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Synthesis, characterization and antiamoebic activity of 1-(thiazolo[4,5-*b*]quinoxaline-2-yl)-3-phenyl-2-pyrazoline derivatives

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Abstract—A new series of 1-*N*-thiocarboxamide-3-phenyl-2-pyrazolines **1**–**6** was synthesized by cyclization of different Mannich bases with unsubstituted thiosemicarbazide. The reaction of cyclized pyrazoline derivatives **1**–**6** with 2,3-dichloroquinoxaline afforded the title compounds **7**–**12**. The structures of the new compounds were confirmed by elemental analyses as well as ^{1}H, ^{13}C NMR, IR and electronic spectral data. The *HM1:IMSS* strain of *Entamoeba histolytica* parasite was cultured in vitro and the sensitivity of the parasite to the synthesized compounds was evaluated using the microdilution method. Among all the pyrazoline derivatives **1**–**6**, none was found to be a better inhibitor as compared to the reference drug, metronidazole. The quinoxaline derivatives, **9**, **11** and **12** were found to be potent inhibitors of *E. histolytica*.

Amoebiasis is a protozoan infection caused by *Entamoeba histolytica*. The prevalence of amoebic colitis, brain and liver abscess is greater in developing world.[1,2] Amoebiasis is the second leading cause of death worldwide.[3] More than 50 million people are infected and up to 110,000 of these die per annum.[4] There are numerous antiamoebic compounds used in medical practice such as nitroimidazoles. Metronidazole [1(β-hydroxyethyl)-2-methyl-5-nitroimidazole] is the drug of choice for the treatment of anaerobic protozoan and bacterial infections.[5–10] Treatment failures among patients with amoebiasis often raise the possibility of drug resistance.[11] Therefore, it is desirable to search for new leads as amoebicidal.

Pyrazolines, bicyclic pyrazolines and quinoxalines are nitrogen-containing heterocyclic compounds, well known for their pronounced anti-inflammatory activity.[12–20] These compounds have been developed as non-steroidal anti-inflammatory drugs. They block the formation of prostaglandins and have analgesic, antipyretic and anti-inflammatory activity.[21] The quinoxaline antibiotics are a family of drugs that include the naturally occurring triostin A and the synthetic derivative [*N*-MeCys3,*N*-MeCys7]TANDEM.[22] In view of the above considerations and as part of our continuous efforts towards the identification of more potent amoebicidal,[23–25] we report herein the synthesis of new 1-*N*-thiocarboxamide-3-phenyl-2-pyrazolines **1**–**6**, their quinoxaline derivatives, 1-(thiazolo[4,5-*b*]quinoxaline-2-yl)-3-phenyl-2-pyrazolines **7**–**12** and in vitro screening of these compounds against *HM1:IMSS* strain of *E. histolytica*. To the best of our knowledge, this is the first report of cyclized pyrazoline derivative having a quinoxaline moiety and showing very encouraging results as regards in vitro activity against *E. histolytica*.

Mannich bases of different ketones (0.2 mol) were prepared by the reaction with paraformaldehyde (0.26 mol) and dimethylamine hydrochloride (0.26 mol) under reflux in a mixture of 35 mL of ethanol and 0.5 mL of concd HCl. The reaction works best when a minimum amount of ethanol and 2 mL of acid/mol ketone are added. After cooling, 200 mL of acetone was added. The crystals formed were collected, washed with acetone and dried in vacuo. The methyl phenyl ketone and ethyl phenyl ketone gave high yields of about 60–90%, while the yields for 3-bromo and 3-chloro acetophenone and propeophenone in the Mannich reaction were lower in the range of 30–60%. The Mannich bases have been reported earlier.[23,26] Mannich reaction product (0.5 mmol) was cyclized with unsubstituted thiosemicarbazide (0.5 mol) under basic conditions in methanol (5 mL) to give 1-*N*-thiocarboxamide-3-phenyl-2-pyrazo-

Keywords: Mannich bases; Thiocarboxamide; Pyrazolines; Quinoxaline; Antiamoebic activity.
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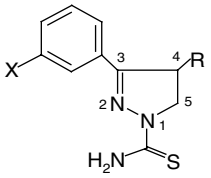
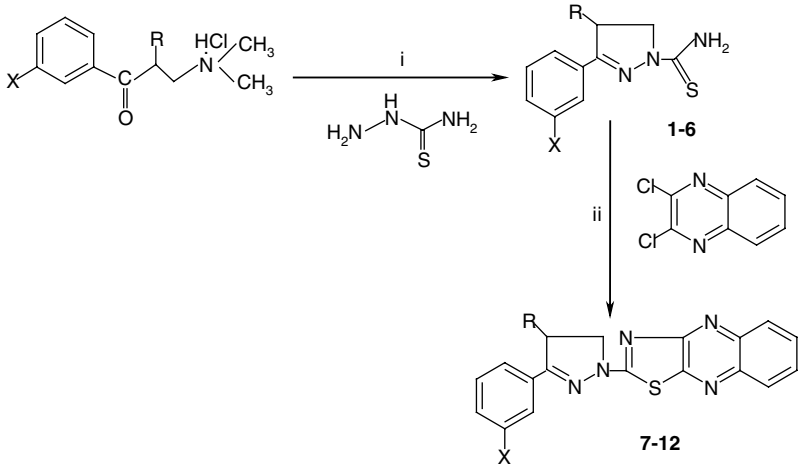
lines **1**–**6**. According to the currently accepted mechanism[27,28] the formation of the cyclized pyrazoline derivatives is favoured via thiosemicarbazone formation, which undergo cyclization under basic conditions to form the desired pyrazoline ring in all the compounds. The product mixture contained only unreacted starting material and the cyclization product, which was purified by column chromatography using silica gel 60 F_{254}, eluted with dichloromethane–methanol (98:2) to give crystalline solid compounds but in low yield. All the cyclized pyrazoline compounds were obtained in moderate yields (30–50%). The refluxing of 1-*N*-thiocarboxamide-3-phenyl-2-pyrazolines **1**–**6** (0.01 mol) with 2,3-dichloroquinoxaline (0.01 mol) in absolute ethanol (15 mL) gave the corresponding fused 1-(thiazolo-[4,5-*b*]quinoxaline-2-yl)-3-phenyl-2-pyrazolines **7**–**12** (Scheme 1). All the compounds were obtained in good yields and are stable in the solid as well as in the solution state. Analytical and spectral data (IR, electronic, and ^{1}H and ^{13}C NMR) are in good agreement with the composition of the compounds.[29] Other analytical and physicochemical data of the compounds are presented in Table 1. The purity of the compounds was established by thin-layer chromatography (TLC) and elemental analyses. Selected diagnostic bands of the IR spectra of 1-*N*-thiocarboxamide-3-phenyl-2-pyrazolines **1**–**6** and 1-(thiazolo[4,5-*b*]quinoxaline-2-yl)-3-phenyl-2-pyrazolines **7**–**12** were very informative and provided evidence for the formation of the expected structures. The compounds **1**–**6** showed intense bands at 1021–1098 cm^{-1} due to the *v*(C=S) stretch of the thiocarboxamide group, while the compounds **7**–**12** showed this band at 991–1094 cm^{-1}. A strong band due to *v*(C=N) stretch was observed in the compounds **1**–**6** at 1530–1598 cm^{-1} because of the ring closure. The compounds **7**–**12** showed two strong bands at 1558–1663 and 1507–1593 cm^{-1} due to *v*(C=N) stretch of azomethine nitrogen of pyrazoline ring and quinoxaline ring, respectively. In addition, the absorption band at 1125–1187 cm^{-1} in compounds **1**–**6** and at 1140–1213 cm^{-1} in compounds **7**–**12** was attributed to the *v*(C–N) stretch vibrations, which also confirm the formation of desired pyrazoline ring in all the compounds. The compounds **1**–**6** showed additional sharp bands in the region 3197–3334 cm^{-1} due to the *v*(NH)stretch. The electronic spectra of the compounds **1**–**6** studied in the UV region exhibited three absorption bands at 369.2–293.3, 274.6–237 and 228–204 nm assignable to n $\rightarrow\pi^*$, $\pi \rightarrow \pi^*$ and n $\rightarrow \sigma^*$ transitions, respectively. The UV spectral data of quinoxaline derivatives **7**–**12** were also studied which showed the same type of transitions as observed in compounds **1**–**6**. They showed three spectral bands at 388.3–299, 287–248 and 239–204 nm assigned to n $\rightarrow \pi^*$, $\pi \rightarrow \pi^*$ and n $\rightarrow \sigma^*$ transitions of thiocarboxamide group (C=S), aromatic ring and azomethine nitrogen, respectively. In the ^{1}H NMR spectra, the pyrazoline protons at C_4 and C_5 carbons in compounds **1**–**3** and **7**–**9** appeared as broad triplets at 3.2–3.36 (J = 7.14–9.0 Hz) and 4.18–4.42 (J = 6.87–9.0 Hz) ppm, respectively. The CH_2 protons of the pyrazoline ring in compounds **4**–**6** and **10**–**12** were resonated as a pair of doublets of doublets at 4.43–4.99 (J = 4.5–6.82, 8.5–11.93 Hz) and 4.06–4.64 (J = 4.5–6.82, 8.5–11.9 Hz). The CH proton of the pyrazoline ring in the same compounds was observed as a multiplet at 3.5–3.87 ppm. The strong deshielding of the C_5 protons compared with that of the C_4 protons of the pyrazoline ring can be assumed due to its conformation A.[30]



A

The CH_3 protons at C_4 carbon of the pyrazoline ring in compounds **4**–**6** and **10**–**12** showed a doublet at 1.12–1.36 (J = 5.01–6.66 Hz). The NH proton of thiocarboxamide group of the compounds (**1**–**6**) showed a singlet at 7.88–8.90 ppm. The protons belonging to the aromatic ring and quinoxaline ring showed multiplet at 6.09–8.23 ppm. In the ^{13}C NMR spectra, the C_4 and C_5 carbons of the pyrazoline ring in compounds **1**–**12** resonate at 42.8–53.2 and 71.2–77.4 ppm, respectively. The com-



Scheme 1. Reagents and conditions: (i) methanol, NaOH, reflux; (ii) absolute ethanol, reflux.

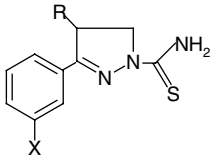
Table 1. Analytical and physicochemical data of pyrazoline and quinoxaline derivatives (**1**–**12**)

S. No.	Compound/stoichiometry	Colour	Yield (%)	Mp (°C)	Found (Calcd)		
					C	H	N
1	3-Ph-2-Pz-1-TC/$C_{10}H_{11}N_3S$	Dark yellow	45	118	58.49 (58.53)	5.37 (5.36)	20.62 (20.48)
2	3-3-BrPh-2-Pz-1-TC/$C_{10}H_{10}N_3SBr$	Light yellow	38	141	42.29 (42.25)	3.47 (3.52)	14.62 (14.78)
3	3-3-ClPh-2-Pz-1-TC/$C_{10}H_{10}N_3SCl$	Cream	35	175	50.15 (50.10)	4.15 (4.17)	17.48 (17.54)
4	3-Ph-4-Me-2-Pz-1-TC/$C_{11}H_{13}N_3S$	Pale yellow	51	97	60.54 (60.27)	5.88 (5.93)	18.97 (19.20)
5	3-3-BrPh-4-Me-2-Pz-1-TC/$C_{11}H_{12}N_3SBr$	Brownish yellow	44	92	44.35 (44.29)	4.11 (4.02)	14.01 (14.09)
6	3-3-ClPh-4-Me-2-Pz-1-TC/$C_{11}H_{12}N_3SCl$	Light yellow	38	107	52.19 (52.07)	4.47 (4.73)	16.62 (16.57)
7	3-Ph-2-Pz-1-Tz-Qz/$C_{18}H_{13}N_5S$	Pale yellow	70	117	65.29 (65.26)	3.81 (3.93)	21.08 (21.15)
8	3-3-BrPh-2-Pz-1-Tz-Qz/$C_{18}H_{12}N_5SBr$	Creamish yellow	55	174	52.55 (52.68)	2.91 (2.93)	17.02 (17.10)
9	3-3-ClPh-2-Pz-1-Tz-Qz/$C_{18}H_{12}N_5SCl$	Dark brown	49	167	59.06 (59.10)	3.22 (3.28)	19.14 (19.15)
10	3-Ph-4-Me-2-Pz-1-Tz-Qz/$C_{19}H_{15}N_5S$	Yellow solid	63	147	66.11 (66.10)	4.29 (4.35)	20.22 (20.30)
11	3-3-BrPh-4-Me-2-Pz-1-Tz-Qz/$C_{19}H_{14}N_5SBr$	Light yellow	46	179	53.69 (53.78)	3.32 (3.30)	16.45 (16.51)
12	3-3-ClPh-4-Me-2-Pz-1-Tz-Qz/$C_{19}H_{14}N_5SCl$	Yellow	41	184	60.05 (60.10)	3.61 (3.70)	18.39 (18.45)

pounds **1**–**6** showed a signal at 153.1–156.4 ppm was assigned due to the azomethine carbon of pyrazoline ring. The compounds **7**–**12** showed two signals at 151.1–155.9 and 141.7–146.2 ppm due to azomethine carbon of the pyrazoline ring and quinoxaline ring, respectively. Thiocarboxamide carbon (C═S) displayed a signal at 169.2–181.2 ppm in all the compounds. The signals from 120.2–140.4 ppm were assumed due to the aromatic carbons in compounds **1**–**12**.

All the compounds (**1**–**12**) were screened in vitro for antiamoebic activity against *HM1:IMSS* strain of *E. histolytica* by the microdilution method.[31] *E. histolytica* trophozoites were cultured in TYIS-33 growth medium as described previously in wells of 96-well microtitre plate.[32] All the compounds were dissolved in DMSO (40 μL) at which level no inhibition of amoeba occurs[33,34] and the stock solutions of the compounds were prepared freshly before use at a concentration of 1 mg/mL. The IC_{50} values in μM are given in Tables 2 and 3. Metronidazole was used as the reference drug and had a 50% inhibitory concentration (IC_{50} 1.69–1.82 μM) in our experiments. The results were estimated as the percentage of growth inhibition compared with the untreated controls and plotted as probit values as a function of the drug concentration. The IC_{50} and 95% confidence limits were interpolated in the corresponding dose–response curve. The pyrazoline derivatives **1**–**6** showed an IC_{50} value in the range 17.2–4.4 μM. Out of six compounds in pyrazoline series, compound **5** with 3-bromo and 4-methyl substitution and compound **6** with 3-chloro and 4-methyl substitution showed the low IC_{50} values (**5**, IC_{50} = 5.9 μM; **6**, IC_{50} = 4.4 μM). The conversion of compounds **1**–**6** into quinoxaline derivatives **7**–**12** results in the increase of their antiamoebic activity. They showed IC_{50} in the range 6.76–0.72 μM. Among all the quinoxaline derivatives, the compounds having 3-chloro (**9**, IC_{50} = 1.09 μM), 3-bromo-4-methyl (**11**, IC_{50} = 1.45 μM) and 3-chloro-4-methyl (**12**, IC_{50} = 0.72 μM) substitutions on the pyrazoline ring were distinctly more potent. The compound **12** is the most active among the series. The results were statistically evaluated by analysis of variance. The null hypothesis was tested using *t* test. The significance of the difference between the IC_{50} values of metronidazole and the compounds **9**, **11** and **12** was evaluated by *t* test. The values of the calculated *t* were found to be higher than the table value of *t* at 5% level, thus concluding that the character under study is said to be significantly influenced by the treatment. All the 3-bromo and 3-chloro substituted cyclised pyrazoline derivatives were found to be more active than their respective unsubstituted analogues. It was concluded that the presence of 3-bromo or 3-chloro substituents

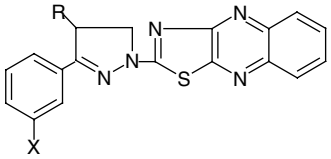
Table 2. In vitro antiamoebic activity of thiocarboxamide-3-phenyl-2-pyrazolines derivatives against *HM1:IMSS* strain of *Entamoeba histolytica*



Compound	X	R	IC_{50} (μM)	SD[a]
1	H	H	17.2	0.15
2	Br	H	13.2	0.18
3	Cl	H	8.7	0.04
4	H	CH_3	10.2	0.11
5	Br	CH_3	5.9	0.65
6	Cl	CH_3	4.4	0.37
Metronidazole			1.82	0.31

[a] Standard deviation.

Table 3. In vitro antiamoebic activity of 1-(thiazolo[4,5-*b*]quinoxaline-2-yl)-3-phenyl-2-pyrazolines derivatives against *HM1:IMSS* strain of *Entamoeba histolytica*



Compound	X	R	IC_{50} (μM)	SD[a]
7	H	H	6.76	0.20
8	Br	H	4.98	0.11
9	Cl	H	1.09	0.08
10	H	CH_3	2.34	0.23
11	Br	CH_3	1.45	0.14
12	Cl	CH_3	0.72	0.10
Metronidazole			1.69	0.24

[a] Standard deviation.

on the phenyl ring and 4-methyl group on the pyrazoline ring greatly effects antiamoebic activity. Detailed studies of the toxicity, in vivo and mechanism of action of these compounds are in progress.

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29. All the new compounds (**1**–**12**) gave satisfactory spectral data consistent with their proposed structures. Selected spectral data for compounds **1**–**12**. Compound **1** λ_{max} (nm): 367.7, 231.6, 319.8, 227, 220.2, 208; ν_{max} (cm^{-1}): 3273 (NH), 1588 (C=N), 1161 (C–N), 1096 (C=S); ^1H NMR ($CDCl_3$): (δ, ppm) 7.74 (s, 2H, NH_2), 7.21–7.67 (m, 5H, Ar-*H*), 4.41 (t, 2H, CH_2, J = 7.89 Hz), 3.35 (t, 2H, CH_2, J = 7.89 Hz); ^{13}C NMR ($CDCl_3$): (δ, ppm) 181.2 (C=S), 155.5 (C=N), 135.4, 130.1, 127.7, 125.4, 123.8, 121.2 (Ar-C), 75.3 (CH_2), 53.2 (CH_2).
Compound **2** λ_{max} (nm): 324.7, 304, 237, 210.1; ν_{max} (cm^{-1}): 3334 (NH), 1570 (C=N), 1166 (C–N), 1071 (C=S); ^1H NMR ($CDCl_3$): (δ, ppm) 7.88 (s, 2H, NH_2), 7.26–7.75 (m, 4H, Ar-*H*), 4.42 (t, 2H, CH_2, J = 8.45 Hz), 3.36 (t, 2H, CH_2, J = 8.45 Hz); ^{13}C NMR ($CDCl_3$): (δ, ppm) 176.3 (C=S), 156.1 (C=N), 133.6, 130.3, 125.3–122.9 (Ar-C), 77.4 (CH_2), 48.4 (CH_2).
Compound **3** λ_{max} (cm^{-1}): 369.2, 328, 239, 227, 204; ν_{max} (cm^{-1}): 3267 (NH), 1565 (C=N), 1187 (C–N), 1098 (C=S); ^1H NMR ($CDCl_3$): (δ, ppm) 7.96 (s, 2H, NH_2), 7.21–7.77 (m, 4H, Ar-*H*), 4.31 (t, 2H, CH_2, J = 6.87 Hz), 3.31 (t, 2H, CH_2, J = 7.14 Hz); ^{13}C NMR ($CDCl_3$): (δ, ppm) 177.2 (C=S), 153.1 (C=N), 132.8, 131.3, 124.3–120.2 (Ar-C), 75.3 (CH_2), 49.1 (CH_2).
Compound **4** λ_{max} (cm^{-1}): 358, 326, 249, 228, 204; ν_{max} (cm^{-1}): 3289 (NH), 1530 (C=N), 1125 (C–N), 1021 (C=S); ^1H NMR ($CDCl_3$): (δ, ppm) 8.54 (s, 2H, NH_2), 7.26–7.74 (m, 4H, Ar-*H*), 4.43 (dd, 1H, C*H*, J = 4.5, 11.4 Hz), 4.19 (dd, 1H, C*H*, J = 4.5, 11.4), 3.82 (m, 1H, C*H*), 1.36 (d,3H, CH_3, J = 6.4); ^{13}C NMR ($CDCl_3$): (δ, ppm) 177.8 (C=S), 156.4 (C=N), 133.5, 132.3, 127.5, 124.8–122.9 (Ar-C), 77.4 (CH_2), 51.2 (CH), 11.6 (CH_3).
Compound **5** λ_{max} (cm^{-1}): 341, 322.9, 307.9, 293.1, 274.6, 248.9, 228, 216, 206.1; ν_{max} (cm^{-1}): 3197 (NH), 1597 (C=N), 1139 (C–N), 1094 (C=S); ^1H NMR ($CDCl_3$): (δ, ppm) 8.07 (s, 2H, NH_2), 7.18–7.87 (m, 4H, Ar-*H*), 3.5–3.8 (m, 1H, C*H*), 4.93 (dd, 1H, C*H*, J = 5.07, 10.22 Hz), 4.21 (dd, 1H, C*H*, J = 5.07, 10.22 Hz), 1.12 (d, 3H, CH_3, J = 5.01 Hz); ^{13}C NMR ($CDCl_3$): (δ, ppm) 177.4 (C=S), 155.2 (C=N), 131.8, 129.4, 127.8–122.2 (Ar-C), 71.2 (CH_2), 42.8 (CH), 12.6 (CH_3).
Compound **6** λ_{max} (cm^{-1}): 349.8, 321, 308, 293.3, 274.6, 249, 228, 216, 206; ν_{max} (cm^{-1}): 3205 (NH), 1598 (C=N), 1138 (C–N), 1078 (C=S); ^1H NMR ($CDCl_3$): (δ, ppm) 8.90 (s, 2H, NH_2), 7.20–7.84 (m, 4H, Ar-*H*), 3.5–3.78 (m, 1H, C*H*), 4.68 (dd, 1H, C*H*, J = 6.82, 11.93 Hz), 4.06 (dd, 1H, C*H*, J = 6.82, 11.93 Hz), 1.16 (d, 3H, CH_3, J = 6.66 Hz); ^{13}C NMR ($CDCl_3$): (δ, ppm) 176.9 (C=S), 156.1 (C=N), 132.6, 129.7, 125.4–121.2 (Ar-C), 74.1 (CH_2), 44.7 (CH), 11.4 (CH_3).
Compound **7** λ_{max} (nm): 388.3, 352.9, 309.8, 237.1, 213.8; ν_{max}/cm^{-1}: 3052 (arom. C–H), 1602 (C=N), 1534 (C=N), 1138 (C–N), 1077 (C=S); ^1H NMR ($CDCl_3$): δ 7.04–8.06 (9H, m, aryl-*H*), 4.18 (2H, t, CH_2, J = 7.5 Hz), 3.23 (2H, t, CH_2, J = 7.5 Hz); ^{13}C NMR ($CDCl_3$): (δ, ppm) 176.3 (C=S), 155.9 (C=N), 144.2 (C=N), 136.2–122.8 (Ar-C), 77.4 (CH_2), 48.4 (CH_2).

Compound **8** λ_{max} (nm): 338, 321, 244, 239, 225, 218, 204;ν_{max}/cm^{-1}: 3118 (arom. C–H), 1663 (C═N), 1593 (C═N), 1140 (C–N), 1078 (C═S); ^{1}H NMR ($CDCl_3$): δ 6.09–7.90 (8H, m, aryl-*H*), 4.3 (2H, t, CH_2, J = 9.0 Hz), 3.2 (2H, t, CH_2, J = 9.0 Hz); ^{13}C NMR ($CDCl_3$): (δ, ppm) 175.2 (C═S), 154.2 (C═N), 145.2 (C═N), 140.2-121.5 (Ar-C), 75.1 (CH_2), 48.6 (CH_2).

Compound **9** λ_{max} (nm): 357.1, 343, 299, 239.5, 209.5, 206.8; ν_{max}/cm^{-1}: 3118 (arom. C–H), 1620 (C═N), 1563 (C═N), 1140 (C–N), 1080 (C═S); ^{1}H NMR (DMSO-d_6): (δ, ppm) 7.11–8.23 (8H, m, aryl-*H*), 4.3 (2H, t, CH_2, J = 8.17 Hz), 3.3 (2H, t, CH_2, J = 8.17 Hz); ^{13}C NMR (DMSO-d_6): (δ, ppm) 170.4 (C═S), 152.2 (C═N), 141.7 (C═N), 138.2–120.8 (Ar-C), 72.3 (CH_2), 46.5 (CH_2).

Compound **10**. λ_{max} (nm): 336.2, 322.2, 211, 209; ν_{max}/cm^{-1}: 3042 (arom. C–H), 1622 (C═N), 1544 (C═N), 1147 (C–N), 1081 (C═S); ^{1}H NMR ($CDCl_3$): δ 7.19–8.01 (8H, m, aryl-*H*), 3.54–3.67 (m, 1H, C*H*), 4.92 (1H, dd, C*H*, J = 5.5, 10.7 Hz), 4.64 (1H, dd, C*H*, J = 5.8, 10.7 Hz), 1.19 (3H, d, CH_3, J = 6.6 Hz); ^{13}C NMR ($CDCl_3$): (δ, ppm) 169.2 (C═S), 155.2 (C═N), 142.7 (C═N), 138.2–124.9 (Ar-C), 76.5 (CH_2), 47.2 (CH), 12.3 (CH_3).

Compound **11** λ_{max} (nm): 335, 321, 309, 287, 248, 226, 216, 207; ν_{max}/cm^{-1}: 2973 (arom. C–H), 1558 (C═N), 1507 (C═N), 1213 (C–N), 1094 (C═S); ^{1}H NMR ($CDCl_3$): δ 7.16–7.93 (8H, m, aryl-*H*), 3.5–3.7 (m, 1H, C*H*), 4.95 (1H,dd, C*H*, J = 5.66, 8.5 Hz), 4.64 (1H, dd, C*H*, J = 5.66, 8.5 Hz), 1.12 (3H, d, CH_3, J = 5.8 Hz); ^{13}C NMR($CDCl_3$): (δ, ppm) 179.5 (C═S), 151.1 (C═N), 145.2 (C═N), 139.7–122.1 (Ar-C), 72.2 (CH_2), 48.3 (CH), 11.2 (CH_3).

Compound **12** λ_{max} (nm): 341, 308, 283, 258, 237, 214, 206; ν_{max}/cm^{-1}: 3042 (arom. C–H), 1630 (C═N), 1584 (C═N), 1179 (C–N), 991 (C═S); ^{1}H NMR ($CDCl_3$): δ 7.27–8.05 (8H, m, aryl-*H*), 3.63-3.87 (m, 1H, C*H*), 4.99 (1H, dd, C*H*, J = 5.6, 8.5 Hz), 4.21 (1H, dd, C*H*, J = 5.6, 8.5 Hz), 1.24 (3H, d, CH_3, J = 4.8 Hz); ^{13}C NMR ($CDCl_3$): (δ, ppm) 179.5 (C═S), 152.1 (C═N), 146.2 (C═N), 140.4–122.8 (Ar-C), 74.1 (CH_2), 49.2 (CH), 10.5 (CH_3).

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