Orbital
The electronic journal of chemistry

the electronic journal of chemistry

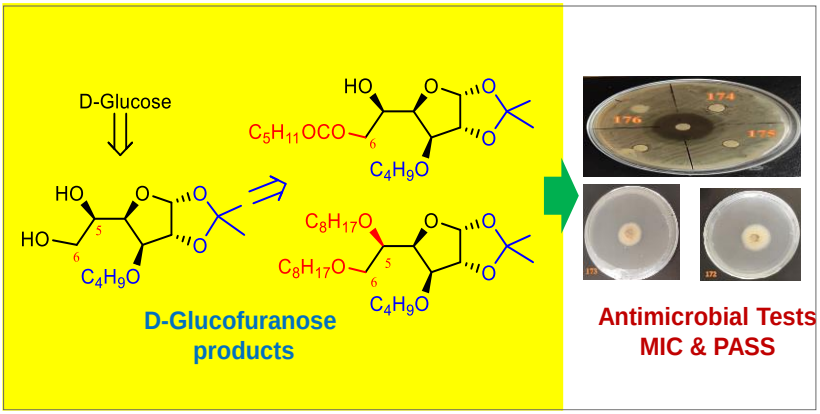
Full Paper | http://dx.doi.org/10.17807/orbital.v15i2.18152

1,2-*O*-Isopropylidene-3-*O*-butylglucofuranose-derived Ester, and Ether: Synthesis, Characterization, and Antimicrobial Study

Tasnim Rahman Anisa [a], Md. Badrul Islam [a], Md. Inshaful Islam [a], Mohammed Mahbubul Matin* [a]

Sugar-fatty acid esters (SFAEs, especially sucrose- and glucose-based ones) have dominated the chemical industries for more than 50 years. In comparison to other carbohydrate products, SFAEs serve essential roles in a variety of industries, including the food, pharmaceutical, and cosmetic industries. In this context, the 6-*O*-hexanoyl ester of 3-*O*-butyl-1,2-*O*-isopropylidene-α-D-glucofuranose was synthesized from commercially available D-glucose in a few steps. For a comparative biological study, the 5,6-di-*O*-benzyl ether of 3-*O*-butyl-1,2-*O*-isopropylidene-α-D-glucofuranose was also prepared and characterized. An *in vitro* antimicrobial test of all the ester and ether compounds indicated that these compounds are more susceptible to fungi than bacteria. Also, they have more potential for *A. niger* than *A. flavus*. According to Prediction of Spectra for Substances (PASS), the chemicals found in the current investigation have a variety of potential biological functions.

Graphical abstract



Keywords

Antimicrobials
Glucofuranose
PASS analysis
Sugar fatty acid esters
Zone of inhibition

Article history

Received 08 Mar 2023
Revised 11 Jun 2023
Accepted 12 Jun 2023
Available online 13 Jul 2023

Handling Editor: Jamal Rafique

1. Introduction

Sugar-fatty acid esters (SFAEs) are environmentally friendly and biocompatible surfactants used in meals, personal care products, and pharmaceuticals [1-4]. They are value-added products made from cheap renewable feedstocks (sugars and fatty acids). Due to their effective surface activities and strong emulsifying, stabilizing, and detergency policies, they are of great interest to researchers and chemical companies [5,6]. They can inhibit biofilm formation [7,8], possess antimicrobial properties, and engage in other biological activities [9, 10]. They are promising anti-tumor drugs and can potentially be effective against some viruses and fungi [11, 12].

[a] Bioorganic and Medicinal Chemistry Laboratory, Department of Chemistry, University of Chittagong, Chittagong, 4331, Bangladesh. *Corresponding author. E-mail: **mahbubchem@cu.ac.bd**

Glycolipids, in particular sugar-based esters, are environmentally friendly, odorless, nontoxic, and tasteless substances [13-15]. SFAEs have both hydrophilic and hydrophobic parts. Its hydrophilic head moiety is a mono-, di-, or oligosaccharide. One or more alkyl/ester moieties with various chain lengths make up the hydrophobic tail most frequently [16]. The hydrophile/lipophile balance greatly contributes to their various physical (such as shelf life, taste, odor, non-toxicity, biodegradability, etc.) and biological (interactions with the target enzymes) activities [17, 18].

Site-selective mono-acyl SFAEs production, however, has long been plagued by a number of fundamental difficulties [19-21]. Three main difficulties encountered during SFAEs synthesis are (i) the inability/poor ability of the acyl donor and acceptor reactants to mix well, (ii) managing water activity throughout the reaction's time course, and (iii) regulating the selectivity of the hydroxyl group(s) [22,23]. In this respect, several esterification methods (such as – direct method, blocking-deblocking, catalyst, microwave, enzymatic method, etc.) have been developed [24-28]. Mechanochemical and biocatalytic approaches to developing greener processes were also reported recently [29]. Considering the improved yield and regioselectivity [30], direct acylation method was used for protected D-glucofuranose in the current study. Synthetic SEs-protected glucose esters have been discovered to be suited to boost therapeutic characteristics in hyper-proliferative and inflammatory drugs and open the door to the synthesis of newer bioactive compounds by regulating hydrophobic alkyl chains [31-34]. For instance, it has been discovered that 3-*O*-acyl glucofuranoses (**1a–c**; Figure 1) significantly regulate unchecked cell development in erythroid tumor cells [35]. The ester moiety, in particular the palmitoyl group found in glucopyranoside **2**, is related to papulacandin D (a common antifungal medication) and has shown antifungal activity against *Candida albicans* and *C. tropicalis* [36].

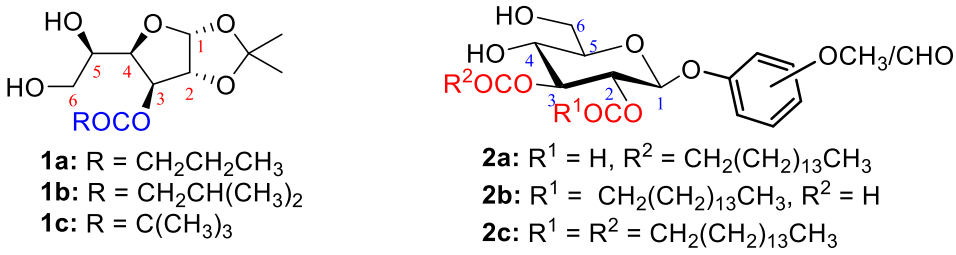


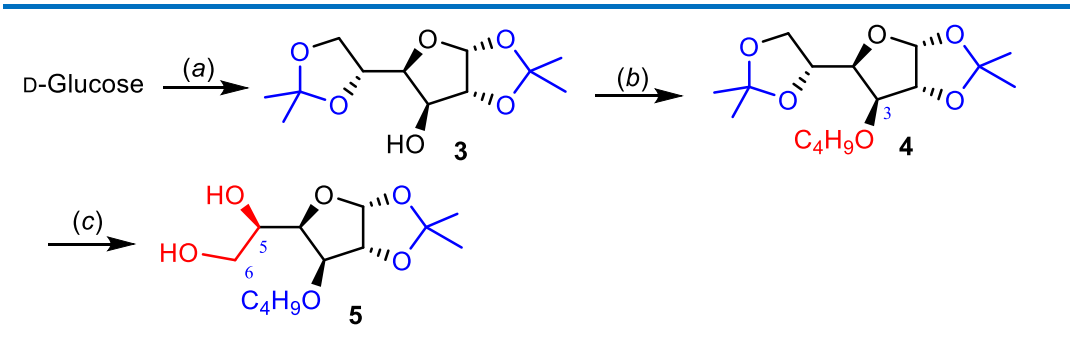
Fig. 1. Structures of glucose-based ester **1** and **2**.

The present study's primary goal is to prepare regioselective 6-*O*-acyl-3-*O*-butyl-1,2-*O*-isopropylidene-α-D-glucofuranose from easily available D-glucose, followed by an antimicrobial study. In this respect, diacetone D-glucose **3**, derived from D-glucose, which was first transformed into 3-*O*-butyl ether **4**. Selective deprotection of the 5,6-acetonide group in compound **4** furnished diol **5**, which upon unimolar hexanoylation at low temperature furnished 6-*O*-hexanoate **6** in good yield. For a comparative study, 5,6-di-*O*-octyl ether **7** from diol **5** containing the same α-D-glucofuranose skeleton was also prepared and characterized. The *in vitro* results of the synthesized glucofuranose compounds **4**-**7** indicated a greater potential for fungal infections than bacterial ones.

2. Results and Discussion

2.1. Synthesis of 3-*O*-butyl-1,2-*O*-isopropylidene-α-D-glucofuranose (5)

To fulfill our objectives, glucofuranose-based 5,6-diol **5** was synthesized from commercially available D-glucose in three steps. Initially, bisacetone D-glucose **3** was synthesized from available D-glucose with acetone in the presence of catalytic H_2SO_4 and $CuSO_4$, followed by recrystallization as a solid, mp 108-110 °C (lit [37] mp. 110-111 °C) in a 46% yield (Scheme 1).



Scheme 1. *Reagents and conditions:* (*a*) Dry acetone, oven dry $CuSO_4$, conc. H_2SO_4, rt, 24 h, 46% [37]; (*b*NaH, C_4H_9Br (BuBr), THF, TBAI, 0 °C, 30 min, rt, 4 h, 92%; (*c*) 10% H_2SO_4, MeOH-H_2O (1:1), rt, 4-5 h, K_2CO_3, ~95%.

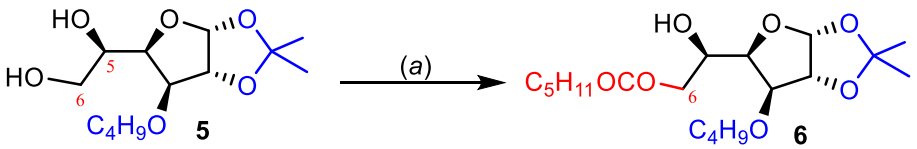
This bisactonide **3** has a free hydroxyl group at the C-3 position. As per our retrosynthetic analysis, this group was protected as the butyl ether group. Thus, treatment of **3** with butyl bromide in dry THF with NaH for 4 h furnished the product as a clear syrup in 92% yield (Scheme 1). This syrup showed the absence of hydroxyl stretching in its FT-IR spectrum (Figure S1) and hence indicated the attachment of a butyl group in the molecule. The fact was supported by its

^{1}H NMR (Figure S2), where additional nine aliphatic protons were observed at δ 3.60-3.65 [m, O-C*H*$_A$H$_B$(CH$_2$)$_2$CH$_3$], 3.51-3.56 [m, *O*-CH$_A$*H*$_B$(CH$_2$)$_2$CH$_3$], 1.55-1.60 [m, CH$_2$C*H*$_2$CH$_2$CH$_3$], 1.38-1.42 [m, (CH$_2$)$_2$C*H*$_2$CH$_3$], and 0.94 [t, 3H, *J* = 7.6 Hz, (CH$_2$)$_3$C*H*$_3$]. These nine protons corresponded to one butyl group in the molecule. Finally, analysis of its ^{13}C NMR spectra (Figure S3) provided proof of the fact, where four additional carbon signals were observed in the aliphatic region. The ^{13}C NMR spectrum showed signals at δ 111.7 [*C*(CH$_3$)$_2$], 108.9 [*C*(CH$_3$)$_2$], 105.3 (C-1), 82.6 (C-2), 82.1 (C-3), 81.2 (C-4), 72.6 (C-5), 70.4 (*O*-*C*H$_2$), 67.2 (C-6), 31.8 (*O*-CH$_2$*C*H$_2$CH$_2$CH$_3$), 26.9 [C(*C*H$_3$)$_2$], 26.8 [C(CH$_3$)$_2$], 26.3 [C(*C*H$_3$)$_2$], 25.4 [C(CH$_3$)$_2$], 19.2 [*O*-(CH$_2$)$_2$*C*H$_2$CH$_3$] and 13.8 [*O*-(CH$_2$)$_3$*C*H$_3$]. With the help of scanning and analyzing its DEPT-135, 2D COSY, 2D HSQC, and 2D HMBC (Figure S4) experiments, the positions of the signals were established for this 3-*O*-butyl product. Consequently, the compound's structure was determined to be 3-*O*-butyl-1,2:5,6-di-*O*-isopropylidene-α-D-*gluco*-1,4-furanose (**4**).

At this stage, selective acetonide removal at the C-5 and C-6 positions was achieved by treating fully protected compound **4** with 10% H_2SO_4 in MeOH-H_2O (1:1) for 4-5 h. Usual work-up and chromatography furnished a slower-moving single component as a thick liquid in 95% (Scheme 1). Its FT-IR spectrum (Figure S5) exhibited the appearance of OH stretching at 3230-3580 cm^{-1} (in the precursor compound **4**, OH stretching was absent). The compound showed only three three-proton singlets corresponding to one acetonide group and one butyl methyl protons in its ^{1}H NMR spectrum (Figure S6). The absence of one acetonide-related six protons (compared to its precursor **4**) clearly indicated the removal of one acetonide group. The selective removal of 5,6-acetonide functionality was previously reported from our laboratory on other substrates [38]. Its ^{13}C NMR spectrum also showed the absence of three carbons (one acetonide group) compared to its precursor, diacetonide **4**. With the help of scanning and analyzing its DEPT-135, 2D COSY, 2D HSQC (Figure S7), and 2D HMBC experiments, the positions of the signals were established for this 5,6-diol product. Thus, the compound was named 3-*O*-butyl-1,2-*O*-isopropylidene-α-D-*gluco*-1,4-furanose (**5**).

2.2. Synthesis and characterization of 3-*O*-butyl-6-*O*-hexanoyl-1,2-*O*-isopropylidene-α-D-*gluco*-1,4-furanose (6): Selective C-6 hexanoylation

To fulfill our objectives, 6-*O*-hexanoylation of diol **5** was conducted. To get the regioselectivity, diol **5** was treated slowly with an unimolar amount of hexanoyl chloride in dry pyridine for 11 h, and after chromatographic purification, it gave a semi-solid in good yield that resisted crystallization (Scheme 2).

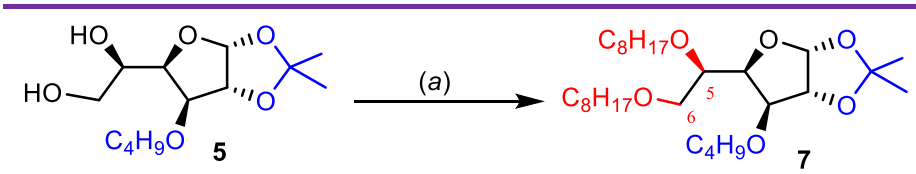


Scheme 2. *Reagents and conditions:* (*a*) *n*-C$_5$H$_{11}$COCl, dry pyridine, 0 °C, 30 min, rt, 10 h, 86%.

In the FT-IR spectrum (Figure S8), the semi-solid showed bands at 3120-3550 (br, OH), 2957, 2921, 2853 (C-H), 1710 (CO), 1376 [C(CH$_3$)$_2$], 1069 cm^{-1} (furanose ring). Of them, characteristic bands at 3120-3550 (br, OH) and 1710 cm^{-1} (CO) clearly informed the partial hexanoylation of the molecule. In the ^{1}H NMR spectrum (Figure S9), additional 11 protons (compared to diol **5**) resonated at δ 2.34-2.39 (2H), 1.55-1.70 (2H), 1.32-1.43 (4H), and 0.88-0.96 (3H). These protons corresponded to one hexanoyl group. Significantly, H-6 protons shifted considerably further down field (i.e., to higher frequencies) at δ 4.39 (as dd) and 4.17-4.22 (as m) than its precursor **5** (δ 3.75 and 3.85 ppm). The shift confirmed the attachment of a hexanoyl group at C-6 position. The appearance of an acyl carbonyl carbon signal at δ 174.2 ppm in its ^{13}C NMR spectrum confirmed the attachment of only one hexanoyl group in the molecule. A complete analysis of all the spectra and positional assignments by DEPT-135 (Figure S10), 2D COSY, 2D HSQC, and 2D HMBC experiments confirmed the attachment of one hexanoyl group at the C-6 position of the glucofuranose skeleton. Thus, it was established as 3-*O*-butyl-6-*O*-hexanoyl-1,2-*O*-isopropylidene-α-D-*gluco*-1,4-furanose (**6**). The formation of mono-*O*-hexanoate **6** is very reasonable due to the higher reactivity of the primary hydroxyl group.

2.3. Synthesis of 3-*O*-butyl-1,2-*O*-isopropylidene-5,6-di-*O*-octyl-α-D-*gluco*-1,4-furanose (7)

To compare biological tests between ester **6** and ether, we explored glucofuranose-derived 5,6-diol **5** for the octyl ether synthesis. Thus, treatment of diol **5** with dimolar octyl bromide in THF with NaH for 4.5 h followed by work-up and chromatographic purification, furnished a product in good yield (87%, Scheme 3).



Scheme 3. *Reagents and conditions:* (*a*) NaH, *n*-C$_8$H$_{17}$Br, dry THF, TBAI, 0 °C, 30 min, rt, 4 h, 87%.

Its FT-IR (neat) spectrum (Figure S11) showed peaks at 2952, 2921, 2853 (C-H), 1253 [C(CH$_3$)$_2$], and 1082 cm^{-1} (furanose ring). The spectrum showed the absence of hydroxyl stretching and indicated complete octanylation of

the molecule. Its ^{1}H NMR spectra (Figure S12) provided evidence for this, where an additional 34 protons appeared in the aliphatic region. It was further confirmed by analyzing its ^{13}C NMR spectrum, which showed the signals at δ 111.6 [*C*(CH$_3$)$_2$], 105.1 (C-1), 81.9 (C-3), 81.8 (C-2), 79.0 (C-4), 75.4 (C-5), 71.6, 71.5, 70.8 (3×*O*-*C*H$_2$), 70.0 (C-6), 32.1, 31.8, 31.7 [*O*-CH$_2$*C*H$_2$CH$_2$CH$_3$ and 2×*O*-CH$_2$*C*H$_2$(CH$_2$)$_5$CH$_3$], 30.4, 29.8, 29.7, 29.6, 29.5, 29.3, 29.2, 29.0 [2×*O*-(CH$_2$)$_2$(*C*H$_2$)$_3$(CH$_2$)$_2$CH$_3$], 26.8 [C(*C*H$_3$)$_2$], 26.3 [C(*C*H$_3$)$_2$], 26.2, 26.1 [2×*O*-(CH$_2$)$_5$*C*H$_2$CH$_2$CH$_3$], 22.7, 22.6 [2×*O*-(CH$_2$)$_6$*C*H$_2$CH$_3$], 19.4 [*O*-(CH$_2$)$_2$*C*H$_2$CH$_3$], 14.1(2), and 13.9 [(CH$_2$)$_3$*C*H$_3$ and 2×*O*-(CH$_2$)$_7$*C*H$_3$]. These spectral data, in corroboration with its DEPT-135, 2D COSY, 2D HSQC, and 2D HMBC (Figure S13) experiments, assigned the structure as 3-*O*-butyl-1,2-*O*-isopropylidene-5,6-di-*O*-octyl-α-D-*gluco*-1,4-furanose (**7**).

2.4. Antimicrobial activities

2.4.1. Antibacterial activities and PASS results

In the present study, test compounds (**4**–**7**) that were 3-*O*-butylglucofuranose derivatives were chosen and their *in vitro* antibacterial properties were checked against six pathogenic bacteria in humans (three Gram-positive bacteria and three Gram-negative, Table 1). The results of the inhibition zone (diameter) of the selected bacteria due to the effect of synthesized chemicals **4**-**7** are presented in Table 1.

Table 1. Inhibition of bacteria by the compounds **4**-**7**.

Drug/ Compd	Diameter of zone of inhibition in mm (75 µg.dw./disc)					
	M. yunnanensis	*M. esteraromaticum*	*S. aureus*	*E. coli*	*S. typhi*	*S. flexneri*
4	NI	NI	NI	NI	NI	NI
5	NI	NI	NI	NI	NI	NI
6	13.5±0.4	15.5±0.7	NI	NI	NI	NI
7	NI	NI	NI	NI	NI	NI
CPC	NI	*24.0±0.1	NI	NI	*27.0±0.1	NI
CFC	*31.0±0.14	NI	*26.0±0.1	*34.0±0.14	NI	*25.0±0.1

NI = inhibition; dw = dry weight; CPC = Chloramphenicol; CFC = Ciprofloxacin; * shows good inhibition; SD = standard deviation indicated by ± sign (*n* = 3)

It is easily understood that these protected glucofuranose compounds are inactive against bacterial pathogens compared to standard antibiotics. To verify this fact, a minimal inhibitory concentration (MIC) of **4**-**7** was determined and showed MIC values >400 µg/mL for most of the compounds. 6-*O*-Hexanoate **6** showed MIC 50 µg/mL and 25 µg/mL against *M. yunnanensis* and *M. esteraromaticum,* respectively. Thus, the *in vitro* results are in agreement with the MIC values. Also, this lower antibacterial potential of **4**-**7** is in agreement with the PASS (Prediction of Activity Spectra for Substances) analyzed results, as shown in Table 2. Table 2 shows that 3-*O*-butylglucofuranose derivatives **4**-**7** have 0.32<Pa<0.50 in antibacterial and 0.55<Pa<0.66 in antifungal. According to these findings, synthetic chemicals are more effective against phytopathogenic fungi than they are against bacterial pathogens. Additionally, according to the PASS study, the chemicals found in the current investigation have a variety of potential biological actions.

Table 2. Predicted biological activity of 3-*O*-butylglucofuranose-derived **4**-**7** using PASS online software.

Drug/ Compd	Biological Activity							
	Antibacterial		Antifungal		Anti-cancer		Antiviral (Herpes)	
	Pa	Pi	Pa	Pi	Pa	Pi	Pa	Pi
4	0.424	0.025	0.615	0.017	0.263	0.076	0.468	0.014
5	0.495	0.017	0.619	0.017	0.305	0.056	0.433	0.023
6	0.471	0.019	0.667	0.012	0.288	0.063	0.439	0.021
7	0.327	0.050	0.646	0.014	-	-	0.397	0.037
CPC	0.507	0.016	0.460	0.038	-	-	-	-
CFC	0.588	0.009	-	-	-	-	-	-
FCZ	-	-	0.776	0.008	0.646	0.014	-	-
VCZ	-	-	0.722	0.009	-	-	-	-

Pa = probability 'to be active'; Pi = probability 'to be inactive'; CPC = chloramphenicol; CFC = ciprofloxacin; FCZ = fluconazole; VCZ = voriconazole; for antiviral prediction herpes virus was used.

2.4.2. Antifungal activities

The percentages of mycelial growth that were inhibited as a result of the 3-*O*-butylglucofuranose derivatives against two pathogenic fungi, viz., *Aspergillus flavus* ATCC 16875 and *Aspergillus niger* ATCC 16404, are displayed in Table 3.

It is evident from Table 3 that both **6** (6-*O*-hexanoate) and **7** (5,6-di-octyl ether) are active against *A. niger* (Figure 2) but inactive against *A. flavus*. Overall, butylglucofuranoses are found to be more active against fungal organisms than bacteria. Additionally, the literature [39, 40] reported on sugar esters' higher antifungal potential. However, the precise process for such an observation is not known. Due to its direct connection to membrane permeability, a material's hydrophobicity is a crucial factor when determining its bioactivity, such as toxicity or changes to the integrity of membranes [41]. With the addition of acyl or alkyl moieties, the lipophilicity of butylglucofuranose molecules increased steadily from **4** to **7**. More non-bonding interactions between these hydrophobic substances and CYP51A1 (an enzyme found in the fungal cell wall) are produced. Thus, ergosterol, a crucial component of the fungal cell wall, cannot be biosynthesized by the enzyme [42]. As a result, microbiological organisms suffer damage to their cytoplasmic membranes, which reduces their membrane permeability and ultimately results in the death of the

organism.

Table 3. Inhibition of bacteria by the compounds **4**-**7**.

Drug/ Compd	Percentage of zone of inhibition	
	A. flavus	***A. niger***
4	NI	NI
5	*67.0±1.00	38.7±1.53
6	NI	51.3±0.58
7	NI	46.7±1.53
FCZ	*84.6±0.64	NI
VCZ	NI	*88.2±0.92

NI = indicates no inhibition; FCZ = fluconazole; VCZ = voriconazole; dw = dry weight; * = good inhibition, SD = standard deviation indicated by ± sign (*n* = 3).

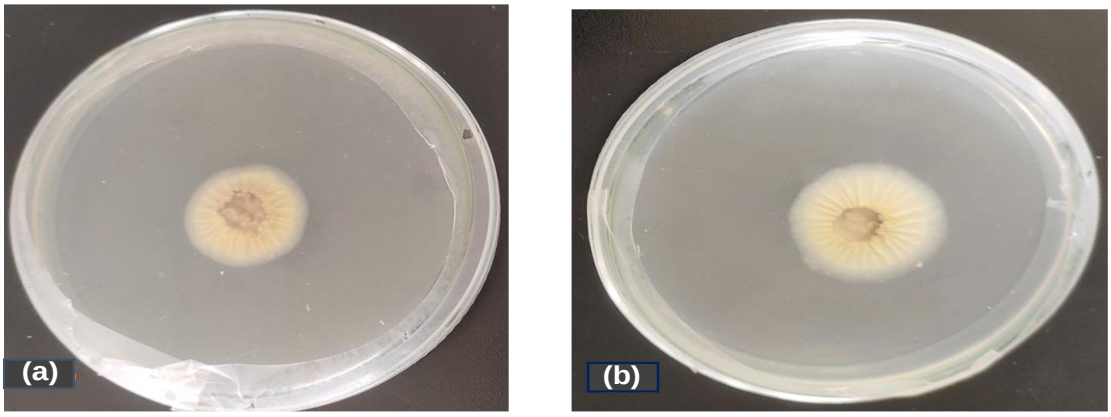


Fig. 2. Zone of inhibition (%) against *A. niger* ATCC 16404 for compound (*a*) **6** and (*b*) **7**.

3. Material and Methods

3.1. Materials and Instrumentation

Except as otherwise noted, all reagents were purchased from Aldrich and used exactly as received. Solvents were filtered or used straight from the store using traditional methods. Thin-layer chromatography (TLC) was performed on Kieselgel GF_{254} plates, and the plates were heated at 150–200 °C while being misted with 1% methanolic sulphuric acid until color appeared. In a Buchi rotary evaporator (R-100, Flawil, Switzerland), evaporations were conducted under reduced pressure at temperatures below 40 °C. Column chromatography (CC) was done using silica gel G_{60}. The solvent solutions for the TLC and CC were made up of varying ratios of *n*-hexane-ethyl acetate. Spectral analyses were conducted with an FT-IR spectrophotometer (Shimadzu, IR Prestige-21, Kyoto, Japan), followed by ^{1}H (400 MHz) and ^{13}C (100 MHz) NMR spectra (Bruker DPX-400 spectrometer, Billerica, USA).

3.2. Synthesis

1,2:5,6-di-*O*-isopropylidene-α-D-*gluco*-1,4-furanose (3)

The title compound **3** was prepared from D-glucose and dry acetone in 46% yield using literature procedures [37].

3-*O*-Butyl-1,2:5,6-di-*O*-isopropylidene-α-D-*gluco*-1,4-furanose (4)

Sodium hydride (0.553 g, 23.043 mmol) was washed with dry hexane (15 mL). Diacetone D-glucose **3** (1.5 g, 5.763 mmol) with a free OH group was dissolved in dry THF (5 mL) and was added dropwise to the reaction mixture containing NaH in THF (20 mL) at 0 °C. After stirring the reaction mixture for 30 min., butyl bromide (0.948 g, 6.919 mmol) in dry THF (1 mL) was slowly added, after which the addition of tetrabutylammonium iodide (TBAI, 0.25 g, 0.677 mmol). The reaction mixture was added to ice water after 4 hours of stirring at room temperature. Then the THF layer was evaporated on rotavapour. The residual aqueous layer was extracted with ether (5×3 mL), the ethereal layer was washed with brine, dried (Na_2SO_4), and concentrated to afford a thick liquid, which on purification by column chromatography (*n*-hexane/ethyl acetate = 9/1) gave the pure butylated product **4** (1.677 g, 92%) as clear syrup. R_f = 0.52 (*n*-hexane/EA = 7/1). FT-IR (neat) υ_{max}: 3019, 2945, 2915 (C-H), 1216 [$C(CH_3)_2$], 1082 cm^{-1} (furanose ring). ^{1}H NMR (400 MHz, $CDCl_3$): δ_H 5.89 (d, *J* = 3.6 Hz, 1H, H-1), 4.54 (d, *J* = 3.6 Hz, 1H, H-2), 4.32 (dd, *J* = 13.4 and 6.0 Hz, 1H, H-5), 4.15 (dd, *J* = 7.2 and 2.8 Hz, 1H, H-4), 4.09 (dd, *J* = 8.8 and 2.4 Hz, H-6a), 4.00 (dd, *J* = 8.8 and 6.0 Hz, H-6b), 3.87 (d, *J* = 3.2 Hz, 1H, H-3), 3.60-3.65 [m, 1H, *O*-$CH_AH_B(CH_2)_2CH_3$], 3.51-3.56 [m, 1H, *O*-$CH_AH_B(CH_2)_2CH_3$], 1.55-1.60 [m, 2H, $CH_2CH_2CH_2CH_3$], 1.53 [s, 3H, $C(CH_3)_2$], 1.44 [s, 3H, $C(CH_3)_2$], 1.38-1.42 [m, 2H, $(CH_2)_2CH_2CH_3$], 1.37 [s, 3H, $C(CH_3)_2$], 1.34 [s, 3H, $C(CH_3)_2$], 0.94 [t, 3H, *J* = 7.6 Hz, $(CH_2)_3CH_3$]. ^{13}C NMR (100 MHz, $CDCl_3$): δ_C 111.7 [$C(CH_3)_2$], 108.9 [$C(CH_3)_2$], 105.3 (C-1), 82.6 (C-2), 82.1 (C-3), 81.2 (C-4), 72.6 (C-5), 70.4 (*O*-CH_2), 67.2 (C-6), 31.8 (*O*-$CH_2CH_2CH_2CH_3$), 26.9 [$C(CH_3)_2$], 26.8 [$C(CH_3)_2$], 26.3 [$C(CH_3)_2$], 25.4 [$C(CH_3)_2$], 19.2 [*O*-$(CH_2)_2CH_2CH_3$], 13.8 [*O*-$(CH_2)_3CH_3$].

3-*O*-Butyl-1,2-*O*-isopropylidene-α-D-*gluco*-1,4-furanose (5)

3-*O*-Butyldiacetone-D-glucofuranose **4** (1.5 g, 4.741 mmol) was dissolved in 15 mL of methanol, 7.5 mL of water, and 0.8 mL of 15% H_2SO_4 at room temperature. After 5 h of continuous stirring at this temperature, saturated potassium carbonate (K_2CO_3) solution was added to bring the reaction mixture's pH

value down to 7–8. TLC of this reaction indicated the formation of a slower-moving single component. Then the solvent (methanol-water) was evaporated in a vacuum, and the residue was extracted with ethyl acetate (EA, 6×3 mL). The organic layer (EA) was dried over Na_2SO_4 and evaporated under reduced pressure to give a thick liquid, which was purified by silica gel column chromatography with *n*-hexane/ethyl acetate (1/1) and afforded pure 5,6-diol **5** as a thick liquid (1.245 g, 95%). R_f = 0.44 (*n*-hexane/EA = 1/4). FT-IR (neat) υ_{max}: 3230-3580 (br, OH), 2957, 2921, 2872 (C-H), 1376 [$C(CH_3)_2$], 1082 cm^{-1} (furanose ring). ^1H NMR (400 MHz, $CDCl_3$): δ_H 5.95 (1H, d, *J* = 3.6 Hz, H-1), 4.59 (d, *J* = 3.6 Hz, 1H, H-2), 4.15 (dd, *J* = 7.2 and 3.6 Hz, 1H, H-4), 4.00-4.08 (m, 1H, H-5), 4.00 (d, *J* = 3.2 Hz, 1H, H-3), 3.85 (dd, *J* = 11.6 and 3.4 Hz, H-6a), 3.75 (dd, *J* = 11.6 and 5.6 Hz, H-6b), 3.66-3.72 [m, 1H, O-$CH_AH_B(CH_2)_2CH_3$], 3.48-3.55 [m, 1H, *O*-$CH_AH_B(CH_2)_2CH_3$], 1.85-1.94 (br s, 2H, 2×O*H*), 1.55-1.63 (m, 2H, $CH_2CH_2CH_2CH_3$), 1.52 [s, 3H, $C(CH_3)_2$], 1.36-1.43 [m, 2H, $(CH_2)_2CH_2CH_3$], 1.35 [s, 3H, $C(CH_3)_2$], 0.95 [t, 3H, *J* = 7.6 Hz, $(CH_2)_3CH_3$]. ^{13}C NMR (100 MHz, $CDCl_3$): δ_C 111.7 [$C(CH_3)_2$], 105.3 (C-1), 83.3 (C-3), 82.0 (C-2), 79.8 (C-4), 70.2 (*O*-CH_2), 69.9 (C-5), 64.5 (C-6), 31.8 (*O*-$CH_2CH_2CH_2CH_3$), 26.8 [$C(CH_3)_2$], 26.2 [$C(CH_3)_2$], 19.3 [*O*-$(CH_2)_2CH_2CH_3$], 13.8 [*O*-$(CH_2)_3CH_3$].

3-*O*-Butyl-6-*O*-hexanoyl-1,2-*O*-isopropylidene-α-D-*gluco*-1,4-furanose (6)

5,6-Diol **5** (0.1 g, 0.362 mmol) was dissolved in anhydrous pyridine (1 mL) with stirring and cooled in an ice bath (0 °C). Unimolar hexanoyl chloride ($C_5H_{11}COCl$) (0.063 g, 0.468 mmol) was added to the reaction mixture at this temperature. The reaction mixture was stirred at this temperature (0 °C) for half an hour and then at room temperature for 10 h. The progress of the reaction was monitored by TLC, which indicated the full conversion of the starting material into a faster-moving single product (R_f = 0.56). A few ice cubes were placed in the flask before the product was extracted using 3 mL of dichloromethane (DCM). Brine, saturated aqueous sodium hydrogen carbonate ($NaHCO_3$) solution, and diluted hydrochloric acid (5% each) were used to wash the mixed DCM layer in turn. After drying over MgSO4, the organic layer was filtered, and the filtrate was then concentrated under lower pressure to yield a syrup. The syrup mass was run through a silica gel column and eluted with n-hexane/ethyl acetate (8/1), producing the 6-*O*-hexanoate **6** (0.117 g, 86%) as a semi-solid, which resisted crystallization. R_f = 0.56 (*n*-hexane/EA = 3/1). FT-IR (neat) υ_{max}: 3120-3550 (br, OH), 2957, 2921, 2853 (C-H), 1710 (CO), 1376 [$C(CH_3)_2$], 1069 cm^{-1} (furanose ring). ^1H NMR (400 MHz, $CDCl_3$): δ_H 5.95 (1H, d, *J* = 4.0 Hz, H-1), 4.58 (d, *J* = 3.6 Hz, 1H, H-2), 4.39 (dd, *J* = 14.0 and 5.6 Hz, H-6a), 4.17-4.22 (m, 2H, H-5 and H-6b), 4.14 (dd, *J* = 6.8 and 3.6 Hz, 1H, H-4), 4.00 (d, *J* = 3.2 Hz, 1H, H-3), 3.67-3.71 [m, 1H, *O*-$CH_AH_B(CH_2)_2CH_3$], 3.52-3.56 [m, 1H, *O*-$CH_AH_B(CH_2)_2CH_3$], 2.34-2.39 [m, 2H, $CH_3(CH_2)_3CH_2CO$], 1.55-1.70 [m, 4H, $CH_2CH_2CH_2CH_3$ and $CH_3(CH_2)_2CH_2CH_2CO$], 1.51 [s, 3H, $C(CH_3)_2$], 1.32-1.43 [m, 9H, *O*-$(CH_2)_2CH_2CH_3$, $C(CH_3)_2$ and $CH_3(CH_2)_2(CH_2)_2CO$, 0.88-0.96 [m, 6H, $(CH_2)_3CH_3$ and $CH_3C_4H_8CO$]. ^{13}C NMR (100 MHz, $CDCl_3$): δ_C 174.2 ($C_5H_{11}CO$), 111.8 [$C(CH_3)_2$], 105.1 (C-1), 83.0 (C-3), 82.0 (C-2), 79.2 (C-4), 70.3 (*O*-CH_2), 68.3 (C-5), 66.4 (C-6), 34.2 [$CH_3(CH_2)_3CH_2CO$], 31.8 (*O*-$CH_2CH_2CH_2CH_3$), 31.3 [$CH_3(CH_2)_2CH_2CH_2CO$], 26.8 [$C(CH_3)_2$], 26.3 [$C(CH_3)_2$], 24.6, 22.3 [$CH_3(CH_2)_2(CH_2)_2CO$], 19.3 [*O*-$(CH_2)_2CH_2CH_3$], 13.9, 13.8 [*O*-$(CH_2)_3CH_3$ and $CH_3C_4H_8CO$].

3-*O*-Butyl-1,2-*O*-isopropylidene-5,6-di-*O*-octyl-α-D-*gluco*-1,4-furanose (7)

At first, sodium hydride (0.069 g, 2.875 mmol) was washed with dry hexane (5 mL) and kept in a round bottom flask. 5,6-Diol **5** (0.1 g, 0.362 mmol) with two free OH groups was dissolved in dry THF (5 mL) and was slowly added at ice-cooled temperature to the reaction mixture. After stirring the reaction mixture for 30 min, octyl bromide (0.174 g, 0.901 mmol) in dry THF (2 mL) was added slowly, followed by the addition of catalytic tetrabutylammonium iodide (TBAI, 0.25 g, 0.677 mmol). After 4 hours of stirring at room temperature, the reaction mixture was put into 1 mL of ice water, followed by evaporation of the THF layer on a rotavapour. The residual aqueous layer was extracted with ether (5×3 mL), the ethereal layer was washed with brine, dried (Na_2SO_4), and concentrated to afford a thick liquid that was purified by silica gel column chromatography (*n*-hexane/ethyl acetate = 9/1). This resulted in the pure di-octyl ether product **7** (0.158 g, 87%) as a clear syrup. R_f = 0.56 (*n*-hexane/EA = 5/1). FT-IR (neat) υ_{max}: 2952, 2921, 2853 (C-H), 1253 [$C(CH_3)_2$], and 1082 cm^{-1} (pyranose ring). ^1H NMR (400 MHz, $CDCl_3$): δ_H 5.87 (1H, d, *J* = 3.6 Hz, H-1), 4.53 (d, *J* = 4.0 Hz, 1H, H-2), 4.14 (dd, *J* = 9.6 and 2.8 Hz, 1H, H-4), 3.88 (d, *J* = 2.8 Hz, 1H, H-3), 3.72-3.82 (m, 3H, H-5 and H-6), 3.59-3.64, 3.38-3.54 (2×m, 6H, 3×*O*-CH_2), 1.53-1.60 [m, 6H, *O*-$CH_2CH_2CH_2CH_3$ and 2×*O*-$CH_2CH_2(CH_2)_5CH_3$], 1.48 [s, 3H, $C(CH_3)_2$], 1.26-1.42 [m, 25H, $C(CH_3)_2$, *O*-$(CH_2)_2CH_2CH_3$ and 2×*O*-$(CH_2)_2(CH_2)_5CH_3$], 0.86-0.96 [m, 9H, $(CH_2)_3CH_3$ and 2×*O*-$(CH_2)_7CH_3$]. ^{13}C NMR (100 MHz, $CDCl_3$): δ_C 111.6 [$C(CH_3)_2$], 105.1 (C-1), 81.9 (C-3), 81.8 (C-2), 79.0 (C-4), 75.4 (C-5), 71.6, 71.5, 70.8 (3×*O*-CH_2), 70.0 (C-6), 32.1, 31.8, 31.7 [*O*-$CH_2CH_2CH_2CH_3$ and 2×*O*-$CH_2CH_2(CH_2)_5CH_3$], 30.4, 29.8, 29.7, 29.6, 29.5, 29.3, 29.2, 29.0 [2×*O*-$(CH_2)_2(CH_2)_3(CH_2)_2CH_3$], 26.8 [$C(CH_3)_2$], 26.3 [$C(CH_3)_2$], 26.2, 26.1 [2×*O*-$(CH_2)_5CH_2CH_2CH_3$], 22.7, 22.6 [2×*O*-$(CH_2)_6CH_2CH_3$], 19.4 [*O*-$(CH_2)_2CH_2CH_3$], 14.1(2), 13.9 [$(CH_2)_3CH_3$ and 2×*O*-$(CH_2)_7CH_3$].

3.3. Antimicrobial tests

Test tube cultures of bacteria and fungi that cause disease were obtained from the University of Chittagong in Bangladesh's Department of Biochemistry. Six bacterial and two fungal organisms were used in this study. Gram-positive organisms were *Micrococcus yunnanensis* ATCC 7468, *Microbacterium esteraromaticum* ATCC 8091, and *Staphylococcus aureus* ATCC 25923. Gram-negative organisms were *Escherichia coli* ATCC 25922, *Salmonella typhi* ATCC 14028, and *Shigella flexneri* ATCC 12022. Fungal organisms are *Aspergillus flavus* and *Aspergillus niger*.

(a) Screening of antibacterial efficacy. One of the known *in vitro* antibacterial screening methods, the "disc diffusion" approach, was used for pure compounds 4 through 7 in 2% DMF solutions. It was kept up to date to adhere to the standards set out by the Clinical and Laboratory Standards Institute (CLSI) [43]. On Mueller-Hinton (agar and broth) medium, bacteria were grown. At 37 °C for 48 hours, test microorganisms were injected onto agar plates. The synthesized chemical is deposited on the agar surface in the form of filter paper discs (about 6 mm in diameter), at the desired concentration. The experiment substance(s) dissolved into the agar. The diameter of the inhibition zone was then used to measure the inhibition of germination and growing organisms (s). Three suitable controls were used in each experiment (only with DMF). Chloramphenicol and ciprofloxacin (standard antibiotics) are used for comparison and validation.

(b) Evaluation of the minimal inhibitory concentration (MIC). The minimum inhibitory concentration, or MIC, is the lowest amount of an antibacterial agent expressed in mg/L (g/mL),

which, under carefully monitored in vitro circumstances, entirely stops the test strain of an organism from exhibiting any observable growth. The microdilution method [44] is used in this regard. At first, the antibacterial activity of the compounds was determined using sterile 2 mL 96-well plates. 0.5 mL of sterilized Mueller Hinton agar was placed in each of the 12 wells of each row. Sequentially, wells 2–11 received an additional 0.5 mL of a mixture of culture medium and compounds serially diluted to create a concentration sequence from 0.512 mL to 0.008 mL. Well 1 served as growth control, and well 12 as antibiotic control. Chloramphenicol (0.1 µg/mL) and ciprofloxacin (0.1 µg/mL) were used as controls. At 37 °C, the deep wells were cultured for 24 hours. After seeing the turbidity that resulted, the MIC was identified as the point when growth was no longer evident using optical density measurements taken at 600 nm using a Beckman DU-70 UV-VIS Spectrophotometer. For each experiment, at least three repeats were performed. MIC <5 g/mL was used to characterize strong activity. The results of the MIC values are presented in Table 4.

Table 4. MIC of the tested compounds against Gram-positive and Gram-negative pathogens.

Drug/ Compd	Minimum inhibitory concentration (MIC) values in µg/mL					
	M. yunnanensis	*M. esteraromaticum*	*S. aureus*	*E. coli*	*S. typhi*	*S. flexneri*
4	> 400	> 400	>400	> 400	> 400	> 400
5	25	> 400	**50**	> 400	> 400	> 400
6	> 400	> 400	> 400	> 400	> 400	> 400
7	> 400	> 400	> 400	> 400	> 400	> 400
CPC	6.25	-	-	6.25	-	-
CFC	-	6.25	6.25	-	-	6.25

NI = inhibition; dw = dry weight; CPC = Chloramphenicol; CFC = Ciprofloxacin; * shows good inhibition; SD = standard deviation indicated by ± sign (*n* = 3)

(c) Evaluation of antifungal efficacy. Antifungal susceptibility was evaluated using the "Poisoned Food" approach [43,44,45]. In a nutshell, the sabouraud (agar and broth, PDA) medium was used to cultivate the fungi. After 3-5 days of incubation, the fungus' linear mycelial growth was quantified. The following formula is typically used to calculate a fungus species' sensitivity to radial mycelial growth.

$$I = \left\{\frac{(C-T)}{C}\right\} \times 100$$

Where, I = percentage of inhibition, C = diameter of the fungal colony in control (DMF), and T = diameter of the fungal colony in treatment. The common antifungal antibiotics fluconazole and voriconazole were examined in a comparable manner to validate and compare antifungal efficacy.

3.4. PASS analysis

Initially, the purpose of PASS (Prediction of Activity Spectra for Substances) development was to estimate choosing the most promising compounds for biological testing based on the biological activity of chemical compounds submitted to the State Registration System. But at present, it is a very convenient way to find the properties & structure of the compound [47,48]. A software program, Chemdraw, was used to picture and determine the structures & molecular formulas of the target substances **4**-**7**. These were then converted into their respective SMILES (Simplified Molecular Input Line Entry System), and these SD file(s) were separately used to predict biological spectrum using the PASS (prediction of activity spectra for substances) online version. The activity was expressed as Pa (probability to be active) and Pi (probability to be inactive).

4. Conclusions

Initially, 3-*O*-butyl-1,2-*O*-isopropylidene-α-D-glucofuranose (**5**) was prepared from available D-glucose in just three economical steps. Unimolar hexanoylation of 5,6-diol **5** indicated selectivity at the C-6 position with the formation of 6-*O*-hexanoate **6** in 86% yield. The presence of a butyl group at the C-3 position might help with the higher selectivity at the C-6 position. To compare biological tests, octyl ether **7** (from compound **5**) was also prepared via dimolar octylation of compound **5**. The fact that the butylglucofuranose derivatives **4**–**7** displayed superior potentiality for fungal infections than that of bacterial organisms was a significant observation. Thus, our empirical findings concur with the predictions made by PASS studies. The compounds showed better inhibition against *A. niger* than *A. flavus*, and ester **6** had slightly better inhibition than ether **7**. The study may help the establishment of sugar-based biodegradable bioactive agents in the near future.

Supporting Information

Supplementary information is annexed with this article in the journal website.

Acknowledgments

We are thankful to the Research and Publication Cell of the University of Chittagong, Bangladesh for a financial grant to accomplish this work (2023, 136/Ka).

Author Contributions

All authors contributed to the design and study conception. Material synthesis, data collection and analysis were performed by Tasnim Rahman Anisa, Md. Badrul Islam, and Mohammed Mahbubul Matin. Antimicrobial evaluation was performed by Tasnim Rahman Anisa, and Md. Inshaful Islam. The first draft of the manuscript was written by Mohammed Mahbubul Matin and all authors commented on previous versions and approved final version of the manuscript.

References and Notes

[1] Pyo, S. -H.; Chen, J.; Ye, R.; Douglas G. Hayes, D. G. In:

Biobased Surfactants. Hayes, D. G.; Solaiman, D. K. Y., Ashby, R. D., eds. Illinois: AOCS Press, 2019, chapter 10, pp 325-363. [Crossref]

[2] Tracy, P.; Dasgupta, D.; More, S. *Ind. Crops and Products* **2023** *193*, 116170. [Crossref]

[3] Dhavale, D. D.; Matin, M. M. *Tetrahedron* **2004**, *60*, 4275. [Crossref]

[4] Szuts, A.; Szabó-Révész, P. *Int. J. Pharm*. **2012**, *433*, 1. [Crossref]

[5] Kroumova, A. B. M.; Zaitlin, D.; Wagner, G. J. *Phytochem*. **2016**, *130*, 218. [Crossref]

[6] Ren, K.; Lamsal, B. P. *Food Chem*. **2017**, *214*, 556. [Crossref]

[7] Rodrigues, L. R.; Banat, I. M.; Mei, H. C.; Teixeira, J. A.; Oliveira, R. *J. Appl. Microbiol*. **2006**, *100*, 470. [Crossref]

[8] Aleksic, I.; Petkovic, M.; Jovanovic, M.; Milivojevic, D.; Vasiljevic, B.; Nikodinovic-Runic, J.; et al. *Front. Microbiol*. **2017**, *8*, 2454. [Crossref]

[9] Okabe, S.; Suganuma, M.; Tada, Y.; Ochiai, Y.; Sueoka, E.; Kohya, H.; et al. *Jpn. J. Cancer Res.* **1999**, *90*, 669. [Crossref]

[10] Puterka, G. J.; Farone, W.; Palmer, T.; Barrington, A. *J. Econ. Entomol.* **2003**, *96*, 636. [Crossref]

[11] Adu, S. A.; Twigg, M. S.; Naughton, P. J.; Marchant, R.; Banat, I. M. *Pharmaceutics* **2022**, *14*, 360. [Crossref]

[12] Banat, I. M.; Carboué, Q.; Saucedo-Castañeda, G.; de Jesús Cázares-Marinero, J. *Bioresour. Technol*. **2021**, *320*, 124222. [Crossref]

[13] Lima, T. M. S.; Procópio, L. C.; Brandão, F. D.; Carvalho, A. M. X.; Tótola, M. R.; Borges, A. C. *Biodegradation* **2011**, *22*, 585. [Crossref]

[14] Grüninger, J.; Delavault, A.; Ochsenreither, K. *Process Biochem*. **2019**, *87*, 45. [Crossref]

[15] Voutsas, E.C.; Tsavas, P.; Magoulas, K.; Tassios, D.; Ferrer, M.; Plou, F. J.; Ballesteros, A. *J. Chem. Eng. Data* **2002**, *47*, 1517. [Crossref]

[16] Sanaullah, A. F. M.; Devi, P.; Hossain, T.; Sultan, S. B.; Badhon, M. M. U.; Hossain, M. E.; et al. *Molecules* **2023**, *28*, 986. [Crossref]

[17] Zhang, X.; We, W.; Cao, X.; Feng, F. *J. Sci. Food Agric*. **2015**, *95*, 1631. [Crossref]

[18] Matin, M. M.; Nath, A. R.; Saad, O.; Bhuiyan, M. M. H.; Kadir, F. A.; Abd Hamid, S. B.; Alhadi, A. A.; Ali, M. E.; Yehye, W. A. *Int. J. Mol. Sci*. **2016**, *17*, 1412. [Crossref]

[19] Demchenko, A. V.; Pornsuriyasak, P.; De Meo, C. *J. Chem. Edu*. **2006**, *83*, 782. [Crossref]

[20] Crich, D. *J. Carbohydr. Chem*. **2012**, *21*, 663. [Crossref]

[21] Matin, M. M.; Bhattacharjee, S. C.; Chakraborty, P.; Alam, M. S. *Carbohydr. Res*. **2019**, *485*, 107812. [Crossref]

[22] Marathe, S. J.; Dedhia, N.; Singhal, R. S. *Cur. Opin. Food Sci*. **2022**, 43, 163. [Crossref]

[23] Ibinga, S. K.; Fabre, J-F.; Bikanga, R.; Mouloungui, Z. *Front. Chem*. **2019**, *7*, 587. [Crossref]

[24] Matin, P.; Rahman, M. R.; Huda, D.; Bakri, M. K. B.; Uddin, J.; Yurkin, Y.; et al. *BioResources*, **2022**, *17*, 3025. [Crossref]

[25] Matin, M. M.; Bhuiyan, M. M. H.; Kabir, E.; Sanaullah, A. F. M.; Rahman, M. A.; Hossain, M. E.; Uzzaman, M. *J. Mol. Struct*. **2019**, *1195*, 189. [Crossref]

[26] Ren, B.; Zhang, L.; Zhang, M. *Asian J. Org. Chem*. **2019**, *8*, 1813. [Crossref]

[27] Richel, A.; Laurent, P.; Wathelet, B.; Wathelet, J.P.; Paquot, M. *Comptes Rendus Chimie* **2011**, *14*, 224. [Crossref]

[28] de Sousa, R. R.; da Silva, A. S.; Fernandez-Lafuente, R.; Ferreira-Leitão, V. S. *Catalysts* **2021**, *11*, 1357. [Cossref]

[29] Hollenbach, R.; Delavault, A.; Gebhardt, L.; Soergel, H.; Muhle-Goll, C.; Ochsenreither, K.; Syldatk, C. *ACS Sustainable Chem. Eng*. **2022**, *10*, 10192. [Crossref]

[30] Matin, P.; Hanee, U.; Alam, M. S.; Jeong, J. E.; Matin, M. M.; Rahman, M. R.; Mahmud, S.; Alshahrani, M. M.; Kim, B. *Molecules* **2022**, *27*, 4125. [Crossref]

[31] Rong, Y. W.; Zhang, Q. H.; Wang, W.; Li, B. L. *Bull. Korean Chem. Soc*. **2014**, *35*, 2165. [Crossref]

[32] Luginina, J.; Vasiljevs, D.; Ivanovs, I.; Mishnev, A.; Turks, M. *Monatshefte für Chemie*. **2019**, *150*, 21. [Crossref]

[33] Rahman, M.A.; Chakma, U.; Kumer, A.; Rahman, M.R.; Matin, M.M. *Biointerface Res. Appl. Chem*. **2023**, *13*, 52. [Crossref]

[34] Sanaullah, A. F. M.; Bhuiyan, M. M. H.; Matin, M. M. *Egyptian J. Chem*. **2022**, *65*, 329. [Crossref]

[35] Catelani, G.; Osti, F.; Bianchi, N.; Bergonzi, M. C.; D'Andrea, F.; Gambari, R. *Bioorg. Med. Chem. Lett*. **1999**, *9*, 3153. [Crossref]

[36] de Souza, T. B.; Bretas, A. C. O.; Alves, R. J.; Magalhães, T. F. F.; Stoianoff, M. A. R. *Química Nova*. **2015**, *38*, 1282. [Crossref]

[37] Gramera, R. E.; Park, A.; Whistler, R. L. *J. Org. Chem*. **1963**, *28*, 3230.

[38] Devi, P.; Matin, M. M.; Bhuiyan, M. M. H.; Hossain, M.E. *J. Turk. Chem. Soc. Sect. A: Chem*. **2021**, *8*, 1003. [Crossref]

[39] Verboni, M.; Sisti, M.; Campana, R.; Benedetti, S.; Palma, F.; Potenza, L.; Lucarini, S.; Duranti, A. *Pharmaceuticals* **2023**, *16*, 136. [Crossref]

[40] Jumina, J.; Mutmainah, M.; Purwono, B.; Kurniawan, Y.S.; Syah, Y.M. *Molecules* **2019**, *24*, 3692. [Crossref]

[41] Matin, M. M.; Chakraborty, P.; Alam, M. S.; Islam, M. M.; Hanee, U. *Carbohydr. Res.* **2020**, *496*, 108130. [Crossref]

[42] Lepesheva, G. I.; Waterman, M. R. *Biochim. Biophys. Acta* **2007**, *1770*, 467.

[43] CLSI. *Performance Standards for Antimicrobial Disk Susceptibility Tests, Approved Standard*, 7th ed.; CLSI Document M02-A11; Clinical and Laboratory Standards Institute: Wayne, PA, USA, 2012.

[44] Rotilie, C. A.; Fass, R. J.; Prior, R. B.; Perkins, R. L. *Antimicrob. Agents Chemother*. **1975**, *7*, 311. [Crossref]

[45] Gakuubi, M. M.; Maina, A. W.; Wagacha, J. M. *Int. J. Microbiol.* **2017**, *2017*, 8761610. [Crossref]

[46] Matin, M.M.; Bhuiyan, M.H.; Hossain, M.M.; Roshid, M.H.O. *Orbital: Electron. J. Chem.* **2015**, *7*, 160. [Crossref]

[47] Druzhilovskiy, D. S.; Rudik, A. V.; Filimonov, D. A.; Gloriozova, T. A.; Dmitriev, A. V.; Pogodin, P. V. et al. *Russ. Chem. Bull.* **2017**, *66*, 1832. [Crossref]

[48] Matin, M. M.; Bhuiyan, M. M. H.; Kibria, S. M.; Hasan, M. S. *Pharm. Chem. J.* **2022**, *56*, 627. [Crossref]

How to cite this article

Anisa, T. R.; Islam, B.; Islam, I.; Matin, M. M. *Orbital: Electron. J. Chem.* **2023**, *15*, 86. DOI: **http://dx.doi.org/10.17807/orbital.v15i2.18152**