PARENTAL STRESS AS A CO-MORBIDITY OF SEVERE EARLY CHILDHOOD CARIES

MASTERS THESIS

Presented in Partial Fulfillment of the Requirements for the Degree of Masters of Science in the Graduate School of The Ohio State University

By

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ABSTRACT

Severe early childhood (S-ECC) caries has classically been studied as a disease, whose initiation and progression is determined by biological risk factors. In recent years S-ECC has begun to be associated to numerous social and psychosocial environmental conditions, one of which is parental stress. Our study set out to investigate the association of parental/caregiver stress as a co-factors to S-ECC prevalence. Using the Parental Stress Index-Short Form we determined the stress of primary caregivers of young children who were classified under clinical exam as either having no caries or S-ECC. Other social economic status determinants and classic biological and social co-factors were accounted for. Our results indicate a weak but positive association (p=.05) between parental stress and the presence and extent of S-ECC. Parents with high stress often share risk factors common to parents of children with S-ECC. Practitioners should be aware of this possible relationship and be prepared to provide appropriate intervention.
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PUBLICATIONS

Research Publications

1. Bradley JD, Cleverly DG, Burns AM, Helm NB, Schmid MJ, Marx DB, Cullen 
DM, Reinhardt RA. Cyclooxygenase-2 inhibitor reduces simvastatin-induced bone 
morphogenetic protein-2 and bone formation in vivo. J Periodontal Res. 2007 
Jun;42(3):267-73.

FIELDS OF STUDY

Major Field: Dentistry 
Pediatric Dentistry
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INTRODUCTION

Early Childhood Caries (ECC) is defined by the American Academy of Pediatric Dentistry as “the presence of 1 or more decayed (noncavitated or cavitated lesions), missing (due to caries), or filled tooth surfaces in any primary tooth in a child 71 months of age or younger”\(^1\). This definition is widely accepted and has been used in many other scientific studies\(^2,3\). ECC is a major public health epidemic and has risen 4% over the last decade in the United States\(^4\). Nearly 1/3 of all children in the U.S. (28%) have ECC. It is the most common chronic childhood disease and the nation’s most unmet health care need with over 4 million children affected\(^5,6\). A more severe form of ECC is Severe Early Childhood Caries (S-ECC) which is diagnosed when a child younger than 3 years of age has any sign of caries. It also includes children of ages 3 to 5 with 1 or more cavitated, missing (due to caries), or filled smooth surface in primary anterior teeth or a decayed, missing or filled teeth (dmft) score of \(>3\) (age 3), \(>4\) age 4 or \(>5\) (age 5)\(^1\).

**Biologic Mechanism of Caries**

Three major hypotheses have been described as the etiology of dental caries: the specific plaque hypothesis, the nonspecific plaque hypothesis, and the ecological plaque hypothesis\(^7,8,9\). While Each theory presents different mechanisms of the decay process, they all demonstrate that caries is a sequelae to bacterial infection which is modulated by
a plaque biofilm and exposure to fermentable carbohydrates. The specific plaque hypothesis claims that *S. Mutans* and *S. Sobrinus* are the primary causative agents. Other research suggest the other bacterial species such as *Veillonella*, *Lactobacillus*, *Bifidobacterium*, and *Propionibacterium*, low-pH non-*S. mutans* streptococci, *Actinomyces* spp. and *Atopobium* spp may play major roles in both the initiation and progression of the caries process\(^{10}\).

**Non-Biologic Pathogenesis of ECC**

With this heavy focus on the microbiological pathophysiology of ECC, researchers and public health officials have begun to recognize that there are many non-biologic factors that contribute to the progression of ECC. Literature in recent years has begun to examine extra-biological co-factors on the community, family and child-level\(^{21}\). This relatively new conceptual model has taken our understanding of ECC beyond the conventional Keyes Model of traditional infectious disease processes to include the understanding that ECC is often a “family disease”\(^{22}\). The recommendations of this literature state psychosocial intervention should be examined as a possible means of decreasing ECC, augmenting traditional methodologies.

The effects of community and other ECC related social factors were highlighted in The Oral Health in America: A report of the Surgeon General in 2000. This report stated
several social influences to oral health including data that reported children in poverty have a caries rate twice that of their non poverty counterparts and uninsured children are 2.5 times more likely to suffer from dental decay\textsuperscript{5}. Other research has supported this report by demonstrating that both income and education level are inversely related to ECC prevalence\textsuperscript{23,24,25}.

The most severe manifestations of familial stresses and their link to ECC have also begun to be studied. In 2008, Valencia-Rojas et al. reported that a history of maltreatment and/or neglect in the home was a positive indicator for ECC\textsuperscript{26}. They also discovered that child protective services intervention actually resulted in a positive effect on a child’s oral health. They demonstrated that children with a history of neglect or abuse who had a history of long term state custody were 87\% less likely to develop S-ECC than children in short term custody who returned to their original home. This study did contain several design weaknesses such as a small sample size and the study being conducted in an isolated location with high potential for recall bias but nonetheless suggests that dysfunctional family life can contribute significantly to ECC and overall decrease in childhood Quality of Life (QoL).

QoL is defined as “The patient's ability to enjoy normal life activities” by Webster’s Medical Dictionary. QoL also has been shown to be significantly affected by ECC. Not
only has ECC been shown to decrease measurable QoL, treatment of ECC has improved QoL\textsuperscript{27}. In a 2003 study, 22-70 month old children with ECC who received dental treatment had a significantly improved oral health-related QoL at the follow-up assessment when compared with their baseline measurement as measured by the Michigan Oral Health Related Quality of Life Scale. A key component of quality of life for a child is stability of family function. Studies have shown family dysfunction to contribute to illness’ of childhood. For example, respiratory illness and asthma has been linked to family dysfunction and maternal coping skills demonstrating a positive correlation with pathophysiology of disease and these psychosocial factors\textsuperscript{28,29}.

Other studies have attempted to link additional psychosocial factors to ECC. Some of these factors include social class and dental health locus of control (internal vs. external)\textsuperscript{30,31,32}. Each of the aforementioned factors has a small but growing body of literature behind it showing attitudes such as an external locus of control as a contributing factor to ECC development. Another major factor cited in recent literature is the interaction between the levels of a parents stress and their offspring’s caries experience\textsuperscript{33}.

\textit{Parental Role In Early Childhood Caries}

Existing literature has established that parents have direct impact on caries activity in children. While no single bacterial etiology has been identified as the causative agent of
ECC, it has been reported a vertical transmission fidelity as high as 80% between mother/child bacterial strains\textsuperscript{11}. Fathers have also demonstrated the ability to pass their bacterial flora onto their children\textsuperscript{12}. In fact, familial inheritance of oral bacterial strains and general oral health is well studied and parent oral flora is currently considered a major determination in risk factor assessment\textsuperscript{13}. The exact mechanism of this process is currently the focus of a large body of craniofacial and microbiology research. For example, mothers who receive dental rehabilitation demonstrated lower levels of ECC in their children\textsuperscript{14}. It has also been shown that parents who take an active role in their child’s oral health, who have good oral hygiene habits themselves and have a balanced nutritional diet tend to have children with low caries activity compared to parents who do not\textsuperscript{15}.

**ECC Co-Factors**

Several other entities have been cited as co-factors for ECC. For example, sub-optimal oral hygiene practices such as brushing less than twice a day with fluoridated toothpaste has a strong negative effect on caries control\textsuperscript{16}. Caplan also demonstrated that frequent bottle, or ad-lib feeding is a major contributory factor to ECC\textsuperscript{17}. This study also showed that children who bottle fed more than 1.5 years, had a mean dmft score 4 times higher than those fed by bottle less than one year. Furthermore, use of a baby bottle at night was a significant factor in caries risk\textsuperscript{18}. In another study, Reisine determined the children of
mothers with an external locus of control and higher tooth decay knowledge had a greater caries risk\textsuperscript{19}. Sub-optimal fluoride exposure has also been shown to be a high risk factor for ECC. Areas with high to optimal fluoride levels in turn have children with 4-10 times fewer caries than those with sub optimal fluoride in the drinking water\textsuperscript{20}. These areas of less than adequate fluoridated drinking water are shrinking. This can be accredited to the increasing areas of municipal water fluoridation and the “halo” effect of these municipalities to neighboring communities. The Academy of Pediatric Dentistry continues to focus on ECC prevention by providing guidance on each of these co-factors\textsuperscript{1}.

*Parental Stress and Quality of Life*

Parental stress has been proven to play a significant role on the QoL of families with a child suffering from a chronic illness. This includes a host of significant diseases with high morbidity and mortality. One such disease is childhood cancers. Parents of affected children have been shown to have a significantly higher level of stress versus the parents of children with physical disabilities such as cerebral palsy\textsuperscript{34}. It has also been shown that parents of children with heart disease were more likely than the healthy population to report excessive parenting stress\textsuperscript{35}. This study also reported that Approximately 1 in 5 parents expressed clinically significant levels of stress. These parents also expressed difficulty with setting limits or discipline of the child with heart disease. Another
longitudinal study of preschool age children with a diagnosis of developmental delay also demonstrated that over the course of 4 years their parents developed clinically high levels of parental stress\textsuperscript{36}. Another study even investigated parental stress between two subgroups of chronic childhood disease, asthma and epilepsy\textsuperscript{37}. They discovered that while both groups had stress levels above normal that the parents of epileptics had overall higher parental stress. Finally parental stress has also been studied as a factor impacting initial infection leading to chronic respiratory disease by elevating the risk of infantile wheezing and asthma\textsuperscript{38}.

The parental stress surrounding certain childhood chronic illness has also been shown to lead to poorer nutrition and meal planning abilities\textsuperscript{39,40,41}. LaValle et al. demonstrated that increased parental stress led to poorer overall oral health in children\textsuperscript{42}. Although no study to date has been published on the topic, it is not unreasonable to conclude that if parental stress can contribute to poor meal planning or a less nutritious diet containing high amounts of fermentable carbohydrates; it may also contribute to an increased risk for childhood caries. Dental caries in children is a chronic, steady-state disease with a high rate of recurrence. Its impact on the psyche and abilities of a parent or caregiver has been poorly studied and is poorly understood.
Study of Parental Stress and Oral Health

Measurement of parental stress has been evaluated extensively since the late 1970’s. In 1983 Abidin first developed and published the Parental Stress Index (PSI)\(^{43}\). This instrument has since been widely used and recognized as a test of chronic parental stress in many different social and health care settings. One of the practical weaknesses of the PSI was its length, with an average 30-45 minute time of administration. The Parental Stress Index Short Form (PSI-SF) was introduced as an alternative to the original PSI in 1995\(^{44}\). It requires much less time to complete making it more attractive to clinical research settings. It has since been validated as a reliable representation of the original PSI\(^{45,46}\). A handful of studies have included parental stress as one of the psychosocial spectrum of variables to ECC prevalence. To date this research has yielded inconclusive and conflicting results. Finlayson et al. reported on how a host of psychosocial factors contributed to ECC such as health belief scales of mothers' self-efficacy, psychosocial measures of depressive symptoms, and availability of instrumental social support\(^{33}\). They also showed parental stress was inversely proportional with ECC incidence. For every unit increase of stress in the scale used the chances of ECC prevalence decreased by one third. Using another indicator of stress, the Holmes and Rahe Life Event Questionnaire\(^{47}\) Litt et al., also found similar findings and correlated increased stressful life events to decreased ECC\(^{32}\). The efforts of Reisine showed a non-significant trend towards parents with lower life stresses and higher knowledge as having children with
higher caries risk$^{48}$. The weaknesses of these studies was their lack of parental stress specific research design. The bias of the measured parental stress was also high by using self reporting or less validated instruments than the PSI.

Conversely, other studies have been more specifically designed for parental stress and have at times used the PSI-SF. Tang showed total parental stress to be positively related to ECC$^{49}$. Their study demonstrated a significant bivariate association between parenting stress and ECC experience as measured by dmft. This confirmed earlier work by Quinonez and LaValle who also found a similar positive relationship between parental stress and childhood caries levels. All of these studies called for more longitudinal studies and further research designs targeted to isolating parental stress as a co-factor of of ECC$^{50,51}$. It is yet undetermined what role the level of parental stress plays in determining childhood caries risk and its presentation.

Teasing out the relevance of stress to dental disease sounds intuitively difficult due to the many other variables that are proven to contribute to ECC. Other well studied socio-economic factors that put children at risk for ECC have usually overshadowed the results of the parental stress correlation. Tang et al., while showing a positive Parental Stress-ECC correlation, stated, “If indeed, an association exists, as we first hypothesized, perhaps its contribution was overwhelmed by the much stronger effect of classic social
and behavioral determinants such as childhood nutrition, ethnicity, and SES” ⁴⁹. A research design in which children with the most severe levels of ECC (as in S-ECC) are matched against non-ECC and controlling for classic ECC predictors, one could potentially distinguish if parental stress in the home relates to caries prevalence. A clinical follow-up would also be useful to determine if the treatment of ECC correlates to a change in parental stress.

The objective of this study was to assess if there is any correlation between parental stress levels and ECC using a prospective, longitudinal case-control design.
METHODS

Introduction

This prospective case-control study (single-blind) was conducted at Nationwide Children’s Hospital (NCH) in Columbus, Ohio. All research was completed in the pediatric dental clinic. The population was randomly selected as they met inclusion criteria and agreed to study participation. The Institutional Review Board at NCH approved this study and its protocol. This study was supported by the Nationwide Children’s Hospital Research Institute Intramural Funding Program.

This investigation targeted families with a young child having either a caries free history or severe early childhood caries history. The parental stress of the caretaker was evaluated via a well validated psychological assessment instrument. Our design goal was to correlate parental stress to caries activity.

Power analysis using data from previous PSI-SF research helped determine our sample size. Using and alpha or 0.05 and a power of 0.95, it was determined that 100 participants in each group would be required (Total N=200).
**Inclusion Criteria**

Criteria for inclusion into the study were determined by several factors. To ensure the diagnosis of ECC per AAPD guidelines and keep oral examinations limited to primary dentition, the age range of 18-60 months was selected for child cohorts. To eliminate potential bias of parental stress due to chronic childhood illness we only allowed for pediatric subjects who fell within an ASA 1 or well controlled 2 classification. Assessment of a well controlled ASA class 2 status was a subjective clinical decision done by the examiner as a child with any reportable disease not requiring significant or frequent medical intervention beyond medication or well child checks.

In addition, we excluded more than one child per family to be allowed into the study. This decreased bias by not allowing on parents stress results to be weighted more than another and disrupting data normality. In cases where choosing between siblings to participate occurred we randomly selected the child prior to chart review or knowing the chief complaint. Although we allowed any cultural or ethnic group to participate, we only allowed for English speaking families to facilitate in ease of administration of the study. Finally, patients either fell into one of two categories based on a clinical exam, Caries Free (CF) or Caries Active (CA).
The CF group had no history of or current diagnosis of dental decay. The child’s primary dentition was complete or age appropriate unless there was a confirmed previous diagnosis of congenitally missing teeth. The CA group had to have at least 4 carious lesions found in at least 2 sextants upon the day of examination. They could also present with a history of previous treatments including restorative care or extractions. All child subjects were patients of record of the clinic or considered new patients presenting for an initial comprehensive or emergency exam.

Early Childhood Caries Evaluation

Upon meeting the inclusion criteria patients were evaluated for dental caries using a mirror and explorer by a calibrated examiner. A plaque index was recorded on all patients using the Oral Hygiene Index by Greene. If the patients exhibited no caries history or no current decay they were included into the CF group. CA patient’s oral health was charted by sextants. Since radiographs were not included in the diagnosis the decay had to be seen visibly and detected with an explorer upon examination. Taking into consideration the age of the patient, their likely temperament and likelihood of open contacts we decided utilize conventional radiography in our diagnosis.

The number of carious teeth, visual presence of infection/abscess and a chief complaint of pain was also noted. Patients meeting the CA criteria equally met the indications for
general anesthesia therapy per the local NCH dental clinic policies. These include pre-
cooperative behavior, treatment requiring more working time than a single mild/moderate
sedation affords (30-45 minutes) and local anesthesia demands that would approach
maximum allowable levels. Other potential contributing factors to general anesthesia
may have included: inability to obtain good diagnostic films due to behavior, distance of
travel for family to obtain routine care, technique sensitive treatment requiring more
treatment time.

Parental Stress Index-Short Form (PSI-SF)
The PSI-SF, revised by Abidin in 1995, is a 36 question instrument that has been well
validated to its original form. Its purpose is to determine parental stress relating to a
three-factor model revolving around the parent, child and their relationship. It is
administered in a 5 point Likert type scale. The three factors it evaluates are 1)Parental
Distress, 2)Parent-Child Dysfunctional Interaction and 3)Difficult Child. The original
PSI has been validated in a broad range of clinical settings with a very reliable internal
consistency alpha of 0.91. Its test-retest value up to 6 months is also well proven with a
correlation of 0.84. In addition to measuring total stress and the three subscales it also
has a built in defensive responding detector. It is validated in all child age groups
including the ages of the subjects in this study. In at least two investigations the PSI-
SF has shown high consistency as a valid marker of the original PSI.
The PSI-SF has a 5th grade health literacy level and takes approximately 10 minutes to complete. Caregivers were allowed to complete the written instrument independently with staff available for questions. After completion of the PSI-SF it was scored as per the instrument guidelines. If a participant scored above the 90th percentile for Total Parental Stress a referral was given to the caregiver to a behavioral and family health service in the hospital as per the instrument scoring guidelines. The proper management of these families was based on direction from a physician psychologist from the Psychology Department at NCH.

Data Collection

Patients were screened through three distinct internal sites within the dental clinic. These included; the Baby Clinic (BC), Hygiene Clinic (HC) or the Emergency Clinic (EC). They were classified as either a new patient or as a recall patient upon arrival in their respective clinic. Patients under the age of 36 months coming for an examination and prophylaxis were scheduled to the BC. Patients between 36 and 60 months of age were seen in the HC. The EC provided data from all included ages. Trained dental staff identified the subjects based on the inclusion criteria. They recruited families by explaining the scope of the research and obtaining a verbal consent. For parents with additional questions a informational sheet on the study and contact information for the study coordinator was provided. Parents completed a brief questionnaire including
demographic information on themselves and the child subject. The parent then completed the PSI-SF while examiners were blinded to the family demographics and results of the instrument.

A trained staff member conducted the oral exam and data collection for the child subject. They also ensured all the data was properly collected from the parent. If treatment that day was warranted it was then performed. Parents were informed they would be contacted in 90 days to be scheduled to retake the PSI-SF and received a participation incentive of $25. If dental treatment had been warranted at the initial exam a follow up on its completion was also recorded at the 90 day follow up.

All data collected was entered into a database maintained by the study coordinator. All information including contact information and personal identification was kept confidential and was solely accessible to the research staff. Upon completion of the study all information was disposed of.

**Statistical Evaluation**

Statistics were analyzed using Jmp 2.1 and SAS 9.13 (Cary, N.C.). Statistical analysis was completed using a logistic regression for the nominal and ordinal dependant variables. In addition a Wilcoxon Rank Sum test was also conducted. This test was used
to isolate the nominal and ordinal oral health/behavior indices against social history in subtracting out the confounding variables. Statistical significance was based on a type one error of $p < .05$. 
RESULTS

Caregiver and Patient Demographics

A total of 163 patients qualified for this study (84 males, 79 females). One hundred
patients were in the caries free group with 63 in the caries active. Of the parents who
completed the initial questionnaire, 151 were female and 12 were male. The mean age of
caregivers was 28 years (sd 6.8). The ethnic breakdown of responders was as follows;
85/52.1% were Caucasian, 71/43.6% were African American and 3/1.8% were Hispanic.
A remaining 4/2.5% responded as ‘other’.

Sixty-five percent (106) of the households had 2 or more caregivers. The majority of
caregivers surveyed (92/56.4%) were high school graduates followed by 45/27.6% who
where college graduates, and 12/7.4% who had completed post-graduate work. The
remaining 14/8.6% had not completed high school. When asked about annual household
income; 78/49.8 % reported <$20,000, 46/29.4% reported between $20,000 and $40,000,
17/10.9 % reported $40,000-$60,000 and 22/15.6% reported $60,000+. The mean age of
the 163 children who qualified was 27 months (sd 10) with 51.5% (84) males and 48.5%
(79) females. When the medical histories were reviewed, it was determined that
138/84.7% were class I according to the American Society of Anesthesiologists rating
scale. The remaining 25/17.8% were ASA II. The children had a mean of 1.5 siblings in
their households. The majority of patients (89%) were recruited from the Nationwide Children’s Hospital Infant Oral Health Clinic. When the average plaque of the children was measured using the Oral Hygiene Index by Greene\textsuperscript{53}, 27.6% of the subjects had no plaque, 40.5% had plaque confined to the gingival 1/3, 12.3% had plaque on less than half the anterior teeth and 19.6% had plaque covering more than half. The entire cohort had a mean of 2.9 carious teeth (sd 4.7) and 1.4 carious sextants (sd 2.1).

When the cohort was split by caries status, it was noted that the mean age of the caries-free children was significantly lower (24.1m/7.9) when compared to the caries-active children (33.1m/11.7)(p=.003). There was no significant difference in the number of single-caregiver households. However, 58.6% (34) of the caries active children lived in households with an annual income of <$20,000, compared to 43.5% of the caries-free households. Children in the caries-active group were significantly more likely to have detectable plaque when compared to the caries-free group (p<.0001). Children in the caries-active group had a mean of 7.8 carious teeth (4.6).

*Parental Stress Scores: Complete Cohort*

The PSI:SF scores for the entire qualifying cohort are presented in Table 1. Presented scores are for the four sub-categories (defensive scoring, parental distress, parent-child dysfunctional interaction, difficult child) as well as the total scoring. Due to the repeated
nature of the variables and the fact that the populations were not normally distributed, data were analyzed using the Wilcoxon Signed Rank Test. The Wilcoxon test allows for the abnormality of variables. There were no significant associations between the sub-categorical PSI:SF measurements. However, when testing the initial and final total scores, statistical significance was noted (p=.002). This can be interpreted as significant as the differences in scores are not distributed symmetrically. A total of 21/13% of the respondents exceeded the high-risk threshold of 90 for the PSI-total score during the initial survey. When assessing the PSI:SF total scores at the 3 month follow-up, 15 exceeded 90.

<table>
<thead>
<tr>
<th>PSI SCORING</th>
<th>Defensive Distress</th>
<th>Parental Dysf</th>
<th>Difficult Child</th>
<th>TOTAL</th>
</tr>
</thead>
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<tr>
<td>INITIAL</td>
<td>13.2 (4.9)</td>
<td>22.3 (8.4)</td>
<td>16.9 (6.7)</td>
<td>61.3 (20.8)</td>
</tr>
<tr>
<td>Follow-up</td>
<td>13.9 (5.3)</td>
<td>23.6 (8.7)</td>
<td>17.7 (6.2)</td>
<td>62.6 (32.9)</td>
</tr>
<tr>
<td>Wilcoxon Rank p values</td>
<td>0.143</td>
<td>.094</td>
<td>.141</td>
<td>.061</td>
</tr>
</tbody>
</table>

Table 1. PSI:SF Values for entire cohort (N=163)

**PSI:SF Scores: Caries Free Group**

As previously mentioned, 100 subjects were in the caries free group. The differences between initial and 3 month follow-up PSI:SF scores are outlined in Table 2. The caries-
free (CF) group scored a mean of 1.3 points below the entire cohort for the initial score total. There were no significant differences between the initial and final stress levels according to these data.

<table>
<thead>
<tr>
<th>PSI SCORING</th>
<th>Defensive</th>
<th>Parental Distress</th>
<th>Parent-Child Dysf</th>
<th>Difficult Child</th>
<th>TOTAL</th>
<th>Change in PSI from Initial to Follow-up**</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial</td>
<td>13(5.1)</td>
<td>22.1(8.8)</td>
<td>16.5 (6.7)</td>
<td>21.2 (8.4)</td>
<td>60 (20.8)</td>
<td>+2.5</td>
</tr>
<tr>
<td>Follow-up</td>
<td>13.4 (5.3)</td>
<td>23 (9)</td>
<td>17.3 (5.6)</td>
<td>22.5 (8.3)</td>
<td>62.5 (20.3)</td>
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</tr>
<tr>
<td>Wilcoxon Rank Signed</td>
<td>.356</td>
<td>.148</td>
<td>.093</td>
<td>.060</td>
<td>.063</td>
<td></td>
</tr>
</tbody>
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Table 2. PSI:SF Values for the Caries Free Group (N=100)

**PSI:SF Scores: Caries Active Group**

The mean PSI:SF initial score total for the 63 respondents in the caries-active group was 64.7, this is 4.7 points higher when compare to the entire cohort of 163. The values for this and the sub-category scores are presented in Table 3.

<table>
<thead>
<tr>
<th>PSI SCORING</th>
<th>Defensive</th>
<th>Parental Distress</th>
<th>Parent-Child Dysf</th>
<th>Difficult Child</th>
<th>TOTAL</th>
<th>Change in PSI from Initial to Follow-up**</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial</td>
<td>13.8 (4.6)</td>
<td>23.3 (7.9)</td>
<td>18.0 (6.9)</td>
<td>23.4 (9)</td>
<td>64.7 (21.2)</td>
<td>+2.3</td>
</tr>
<tr>
<td>Follow-up</td>
<td>14.7 (5.1)</td>
<td>24.5(8.5)</td>
<td>18.4 (7.1)</td>
<td>24 (7.7)</td>
<td>67 (19.8)</td>
<td></td>
</tr>
<tr>
<td>Wilcoxon Signed Rank</td>
<td>.176</td>
<td>.411</td>
<td>.881</td>
<td>.464</td>
<td>.008*</td>
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</tr>
</tbody>
</table>

Table 3. PSI:SF Values for Caries-Active group (N=63)
There was a significant difference between the initial and final PSI:SF scores for the caries active group (p=.008). The caries-active group had a mean of 7.8 carious teeth per child. A total of 66.7% of the caries active patients who qualified completed follow-up. During the 3 months between the initial and final PSI: SF administration, 69.4% of these caries-active children had dental treatment completed under general anesthesia. The caries-active respondents whose children had treatment completed prior to the follow-up assessment went down a mean of 3.6 points. The caries-active subjects who did not have treatment completed at time of follow-up went scored a mean of 3.1 points higher on the follow-up assessment. Table 4 outlines the analysis of the caries-active patients who completed follow-up and furthermore, those who received dental care.

<table>
<thead>
<tr>
<th>PSI SCORING</th>
<th>Defensive Parental Distress</th>
<th>Parent-Child Dysf</th>
<th>Difficult Child</th>
<th>TOTAL</th>
<th>Change in PSI Initial to Follow-up**</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial</td>
<td>14.1 (4.9)</td>
<td>24 (8.3)</td>
<td>18.3 (7.4)</td>
<td>23.5 (9.7)</td>
<td>65.8 (22.4)</td>
</tr>
<tr>
<td>Follow-up</td>
<td>14.8 (5.1)</td>
<td>24.7 (8.3)</td>
<td>18.7 (7.5)</td>
<td>24.1 (7.9)</td>
<td>64.2 (24.4)</td>
</tr>
<tr>
<td>Wilcoxon For Total CA Follow-ups</td>
<td>.228</td>
<td>.473</td>
<td>.975</td>
<td>.232</td>
<td>.851</td>
</tr>
<tr>
<td>Follow-up Tx completed N=29</td>
<td>15.4 (5.2)</td>
<td>25.9 (8.1)</td>
<td>19.1 (7)</td>
<td>25.1 (8.3)</td>
<td>65.2 (26.2)</td>
</tr>
<tr>
<td>Wilcoxon for TX complete</td>
<td>.389</td>
<td>.396</td>
<td>.986</td>
<td>.471</td>
<td>.855</td>
</tr>
<tr>
<td>Follow-up Tx NOT completed N=13</td>
<td>13.4 (5)</td>
<td>22.3 (8.5)</td>
<td>17.7 (8.7)</td>
<td>22.1 (7)</td>
<td>62.1 (20.7)</td>
</tr>
<tr>
<td>Wilcoxon for tx not complete</td>
<td>.850</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 4. PSI:SF Value for Caries-Active group with Follow-up (N=43)
Linear Regression Analysis: Caries-Status

A regression analysis was completed to determine which associations may be predictive of whether a child would fall into the caries-active or caries-free group. The results are presented in Table 5. The only significant predictors were patient age (p=.003) with younger children, more likely to be caries-free, and plaque-index (p=.002) with children with higher plaque indices more likely to be caries-active.

<table>
<thead>
<tr>
<th>Predictor Variable</th>
<th>p-value</th>
<th>interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Caregiver Age</td>
<td>.498</td>
<td></td>
</tr>
<tr>
<td>Caregiver gender</td>
<td>.220</td>
<td></td>
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<tr>
<td>Caregivers in Home</td>
<td>.355</td>
<td></td>
</tr>
<tr>
<td>Caregiver Education</td>
<td>.155</td>
<td></td>
</tr>
<tr>
<td>Annual Income</td>
<td>.489</td>
<td></td>
</tr>
<tr>
<td>Caregiver Ethnicity</td>
<td>.336</td>
<td></td>
</tr>
<tr>
<td>Patient Age</td>
<td>.003</td>
<td>Younger patients more likely to be in caries free</td>
</tr>
<tr>
<td>Patient Gender</td>
<td>.288</td>
<td></td>
</tr>
<tr>
<td>Siblings in House</td>
<td>.499</td>
<td></td>
</tr>
<tr>
<td>ASA</td>
<td>.455</td>
<td></td>
</tr>
<tr>
<td>Plaque Index</td>
<td>.002</td>
<td>Higher plaque index in caries active group</td>
</tr>
</tbody>
</table>

Table 5. Regression Analysis for Predictors of Caries-Status

Linear Regression Analysis: Initial PSI:SF score (total)

When the initial PSI:SF score was analyzed for predictors, the only significant associations noted were annual income, with lower income respondents significantly associated with higher initial PSI:SF scores (p=.003) and number of carious teeth (p=.005). Data for these associations are presented in Table 6.
<table>
<thead>
<tr>
<th>Predictor Variable</th>
<th>p-value</th>
<th>interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Caregiver Age</td>
<td>.315</td>
<td></td>
</tr>
<tr>
<td>Caregiver Gender</td>
<td>.150</td>
<td></td>
</tr>
<tr>
<td>Caregivers in Home</td>
<td>.142</td>
<td></td>
</tr>
<tr>
<td>Caregiver Education</td>
<td>.226</td>
<td></td>
</tr>
<tr>
<td>Annual Income</td>
<td>.013</td>
<td>Lower income respondents scored higher on PSI</td>
</tr>
<tr>
<td>Caregiver Ethnicity</td>
<td>.415</td>
<td></td>
</tr>
<tr>
<td>Patient Age</td>
<td>.339</td>
<td></td>
</tr>
<tr>
<td>Patient Gender</td>
<td>.395</td>
<td></td>
</tr>
<tr>
<td>Siblings in House</td>
<td>.180</td>
<td></td>
</tr>
<tr>
<td>ASA</td>
<td>.389</td>
<td></td>
</tr>
<tr>
<td>Plaque Index</td>
<td>.229</td>
<td></td>
</tr>
</tbody>
</table>

Table 6. Regression Analysis and Associations with PSI-Initial Total Score

A graphical representation of the relationship between PSI: SF score and number of carious teeth is presented in Figure 1. The graphical depiction of our caries active and caries free participants as to where they fell into the PSI:SF percentiles are found in Figure 2.
Relationship of Number of Carious Teeth to Initial Score on PSI:SF

Fig. 1  Relationship of Number of Carious Teeth to Initial Score on PSI:SF
Fig. 2  PSI:SF Percentile by Caries Status
Regression Analysis: Caries-Active Respondents Exceeding Threshold of 90

When scoring the PSI:SF, a total score of 90 is considered to be strongly indicative of ‘potential parental-child dysfunctional interaction’. Families who scored above 90 on either the initial or follow-up were offered referral services to the Nationwide Children’s Hospital Department of Psychiatry. We analyzed these subjects and their families more in-depth, and although only 13% (21) of the entire enrolled population exceed this threshold, it should be noted that this represented 15.8% of the caries active group. Regression analysis of the “over-90” population revealed significant associations between scoring higher than 90 on the initial PSI:SF test and; annual household income (p=.032), caregiver education level (p=.018) and the numbers of caregivers in the household (p=.014). Thirty-eight percent (8) of these families exceeded 90 on the PSI: SF follow-up exam 3 months later. These 8 were in addition to 7 from the caries-free group who had not exceeded 90 initially but did on follow-up examination. When these 15 families were examined; there were significant associations between ASA status (p=.006) as well as the how high the families scored on the initial PSI: SF (p=.016).
DISCUSSION

The Multi-Cofactor Model and Stress

The movement in describing ECC has moved from a purely biological study to a model of population health and burden of illness. This has complicated the conceptualization of the caries process from a simple cause and effect analysis to a multi-level series of relationships. The true challenge in taking on such an approach is teasing out the individual variable while excluding a host of other potentially confounding variables. In 2008, Fisher-Owens provided perhaps the most all-inclusive model to date by categorizing co-factors into three major influencing levels: Community, Family and Child. All three of these levels are bound by time and create the environment that dictates the social risk factors of ECC. Stress itself can be found and perpetuated in each of these areas of community, family and child and can also cause several interactions between these levels.

Stress, defined as “forces from the outside world impinging on the individual” has been recognized as having pathophysiologic consequences for the individual. This has led to a paradigm shift causing global changes in health care administration, policy and even marketing in the last few decades such as the current standards of care to pain
management. However, the study of stress as it relates to chronic disease in children and more specifically ECC is poorly understood.

With nearly every aspect of a young child’s oral health being managed by a parent or caregiver, it stands to reason that stresses in the family environment, and more specifically stress between the parent and child should be considered in childhood caries risk assessment. For example, parents with high stress may find less priority in proper oral hygiene when other basic needs are not being met. There is a large body of literature showing stressed families tend to eat less nutritious diets. Financial stressors may also lead to access to care and inadequate resources to maintain good oral health. Finally, a parent with high stress in their parent child relationship could suffer from poor coping abilities and thus may lack the motivation to or abilities to properly care for their child’s teeth. Conversely, parents stressed by inadequacy may be more likely to be vigilant. This longitudinal study set out to determine if there exists an interaction between parental stress and childhood caries.

Our other goal was to help determine the true nature of this interaction if present. With a good deal of medical literature demonstrating a relationship between chronic parental and development of a chronic childhood disease on could believe that ECC is no different. However, we also hypothesized that perhaps it is the presence of caries itself was
increasing a parent’s stress. We hope to determine if this association was cyclical, directional or some combination.

Anticipated Findings

In many ways our findings support many common beliefs and proven facts about the social factors of ECC. Many of the well established correlations to low socio-economic status (SES) held true in our study. We demonstrated inverse relationships of parent education, family income and the number of caregivers in the home to ECC (p=.032, p=.018, p=.014). While skewed to a lower SES represented by our population at a larger urban hospital, our cohort groups included a good range of SES data. This enabled us to further examine the notions that co-factors of low SES put a child at increased risk for ECC.

In addition to social factors we also upheld other common biological causative risk factors. This includes the regression results that younger children tend to have less decay than their older counterparts (p=.003). This stands to reason with the chronic nature of dental caries and the time that is essential to caries development. Another classic discovery was that a child’s plaque index has a strongly significant positive correlation to ECC (p=.002). This finding somewhat typifies the model of caries we currently adopt;
that complex bacterial biofilms and fermentable carbohydrates are the direct cause of decay. However more indirect factors can also contribute such as parental stress.

A subtle finding of our regression analysis was that as parental stress increased so did ECC \((p=.05)\). This supports the literature of this decade by Tang, Quinonez and LaValle who all showed the same interaction using other methods of study and scales but who set out with the same purpose as we had. While their studies were similar to ours, to date this study was the first to our knowledge that used the PSI:SF against two study groups solely trying to elucidate parental stress and S-ECC. Other previous studies disagree with our findings and demonstrated an inverse relationship of caries activity to parental stress. The research design of these studies included parental stress being scored by other methods such as self reporting or other stress indexes. Also two of these studies primary focus was on low income status to caries incidence and the other to bacterial strain fidelity amongst family groups.

Another important subtle finding was among our most “at risk” parents. When a parent scores above the 90\(^{th}\) percentile on the PSI:SF they are considered to have clinically significant stress according to the instrument guidelines and be as risk for high “parent to child relationship dysfunction”. Our data demonstrated a general trend that correlated this “high risk” group to caries activity. In percentiles below 75 we generally found a 2
or 3 to one relationship of CF to CA children. However, upon reaching the 75 percentile and up, the distribution of CF to CA families became more equal. Due to the lack of CA cohorts we cannot state if this trend would have continued but in all likelihood it would have. This would demonstrate perhaps a spike in S-ECC amongst the most at risk families for severe dysfunction.

**Acuity of ECC**

The PSI:SF was designed is an indicator of chronic stress and has been used in numerous studies on chronically ill children. While ECC is a chronic disease there are certainly aspects of acuity to its effect on the family. If a child develops pain or abscess, ECC could easily transform from a “silent” chronic problem to an acute problem in the families eyes. We attempted to account for this and collected data on our CA patients as to the presence of pain or infection. Unfortunately, not only did we have a low n value but determining pain or infection on such a young population was likely unreliable. This was due to the patient’s ability to effectively communicate and a lack of diagnostic clinical evidence of infection even if present. Parents generally reported the pain or infection symptoms and followed no set criteria thus again adding to the bias of pain/infection reporting.
In addition, the diagnosis of ECC in a child may also raise the stress of a parent acutely. The actual time of the PSI:SF administration could then be deemed a weakness to our study. Some caregivers completed the PSI:SF before examination while others after results from the clinical exam were revealed. We did not account for the completion time. It certainly would have been interesting to maintain such data and see if the PSI:SF actually did reveal results in the acute stress increase due to a ECC diagnosis for parents who were ignorant to their child’s caries condition. It would have also been of interest to see if the parents who knew their child needed dental treatment or had been referred had a change in stress when they received information as how bring resolution to their child’s condition.

It seems with so many potential acute stressors that perhaps solely using a chronic stress indicator did not adequately assess total parental stress. Our study then is a perhaps a better marker of the indirect chronic stress effects and relationships to ECC rather than the direct and short term effects of ECC to parental stress. Certainly in hindsight we would design our study to more strategically time the administration of the PSI:SF.

**Other Causative Stress Factors**

Another subgroup of our sample population we did not assess were patients that had previous treatment completed but a new diagnosis of caries. These cohorts were included
simply in the CA group. The value in assessing these patients would have helped us address a larger potential causative stress factor: access to care. Difficulty in obtaining access to dental care has traditionally been a finding among populations with elevated ECC levels, especially in areas of low SES such as our population. In fact it could be said that inability to find access to care could develop into a chronic stress itself. Families who had received treatment and had means to access it potentially could demonstrate a different stress level than those who had been unable to find care up until participation in our study.

We did not account for our patient’s ability to find care in this study. We also did not delve into other access to care issues such as payment method (ie. state funded health care, private insurance and self pay). The challenge in deriving such information is that the most at risk group, those who cannot find care, could not be assessed in our study since all participants obviously had found a treatment local. Evaluating the parents’ stress related to accessing care could have helped elucidate if the positive relationship we found of ECC to parental stress could be attributed more to the inability to find routine dental care and the time spent seeking it out. This would provide a definition to parental stress and ECC as more associative and indirect with chronic access to care as the “true primary stressor”. This would be a direction that future studies could be designed; to
account for access to care stress with sequelae to increased parental stress and increased ECC.

*Other Confounding Variables*

Another interesting aspect of the study was the timing in which it took place. As our data collection started, there coincided a general belief that economic times were worsening. By the time data collection concluded the entire country was in an unprecedented economic downturn with widespread job loss, economic slowing and failing of financial markets. This could be an explanation to the overall stress increase we saw between both caries active and caries free cohort caregivers and their unequal distribution. We attempted to determine the stress of the parent/caregiver using the PSI:SF which is designed to assess chronic stress and discount more acute forms of stress. Even still it is hard to believe that the somewhat acute nature of the economic circumstances did not play a role in our results.

There were other potential confounding variables. The most obvious is perhaps the reporting bias of the study itself. It is possible assume that parents felt threatened by the nature of the survey questions and in turn answered untruthfully in hopes to not be singled out in some way. While the PSI:SF does include a “disqualifier” that attempts to assess the parents truthfulness in the survey called the Defensive Parent score, we still
must question the honesty and validity of results generated on the survey. Only a much larger cohort size would be the only way to truly eliminate this bias.

Another consideration is the mean population we sampled. The majority of our participants were from low SES and feel the stress of poverty already. Thus our findings may inject bias as not representing the true population as a whole and only the populations with lower SES.

The Parental Stress Index by Abidin has been used to study a host of personal and environmental stressors. These include children with chronic illness, certain ethnicities, family relationships, etc. It has even been shown to be a good predictor of a parent who is at high risk for child abuse or neglect. Extensive literature has proven decay and poor oral health is high among children with a history of abuse or neglect. This also led us to seek out the incidence of caries activity when a parent exceeded the “high risk for dysfunction” threshold. This threshold level proved to be valuable as a predictive value although non significant as the percentage of the “above threshold” families was less than the percentage of decay they contributed overall.

Another confounding variable that we tried to account for but ended up not providing significant data was the number of siblings or maternal age (p=.499, p=.498).
Interestingly while caries increased in our high risk families with a single parent in the home it did not matter the number of siblings. We had assumed that increasing the number of children in the home might increase parental stress. This included the belief that new young mothers that lack parenting experience or who have not yet obtained maternal coping skills might score high also. Our data determined that neither of these scenarios proved to be the case.

Population health studies require large cohort groups. Patient recruitment proved to be a challenge in this study and ideally would have included an additional 30% of participation in our CA group. While we offered an incentive we still had many families turn down the offer to randomly participate. This was often due to time constraints, a sense of privacy invasion and the general nature of the clinic where data collection took place. With so many confounding variables it would take a monumental study to begin to fully elucidate all categories of the study.

*Future Studies*

Perhaps our most interesting finding came in our caries active group between those who received dental treatment and those who did not between the initial and final surveys. Up to this point we had assumed that stress is more of an indirect causation to ECC sharing the same host of co-factors that ECC has. However, we demonstrated a trend, although
not significant, that caregivers of children who received treatment between surveys tended to have a decrease in less stress (-3.6(2.6)). The opposite trend was found to those who did not receive treatment as their stress actually increased (+3.1 (2.2)). This begins to demonstrate that perhaps there is more of a direct link between stress and caries as opposed to indirect.

In any case there is good evidence that an assessment of parental stress or coping should be part of the anticipatory guidance of a child’s dental needs. This requires the practitioner to recognize; warning signs of stress through conversation with the parent and stressful circumstances in the families’ lives. This may help dictate treatment options and planning when trying to understand a child’s dental needs. Findings of S-ECC might also instigate appropriate conversation and a referral to social services available to decrease or alleviate a parent’s stress. A practitioner should be ready and prepared to offer assistance in obtaining such a referral in his or her area of practice.
This longitudinal, case controlled, single blind study demonstrated several classic hypotheses to ECC. These include patient age (p=.003) and plaque index (p=.002) as biologic risk factors for ECC. Equally, we determined that annual income (p=.013) was a significant inverse determining factor in total parental stress. We also found that annual income (p=.032), caregiver education level (p=.018) and number of caregivers in the home (p=.014) are socio-economic status risk factors to high parent-child dysfunction as rated by the Parental Stress Index Short Form. Finally, our linear regression analysis demonstrated a weak but significantly positive association between ECC and total parental stress (p=.05). This indicates that primary caregiver stress and the number of carious primary teeth in the associated cared for children rise simultaneously. The true relationship of parental stress and childhood requires more study to be fully understood.
BIBLIOGRAPHY


