

Original Research Article

Oral *Candida albicans* in patients in the ICU of an Brazilian hospital school and in vitro susceptibility of isolated yeasts to extra-virgin coconut oil

ABSTRACT

Aims: To verify the presence and growth of *Candida* in oral samples from hospitalized patients in Intensive Care Unit (ICU) and to detect the *in vitro* susceptibility of isolated yeasts to extra-virgin coconut oil.

Study design: This is a Cross-sectional study used to oral *Candida* collect from hospitalized patients in Intensive Care Unit in specific time interval.

Place and Duration of Study: Sample: ICU of the Hospital das Clínicas (HC - hospital school) of the Universidade Federal de Goiás (UFG) (ICU/HC/UFG), between May to October 2015.

Methodology: Isolation and the identification in cultures of oral *Candida* derived from patients of the ICU/HC/UFG were performed according to Kreeger van-RIJ. In triplicate, these *C. albicans* yeasts were seeded in Sabouraud dextrose agar (SDA) with chloramphenicol with the addition of commercial use extra-virgin coconut oil, and filtered at concentrations from 5 to 50 %. Petri dishes with culture medium and varied concentrations of coconut oil were subjected to 37°C / 24 h, and those that did not exhibit growth of fungal colonies were considered positive.

Results – There was a significant increase in the number of patients colonized with *Candida* yeasts in the first 72 hours after ICU admission, followed by stabilization at 96 hours. The *Candida* species isolated was *Candida albicans*. All cultures of oral *C. albicans* from ICU / HC / UFG patients, regardless of patient length of stay, were susceptible to the concentration of 30% coconut oil present in the culture medium of SDA plus antibiotic.

Conclusions – Keeping patients in the ICU/HC/UFG influenced the number of individuals with *Candida* yeasts in the buccal cavity, particularly in the first 72 hours. *Albicans* is the *Candida* species most commonly detected in the buccal mucosa of patients undergoing hospital intensive care. Extra-virgin coconut oil proved to have a natural antifungal effect in inhibiting oral *C. albicans* isolates

Keywords: *Candida albicans*; Intensive Care Unit; Coconut oil

INTRODUCTION

Frequent reports of oral candidiasis in patients hospitalized in intensive care units (ICUs) have stimulated the search for new supplemental therapeutic alternatives, such as plant therapies, which can spur therapeutics, such as new pharmacological resources in mitigating or curing candidiasis.

Coconut oil is a plant substance consisting of fatty acids, among which approximately 90% are saturated acids [1]. It is obtained through pressing the pulp or core of coconut (*Cocos nucifera* L.). It is a natural product commonly used in the cosmetics industry for preparing soaps and creams and also in food preparation in some Asian countries. It acts as a complementary food, with various properties beneficial to health, strengthening the immune system and easing digestion, nutrient absorption and reduction in risk factors for cardiovascular disease [1,2].

An antimicrobial capacity involving an antifungal effect has been associated with coconut oil. Such pharmacological properties are a result of the biochemical composition of coconut oil, which includes lauric acid, myristic acid, and caprylic acid [1-3]. These acids hold medium-chain saturated fatty acids that act together to ensure rapid elimination of fungal infection through destructuring the cell wall [4].

A beneficial association has been observed between topical and oral administration of coconut oil and treatment of candidiasis [5,6]. *Candida* is a yeast-form fungus present in microbiota of the skin, mucous, and folds of the human body as of birth. Clinical manifestation is more common when the patient has low cell immunity, becoming vulnerable to a series of microbiological infections, as occurs in patients hospitalized in intensive care units [7]. White patches covering all internal areas of the oral cavity characterize high yeast-form colonization by *Candida*, which predisposes the ICU patient to the condition of pseudomembranous candidiasis [7,8]. Yeasts of the *Candida* genus are highly opportunistic microorganisms, especially *Candida albicans*, due to the high capacity for manifestation of virulence factors (adherence, morphological dimorphism, phenotypic variability – switching enzymes: aspartyl proteinase and phospholipases, and toxins: toxic glycoproteins and canditoxins) when exposed to a pathogenic condition [8-10].

The aim of this study was to detect the presence and growth of *Candida* isolates in oral samples from patients hospitalized in the ICU of the *Hospital das Clinicas* of the Universidade Federal de Goias (ICU/HC/UFG) and verify the *in vitro* sensitivity of the isolated yeasts to extra-virgin coconut oil.

METHODOLOGY

Candida samples

Ninety samples of salivary secretion were collected at time zero, 72 hours, and 96 hours after admission, from 30 patients in the ICU/HC/UFG in the period from May to October 2014. Salivary specimens came from adults subjected to mechanical ventilation, tracheostomies, and non-invasive ventilation, regardless of age, gender, and clinical

condition, with or without use of antibiotherapy. A sterilized swab was used to collect each saliva sample from the patient, which was placed in a test tube containing 4 mL of sterilized water for transport to the Laboratory of Medical Microbiological Analysis of the Instituto de Patologia Tropical e Saúde Pública of the Universidade Federal de Goiás (LAMSA/IPTSP/UFG) for fungal isolation and identification.

This study protocol was performed according to the Helsinki declaration and approved by ethics committee of the Clinical Hospital of Federal University of Goiás (Hospital das Clínicas of the Universidade Federal de Goiás - CEPMHA/HC/UFG), number protocol no. 634.432/2014. Written informed consent was obtained from each participant.

Isolation and identification of yeasts

The yellowish white colonies that developed up to 15 days in test tubes containing 4 mL of Sabouraud dextrose agar (SDA) medium with chloramphenicol were identified through formation of germination tubes in fetal bovine serum, formation of chlamydospores in cornmeal agar with the addition of tween 80, and carbohydrate assimilation and fermentation test [7,8].

SDA with the addition of coconut oil

Petri dishes of 20 mL volume were prepared with liquefied Sabouraud dextrose agar at a temperature of 30°C with the addition of chloramphenicol and filtered extra-virgin coconut oil, for commercial use, at different concentrations from 5 to 50 % at 5% intervals. These Petri dishes, after preparation of the defined culture medium, were subjected to the sterilization test at 37°C / 24h [10].

Suspension and subjection of Candida to SDA with coconut oil

Candida-test suspensions, in distilled and sterilized water, McFarland scale 3, were carried out and seeded with sterilized swabs in Petri dishes with SDA and chloramphenicol at different concentrations of extra-virgin coconut oil, in triplicate, and kept at 37°C. Petri dishes with the culture medium used that did not exhibit macroscopic growth of *Candida* colonies were considered positive.

Statistics

The chi-square test (X^2) was used to compare the analyzed groups. The values obtained were considered statistically significant when $p < 0.05$. The Sperman Correlation Coefficient was used correlate the numbers of samples of *Candida* isolated from salivary

secretion of individuals hospitalized in the ICU/HC/UFG at different time intervals (group I – time zero, group II – 72 h, and group III – 96 h) for identification of association between the time period studied and the number of positive cases.

RESULTS AND DISCUSSION

The occurrence of yeasts of *Candida albicans* was observed in 61/90 (67.8%) of the samples of salivary secretion collected from patients hospitalized in the ICU/HC/UFG, without reference to the time the individual remained in the hospital unit considered in this study. Considering the interval of hospitalization time of the patient in this unit, manifestation of *Candida* isolates was detected in 13/30 (43.3%) of patients (saliva samples) at time zero; in 22/30 (73.3%) at 72 h; and in 26/30 (86.7%) at 96 h (Table 1). Statistical test using the Spearman correlation coefficient ($p=0,976$) indicated a positive correlation between length of ICU stay (in hours) and the increase in the number of cases of *C. albicans*.

Insert Table 1

All the oral samples of *Candida albicans* identified in this study, regardless of the hospitalization time of the patients in the ICU/HC/UFG, showed *in vitro* susceptibility to a concentration greater than or equal to 30% extra-virgin coconut oil present in Petri dishes containing SDA with the addition of chloramphenicol (Table 2).

Insert Table 2

Yeasts of *Candida albicans* continue to be the yeast-form fungus predominantly detected in the saliva of patients hospitalized in the ICU/HC/UFG. The high rate of fungi in the buccal mucosa of these individuals is due to microorganism carrying capacity as a compositional element of the oral microbiota since birth, the immediate clinical-laboratorial situation of each patient, and the use of therapeutic procedures with or without antibiotics, depending on the disease of the patient [7,10]. A decline in the health of these patients was also observed mycologically; in the interval from zero (Group I) to 72 h (Group II) and then to 96 h (Group III) of hospitalization in the ICU/HC/UFG, the number of patients from whom *Candida* in salivary secretion was isolated doubled; therefore, a statistically significant ($p < 0.05$) increase occurred among the groups considered. This fungal and statistical ($p > 0.05$) reality was not observed in these same patients in the time interval from 72 to 96 h in the ICU. Siqueira *et al.* [11] evaluated the oral microbiota of patients in Brazilian ICU units and detected continuity of *Candida albicans* as a basic agent of healthcare associated infections (HAIs). In another study conducted in the ICU of a hospital in England, *Candida* colonization was detected in 75% of the patients in one or more anatomical sites, such as the mouth and

130 groin [12]. Likewise, in a hospital in the south of Iran, 63.2% of the patients exhibited
131 colonization of *Candida* in different anatomical locations during hospitalization [13]. Bajwa
132 and Kulshrestha [14] affirmed that with the advances in intensive medicine and introduction
133 of broad spectrum antibiotics, the occurrence of invasive fungal infections in intensive care is
134 rising, especially in patients with immunosuppression.

135 *Candida albicans* is responsible for up to 78 % of nosocomial candidiasis, however, reports
136 demonstrating infections of *Candida non-albicans* species have been also documented [15].
137 For *C. albicans* identification, we used tests of germination tubes formation in fetal bovine
138 serum, formation of chlamydospores in corn-meal agar with the addition of tween 80, and
139 biochemical tests (carbohydrate assimilation and fermentation) [7,8]. However, due to the
140 high degree of phenotypic similarity between *Candida* species (*Candida non-albicans*), and
141 due some mutations in *C. albicans* that may inhibit the formation of germ tubes or alter
142 colony, the phenotypic and biochemical identification problems are imminent. [15,16].
143 Consequently, it is important to identify the causative organism to the species level correctly
144 by molecular methods. Therefore, is highly recommended the identification and
145 discrimination of ethological agents for early treatment, and preventing the invasion.
146 Therefore, alternative molecular assay with high specificity, reproducibility and sensitivity are
147 necessary [15]. Despite being a future perspective, in our work, we did not carry out
148 molecular identification and therefore did not eliminate the possibility of *Candida non-*
149 *albicans* among isolates.

150 *Candida* and *Aspergillus*, followed by *Cryptococcus* and *Histoplasma*, in endemic areas
151 throughout the world, are the main fungi involved in attacking individuals in intensive
152 therapy. In our study, compatibility was observed regarding the occurrence of colonization of
153 buccal mucosa by *Candida* yeast-form fungi in patients undergoing intensive therapy. Amiri
154 *et al.* [17] examined an increase in the number of patients afflicted by *Candida* yeasts in
155 relation to length of stay in the hospital ICU, and they analyzed risk factors leading to
156 hospitalization in intensive therapy in France, such as diabetic individuals and those afflicted
157 by chronic renal disease. They found an increase in the number of patients that were
158 carriers of oropharyngeal colonization by *Candida* when they were placed in the ICU. The
159 difficulty of adequate oral hygiene, associated with the weakened state of the immunological
160 system of the patient, leading to the dental conditions of periodontitis and gingivitis, among
161 others, seems to contribute to highly increased proliferation of *Candida* isolates in patients in
162 hospital ICUs [7-11]. This fungal condition was observed in the first 72 h of patients in the
163 ICU/HC/UFG. Stabilization in the following 24 h (72 to 96 h) of hospitalization is probably
164 due to the microbiological relation of oral microorganisms among themselves, without
165 considering the immediate situation of the patient and the therapy used [7,9].

166 Susceptibility of *Candida* yeasts to natural products has been observed, as in the case
167 of the use of coconut oil [1-5]. This antifungal activity presents an alternative for refining
168 natural medications for treatment of infections brought about by fungi. Studies, such as ours,
169 following the model of the agar diffusion method advocated by the Clinical and Laboratory
170 Standards Institute (CLSI) of the Centers for Disease Control and Prevention (CDC) of the
171 United States [11], have shown the gradual sensitivity of oral samples of *Candida albicans*
172 isolates from patients in the ICU/HC/UFG to variation in concentrations from 10 to 30 % of
173 extra-virgin coconut oil added to the SDA medium with the addition of antibiotics. The 10%
174 coconut oil susceptible strain was isolated from the same patient at the different collection
175 times. However, at other concentrations it was evident that the susceptibility to coconut oil
176 may suffer interference from the adaptability of the sample to the buccal microbiota and the
177 momentary state of the patient's immune status at the time of collection. Thus, time
178 interferes with susceptibility to the fungus, that is, over time it adapts to the situation of the
179 oral microbiota and becomes more resistant. This was evident at 25% concentration where
180 strains isolated at time 0 were susceptible 10 (76.9%) while most strains isolated at 72h and
181 96h times were resistant, requiring a concentration of 30% oil to reach Susceptibility in most
182 strains. A study conducted in Ibandan, Nigeria [18] exhibited 52 isolates of the *Candida*
183 species, derived from diverse human biological materials, as having susceptibility *in vitro* to
184 coconut oil by the agar dilution method, and 17/52 (32.7%) cultures of *Candida albicans*
185 identified were completely sensitive to the plant extract used with a minimum inhibitory
186 concentration of 25%. A similar result was observed in our study. The effectiveness of
187 coconut oil has also been shown in experiments [19], in control of oral microbial populations
188 of *Streptococcus mutans* and *Candida albicans*, reducing the number of dental cavities and
189 oral infections in humans. Early colonization and a high number of carcinogenic
190 microorganisms present in the oral microbiota of Egyptian children [20], such as yeasts of
191 *Candida albicans*, have encouraged the use of coconut oil as an alternative chemotherapy
192 agent, obtaining laboratory results compatible with the use of azole antifungal drugs, such as
193 ketoconazole. Daily oral hygiene with coconut oil in Nigeria adolescents [21], in the 16-18
194 age range, reduced the occurrence of gingivitis, showing that the presence of lauric acid
195 stimulates the anti-inflammatory and antimicrobial effect of the plant extract used. Takahashi
196 et al. [22], working with experimental models of mice infected with oral candidiasis, found
197 antifungal activity from the four fatty acids of coconut oil (caproic acid, caprylic acid, capric
198 acid, and lauric acid), inhibiting the morphological dimorphism of *Candida* yeasts from yeast-
199 form (infectant) to mycelial (parasitic) fungi necessary for establishment of the infectious
200 process. Caprylic acid was the fatty acid with best therapeutic response using 50 µL three
201 times on *Candida* lesions on the tongues of mice. Association of terpineol-4, the main oil of

202 melaleuca [23], a plant native to Australia and known as the tea tree, with capric acid, a fatty
203 acid of coconut oil, has shown to be a potential natural medication *in vitro* and *in vivo*, with
204 synergistic antimycotic activity on the capacity of pathogenic variability of *Candida* isolates.
205 Nevertheless, Cavalcanti [24] showed that extracts of *Eugenia uniflora* (pitanga tree) leaves,
206 bark from the trunk of *Libidibia ferrea* (*pau ferro* or Brazilian ironwood), and *Psidium guajava*
207 (guava tree) leaves, obtained from the Brazilian *Caatinga* biome, constitute plant therapeutic
208 alternatives due to *in vitro* susceptibility of *Candida* to plant extracts, through the fungistatic
209 effect on standard ATCC yeasts of *Candida* species, with minimum inhibitory concentration
210 values from 15.62 to 6.25 µg/mL. Santos *et al.* [25], in a comparative study on alternatives to
211 the use of plant extracts, with the use of an enzymatic oral solution based on
212 lactoperoxidase in patients treated in a hospital ICU, in relation to a control group with use
213 of cetylpyridinium chloride, showed, in clinical evaluation of the simplified oral hygiene index
214 (OHI-S), that there was statistical significance when compared to the study and control
215 groups. The choice of an enzymatic product as an auxiliary method in reduction of oral
216 bacterial plaque arises from the absence of abrasive substances (alcohol, detergent, and
217 stain) in its composition, which hurt even more the already compromised buccal mucosa. It
218 is also important that the enzymatic product contains lactoferrin, which, through its action
219 and interactions on saliva, reduces the occurrence of *Candida albicans* and *Candida krusei*
220 in buccal mucosa. Even so, the antifungal capacity of extra-virgin coconut oil on fungi is
221 broader. In addition to inhibiting proliferation and, consequently, combat the pathogenic
222 action of *Candida* yeasts in infected organic mucosas, it has antimycotic action on isolates of
223 *Cryptococcus neoformans* and filamentous fungi, such as *Aspergillus*, *Penicillium*,
224 *Cladosporium*, *Fusarium*, *Alternaria*, and *Fonsecaea pedrosoi* [26].

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228 CONCLUSION

229 This study reveals that patient stay in the ICU in the hospital environment seems to
230 influence the occurrence of *Candida* yeasts in the buccal mucosa, mainly because of the
231 daily difficulty in carrying out adequate oral hygiene. *Albicans* continues to be the *Candida*
232 species most detected in the buccal cavity of patients in the ICU, particularly in the first 72
233 hours. The use of extra-virgin coconut oil *in vitro*, in a maximum concentration of 30%,
234 added to the SDA medium with the addition of chloramphenicol, could inhibit the proliferation
235 of buccal isolates of *Candida albicans* originating from patients in a hospital ICU. Thus,
236 extra-virgin coconut oil is an alternative plant therapy effective in controlling the proliferation
237 of *Candida* yeasts in buccal mucosa and infection of buccal mucosa by *Candida* yeasts.

CONSENT

It is not applicable.

ETHICAL APPROVAL

Approval for this study was obtained from the Clinical Hospital of Federal University of Goiás (Hospital das Clínicas of the Universidade Federal de Goiás - CEPMHA/HC/UFG), number protocol no. 634.432/2014.

References

1. Eyres L, Eyres MF, Chisholm A, Brown RC. Coconut oil consumption and cardiovascular risk factors in humans. *Nutr Rev*. 2016;74(4):267-80.
2. Yong JW, Ge L, Ng YF, Tan SN. The chemical composition and biological properties of coconut (*Cocos nucifera* L.) water. *Molecules*. 2009;14(12):5144-64.
3. Shino B, Peedikayil FC, Jaiprakash SR, Ahmed Bijapur G, Kottayi S, Jose D. Comparison of Antimicrobial Activity of Chlorhexidine, Coconut Oil, Probiotics, and Ketoconazole on *Candida albicans* Isolated in Children with Early Childhood Caries: An In Vitro Study. *Scientifica* (Cairo). 2016;2016:7061587.
4. Lima EB, Sousa CN, Meneses SN, Ximenes SC, Santos Júnior MA, Vasconcelos GS, et al. *Cocos nucifera* (L.) (Arecaceae): A phytochemical and pharmacological review. *Braz. J. Med. Biol. Res*. 2015;48(11):953-64.
5. Gunsalus KT, Tornberg-Belanger SN, Matthan NR, Lichtenstein AH, Kumamoto CA. Manipulation of Host Diet to Reduce Gastrointestinal Colonization by the Opportunistic Pathogen *Candida albicans*. *mSphere*. 2015;18;1(1).
6. Ogbolu DO, Oni AA, Daini OA, Oloko AP. In vitro antimicrobial properties of coconut oil on *Candida* species in Ibadan, Nigeria. *J Med Food*. 2007;10(2):384-7.
7. Choi JY, Kwak YG, Yoo H, Lee SO, Kim HB, Han SH, et al. Korean Nosocomial Infections Surveillance System (KONIS). Trends in the distribution and antimicrobial susceptibility of causative pathogens of device-associated infection in Korean intensive care units from 2006 to 2013: results from the Korean Nosocomial Infections Surveillance System (KONIS). *J Hosp Infect*. 2016;92(4):363-71.
8. Tamura NK, Negri FMN, Bonassoli LA, Svidzinski TIE. Virulence factors for *Candida* spp recovered from intravascular catheters and hospital workers' hands. *Rev. Soc. Bras. Med. Trop*. 2007;40(1):91-3.
9. Bergsson G, Arnfinnsson J, Steingrímsson O, Thormar H. *In vitro* killing of *Candida albicans* by fatty acids and monoglycerides. *Antimicrob Agents Chemother*. 2001;45(11):3209-12.
10. Höfs S, Mogavero S, Hube B. Interaction of *Candida albicans* with host cells: virulence factors, host defense, escape strategies, and the microbiota. *J Microbiol*. 2016;54(3):149-69.

- 288 11. Siqueira JSS, Batista SA, Júnior AS, Ferreira MF, Agostini M, Torres SR. Oral
289 candidiasis in patients admitted to ICU. Rev. Bras. Odontol. 2014;71(2):176-9.
290
- 291 12. Cliff PR, Sandoe JT, Heritage J, et al. Use of multilocus sequence typing for the
292 investigation of colonisation by *Candida albicans* in intensive care unit patients. J. Hosp.
293 Infect. 2008;69(1):24-32.
294
- 295 13. Badiie P, Alborzi A, Joukar M. Molecular assay to detect nosocomial fungal infections in
296 intensive care units. Eur. J. Intern. Med. European Federation of Internal Medicine. 2011;22
297 (6): 611-5.
298
- 299 14. Bajwa SJ, Kulshrestha A. Fungal Infections in Intensive Care Unit: Challenges in
300 Diagnosis and Management Ann Med Health Sci Res. 2013;3(2):238–44.
301
- 302 15. Rezazadeh E, Moazeni M, Sabokbar A. Use of cost effective and rapid molecular tools
303 for identification of *Candida* species, opportunistic pathogens. Curr Med Mycol. 2016;2(3):1-
304 4
305
- 306 16. Li L, Zhang C, Konopka JB. A *Candida albicans* temperature-sensitive cdc 12-6 mutant
307 identifies roles for septins in selection of sites of germ tube formation and hyphal
308 morphogenesis. Eukaryot Cell. 2012; 11(10):1210-8.
309
- 310 17. Amiri HM, Frandah W, Colmer-Hamood J, Raj R, Nugent K. Risk factors
311 of *Candida* colonization in the oropharynx of patients admitted to an intensive care unit .Ann
312 Med Health Sci Res. 2013;3(2):238–44.
313
- 314 18. Ogbolu DO, Oni AA, Daini OA, Oloko AP. In vitro antimicrobial properties of coconut oil
315 on *Candida* species in Ibadan, Nigeria. J Med Food. 2007;10(2):384-7.
316
- 317 19. Barnabé W, de Mendonça Neto T, Pimenta FC, Pegoraro LF, Scolaro JM. Efficacy of
318 sodium hypochlorite and coconut soap used as disinfecting agents in the reduction of
319 denture stomatitis, *Streptococcus mutans* and *Candida albicans*. J Oral Rehabil. 2004;
320 31(5):453-9
321
- 322 20. Shino B, Peedikayil FC, Jaiprakash SR, Ahmed Bijapur G, Kottayi S, Jose D.
323 Comparison of Antimicrobial Activity of Chlorhexidine, Coconut Oil, Probiotics, and
324 Ketoconazole on *Candida albicans* Isolated in Children with Early Childhood Caries: An *In*
325 *Vitro* Study. Scientifica. 2016:7061-587.
326
- 327 21. Peedikayil FC, Sreenivasan P, Narayanan A. Effect of coconut oil in plaque related
328 gingivitis - A preliminary report. Niger Med J. 2015;56(2):143-7.
329
- 330 22. Takahashi M, Inoue S, Hayama K, Ninomiya K, Abe S. Inhibition of *Candida* mycelia
331 growth by a medium chain fatty acids, capric acid in vitro and its therapeutic efficacy in
332 murine oral candidiasis. Med Mycol J. 2012;53(4):255-61.
333

334

335 23. Ninomiya K, Hayama K, Ishijima S, Takahashi M, Kurihara J, Abe S. Effects of inhibitory
 336 activity on mycelial growth of *Candida albicans* and therapy for murine oral candidiasis by
 337 the combined use of terpinen-4-ol and a middle-chain fatty acid, capric acid. Yakugaku
 338 Zasshi. 2013;133(1):133-40.

339

340 24. Cavalcanti YW Péres, ALA de L, Xavier, GDR, de Almeida LFD, Padilha, WWN.
 341 Antifungal Activity of Brazilian Plant Extracts Against *Candida* Strains. Revista Brasileira de
 342 Ciências da Saúde. 2012;16(1)43-48.

343

344 25. Santos PSS, Mello WR, Wakim RCS, Paschoal MAG. Use of Oral Rinse with Enzymatic
 345 System in Patients Totally Dependent in the Intensive Care Unit. RBTI.2008; 20(2):154-59.

346

347 26. DebMandal M, Mandal S. Coconut (*Cocos nucifera* L.: Arecaceae): In health promotion
 348 and disease prevention. Asian Pacific J. Trop. Med. 2011;4(3):241-47.

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355 **Table 1. Occurrence of *Candida albicans* isolates in saliva samples of patients**
 356 **hospitalized at different time intervals (number of patients = 30)**

Time of hospitalization/Groups (h)/(n)	Positive samples n (%)	Negative samples n (%)
zero (I)	13 (43.3) ^{a,b}	17 (56.7)
72 (II)	22 (73.3) ^c	08 (26.7)
96 (III)	26 (86.7)	04 (13.3)
Total	61 (67,8)	29 (32,2)

357 (a) - Groups I and II – Statistical ratio; $X^2 = 5.54$; $p < 0.05$; (b) - Groups I and III – Statistical
 358 ratio; $X^2 = 12.36$; $p < 0.05$; (c) - Groups II and III – Statistical ratio; $X^2 = 1.64$; $p > 0.05$.
 359 Sperman correlation coefficient - positive ($p=0,976$).

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362 **Table 2. Minimum percentage concentration of extra-virgin coconut oil able to inhibit**
 363 **the proliferation of *Candida albicans***

SDA with extra- virgin coconut oil concentration (%)	<i>C. albicans</i> isolated from saliva of patients of the ICU/HC/UFG at different time intervals, and susceptible in SDA to minimum percentage concentration of extra-virgin coconut oil		
	Time (h)		
	Zero n (%)	72 n (%)	96 n (%)
5	-	-	-
10	01 (7.7)	01 (4.5)	01 (3.9)
15	02 (15.4)	04 (18.2)	03 (11.5)
20	-	03 (13.7)	01 (3.9)
25	10 (76.9)	02 (9.1)	03 (11.5)
30	-	12 (54.5)	18 (69.2)
> 35 to 50	-	-	-

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