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Selective Synthesis of Some Carboxylic Acids Esters

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Abstract—The synthesis of esters of some carboxylic acids was carried out by the oxidative esterification method. It was shown that the proposed method allows the selective esterification of carboxylic acids with hindered carboxyl groups. It was found that the synthesized esters have anti-aggregation activity at the level of acetylsalicylic acid.

Keywords: organic acids, alcohols, glycerol formals, solketal, esterification, esters

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Esters are widely used in medicinal chemistry [1–3]. The main method for their preparation is esterification [4–11] in the presence of mineral acids (sulfuric, hydrochloric, boric, orthophosphoric, etc.) at elevated temperatures, which often leads to undesirable side products [12, 13].

Herein, we reported the synthesis of esters by selective esterification of aromatic carboxylic acids with alcohols under mild conditions in the presence of triphenylphosphine, iodine, and imidazole (Scheme 1). Benzoic (**1a**), phenoxyacetic (**1b**), 2,4-dichlorophenoxyacetic (**1c**), and maleopimaric (**1d**) acids were used, as well as primary and secondary alcohols such as butanol **2a**, isobutanol **2b**, *sec*-butyl (**2c**) and tetrahydrofurfuryl (**2d**) alcohols, solketal **2e**, dimerol **2e**, and a mixture of glycerol formals **3a** and **3b**. The reaction was carried out in methylene chloride for 24 h at a molar ratio of reagents acid : alcohol : Ph_3P : I_2 : Im = 1 : 1 : 1.5 : 1.5 : 3.3 at room temperature (Table 1).

As follows from the results obtained (Table 1), under the chosen conditions, the esterification of primary alcohols occurs in 60–92% yields, while 2-butanol reacts much worse (the yield of ester **3c** is 20%, entry 3).

Under the conditions studied, maleopimaric acid **1d**, containing a sterically hindered carboxyl group, effectively reacts with tetrahydrofurfuryl alcohol (Table 1, entry 12). It should be noted that the esterification of terpene acids is an important process for the modification of natural compounds and usually proceeds at high temperatures [14, 15].

This approach turned out to be effective for the synthesis of 2-ethylhexyl esters of phenoxyacetic (**5a**) and 2,4-dichlorophenoxyacetic (**6a**) acids, industrial pesticides and herbicides. The target esters were isolated in 80 and 83% yields, respectively (entries 6, 9).

Competitive esterification of benzoic acid **1a** with 2-butanol **2c** and solketal **2e** showed that at the initial stage (1 h) only (tetrahydrofuran-2-yl)methylbenzoate **4e** accumulated in the reaction mixture (Scheme 2). Similarly, in the case of esterification of phenoxyacetic acid **1b** with a mixture of glycerol formals **3a** and **3b** (1 : 1), after 1 h, only ester **5d** was present in the reaction mixture, which was the product of the reaction between phenoxyacetic acid **1b** and 4-hydroxymethyl-1,3-dioxolane **3a** (Scheme 3).

Scheme 1.

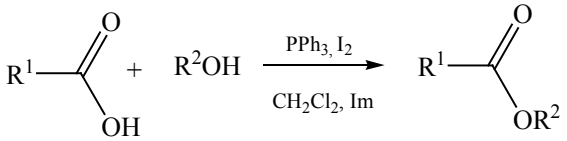
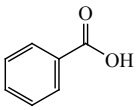
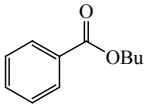
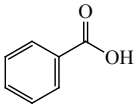
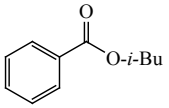
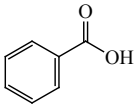
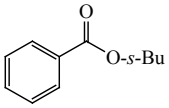
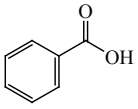
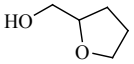
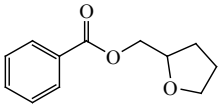
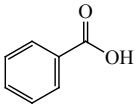
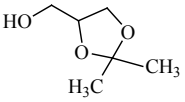
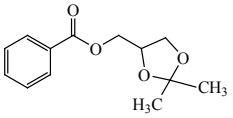
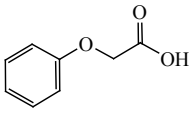
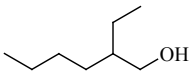
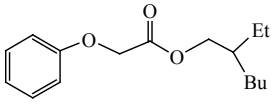
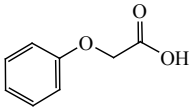
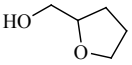
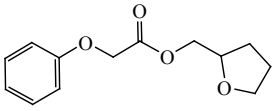
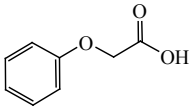
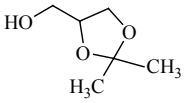
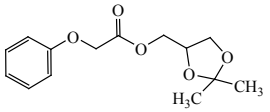
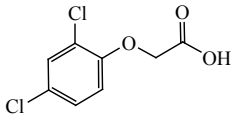
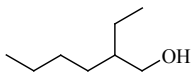
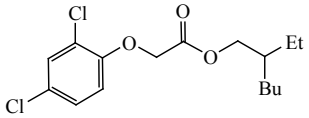
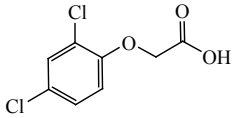
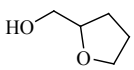
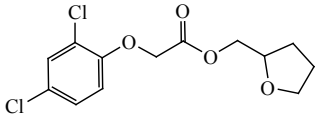
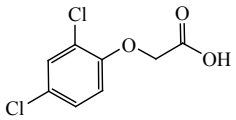
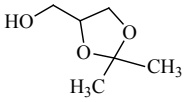
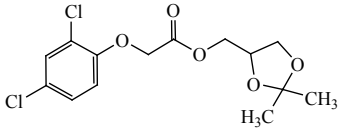
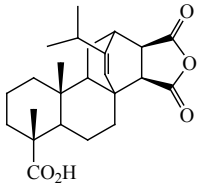
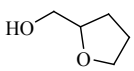
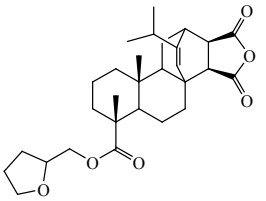
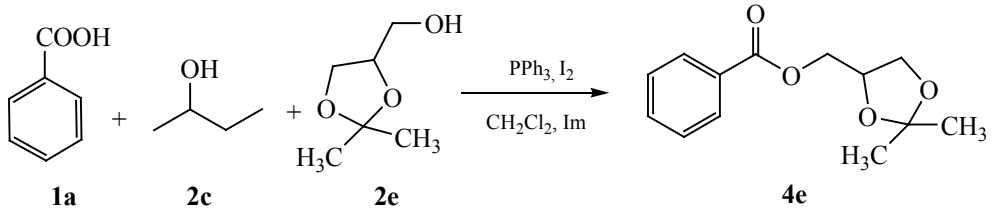


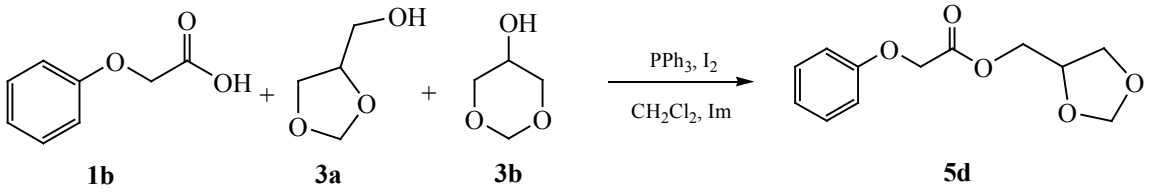
Table 1. Esterification of acids **1a**–**1d** with alcohols **2a**–**2f** (CH_2Cl_2, 24 h, PPh_3, I_2, Im)

Entry	Comp. no	Formula	Comp. no	Formula	Product (yield, %)	Formula
1	**1a**		**2a**	BuOH	**4a** (76%)	22 
2	**1a**		**2b**	*i*-BuOH	**4b** (60%)	
3	**1a**		**2c**	*s*-BuOH	**4c** (20%)	
4	**1a**		**2d**		**4d** (90%)	
5	**1a**		**2e**		**4e** (85%)	
6	**1b**		**2f**		**5a** (80%)	
7	**1b**		**2d**		**5b** (86%)	
8	**1b**		**2e**		**5c** (92%)	
9	**1c**		**2f**		**6a** (83%)	
10	**1c**		**2d**		**6b** (92%)	
11	**1c**		**2e**		**6c** (90%)	
12	**1d**		**2d**		**7** (60%)	

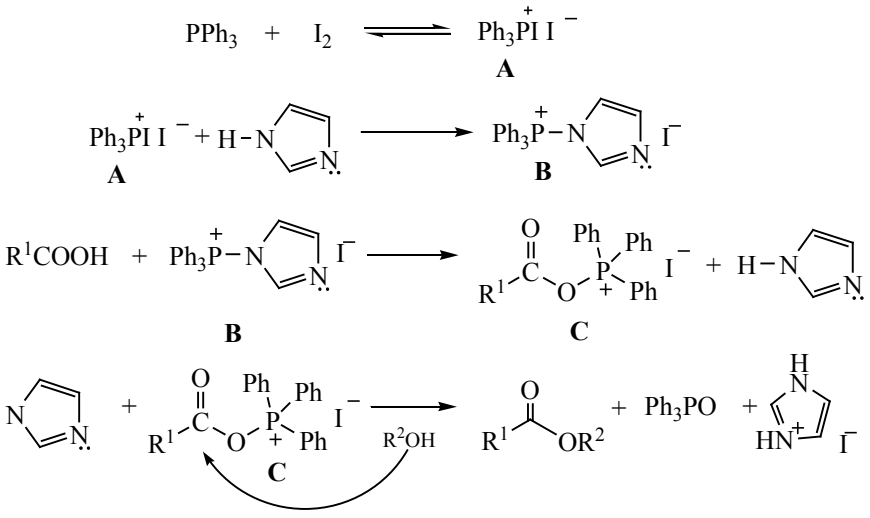
Scheme 2.



Scheme 3.



Scheme 4.



A plausible reaction mechanism includes, at the first stage, the oxidation of triphenylphosphine with the formation of salt **A**, which then reacts with imidazole, leading to complex **B**. Further reaction of salt **B** with carboxylic acid leads to intermediate **C**, which reacts with alcohol to form the target ester (Scheme 4). The sterically hindered intermediate **B** reacts more easily with primary alcohols, and the corresponding esters were obtained in high yields. When more sterically hindered secondary alcohols were used, repulsion occurred between the phosphine ligands and substituents at the OH group, which hinders the nucleophilic addition.

Previously, we have shown that esters of arylacetic acids containing cycloacetal fragments have herbicidal and antibacterial activity [16, 17]. Continuing these studies, we determined the anticoagulation and anti-aggregation activity of the obtained compounds on the model of ADP-induced platelet aggregation in vitro

Table 2. Effect of tested compounds and sodium heparin on parameters of plasma hemostasis *in vitro*, Me (0.25–0.75)[a]

Compound	APTT change, %[b]	APTT change, %[b]	Fibrinogen concentration, %
4e	+9.5 (8.2–11.8)	0.0 (0.0–0.0)	0.0 (0.0–0.0)
4d	+3.1 (2.7–5.2)	0.0 (0.0–0.0)	0.0 (0.0–0.0)
5a	+5.7 (4.5–8.2)	0.0 (0.0–0.0)	0.0 (0.0–0.0)
6a	+8.2 (6.9–10.2)	0.0 (0.0–0.0)	0.0 (0.0–0.0)
Sodium heparin	+20.3 (19.7–21.4)	0.0 (0.0–0.0)	0.0 (0.0–0.0)

[a] The data are significant in comparison with control and heparin at $p < 0.05$, $n = 6$.
[b] APTT—activated partial thromboplastin time, PT—prothrombin time.

according to the method proposed in [18], which is convenient for assessing the inhibition of the platelet aggregation process by various biologically active compounds, including drugs (Tables 2, 3).

The studied compounds demonstrated a different degree of influence on the plasma component of the hemostasis system, manifested by a change in the indicator of the internal pathway of blood coagulation—activated partial thromboplastin time (APTT). These compounds at the specified concentration (5×10^{-4} g/mL) did not affect the fibrinogen concentration and prothrombin time (PT). It was found that the resulting esters exhibit anti-aggregation activity at the level of acetylsalicylic acid, effectively suppressing the reaction of platelet release (lag period in collagen-induced platelet aggregation).

In conclusion, the esterification of carboxylic acids with primary alcohols in the presence of triphenylphosphine, iodine, and imidazole is a fairly simple and effective method for the synthesis of esters. In situ activation of a carboxylic acid makes it possible to exclude the stage of obtaining acid chlorides, and also excludes the use of inorganic acids as catalysts, which makes it possible to involve acid-sensitive heterocyclic alcohols in the reaction. It was found that the synthesized esters have anti-aggregation activity at the level of acetylsalicylic acid and can be considered as promising compounds for studying biological activity.

EXPERIMENTAL

The ^{1}H and ^{13}C NMR spectra were recorded on a Bruker AM-300 spectrometer with an operating frequency of 300.13 and 75.47 MHz, respectively, with Me_4Si as the internal standard. GLC studies were carried out on a Chrom-5 instrument [column length 1.2 m, stationary phase SE-30 silicone (5%) on ChromatonN-AW-DMCS (0.16–0.20 the carrier is helium]. Chromato-mass spectra were recorded on a Kristall-5000 M instrument equipped with a Finnigan DSQII mass spectrometric detector at an ionizing voltage of 70 eV. The temperature of the ion source is 200°C. Separation into components was carried out on a 30 m long ThermoTR-5MS column with an inner diameter of 0.25 mm and a stationary liquid phase thickness of 0.25 μm at a flow rate of 0.7 mL/min. The carrier gas is helium. The electron impact mass spectra were recorded at an energy of ionizing electrons of 70 eV and an ionization chamber temperature of 200°C. TLC was carried out on Sorbfil plates (Russia), eluent is petroleum ether–ethyl acetate. SiO_2 (70–230 mesh, Lancaster, UK) was used for column chromatography.

General procedure for the synthesis of esters. To a solution of 1.5 mmol of iodine in 20 mL of methylene chloride was added 1.5 mmol of triphenylphosphine and 3.3 mmol of imidazole, after which 1 mmol of acid **1a**–**1d** was added. The resulting mixture was stirred at room temperature for 5 min, then 1.5 mmol of alcohol was added. At the end of the reaction (control by TLC, GC/MS), the reaction mixture was washed twice with 2N HCl solution, then with water until neutral and dried with Na_2SO_4. The solvent was removed under reduced pressure. The product was isolated by column chromatography. Data on the yields of esters are given in Table 1. Physico-chemical constants and spectral characteristics of compounds **4a** [20], **4b** [21], **4c** [22], **4d** [23], **4e** [24], and **5d** [16] coincided with those reported earlier.

(2-Ethylhexyl) phenoxyacetate (5a). ^{1}H NMR spectrum ($CDCl_3$), δ, ppm (*J*, Hz): 0.88 t and 0.99 t (2×3H, Et, Bu), 1.19–1.31 m (6H, Bu), 1.55 m (2H, Et), 4.24–4.94 m (2H, $C^1{}'H_2$), 4.99 s (2H, C^2H_2), 6.80–7.40

Table 3. Effect of tested compounds and comparative drugs on platelet aggregation indicators, Me (0.25–0.75)[a]

Compound	Latent period, %[b]	Maximum amplitude, %	Aggregation speed, %	Time to reach MA, %[c]	Disaggregation, %
4d	+13.7 (10.7–17.1)[d,e,f]	–1.8 (0.6–2.5)[e,g]	–13.5 (11.3–17.6)[d,e,f]	+4.7 (2.3–7.5) [e,g]	0.0 (0.0–0.0)
4e	+8.1 (5.4–10.3) [d,e,f]	–16.2 (14.5–20.9)[h,i]	–15.4 (10.1–19.3)[d,e]	+15.3 (10.7–16.1)[d,i]	0.0 (0.0–0.0)
5a	+8.4 (6.9–10.3) [d,e,f]	–8.3 (6.2–10.3)[d,e,f]	–11.8 (10.3–13.5)[d,e]	+9.6 (8.3–12.2)[d,e]	0.0 (0.0–0.0)
6a	+3.2 (2.5–4.7)[e,f]	–13.5 (12.1–15.3)[d,e]	–13.7 (11.4–15.9)[d,e,f]	+10.8 (8.7–11.3)[d,e]	0.0 (0.0–0.0)
Euphylline	+19.8 (16.3–23.1)[d,e,f]	–7.4 (5.6–9.3)[d,e,g]	–21.4 (18.7–23.2)[h,i,g]	+13.4 (11.2–16.7)[d,e]	0.0 (0.0–0.0)
Caffeine sodium benzoate	+23.1 (20.1–25.6)[d,e,g]	–14.7 (10.3–17.9)[d,e]	–30.1 (26.4–34.2)[h,g]	+16.9 (14.3–19.5)[d,e]	0.0 (0.0–0.0)
Acetylsalicylic acid	–2.1 (1.1–2.6)[e]	–13.7 (10.8–16.4)[d,e]	–10.5 (7.6–12.3)[d,e]	+10.5 (8.7–13.4)[d,e]	0.0 (0.0–0.0)
Pentoxifylline	+32.4 (28.7–35.6)[d]	–48.4 (42.7–56.5)[h]	–34.9 (28.7–39.6)[h]	+32.1 (27.6–32.4)[h]	13.6 (11.2–16.8)

[a] Data are presented in comparison with control. [b] Latent period is shown for collagen-induced platelet aggregation, other parameters for ADP-induced platelet aggregation. [c] MA, the maximum amplitude of aggregation. [d] $p \le 0.05$ compared to control. [e] $p \le 0.001$, in comparison with pentoxifylline. [f] $p \le 0.05$, in comparison with acetylsalicylic acid. [g] $p \le 0.001$, in comparison with acetylsalicylic acid. [h] $p \le 0.001$, compared with control. [i] $p \le 0.05$, compared with pentoxifylline.

m (5H, Ph). ^{13}C NMR spectrum ($CDCl_3$), δ_C, ppm: 11.6 ($C^{2'''}$), 14.1 ($C^{6''}$), 23.0 ($C^{5''}$), 23.70 ($C^{1'''}$), 29.30 ($C^{4''}$), 30.80 ($C^{3''}$), 39.60 ($C^{2''}$), 65.30 (C^2), 67.20 ($C^{1''}$), 114.31 (C^2, C^6, Ph), 121.0 (C^4, Ph), 129.72 (C^3, C^5, Ph), (Ph), 158.10 (C^1, Ph), 169.20 (COO).

[(Tetrahydrofuran-2-yl)methyl] phenoxyacetate (5b). ^{1}H NMR spectrum ($CDCl_3$), δ, ppm (*J*, Hz): 1.20 s (3H, C^7H_3), 1.50 s (3H, C^6H_3), 3.70 d. d (1H, C^4H, ^2J 5.6, ^3J 4.2), 4.00 d. d (2H, C^5H_2, ^2J 4.2, ^3J 5.6), 4.23 d. d (2H, C^8H_2, ^2J 6.4, ^3J 5.6), 4.60 d (2H, $C^{12}H_2$, ^2J 17.4), 4.99 s (2H, H^2), 6.80–7.40 m (5H, Ph). ^{13}C NMR spectrum ($CDCl_3$), δ_C, ppm: 25.60 ($C^{4'}$), 27.60 ($C^{3'}$), 65.30 (C^2), 67.60 (C^5), 66.10 ($C^{1''}$), 85.10 ($C^{2'}$), 114.30 (C^2, C^6, Ph), 121.1 (C^4, Ph), 129.70 (C^3, C^5, Ph), 158.10 (Ph), 130.10 (C^1, Ph), 165.90 (COO).

[(2,2-Dimethyl-1,3-dioxolan-4-yl)methyl] phenoxyacetate (5c). ^{1}H NMR spectrum ($CDCl_3$), δ, ppm (*J*, Hz): 1.20 s (3H, C^7H_3), 1.50 s (3H, C^6H_3), 3.70 d. d (1H, C^4H, ^2J 5.6,^3J 4.2), 4.00 d. d (2H, C^5H_2, ^2J 4.2, ^3J 5.6), 4.25 d. d (2H, C^8H_2, ^2J 6.4, ^3J 5.6), 4.60 d (2H, $C^{12}H^2$, ^2J 17.4), 6.80–7.40 m (5H, Ph). ^{13}C NMR spectrum ($CDCl_3$), δ_C, ppm: 25.24 (C^7), 26.61 (C^6), 62.41 (C^8), 63.14 (C^4), 69.77 (C^{12}), 71.12 (C^5), 109.88 (C^2), 114.30 (C^2, C^6, Ph), 121.1 (C^4, Ph), 129.70 (C^3, C^5, Ph), 157.64

(C^1, Ph), 168.79 (COO). Mass spectrum, *m*/*e* (I_{rel}, %): 266 (18) $[M]^+$, 251 (68), 117 (17), 107 (100), 101 (32), 79 (14), 77 (50), 72 (12), 59 (10), 51 (9).

(Tetrahydrofuran-2-yl)methyl (2,4-dichlorophenoxy)acetate (6b). ^{1}H NMR spectrum ($CDCl_3$), δ, ppm (*J*, Hz): 1.20 s (3H, C^7H_3), 1.50 s (3H, C^6H_3), 3.72 d. d (1H, C^4H, ^2J 5.6, ^3J 4.2), 3.99 d. d (2H, C^5H_2, ^2J 4.2, ^3J 5.6), 4.22 d. d (2H, C^8H_2, ^2J 6.4, ^3J 5.6), 4.60 d (2H, $C^{12}H_2$, ^2J 17.4), 4.99 s (2H, H^2), 6.80–7.40 m (3H, Ph). ^{13}C NMR spectrum ($CDCl_3$), δ_C, ppm: 25.60 ($C^{4'}$), 27.60 ($C^{3'}$), 65.30 (C^2), 67.60 (C^5), 66.10 ($C^{1''}$), 85.10 ($C^{2'}$), 114.70 (C^6, Ph), 121.5 (C^2, Ph), 128.0 (C^4, Ph), 129.0 (C^5, Ph), 130.36 (C^3, Ph), 152.70 (C^1, Ph), 169.20 (COO).

(2,2-Dimethyl-1,3-dioxolan-4-yl)methyl (2,4-dichlorophenoxy)acetate (6c). ^{1}H NMR spectrum ($CDCl_3$), δ, ppm (*J*, Hz): 1.34 s (3H, C^7H_3), 1.42 s (3H, C^6H_3), 3.72 d. d (1H, C^4H, ^2J 5.7, ^3J 7.2), 4.19 d. d (2H, C^5H_2, ^2J 7.2, ^3J 5.7), 4.25 d. d (2H, C^8H_2, ^2J 4.9, ^3J 5.7), 4.75 s (2H, $C^{12}H_2$), 6.75–7.45 m (3H, Ph). ^{13}C NMR spectrum ($CDCl_3$), δ_C, ppm: 25.25 (C^7), 26.62 (C^6), 63.13 (C^8), 66.02 (C^5), 66.26 (C^{12}), 73.22 (C^4), 109.99 (C^2), 114.70 (C^6, Ph), 121.5 (C^2, Ph), 128.0 (C^4, Ph), 129.0 (C^5, Ph), 131.4 (C^3, Ph), 152.25 (C^1, Ph), 167.88 (COO), 152.25 (C^{14}), 167.88 (C^{10}). Mass spectrum, *m*/*z* (I_{rel}, %): 334/336/338 (10/6/2), 319/321/323 (100/72/8), 175/177/179 (66/38/8), 145/147/149 (14/9/2), 133/135/137 (8/14/2), 109/111/113 (8/14/2), 101 (44), 73 (28), 57 (14), 43 (96).

(Tetrahydrofuran-2-yl)maleopimaric acid methyl ester (7). ^{1}H NMR spectrum ($CDCl_3$), δ, ppm (*J*, Hz): 0.60 s (3H, $C^{17}H_3$), 1.00 d (3H, $C^{15}H_3$, *J* 6.9), 0.96 d (3H, $C^{16}H_3$, ^3J 6.4), 1.17 s (3H, $C^{18}H_3$), 1.21–1.91 m (16H, C^4H_2, C^5H_2, C^7H_2, C^8H_2, C^9H_2, $C^{10}H_2$, $C^{4'}H_2$, $C^{5'}H_2$), 1.25 t (3H, $C^{23}H_3$, ^3J 7.1), 2.26 quintet (1H, $C^{5a}H$, ^3J 6.7), 2.54 d. t (1H, $C^{9b}H$, ^3J 3.0, ^3J 13.8), 2.73 d (1H, $C^{3a}H$, ^3J 8.7), 3.09 d. d (1H, $C^{11}H$, ^3J 8.6, ^3J 3.0), 3.98 d (1H, $C^{11a}H$, ^3J 9.0), 3.73–3.76 m (2H, $C^{3'}H_2$), 4.30–4.34 m (3H, $C^{1'}H$, OCH_2), 5.54 s (1H, $C^{13}H$). ^{13}C NMR spectrum ($CDCl_3$), δ_C, ppm: 14.28 (C^{17}), 15.56 (C^{18}), 16.71 (C^{24}), 17.02 (C^8), 19.77(C^{23}), 19.96 (C^{16}), 20.57 (C^{15}), 21.56 (C^5), 25.6 ($C^{4'}$), 27.23 (C^{10}), 27.6 ($C^{5'}$), 32.77 (C^{14}), 34.83 (C^4), 35.68 (C^{11}), 36.64 (C^7), 36.94 (C^{9a}), 37.68 (C^{22}), 38.04 (C^9), 40.48 (C^{3b}), 45.67 (C^{11a}), 46.87 (C^6), 49.38 (C^{5a}), 53.07 (C^{3a}), 53.28 (C^{9a}), 65.31 (OCH_2), 66.32 (C^{21}), 85.11 ($C^{1'}$), 124.77 (C^{13}), 148.31 (C^{12}), 171.00 (C^1), 172.78 (C^3), 178.20 (C^{19}).

Antiaggregatory and anticoagulant activity. The experiments were performed in accordance with the requirements of the Rules of Good Laboratory Practice of the Eurasian Economic Union in the field of drug circulation. The assessment of anti-aggregation and anticoagulation activity was carried out in vitro on the blood of 27 healthy male donors aged 18–24 years. The study was approved by the Ethics Committee of the Bashkir State Medical University of the Russian Ministry of Health (No. 1 dated February 20, 2019). Informed consent was obtained from all study participants prior to blood sampling. The effect of the compounds on platelet aggregation was studied according to the method described in [18] on an AT-02 aggregometer (SPF Medtech, Russia). The anti-aggregation activity of the test substances and reference drugs was evaluated at a final concentration of 2×10^{-3} mol/L during incubation for 5 min. Adenosine diphosphate (ADP) at a concentration of 20 μg/mL and collagen at a concentration of 5 mg/mL (TekhnologiyaStandard, Russia) were used as aggregation inducers. The effect of the compounds on the maximum amplitude of aggregation, the rate of aggregation, and the time to reach the maximum amplitude of aggregation during platelet aggregation induced by ADP was studied. In the collagen-induced platelet aggregation test, the latent period of aggregation was evaluated, which corresponds to the reaction of platelet release. Acetylsalicylic acid (powder substance, Shandong Xinghua Pharmaceutical Co. Ltd., China) was used as reference drugs [19]. Determination of anticoagulant activity was carried out by clotting tests [19] using a Solar CGL 2110 natbidimetric hemocoagulometer (SOLAR, Belarus), the final concentration of the test substances and the reference drug was 5×10^{-4} g/mL. The indicators of activated partial thromboplastin time (APTT), prothrombin time (PT) and fibrinogen concentration were studied. Sodium heparin (sodium heparin, 5000 IU/mL solution for injection, 1 mL ampoules, Sintez, Russia) was used as a reference drug. Statistical analysis was performed using the Statistica 10 software package (StatSoftInc, USA). Distribution normality was tested using the Shapiro–Wilk test. To describe the variational series, the median, 25th and 75th percentiles, minimum and maximum values were calculated. One-way analysis of variance was performed (if the dataset obeyed the laws of normal distribution and the variances of all samples were equal; F-test) or the Kruskal–Wallis test (if the dataset did not obey the laws of normal distribution; A-test). The critical significance level p for statistical tests was taken equal to 0.05.

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CONFLICT OF INTEREST

No conflict of interest was declared by the authors.

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