-and-Whisker plot for the frequency of diet soda/pop intake and white spot scores.



5.11B OHF2 with WSL and Plaque Scores

We assessed white spot lesions and plaque scores for Question 2 “How many times a day do you floss your teeth?” and determined the mean scores between subject answers in Table 31.

Table 31: Flossing per day and the corresponding plaque scores and caries severity (DS 1 and 2) of white spot lesions.

OHF Q2			
	2a (n=11)	2b (n=6)	2c (n=3)
White Spot Score	8.09	2.00	1.00
Plaque Score	1.92	1.04	0.62

For the white spot scores differences were statistically significant with a p value of 0.04.

For plaque scores differences were not significant with a p value of 0.09.

Table 32: Box-and-Whisker plot for the frequency of flossing and white spot scores.

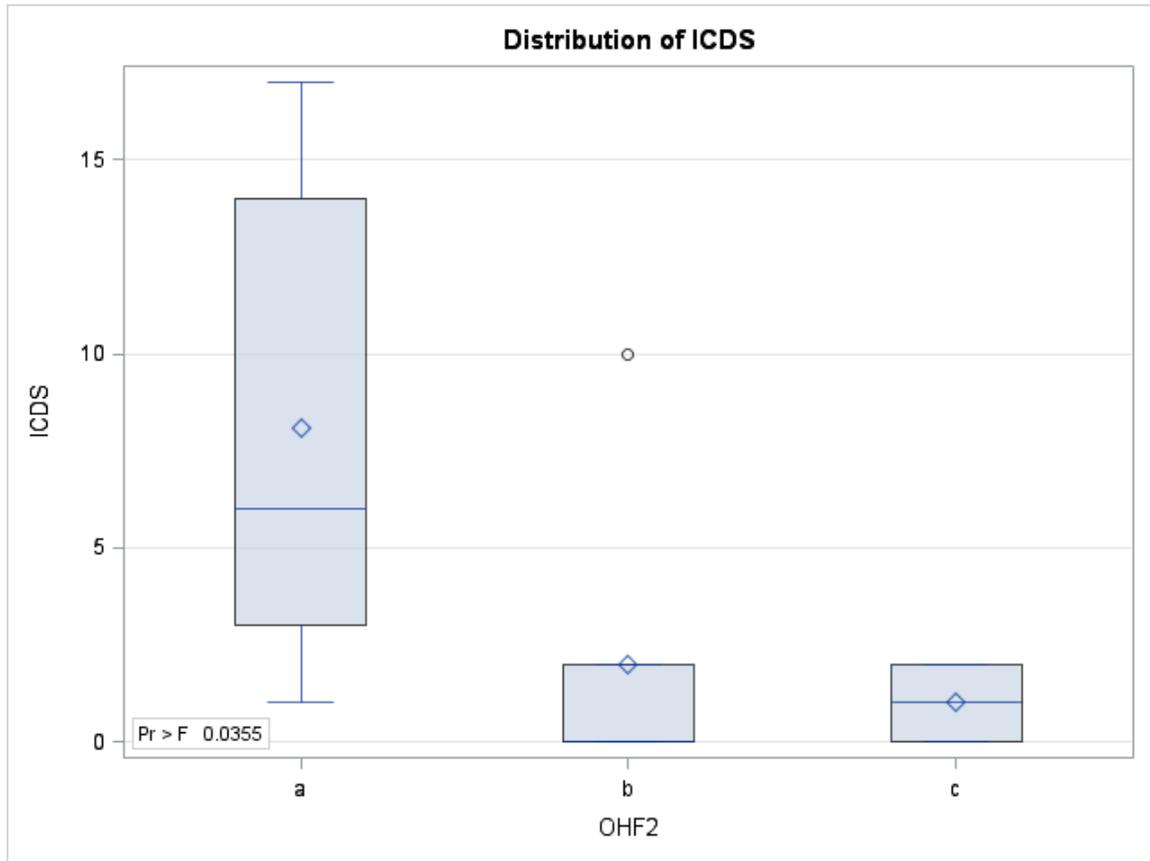
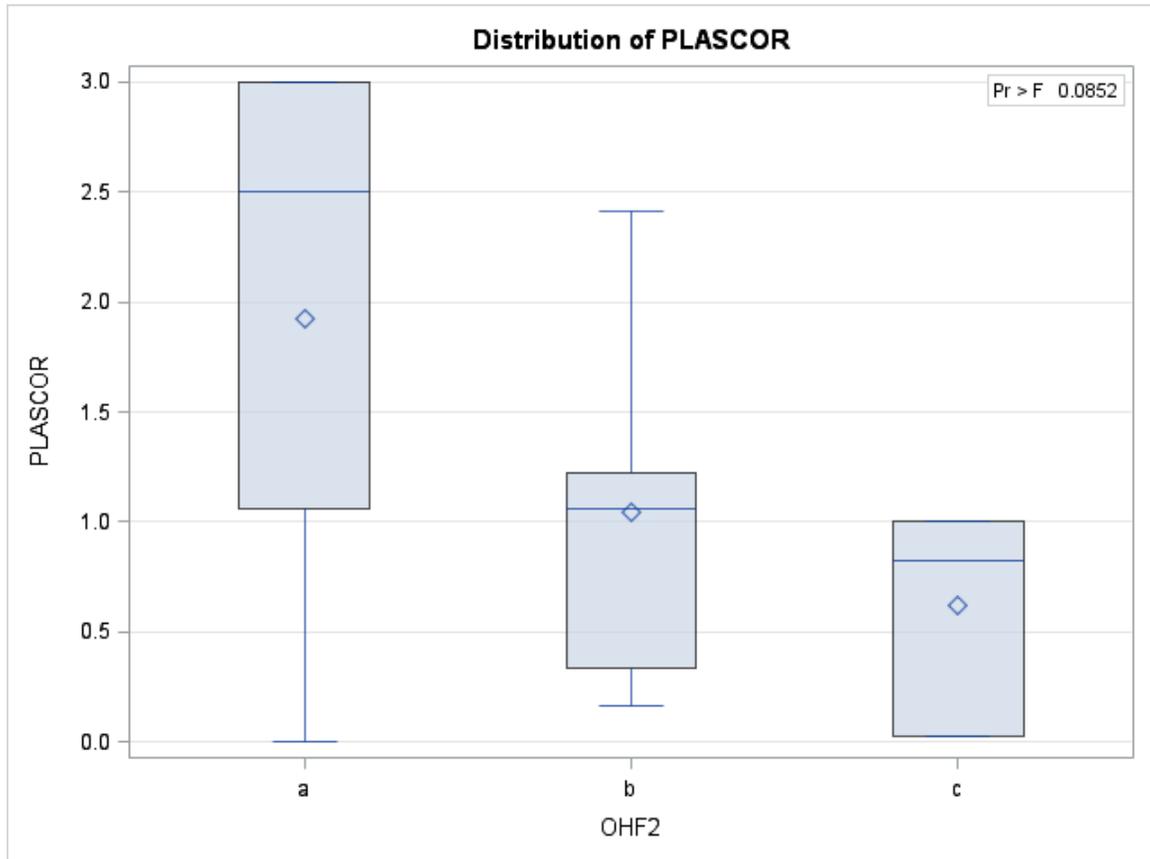


Table 33: Box-and-Whisker plot for the frequency of flossing and white spot scores.



5.11C OHF3 with WSL and Plaque Scores

We assessed white spot lesions and plaque scores for Question 3 “How long do you brush your teeth (minutes)?” and determined the mean scores between subject answers in Table 34.

Table 34: Brushing in minutes and the corresponding plaque scores and caries severity (DS 1 and 2) of white spot lesions.

OHF Q3					
	3b (n=11)	3c (n=9)	3d (n=4)	3e (n=4)	3f (n=2)
White Spot Score	1.00	6.89	4.75	5.50	0.00
Plaque Score	1.45	1.72	1.03	1.38	1.38

For the white spot and plaque scores, differences were not significant. The P-values were 0.61 and 0.91, respectively.

Table 35: Box-and-Whisker plot for brushing time in minutes and white spot scores.

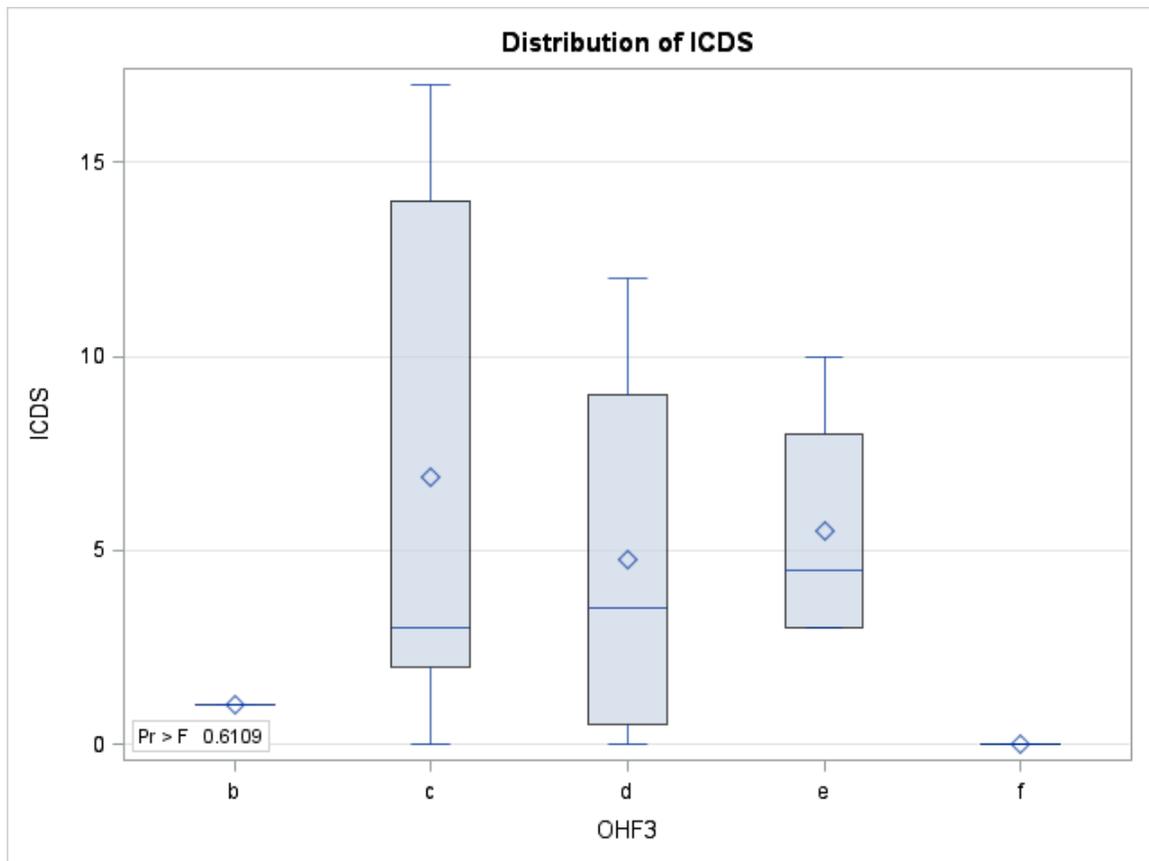
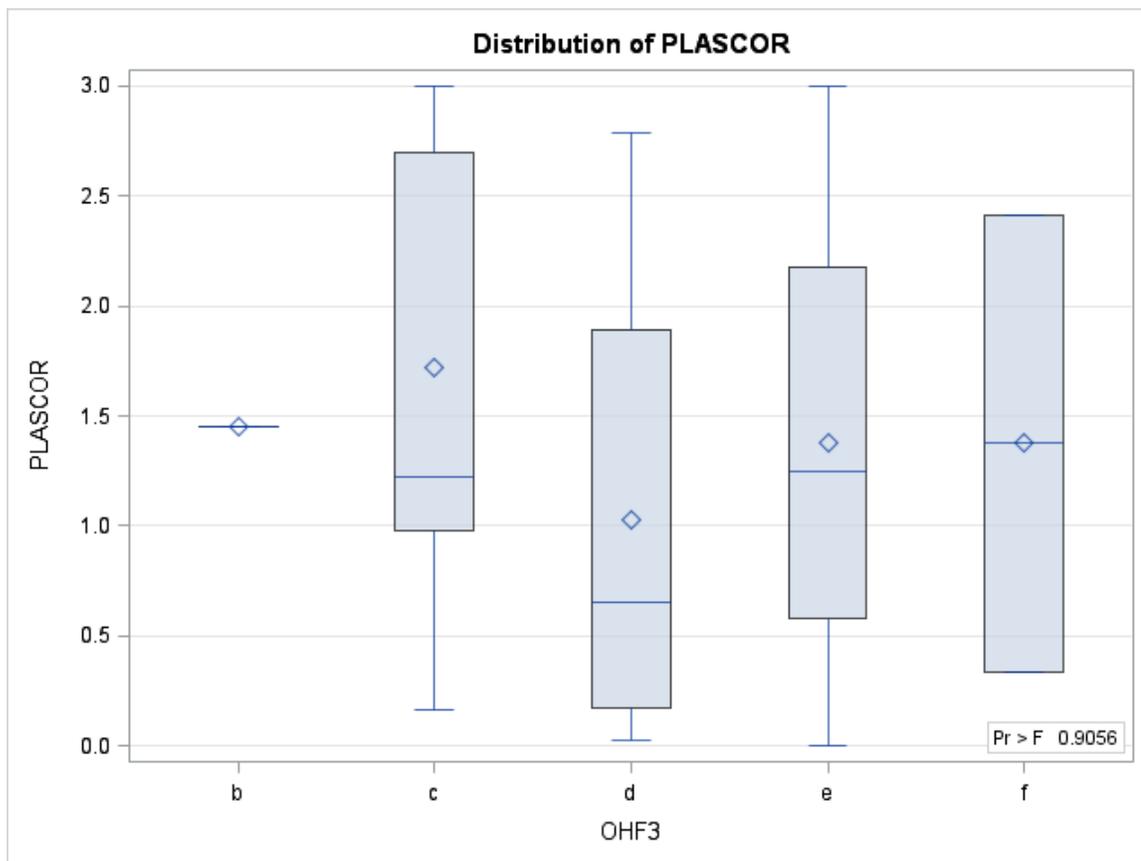


Table 36: Box-and-Whisker plot for brushing time in minutes and plaque scores.



5.11D OHF4 with WSL and Plaque Scores

We assessed white spot lesions and plaque scores for Question 4 “How many times a day do you eat sweets or drink a sweetened drink?” and determined the mean scores between subject answers in Table 33.

Table 37: Daily Consumption of sweets and drinks and the corresponding plaque scores and caries severity (DS 1 and 2) of white spot lesions.

OHF Q4						
	4a (n=4)	4b (n=3)	4c (n=4)	4d (n=1)	4e (n=2)	4f (n=1)
White Spot Score	4.25	3.85	8.25	3.33	0.00	17
Plaque Score	2.00	1.03	1.70	1.41	0.34	2.70

For the white spot and plaque scores, differences were not significant. The P-values were 0.23 and 0.48, respectively.

Table 38: Box-and-Whisker plot for daily sweets consumed.

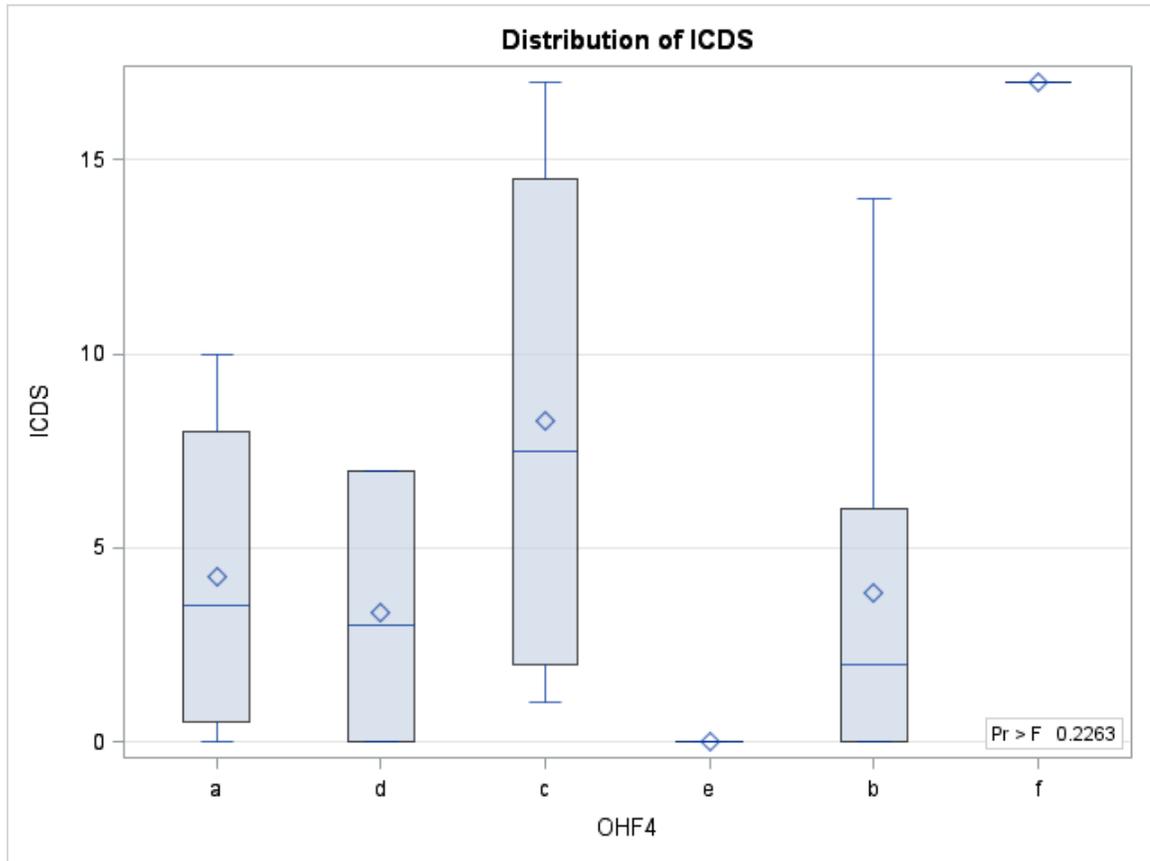
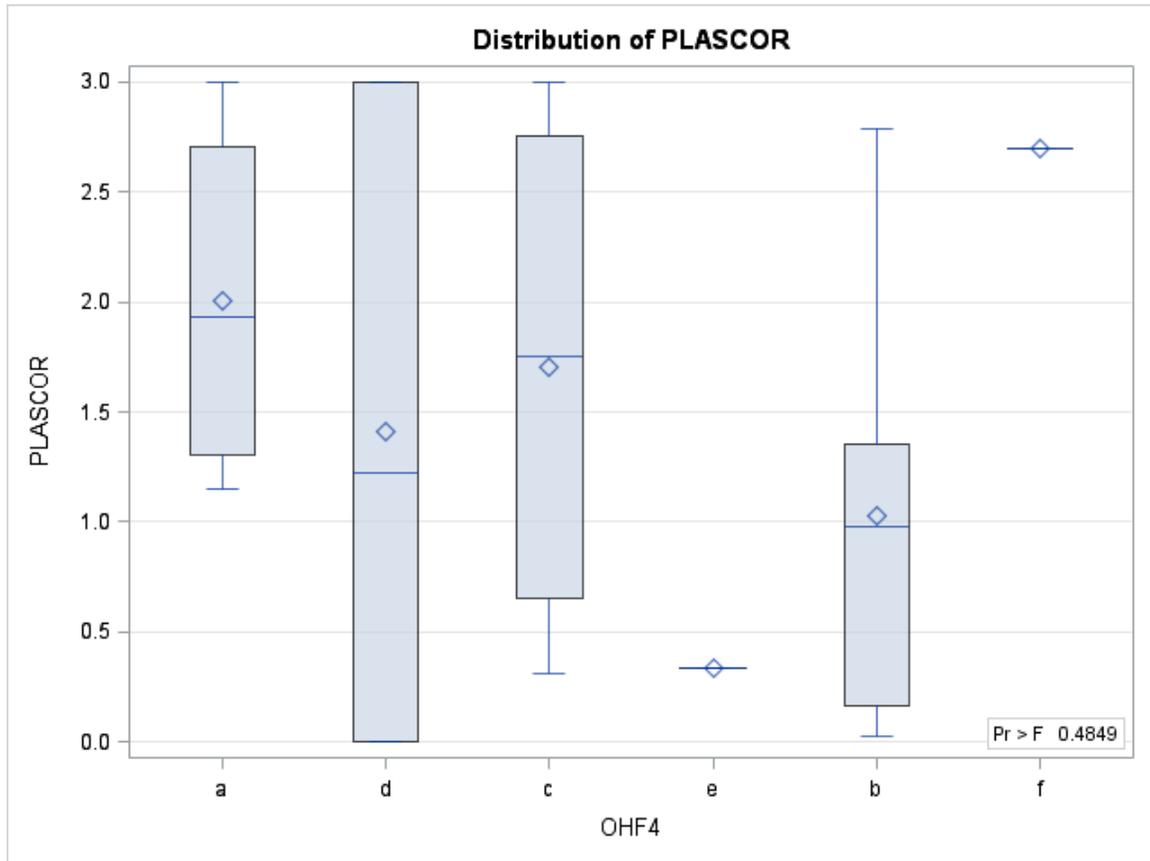


Table 39: Box-and-Whisker plot for brushing for daily sweets consumed.



CHAPTER 6

DISCUSSION

In this pilot study the sample size was limited due to time constraints created by a difficult patient enrollment process. The current small sample size may not be sufficiently powered to detect statistical significance in the tests that were conducted. The prevalence WSL among this patient population was 75% which fits within the normal range of WSL expected in an orthodontic practice (Heymann et al. 2013). Ten percent of the subjects had only the primary stage of WSL (visible after air drying), with thirty-five percent having only the later stages (visible on wet tooth), and thirty percent having a combination of both. Forty percent of the subjects having at least one primary WSL indicates that there is potential for long term incidence evaluation to see if they progress or re-mineralize. If more patients could be enrolled the prevalence of white spot lesions should be similar and better significance may be achievable.

6.1 Feasibility of Patient Population for Long Term Study

The chief purpose of this study was to determine if the graduate orthodontic patient population at Temple University is feasible for a long-term clinical trial on WSL. The prevalence of WSL in this patient population was 75% which when applied to a larger sample size will allow for significant data collection.

After approximately six months, forty patients were enrolled, and T0 could be collected on only twenty of the patients. The problems with this patient population are

numerous however it is the opinion of the author that these do not make the success of a study un-achievable.

The first issue was parent/guardian acceptance. Parents and guardians were hesitant to allow their sons or daughters to participate. Approximately a fifth of the parents who were approached about their child participating were apprehensive about the validity and safety of the study. A second subgroup of parents felt that the time requirement of forty-five minutes to an hour was far too long for them to build into their already busy schedules. Once consents were signed, scheduling the appointment for data collection created further difficulties with many parent's not returning calls or being uninterested in making additional appointments. It was difficult to get resident assistance in notifying the data collectors if their patients that were involved in the study would be coming in. One suggestion would be to pay the parents or children involved in the study a small fee in order to have them compliant with attending appointments.

6.2 Patient age and Number of Untreated Decayed Surfaces Including ICDAS Codes 1 and 2

There was a moderately strong statistically significant correlation that as a patient's age increases so does their prevalence of WSL. This can be explained by one of two theories. First, as a patient ages their enamel structure changes causing them to be more susceptible to white spots (Park et al. 2008). The second explanation is that as patient age, if they maintain the same risk profile, they would accumulate a greater number of lesions (Chapman et al. 2008). This cannot be disproved in this study because no examination for WSL was done prior to bonding. In the subsequent time points of this

study the incidence of WSL can be calculated, and one of these theories may be further supported.

6.3 Patient gender and Number of Untreated Decayed Surfaces Including ICDAS Codes 1 and 2

There was no statistically significant correlation between gender and caries severity (DS codes 1 and 2). These results were most likely due to the small sample size of the study. A multitude of studies with subjects ranging from 37-45 have shown statistical significance for males having increased prevalence of WSL. (Boersman et al. 2004; Chapman et al. 2008; Khalaf et al 2014; Tufekci 2011;). These studies have double the amount of patients, demonstrating a larger sample size may produce statistically significant results. The differences in prevalence is thought to stem from higher hygiene standards in females than males (Ostberg et al. 1999) with most research showing there is little if any structural difference in the enamel of males and females (Astekar 2014).

6.4 BMI Percentile and Number of Untreated Decayed Surfaces Including ICDAS Codes 1 and 2

As shown in figure, the subjects had BMI percentiles that closely parallel the national averages (Odgen et al. 2010).

	Under Weight <5 th percentile	Healthy Weight 5 th to 85 th Percentile	Overweight 85 th to <95 th percentile	Obese ≥95 th Percentile
National averages (percent of population)	2.8%	60.8%	18.9%	17.5%
Patient Population (Percent of population)	10%	50%	20%	20%

Table 40: National and study BMI percentiles

Although our distribution is useful for making comparisons, there was little to no correlation and the results were not statistically significant. This agreed with select studies that found there was no correlation between white spot lesions and a patients BMI percentile (D’Mello 2011; Tramini et al. 2011). The majority disagree, and have found a statistically significant correlation. One study from Germany looked at 2071 primary school students and found correlations between high BMI and higher caries prevalence, as well as low BMI having a lower prevalence of carious lesions (Willerhausen et al 2007). The mechanism by which BMI may cause an increase in caries is debated with some studies linking increased sugar consumption with higher BMIs (Tramini et al. 2011, Marshall et al. 2007), interestingly salivary *S. Mutans* does not appear to be affected by a patients diet (Belstrom et al 2014).

6.5 Plaque *S. Mutans* with Number of Untreated Decayed Surfaces Including ICDAS Codes 1 and 2

S. Mutans showed little to no correlation with caries severity. A high p-value of .99 causes the correlation data to be meaningless. In multiple other studies the quantity of Plaque *S. Mutans* has been shown to be correlated with white spot lesions, with *S. Mutans* levels increasing up to fivefold during orthodontic treatment (Ren et al. 2014; Sudjalim et al. 2006; Mattingly et al. 1983). The fixed appliances of orthodontics create a mechanical difficulty in patient hygiene and foster an environment for biofilm proliferation (Ren et al. 2014).

6.6 Saliva *S. Mutans* with Number of Untreated Decayed Surfaces Including ICDAS Codes 1 and 2

Saliva *S. Mutans* showed an inverse correlation with caries severity. A high p-value of .69 indicated that this was not statistically significant. Studies have shown a direct correlation of salivary *S. Mutans* levels with prevalence of dental caries (Ren et al. 2014; Woo-Sun et al. 2014) because increased acidogenic bacteria will increase the daily challenges to the enamel. Therefore the inverse correlation in this study is likely a result of a small sample size.

6.7 Plaque Scores with Number of Untreated Decayed Surfaces Including ICDAS Codes 1 and 2

Plaque scores showed a low correlation with caries severity (DS codes 1 and 2) which although not statistically significant (p value of .065) is reasonably high for such a

small sample. Previous studies have shown “an almost linear correlation between plaque accumulation and the development of caries” in orthodontic patients (Ren et al. 2014; Zachrisson et al. 1971).

6.8 SOC Scores with Number of Untreated Decayed Surfaces Including ICDAS Codes 1 and 2

Sense of coherence scores showed little if any correlation with caries severity (DS codes 1 and 2). The high p-value of .72 showed that this was not statistically significant. This is not in-line with other studies that demonstrate that other measures of oral health status such as increased prevalence of caries were strongly correlated in adolescents (Friere et al. 2001). A weak sense of coherence increases the likelihood of poor oral health and is tied strongly to a tooth brushing frequency of less than once a day (Savolainen et al., 2005; Bernabe et al. 2010).

6.9 SOC Scores with Plaque Scores

Sense of coherence scores showed little if any correlation with plaque scores. This is opposite of other studies that show increased plaque scores were strongly correlated with high sense of coherence scores and is likely due to the small sample size (p value of .73) (Friere et al. 2001). Low SOC scores increases the likelihood of missing appointments, decreased brushing frequency, and greater prevalence of plaque (Bernabe et al. 2010).

6.10 Frequency of Diet Soda Intake with Number of Untreated Decayed Surfaces Including ICDAS Codes 1 and 2

The frequency of diet soda intake showed little to no correlation with caries severity and the p-value of 0.58 demonstrated this data was not statistically significant. However, as the frequency of intake increased, the caries severity increased as well. Thus, if the sample size was larger for this study, the results may become significant following a similar trend.

6.11 Frequency of Diet Soda Intake with Plaque Scores

The frequency of diet soda intake also showed little if any correlation with the plaque scores. A p-value of 0.53 was achieved with this study, illustrating results that were not statistically significant. For the most part, the results demonstrated that as the intake of diet soda increased, the plaque scores did as well. However, the one patient who drank two bottles per day had a lower mean plaque score than the additional groups. It would make sense for the plaque scores to increase with a higher frequency of soda intake, and the possible cause of the results being not significant may have been due to the small sample size.

6.12 Frequency of Non-Diet Soda Intake with Number of Untreated Decayed Surfaces Including ICDAS Codes 1 and 2

Increased frequency of non-diet soda intake had a slight non-significant correlation with an increased caries severity for WSL (DS 1 + 2). The current literature supports this relationship, and it is believed that a diet high in sugar will cause the

bacteria in plaque such as *S. Mutans* to create acidic challenges to the enamel which lead to white spot lesions (Khalaf et al. 2014; Kidd 2011).

6.13 Frequency of Non-Diet Soda Intake with Plaque Scores

There was little to no correlation between the frequency of non-diet soda intake and plaque scores. There was a p-value of 0.46, which showed that the results were not statistically significant. One would think that the plaque scores would increase with greater exposure to non-diet soda, as sugar can contribute to the formation of plaque by providing a means of adhering to the tooth's surface (Duany et al. 1972). However, due to the small sample size in this study, these trends were not demonstrated.

6.14 Usual Serving Size and Number of Untreated Decayed Surfaces Including ICDAS Codes 1 and 2

The usual serving size of sodas/pop had no correlation with the caries severity (DS codes 1 and 2); it should be kept in mind that these results were not statistically significant and because of the low sample size should not be taken at face value. Although it would make logical sense that since sugar has been shown in multiple studies to be correlated with increased risk of caries, the literature actually supports that serving size has little correlation, and instead the serving frequency is the more important factor (Heller et al. 2001).

6.15 Usual Serving Size and Plaque Scores

In this study, the results were not statistically significant, but the usual serving size of sodas/pop was shown to have no correlation with the patient's plaque scores.

Although it would make logical sense that since sugar has been shown in multiple studies to be correlated with increased risk of caries, the literature actually supports that serving size has little correlation, and instead the serving frequency is the more important factor (Heller et al. 2001).

6.16 Frequency of Sugared Drink and Non-Carbonated Fruit Drink Intake and Number of Untreated Decayed Surfaces Including ICDAS Codes 1 and 2

The frequency of the intake of sugared drinks and non-carbonated fruit drinks had little to no correlation with the caries severity (DS codes 1 and 2). The p-value was 0.06, which was not a statistically significant result, although high for such a small sample size. The results were also varied, possibly due to such a small and diverse patient population. Previous studies have shown direct correlation between frequency of sugared drink consumption and incidence of WSL (Khalaf et al. 2014).

6.17 Frequency of Sugared Drink and Non-Carbonated Fruit Drink Intake and Plaque Scores

For the patient population in this study, the frequency of intake of sugared and non-carbonated fruit drinks had very little if any correlation to the plaque scores. The p-value was 0.17, which was not statistically significant. Again, the results for this study were variable which may have been due to the small sample size. In most studies there has been a direct correlation with frequency of sugared drinks and a patients dental plaque (Khalaf 2014).

6.18 Frequency of Sports Drink or Sugar-Sweetened Vitamin Water Intake and Number of Untreated Decayed Surfaces Including ICDAS Codes 1 and 2

The frequency of intake of sports drinks or sugar-sweetened vitamin water showed little correlation to the caries severity (DS codes 1 and 2). The p-value of 0.83 illustrated the results were not statistically significant. Some of the data seems to follow a general trend where increased frequency of intake leads to higher mean white spot scores. However, the results may seem variable due to the small sample size. It is expected that sports drink which on average contain five teaspoons of sugar per twelve ounces of beverage could easily lead to an increased prevalence of WSL for the same reasons as sugared soda.

6.19 Frequency of Sports Drinks or Sugar-Sweetened Vitamin Water Intake and Plaque Scores

Throughout this study, the frequency of intake of sports drinks or sugar-sweetened vitamin water had little to no correlation with plaque scores. The results were not statistically significant, producing a p-value of 0.36. In addition, the data did not really produce any trend, providing variable results due to such a small sample size.

6.20 Frequency of Intake of Sugar-free or Low Calorie Energy Drinks with the Number of Untreated Decayed Surfaces Including ICDAS Codes 1 and 2

The frequency of intake of sugar-free or low calorie energy drinks had little if any correlation with the caries severity (DS codes 1 and 2). A p-value of 0.19 showed that the results were not statistically significant. The results however did illustrate that with an

increased intake frequency of these drinks, there was a higher prevalence for white spot lesions. This would make sense, as the increased intake would lead to a higher amount of acid produced by plaque bacteria, and result in the formation of white spot lesions.

6.21 Frequency of Intake of Sugar-free or Low Calorie Energy Drinks with Plaque Scores

There was little to no correlation for the frequency of intake and the plaque scores. The p-value was 0.79, which illustrated that the results were not statistically significant. These results seem to show that regardless of the frequency of intake of these types of drinks, the plaque scores are similar.

6.22 Frequency of Intake of Regular Energy Drinks with Number of Untreated Decayed Surfaces Including ICDAS Codes 1 and 2

In this study, the frequency of intake of regular energy drinks had little if any correlation with the caries severity (DS codes 1 and 2). A p-value of 0.26 demonstrated that the results were not statistically significant. The results showed that with an increased intake frequency of these drinks, there was a higher prevalence for white spot lesions. This would illustrate a similar trend as before, where an increased intake of sugar would lead to an increase in demineralization by acid-producing plaque bacteria.

6.23 Frequency of Intake of Regular Energy Drinks with Plaque Scores

There was little to no correlation for the frequency of intake of regular energy drinks and the plaque scores. The results were not statistically significant, producing a p-

value of 0.96. These results seem to show that plaque scores are similar regardless of intake of regular energy drinks.

6.24 Tooth Brushing Per Day and Corresponding Number of Untreated Decayed Surfaces Including ICDAS Codes 1 and 2 and Plaque Scores

From this data it appears that brushing twice per day lowers a patient's caries severity, which although these results were not statistically significant agrees with the current literature on the subject (Chapman et al. 2010). Brushing disrupts a patient's biofilms and prevents plaque from reaching a mass capable of prolonged acidic challenge to the enamel of the tooth (Khalaf et al. 2014).

6.25 Flossing Frequency and Corresponding Number of Untreated Decayed Surfaces Including ICDAS Codes 1 and 2 and Plaque Scores

This study shows interesting results where as a patient increases their flossing they have a statistically significant decrease in caries severity for DS codes 1 and 2, and a close to statistically significant (p value of .09) decrease in plaque on their teeth. This may be because patients that floss have a better overall level of oral hygiene; however it is worth closer examination that flossing may be important in preventing white spot lesions. A review of the literature has shown that no studies on the effect of flossing on caries severity for DS codes 1 and 2 have at this time been completed.

6.26 Duration of Brushing (minutes) and Corresponding Number of Untreated Decayed Surfaces Including ICDAS Codes 1 and 2 and Plaque Scores

The duration of brushing in minutes had no significant impact on the caries severity (DS codes 1 and 2) or the patient's plaque scores. This is further illustrated by the plaque scores ranging from 1.03 for 3 minutes of brushing to 1.72 for two minutes of brushing. This is contrary to what is found in the literature where it is generally supported that duration of brushing has a strong influence on the plaque found (Honkala et al. 1982). The reason for the conflicting data is most likely because of our lower sample size and very high p-values.

6.27 Daily Consumption of Sweets and Drinks and the Corresponding Plaque Scores and Number of Untreated Decayed Surfaces Including ICDAS Codes 1 and 2

Daily consumption of sweets and drinks has been shown in the literature to increase the likelihood of carious lesions and therefore in theory should show an increase in white spot lesions (Leroy et al. 2005). In this study however we found no correlations between the intake of sweets and sugary drinks and a patient's prevalence of white spot lesions or plaque scores. This is most likely due to our small sample size and fits with our insignificant p-value.

CHAPTER 7

CONCLUSIONS

- The Orthodontic program at Temple University has a population that may be feasible for long term studies as long as the study is designed appropriately.
- The prevalence of WSL at Temple is 75% of patients.
- Patient age has a moderately strong direct correlation with white spot lesions.
- Plaque scores may be correlated with caries severity of white spot lesions.
- Frequency of sugary drinks may have no correlation with caries severity of white spot lesions.
- Flossing is correlated with decreased caries severity of white spot lesions, and may be correlated with decreases plaque scores.

CHAPTER 8

FUTURE DIRECTIONS

This study suffered from two main flaws: patient recruitment and patient retention. Patient recruitment involved the researchers screening the patients and finding out pertinent information such as treatment duration, while actively treating their own patients.

Once the patients that fit the enrollment were identified, the next problem was having a parent or guardian present to sign the consents. Many of the parents/guardians of this clinic's patients do not routinely come to their child's appointments, and almost none were willing to change for the sake of enrollment into the TOWs study. The parent/guardian then had to be willing to add extra appointments for data collection, with the value in the study being the identification and treatment of the patient's white spots, which unfortunately swayed few. Once data collection began, it became apparent that the actual duration of each appointment was far past what most patients deemed acceptable, sometimes lasting for upwards of two hours, although it should be noted that by the end of the study the average appointment time was only about sixty minutes. The attrition rate of patients was severe, and most patients never passed the T0 data collection.

It is the opinion of this researcher that with several fundamental changes, this research could be streamlined into a more practical study. Patient recruitment lasted approximately three months longer than originally predicted, and the goal for enrollment was never achieved. This was due to difficulty finding patients that fit the enrollment criteria and had parents willing to show up to the appointments to sign the consents. The

solution to this problem would be to enroll the patient into the study earlier in treatment during the consult appointment. The consult appointment works for several reasons. The parent has to be present for treatment to begin, the patient and parent are both highly motivated (Pramod 2010), and the patient will adjust to having two appointments per month without the feeling of their appointments suddenly doubling.

Offering payment or rewards for participation in the study could motivate parents to allow their children to participate. There are three recognized models for compensating patients to participate in research. The first is the market model, which is used when a study is risky and has little direct benefit to the patient. It often involves a large compensation in order to foster participation. The second model is the reimbursement model, it will pay a patient/guardian enough to cover all their expenses. In this study it would be to pay transit and any parking fees. It is useful when the study offers a large benefit to the patient and has little inconvenience. The final model is the wage payment model. It pays the patient/guardian a small hourly wage for participating in the study. It has been shown to be the most effective method (Dickert 1999), and has the added benefit of compensating patients more the longer their appointments last. This directly addresses our chief issue, appointment duration.

The actual duration of data collection was too long without payment to keep patients motivated to come to these extra appointments. Parents complained about needing to pay for extra parking, and the subjects became restless after approximately forty-five minutes. Parking can be handled in one of two ways: first the researchers could compensate the parent (reimbursement method), or second the researchers could allow the parent to use the dental school valet parking system free of charge. This would

eliminate the parent from needing to find a parking spot, spending money at the meter, or the possibility of parking tickets.

The study should be streamlined to collect data in the most efficient method possible. Although the appointment flow is logical, alterations in the collection order can be tested for shorter appointments. Taking measures to make the collections appointment appear shorter would also benefit. One method would be to have the patient fill out the surveys in the lobby prior to being checked in, which would decrease the time the patient is actually in a clinic chair.

Although this study presents difficulties, they are all of the patient/guardian, and the underlying protocol is sound. If the problems of patient enrollment and attrition can be solved, Temple's Orthodontic clinic offers a viable patient population for further research.

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APPENDICES

APPENDIX A

Condensed Patient Data

Patient ID	Treatment Duration	Age T0	Sex	Height	Weight	BMI	BMI Percentile	Plaque Score	ICDAS CODE 0	ICDAS CODE 1	ICDAS CODE 2	ICDAS CODES 1&2	SOC Scores	Salivary S-Mutans	Plaque S-Mutans
111	405	15	F	176.0	54.6	17.6	16.6	1.45	79	0	1	1	77	2.5	2.63
135	816	14	M	172.0	72.6	24.4	91.6	3.00	65	0	7	7	53	2	2.13
218	205	15	F	163.0	49.9	18.8	31.8	0.31	68	0	12	12	71	0	0.38
302	620	12	F	161.0	48.4	18.7	61.0	3.00	74	4	2	6	53	0	1.00
319	600	12	F	159.0	69.2	27.4	96.2	0.34	80	0	0	0	55	0	0.00
444	82	12	F	166.0	34.8	15.9	14.9	2.41	80	0	0	0	64	3	2.38
470	122	14	M	150.4	33.0	16.8	11.8	2.79	74	3	3	6	66	1	0.38
448	150	11	F	157.0	34.8	14.1	2.0	1.00	79	1	0	1	51	1	1.50
541	241	16	M	172.7	83.0	27.8	95.1	1.35	77	0	3	3	62	2	2.25
641	318	18	F	155.5	74.3	30.7	95.0	1.15	62	0	10	10	60	0.5	1.13
645	314	14	F	155.5	52.7	21.8	76.4	0.16	78	0	2	2	56	1	2.13
662	350	17	M	189.0	93.2	26.1	90.1	2.70	63	3	14	17	60	3	2.88
670	244	17	F	161.0	71.1	27.4	91.4	0.83	78	2	0	2	30	1	1.25
678	1058	17	F	172.5	67.9	22.8	66.4	2.51	77	2	1	3	80	2	1.50
706	734	18	F	163.1	53.0	19.9	29.7	3.00	47	7	10	17	65	2	1.75
711	659	11	F	174.0	73.7	24.3	94.2	0.00	77	0	3	3	59	2	2.25
726	90	14	F	162.5	36.2	15.0	1.1	0.03	80	0	0	0	77	3	1.25
730	561	12	M	179.0	86.8	27.1	97.3	0.98	80	0	0	0	64	1.5	1.63
989	118	14	M	170.0	52.6	18.2	27.6	1.06	66	9	5	14	58	3	2.88
999	363	14	F	154.0	41.4	17.5	22.2	1.23	80	0	0	0	51	3	3.00