Antibacterial and Antifungal Effectiveness of Virgin Coconut Oil (VCO) Mousse against *Streptococcus mutans* and *Candida albicans* Biofilms

Lili Nur Indah Sari[1], Eva Fauziah[2]*, Sarworini Bagio Budiardjo[2], Margaretha Suharsini[2], Heriandi Sutadi[2], Ike Siti Indiarti[2], Mochamad Fahlevi Rizal[2]

1. Pediatric Dentistry Residency Program, Faculty of Dentistry, Universitas Indonesia, Jakarta, Indonesia.
2. Department of Pediatric Dentistry, Faculty of Dentistry, Universitas Indonesia, Jakarta, Indonesia.

Abstract

Early childhood caries (ECC) is a common chronic disease in children <72 months old and is characterized by destruction of tooth tissue caused by multiple factors, including accumulation of biofilm dental plaque, *Streptoccus mutans*, and *Candida albicans*.

We analyzed the effectiveness of virgin coconut oil (VCO) mousse at concentrations of 0.8%, 8%, and 80% against *S. mutans* and *C. albicans* biofilms in children with ECC. Cell viability was analyzed to estimate the growth of biofilm cells using crystal violet 0.5% in 96-well plates and colony-forming units to count *S. mutans* colonies in brain heart infusion broth and *C. albicans* in saborous dextrose agar (SDA) medium. Intergroup trials were analyzed by one-way analysis of variance ($p < 0.05$).

Viability tests of 80% VCO mousse showed different statistical significance results compared with 0.8% and 8% VCO mousse and equal results to casein phosphopeptide–amorphous calcium phosphate. VCO mousse 80% was effective as an antibacterial and antifungal agent, especially on microbes that cause ECC.

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Introduction

According to the American Surgery Journal in 2000, a common chronic disease in children is dental caries, which can compromise their growth and development.[1,2] In 2013, the RI Health Department declared an increase in nearly all provinces of Indonesia's population from 2007 (43.4%) to 2013 (53.2%); even in children aged 1–14 years, the number with caries reaches 64.5%.[1] According to the American Academy of Pediatric Dentistry, early childhood caries (ECC), defined as the presence of one or more carious teeth, is characterized by presence or absence of cavities, caries-induced tooth loss, or restoration of the surface of primary teeth in children aged ≤71 months.[3] Dental caries is an infectious dental disease, characterized by destruction of hard tissue due to multiple factors, including accumulation of biofilm dental plaque and acid produced by aciduric bacteria, *Streptococcus mutans*.[4,5,6] Biofilm dental plaque is a soft deposit that forms a biofilm layer composed of a complex microbial community.[7]

S. mutans is the most prominent anaerobic, facultative gram-positive bacteria and has the highest virulence as a caries-causing organism.[8,9] This is because *S. mutans* can attach to the enamel surface, produce acid metabolites, provide glycogen reserves, and has the ability to synthesize extracellular polysaccharides.[10] In addition to bacteria, a microorganism that has a role in increasing the pathogenicity of children with ECC is *Candida albicans*, the normal commensal organism in the oral cavity of healthy individuals. The number of *C. albicans* was reported to increase in saliva, plaque, and dentin in children with ECC.[6] *C. albicans* also is reportedly able to increase *S. mutans* attachment to oral biofilm *in vitro*.[13]

Therapy is aimed at reducing or eliminating the major etiologic factors of dental plaque biofilm containing caries-causing bacteria.[14] Mechanical and chemical

Corresponding author:
Eva Fauziah
Department of Pediatric Dentistry,
Faculty of Dentistry, Universitas Indonesia,
Jakarta, Indonesia.
E-mail: eva.fauziah@ui.ac.id

maintenance techniques can be performed.[15] Chemicals reported safe to use in children include tooth mousse containing water-based, nondetergent, and sugar-free ingredients.[16] Virgin coconut oil (VCO) is reported to have anti-inflammatory, antifungal, and antibacterial effects, and its popularity in Indonesia has increased over time. VCO has previously been reported to have antibacterial effects on chromogenic bacteria[17–19] and *Streptococcus* species, particularly *S. mutans*, and antifungal effects on *C. albicans* due to the saturated fatty acid and polyphenols content.[20,21] However, to our knowledge, no study has demonstrated antibacterial and antifungal effects of VCO against *S. mutans* and *C. albicans* biofilm in water-based mousse preparations that have been reported safe for use in children.[16]

Materials and methods

This *in vitro* laboratory experiment tested the viability of *S. mutans* and *C. albicans* biofilm after administration of VCO at various concentrations (0.8%, 8%, and 80%). A chemist processed the VCO mousse at Akademi Farmasi IKIFA. This study was approved by the ethics committee of the Faculty of Dentistry, Universitas Indonesia. The study sample consisted of laboratory and clinical strain microorganisms. Subjects were selected by examination of oral hygiene and childhood caries. Inclusion criteria were child age 0–72 months, deciduous dentition, decayed–extracted–filled teeth (deft) index score ≥5, and no systemic disease. Parents provided informed consent for participation in the study. The antibacterial and antifungal effectivity of VCO mousse against *S. mutans* and *C. albicans* biofilm was assessed using a crystal violet test and colony count (colony-forming units [CFU]/mL).

Plaque sampling

Dental plaque was obtained using sterile toothpicks on the entire tooth surface. The plaque was inserted into a 1.5 mL Eppendorf tube containing a solution of brain heart infusion (BHI) medium and Sabouraud dextrose broth (SDB). The Eppendorf tube was placed in a cooler box and taken to the oral biology laboratory to begin the bacterial incubation process. Laboratory strains of *S. mutans* and *C. albicans* were maintained in a freezer at −80 °C at the Laboratory of Oral Biology, Universitas Indonesia.

Microorganism culturing

The Eppendorf tube containing the sample was vortexed. Then, 20 μL *S. mutans* was spread across TYS20B agar and incubated in the anaerobic jar for 24 hours (CO_2 5%, H_2 10%, dan N_2 85%). On the other hand, 10 μL *C. albicans* was spread across CHROMagar (Chromagar Co., Paris, France) and incubated for 48 hours at 37 °C.

Identifying S. mutans and C. albicans clinical strains using polymerase chain reaction (PCR)

After incubation in TYS20B for 24 hours, *S. mutans* was identified by PCR using Dreamtaq Green PCR Master Mix (Thermo Fisher Scientific, Waltham, MA, USA). After amplification, the PCR products were analyzed by electrophoresis on an agarose gel, 60 V, 400 mA for 75 minutes. The newly synthesized DNA fragments were visualized under ultraviolet light.

After incubation in CHROMAgar for 48 hours, a *C. albicans* colony will exhibit a green color. A *C. albicans* colony was extracted from CHROMAgar, placed into SDB, and incubated for 24 hours at 37 °C. Then, the DNA sample was identified by PCR (Dreamtaq Green PCR Master Mix). After amplification, the PCR products were analyzed by electrophoresis at 1.0% (wt/vol) agarose gel 94 V, for 20 minutes.

In vitro biofilm formation

The method of biofilm formation on the bottom of a 96-well-μL plate was used. First, fetal bovine serum (FBS) was diluted in the waterbath and then incubated at 37 °C for 90 minutes while shaking to inactivate the FBS complement ingredients. Second, 100 μL FBS was added in a 96-well plate and incubated for 30 minutes. Next, the wells were washed with phosphate-buffered saline (PBS), 50μL BHI broth and 50μL SDB added, *C. albicans* and *S. mutans* were inserted together into each well (treated or control) and then incubated at 37 °C for 24 hours. After incubation, the wells were washed with 100 μL PBS. Then, 100 μL VCO mousse (concentration 0.8%, 8%, 80%) and mousse base without active ingredient were added. The specimens were prepared with as much as 50 μL each of media BHI broth and SDB, positive control with casein

phosphopeptide–amorphous calcium phosphate (CPP-ACP) and negative control in duplicate, with one 96-well plate for crystal violet assay and another for CFU assay. Then, the wells were reinserted into the incubator at 37 °C for 24 hours, after which, bacterial cells that did not form a biofilm were removed and washed with PBS 100 µL 2 times.

Crystal violet test

As much as 200 µL 0.5% crystal violet was placed in each well, and the wells were incubated at room temperature for 15 minutes. After that, the supernatant-containing residual 0.5% crystal violet solution was removed and rinsed using 200 µL PBS. Then, 200 µL of a 96% ethanol solution was added into each well. Optical density was measured at 490 nm using a microplate reader.

Colony-forming unit (CFU)

Biofilm was obtained by scraping using a micropipette in the 96 incubated wells and transferred into a 1.5 mL Eppendorf tube. In other Eppendorf tubes, 990 µL PBS solution and 10 µL bacteria from initial stock were prepared and vortexed. The process was repeated for further dilution. Then, 5 µL from each dilution was spread onto BHI agar and SDA and incubated at 37 °C for 24 hours. Then, the colonies formed on the agar were counted.

Data analysis

Viability test data on *S. mutans* and *C. albicans* biofilm were analyzed using one-way analysis of variance (ANOVA; $p < 0.05$). Statistical significance between the groups were analyzed using post hoc least significant difference ($p < 0.05$).

Results

Crystal violet test

The Shapiro–Wilk test score showed that the data had a normal distribution. One-way ANOVA was used to test for viability of *S. mutans* and *C. albicans* biofilm after administration of various concentrations of VCO (0.8%, 8%, and 80%) and to identify any discrepancies between each microorganism biofilm viability score.

ANOVA significance was calculated as 0.001 ($p < 0.05$). At least two groups had significantly different mean viability scores (Table 1). Because of the significant differences, a post hoc test was used to determine which groups had differences. Table 2 shows the results of post hoc difference analysis of *S. mutans* and *C. albicans* to VCO mousse in various concentrations. Statistically significant differences from viability of biofilm were found between the positive control group (CPP-ACP) and groups treated with 0.8% and 8%VCO, between the positive group and base without active ingredient, between the 80% and 0.8% VCO groups, and between the 80% VCO group and base without active ingredient ($p < 0.05$).

Treatment group	*n*	Bacterial Viability (OD) Mean (standard deviation)	*p*-value
VCO mousse 0.8%	4	0.456 (0.064)	0.000
VCO mousse 8%	4	0.475 (0.073)	
VCO mousse 80%	4	0.208 (0.047)	
Positive control (CPP-ACP)	4	0.201 (0.045)	
Negative control	4	0.113 (0.068)	
Base without active ingredient	4	0.430 (0.055)	

One-way ANOVA test; significant score based on $p < 0.05$. OD, optical density; VCO, virgin coconut oil.

Table 1. Differences in Viability Tests in *S. Mutans* and *C. Albicans* Biofilm After Administration of VCO Mousse at Different Concentrations (Crystal Violet).

Group	*p*-value
VCO mousse 0.8% vs. 8%	1.000
VCO mousse 0.8% vs. 80%	0.001*
VCO mousse 0.8% vs. base	1.000
VCO mousse 0.8% vs. positive control	0.001*
VCO mousse 0.8% vs. negative control	0.001*
VCO mousse 8% vs. 80%	0.001*
VCO mousse 8% vs. base	1.000
VCO mousse 8% vs. positive control	0.001*
VCO mousse 8% vs. negative control	0.001*
VCO mousse 80% vs. base	0.001*
VCO mousse 80% vs. positive control	1.000
VCO mousse 80% vs. negative control	0.576
Base vs. positive control	0.001*
Base vs. negative control	0.001*
Positive control vs. negative control	0.789

Post hoc Bonferroni test, *significant score based on p < 0.05. VCO, virgin coconut oil.

Table 2. Post hoc Analysis of *S. Mutans* and *C. Albicans* Viability Scores in Treatment Intergroup.

Colony-forming unit (CFU)

The Shapiro–Wilk test for normality of *S. mutans* and *C. albicans* biofilms (Tables 3 and 4, respectively) showed a normal distribution. Comparison of total viable colony counts

(CFU/mL) between various treatment groups was analyzed using one-way ANOVA. Since one-way ANOVA revealed significant differences, a post hoc test was used to determine which groups had differences.

Treatment Group	*n*	Mean CFU (standard deviation)	*P* Value
VCO mousse 0.8%	4	242.5 (25.053)	0.292
VCO mousse 8%	4	190.00 (28.142)	0.382
VCO mousse 80%	4	49.00 (41.777)	0.096
Positive control (CPP-ACP)	4	70.00 (20.265)	0.065
Negative control	4	248.50 (34.424)	0.144
Base without active ingredient	4	224.00 (11.225)	0.366

One-way ANOVA test; significant score based on $P < 0.05$. VCO, virgin coconut oil

Table 3. Differences in Number of Viable Bacterial Colony Counts (*S. Mutans*) After Administration of VCO Mousse at Different Concentrations.

Treatment Group	*n*	Mean CFU (standard deviation)	*p*-value
VCO mousse 0.8%	4	249.5 (26.300)	0.353
VCO mousse 8%	4	178.00 (7.118)	0.405
VCO mousse 80%	4	20.00 (14.697)	0.262
Positive control (CPP-ACP)	4	56.75 (8.995)	0.097
Negative control	4	252.25 (29.216)	0.298
Base without active ingredient	4	257.25 (40.525)	0.191

One-way ANOVA test; significant score based on $P < 0.05$. VCO, virgin coconut oil.

Table 4. Differences in The Number of Viable Fungal Colony Counts (*C. Albicans*) After Administration of VCO Mousse at Different Concentrations.

Tables 5 and 6 present comparisons of *S. mutans* and *C. albicans* biofilms, respectively, between two groups using the Tamhane test. The viable colony counts were statistically different between positive and negative controls, positive control and base without active ingredient, positive control and 0.8% and 8% VCO groups, and between the 80% VCO group and negative controls, base without active ingredient, and 0.8% and 8% VCO groups.

Group	*p*-value
VCO mousse 0.8% vs. 8%	0.271
VCO mousse 0.8% vs. 80%	0.001*
VCO mousse 0.8% vs. base	1.000
VCO mousse 0.8% vs. positive control	0.001*
VCO mousse 0.8% vs. negative control	1.000
VCO mousse 8% vs. 80%	0.001*
VCO mousse 8% vs. base	1.000
VCO mousse 8% vs. positive control	0.001*
VCO mousse 8% vs. negative control	0.144
VCO mousse 80% vs. base	0.001*
VCO mousse 80% vs. positive control	1.000
VCO mousse 80% vs. negative control	0.001*
Base vs. positive control	0.001*
Base vs. negative control	1.000
Positive control vs. negative control	0.001*

Post hoc Tamhane test, *significant score based on $p < 0.05$. VCO, virgin coconut oil.

Table 5. Post hoc Analysis of The Difference on *S. Mutans* Total Viable Colony Counts (CFU/Ml) in Treatment Intergroup.

Group	*p*-value
VCO mousse 0.8% vs. 8%	0.133
VCO mousse 0.8% vs. 80%	0.001*
VCO mousse 0.8% vs. base	1.000
VCO mousse 0.8% vs. positive control	0.004*
VCO mousse 0.8% vs. negative control	1.000
VCO mousse 8% vs. 80%	0.001*
VCO mousse 8% vs. base	0.344
VCO mousse 8% vs. positive control	0.001*
VCO mousse 8% vs. negative control	0.168
VCO mousse 80% vs. base	0.008*
VCO mousse 80% vs. positive control	0.115
VCO mousse 80% vs. negative control	0.001*
Base vs. positive control	0.001*
Base vs. negative control	1.000
Positive control vs. negative control	0.006*

Post hoc Tamhane test, *significant score based on $P < 0.05$. VCO, virgin coconut oil.

Table 6. Post hoc Analysis of The Difference in *C. Albicans* Total Viable Colony Counts (CFU/Ml) in Treatment Intergroup.

Discussion

We analyzed the effectiveness of VCO mousse at concentrations of 0.8%, 8%, and 80% against biofilms of *S. mutans* and *C. albicans* in children with ECC. VCO has been reported to have anti-inflammatory, antioxidant, antifungal, and antibacterial effects.[22,23]

VCO mousse significantly reduced the number of *S. mutans* and *C. albicans* colonies in the group treated with 80% VCO, and results were comparable with those of the positive control group (CPP-ACP). VCO proved to function as an antibacterial and antifungal agent. The antibacterial effect is in line with reports of other studies stating that the lauric acid content in coconut oil has antimicrobial activity, especially

against *Streptococcus* species,[18,20] and that lauric acid showed higher antimicrobial activity compared with chlorhexidine.[24] Kaushik reported a significant decrease in numbers of *S. mutans* after rinsing the mouth with 10 mL coconut oil for 10 minutes.[25] Beena et al. suggested that the antimicrobial effect of VCO against *C. albicans* is as good as that of chlorhexidine and significantly higher than that of ketoconazole.[13] Lui Dwen Tjin reported that VCO has an antifungal as well as a synthetic antifungal effect.[23] Others have reported that lauric acid also is capable of damaging the *C. albicans* cell wall membrane.[21]

This effect may be due to the ingredients contained in the VCO that are saturated with fatty acids, such as lauric(48%–53%), caproic, and caprylic acids.[26] Previous studies have reported that the lauric acid content in VCO damaged the membrane of fat or inhibited protein synthesis, which can kill some bacteria and is capable of inhibiting aggregation of biofilm formation.[20,24] Monolaurin and the other monoglyceride compounds can alter bacterial cell walls, penetrate and inhibit cell membranes, and inhibit enzymes that have a role in energy production and nutrient transfer that can cause death of bacteria.[25]

In addition, the lauric acid content in VCO also is able to inhibit growth of several types of other microorganisms, including *C. albicans*.[13,26,27] Caproic acid can kill *C. albicans* most quickly and effectively, followed by lauric acid and monocaprin. Caprid acid is capable of inhibiting *C. albicans* filaments and reducing Candida adhesion and biofilm formation. Fatty acids enter the lipid bilayer of fungal membranes and physically disturb the fungal cell membrane, resulting in increased membrane fluidity that causes cell membrane disorganization, generally leading to conformational changes in membrane proteins, intracellular release of the compound, cytoplasmic disorders, and ultimately cell disintegration. Also, fatty acids act by inhibiting formation of hyphae, which prevents invasion of host causing reduced pathogenity.[23] 1-Monolaurin has a potential antifungal effect based on susceptibility and biofilm testing.[28]

In this study, the negative and positive controls showed no significant difference in the crystal violet test. The possible cause of this outcome was the cloudy ingredient of the mousse, such as Na-CMC, Na-Sakarin, guar gum, titanium dioxide, glycerol, zinc oxide, propylene glycol, D-sorbitol, Aqua Des, natrium benzoate, phosporic acid, and xylitol. Similar results also were found in the CPP-ACP group compared with the negative controls. Further research is needed.

Conclusions

VCO mousse 80% has antibacterial and antifungal effects in inhibiting biofilms of *S. mutans* and *C. albicans* based on the viability test (crystal violet test and CFU) and as well as positive controls (CPP-ACP).

Declaration of Interest

The authors declare no conflict of interest in this research and this work is supported by Hibah PITTA 2018 funded by DRPM Universitas Indonesia No.2154/UN2.R3.1/HKP.05.00/2018.

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