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One-Pot Formation of 1,3,4-Oxadiazol-2(3*H*)-ones and Dibenzo[*c*,*e*]azepines by Concomitant Cathodic Reduction of Diazonium Salts and Phenanthrenequinones

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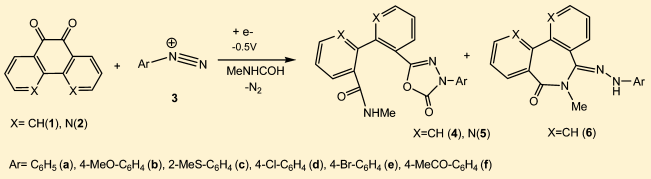
Supporting Information

ABSTRACT: The one-pot concomitant electrochemical reduction of phenanthrenequinones (**1**, **2**) and arenediazonium salts (**3a**−**f**) led to the formation of 1,3,4-oxadiazol-2(3*H*)-ones (**4a**−**f**, **5a**) and dibenzo[*c*,*e*]azepines (**6a**−**f**) when *N*-methylformamide was used as the solvent. A new pathway, different from those previously described with other aprotic solvents, is proposed. The experimental data support a radical mechanism for the electrochemical process followed by an internal rearrangement to give the products.



In a previous work,[1] we found that the concomitant electrochemical reduction of benzenediazonium tetrafluoroborate and 1,2-quinones in DMF solvent afforded the corresponding introduction of an *N*,*N*-dimethylaminocarbonyl group in the quinone (Scheme 1). This radical pathway was supported by the isolation of *N*,*N*-dimethylbenzamide when the reaction was performed in the absence of quinone.

The simultaneous reduction of 1,2-quinones and diazonium salts has been carried out with different solvents such as acetonitrile, 1,2-dichloroethane, dichloromethane, chloroform, chloroacetonitrile, and ethyl acetate with positive results.[1,2] Here we report a similar reaction using *N*-methylformamide as the solvent instead of DMF. The obtained results are significantly different: although the initial process is again a radical reaction, the subsequent radical coupling is followed by an unexpected cleavage and rearrangement that evolves to 1,3,4-oxadiazol-2(3*H*)-ones and/or 1*H*-azepines, depending on the nature of the electrolyte used.

Surprisingly, the literature does not describe one-pot procedures for obtaining substituted 1,3,4-oxadiazol-2(3*H*)-ones. Although a series of them were prepared from aldehydes, ketones, phenylacetic acids, and 1,2- or 1,3-diketones, the conditions for the formation of these oxadiazolones from the *N*-carbamoyl chloride precursors depend on the structure and differ from spontaneous ring closure to those requiring bases.[3] Also, a novel process for the preparation of 3,5-disubstituted 3*H*-1,3,4-oxadiazol-2-ones from the reaction of *N*-*tert*-butyldiacylhydrazines with potassium *tert*-butoxide followed by treatment with phosgene has been reported.[4]

A more recent synthesis of 3-methyl-5-aryl-1,3,4-oxadiazolones has been performed starting from 1-R-3-aryl-5-methyl-6-oxoverdazyl radicals.[5] Substituted 1,3,4-oxadiazol-2-ones are potent hormone-sensitive lipase (HSL) inhibitors. HSL plays an important role in the mobilization of free fatty acids (FFA) from adipocytes. In this sense, the inhibition of HSL may offer a pharmacological approach to reduce FFA levels in plasma and diminish peripheral insulin resistance in type-2 diabetes.[6] 1,3,4-Oxadiazol-2-ones have also been used in therapy as modulators of peroxisome proliferator-activated receptor delta[7] and are useful for treating conditions modulated by a peroxisome proliferator-activated receptor such as diabetes mellitus.[8]

On the other hand, 1*H*-azepines result from spontaneous valence-bond isomerization of azanorcaradienes, which are themselves made by the reaction of arenes with nitrenes.[9] A 1*H*-azepine ring has been described to show interesting pharmacological properties as a potent and highly selective inhibitor of human neuronal nitric oxide synthase.[10]

As indicated above, the concomitant electrochemical reduction of 1,2-dicarbonyl compounds (**1**, **2**) and arenediazonium salts (**3**) in *N*-methylformamide as the solvent gives 1,3,4-oxadiazol-2(3*H*)-ones (**4a**−**f**, **5a**) and dibenzo[*c*,*e*]-azepines (**6a**−**f**) following a reaction pathway completely different from those previously described in another aprotic solvents. The transformation of **1** and **3a** to **4a** and **6a** is indicated in Scheme 2.

The first electrochemical step provides, once again, the radical anion of **1** and the aryl radical of **3**, which abstracts a hydrogen atom from the carbonyl position in the *N*-methylformamide. A further radical coupling produces the alkoxide anion, as indicated in Scheme 3. On the other hand,

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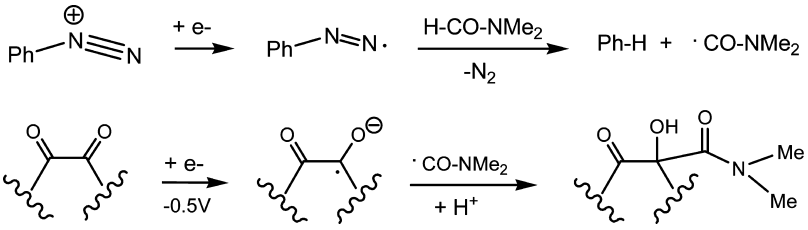
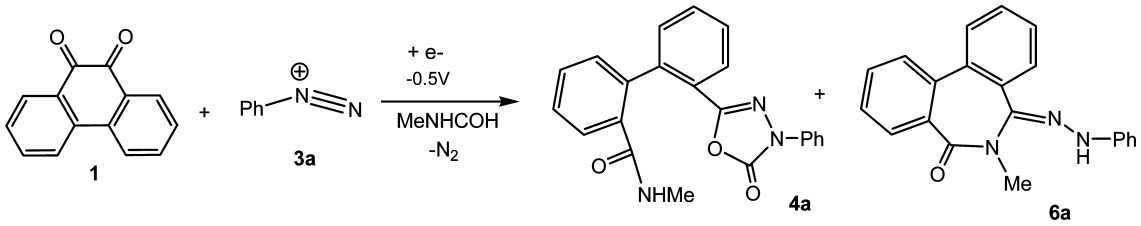
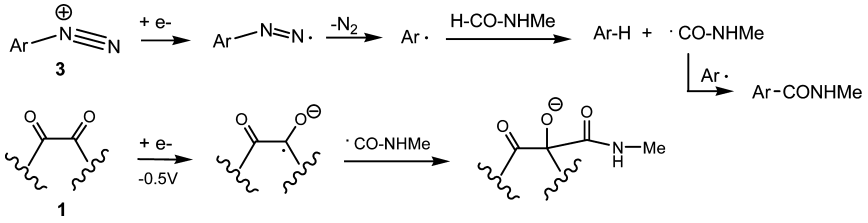
Scheme 1



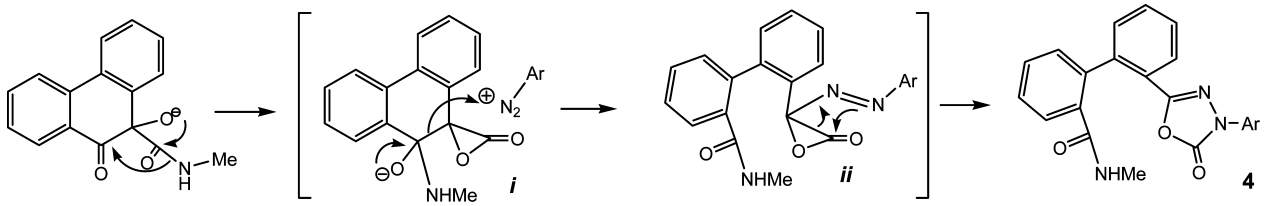
Scheme 2



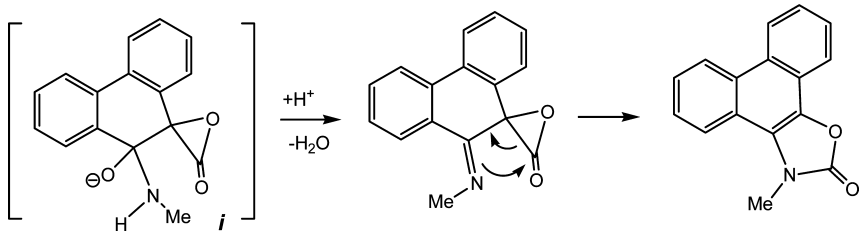
Scheme 3



Scheme 4



Scheme 5



we have observed that in the absence of a dicarbonyl compound, the electrochemical reduction of the arenediazonium salt in *N*-methylformamide produces the corresponding *N*-methylbenzamide. This fact supports the radical nature of this pathway.

Once the radical coupling reaction takes place, as a result of the absence of steric hindrance (present when DMF was used as the solvent), the remaining alkoxide anion is added to the amide carbonyl group. A similar intramolecular nucleophilic attack by the alkoxide was previously observed when the reaction was carried out in 1,2-dichloroethane as the solvent. In that case, a 14% yield of spiroepoxide was obtained as a side product together with the expected 1,2-dichloroethylated derivative.[1]

Next, after the attack by the alkoxide, the following processes should take place simultaneously: (1) migration of the leaving group to the carbonyl group of the ketone, (2) carbon−carbon bond cleavage (assisted by the diazonium salt), and (3) ring formation to afford 3,5-diaryl-1,3,4-oxadiazol-2(3*H*)-one (**4**). This plausible reaction pathway is summarized in Scheme 4. It is important to highlight that, although several steps take place in the electrochemical cell, the products are obtained in a one-pot process. This pathway proposal is supported by the isolation of 3-methylphenanthro[9,10-*d*]oxazol-2(3*H*)-one

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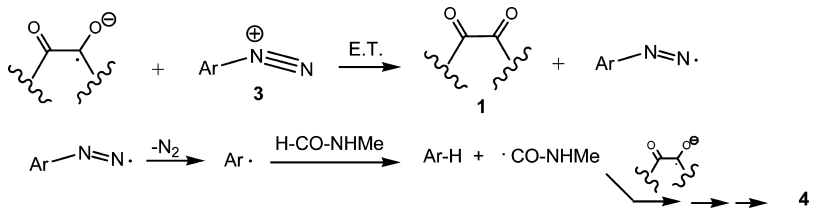
Scheme 6



(<4% yield), which was formed by the reaction of ***i*** with a small amount of water present in the *N*-methylformamide solvent (Scheme 5).

It should be noticed that in the *N*-methylformamide molecule, two possibilities for radical hydrogen abstraction can be considered. The hydrogen bonded to the carbonyl group is abstracted faster than that bonded to the nitrogen atom, as had been already suggested by Cadman et al.[11]

It is well-known that in absence of a diazonium salt, the radical anion of **1** evolves into the starting substrate by electron transfer to oxygen in the air. However, once the radical anion was formed and the potentiostat was switched off, if the diazonium salt was immediately added, some amount of **4** was formed, probably through an electron-transfer process between the radical anion of **1** and the diazonium salt (Scheme 6).

Moreover, when the lithium salt was substituted by a tetrabutylammonium salt as the electrolyte, 1*H*-azepine **6** was also obtained together with **4** or **5**. The results are indicated in Table 1. The formation of **6** can be rationalized through the same intermediate ***ii*** postulated in the formation of **4**. However, **6** is formed after the loss of a carbon dioxide molecule from this intermediate (Scheme 7). The possible provenance of **6** by thermal decomposition of **4** was ruled out when a reflux of **4** in DMF for 24 h did not afford any amount of **6**.

Table 1. Obtained Yields (%) of 4(5) and 6 in the Concomitant Reduction of 1,2-Dicarbonyl 1(2) and ArN_2^+ 3

		with $LiClO_4$[a]		with Bu_4NClO_4[b]	
1,2-dicarbonyl	3: Ar	4(5)	6	4(5)	6
1	**3a**: C_6H_5	91	3	78	13
	3b: 4-$MeOC_6H_4$	60	–	8	62
	3c: 2-$MeSC_6H_4$	92	traces	83	traces
	3d: 4-ClC_6H_4	88	–	–	71
	3e: 4-BrC_6H_4	82	–	1	88
	3f: 4-$MeCOC_6H_4$	83	5	2	84
2	**3a**: C_6H_5	(64)	–	(60)	–

[a]SSE: *N*-methylformamide/$LiClO_4$. [b]SSE: *N*-methylformamide/Bu_4NClO_4.

Finally, the hydrolysis of compound **6** was carried out, providing the corresponding *N*-methyldibenzo[*c*,*e*]azepine-5,7-dione, supporting the proposed structure for **6**. Nevertheless the structures of **4** and **6** were unequivocally determined by the corresponding single-crystal X-ray diffraction studies on **4a**, **5a**, and **6b** (see the Supporting Information).

EXPERIMENTAL SECTION

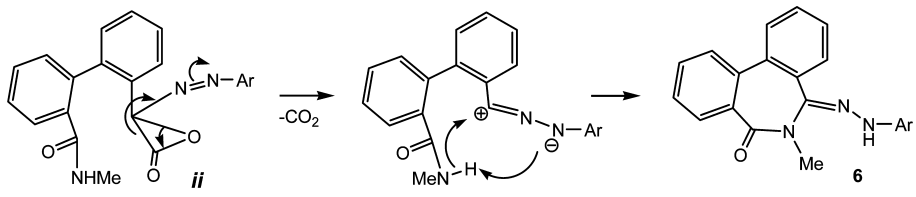
The peak potentials are given in volts versus Ag/Ag^+(3 M). Mass spectra were determined using EI at an ionizing voltage 70 eV. IR spectra of the compounds were recorded as dispersions in KBr or NaCl films. ^1H and ^{13}C NMR spectra were recorded in $CDCl_3$ on a 300 MHz spectrometer with tetramethylsilane (TMS) as the internal standard. The chemical shifts are given in parts per million. All melting points are uncorrected.

General Electrochemical Procedure. The electroactive diazonium tetrafluoroborates were prepared according to conventional methods.[12] The electrolyses were carried out under an argon atmosphere and potentiostatic conditions. Cyclic voltammetry of **1** under aprotic conditions[1] shows two reversible reduction peaks at E_{pc1}= −0.5 V and E_{pc2}= −1.0 V. A concentric cell with two compartments separated by a porous (D4) glass frit diaphragm and equipped with a magnetic stirrer was used. The temperature was maintained constant at 5 °C with a cryostat. A mercury pool was used as the cathode (12 cm^2) and a platinum plate as the anode (2 cm × 2 cm × 0.1 cm). A Ag/Ag^+(3 M) reference electrode was used. The solvent-supporting electrolyte system (SSE) was anhydrous *N*-methylformamide containing 0.1 M lithium perchlorate or tetrabutylammonium perchlorate.

A solution of 9,10-phenanthrenequinone (**1**) or 1,10-phenanthroline-5,6-dione (**2**) (1.0 mmol in 60 mL of SSE) was electrolyzed at a constant potential of −0.5 V vs Ag/Ag^+(3 M). Diazonium salt **3** (2 mmol) was added to the cathode compartment in solid portions during the electrolysis (5 h). The initial current of 100 mA was decreased during the reduction (except when a new diazonium salt portion was added). The complete disappearance of the radical anion red coloration in the cathode solution (once the diazonium salt was completely added) indicated the end of the electrolysis. When the final current was close to 3 mA, a charge consumption of 3 F/mol was achieved.

Once the reduction was finished, the solvent in the cathode solution was removed under reduced pressure. The residue was extracted with ether/water, and the organic phase was dried over Na_2SO_4 and concentrated by evaporation. The resulting products were purified by silica gel 60 (35−70 mesh) in a 30 cm × 3 cm column using CH_2Cl_2/EtOH mixtures as eluents. Spectroscopic description of the obtained compounds is given below. Yields of isolated products in the *N*-methylformamide/$LiClO_4$ SSE (*) or the *N*-methylformamide/Bu_4NClO_4 SSE (**) are given.

Scheme 7



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3-Methylphenanthro[9,10-d]oxazol-2(3H)-one. Mp: 208−210 °C [lit.[13] 210−211 °C]. MS *m*/*z* (relative intensity) EI: 250 (M^++1, 21), 249 (M^+, 100), 234 (7), 220 (40), 206 (18), 192 (34), 177 (12), 165 (56), 151 (15).

N-Methyl-5H-dibenzo[c,e]azepine-5,7(6H)-dione. Mp: 161−163 °C [lit.[14] 165−167 °C]. IR (KBr) ν/cm^{-1}: 3058, 2924, 1596, 1504, 1445, 1281, 1224, 1029, 752, 725. MS *m*/*z* (relative intensity) EI: 237 (M^+, 19), 209 (100), 192 (24), 181 (30), 165 (31), 152 (28) 126 (4), 76 (12).

*5-[2-(2′-(Methylcarbamoyl)phenyl)phenyl]-3-phenyl-1,3,4-oxadiazol-2(3H)-one (**4a**).* (337 mg, 91%)*. Mp: 129−132 °C. IR (KBr) ν/cm^{-1}: 3370, 3062, 2924, 1782, 1649, 1597, 1501, 1373, 1145, 753. ^{1}H NMR (300 MHz, $CDCl_3$) δ: 2.78 (d, 3H, *J* = 5.0 Hz), 5.9 (bs, 1H), 7.1−7.6 (m, 11H), 7.8−7.9 (m, 2H). ^{13}C NMR (75.4 MHz, $CDCl_3$) δ: 26.6, 117.8, 122.0, 126.1, 127.5, 128.1, 128.3, 128.4, 129.7, 130.0, 130.1, 131.2, 131.6, 132.0, 138.3, 140.2, 165.5, 169.7. MS *m*/*z* (relative intensity) EI: 371 (M^+, 18), 314 (24), 313 (100), 269 (15), 204 (21), 194 (35), 167 (16), 151 (14), 91 (8), 77 (5). Anal. Calcd for $C_{22}H_{17}N_3O_3$: C, 71.16; H, 4.58; N, 11.32. Found: C, 70.86; H, 4.80; N, 11.07.

*5-[2-(2′-(Methylcarbamoyl)phenyl)phenyl]-3-(4-methoxyphenyl)-1,3,4-oxadiazol-2(3H)-one (**4b**).* (240 mg, 60%)*. Mp: 62−64 °C. IR (KBr) ν/cm^{-1}: 3401, 3058, 2929, 1771, 1653, 1513, 1380, 1251, 1029, 833, 757. ^{1}H NMR (300 MHz, $CDCl_3$) δ: 2.64 (d, 3H, *J* = 5.0 Hz), 3.81 (s, 3H), 5.8 (bs, 1H), 6.87 (d, 2H, *J* = 9.4 Hz), 7.18−7.22 (m, 1H), 7.4−7.5 (m, 3H), 7.57 (t, 2H, *J* = 8.0 Hz), 7.65−8.0 (m, 4H). ^{13}C NMR (75.4 MHz, $CDCl_3$) δ: 26.6, 55.5, 114.4, 119.7, 127.6, 127.7, 127.9, 128.1, 128.2, 128.3, 128.6, 129.2, 129.8, 129.9, 130.2, 131.2, 131.5, 150.4, 153.2, 157.7, 169.6. MS *m*/*z* (relative intensity) EI: 402 (M^++1, 20), 401 (M^+, 76), 343 (49), 222 (100), 204 (41), 195 (25), 178 (18), 151 (22), 121 (47), 106 (12). Anal. Calcd for $C_{23}H_{19}N_3O_4$: C, 68.83; H, 4.74; N, 10.47. Found: C, 68.57; H, 4.55; N, 10.72.

*5-[2-(2′-(Methylcarbamoyl)phenyl)phenyl]-3-[2-(methylthio)phenyl]-1,3,4-oxadiazol-2(3H)-one (**4c**).* (383 mg, 92%)*. Mp: 92−94 °C. IR (KBr) ν/cm^{-1}: 3421, 3051, 2921, 1786, 1652, 1534, 1479, 1337, 974, 751. ^{1}H NMR (300 MHz, $CDCl_3$) δ: 2.3 (s, 3H), 2.55 (d, 3H, *J* = 5.0 Hz), 6.0 (bs, 1H), 7.12 (t, 2H, *J* = 7.6 Hz), 7.19 (t, 2H, *J* = 8.9 Hz), 7.28−7.36 (m, 4H), 7.40−7.51 (m, 3H), 7.80 (d, 1H, *J* = 7.6 Hz). ^{13}C NMR (75.4 MHz, $CDCl_3$) δ: 15.9, 26.6, 122.3, 125.7, 127.2, 127.7, 127.9, 128.1, 128.8, 129.7, 129.8, 130.1, 131.4, 131.5, 132.2, 136.8, 137.0, 137.7, 140.4, 151.8, 154.0, 170.0. MS *m*/*z* (relative intensity) EI: 418 (M^++1, 4), 417 (M^+, 14), 360 (26), 359 (100), 268 (7), 238 (16), 206 (28), 194 (31), 181 (52), 166 (23), 152 (25), 136 (72), 122 (21), 78 (9). Anal. Calcd for $C_{23}H_{19}N_3O_3S$: C, 66.19; H, 4.56; N, 10.07; S, 7.67. Found: C, 65.87; H, 4.31; N, 9.80; S, 7.95.

*5-[2-(2′-(Methylcarbamoyl)phenyl)phenyl]-3-(4-chlorophenyl)-1,3,4-oxadiazol-2(3H)-one (**4d**).* (356 mg, 88%)*. Mp: 59−61 °C. IR (KBr) ν/cm^{-1}: 3411, 3058, 2921, 1785, 1647, 1534, 1495, 1377, 1095, 974, 934, 830, 736. ^{1}H NMR (300 MHz, $CDCl_3$) δ: 2.56 (d, 3H, *J* = 4.7 Hz), 5.62 (bs, 1H), 7.10−7.16 (m, 1H), 7.17−7.43 (m, 7H), 7.48 (t, 2H, *J* = 6.8 Hz), 7.54−7.63 (m, 1H), 7.83 (d, 1H, *J* = 6.8 Hz). ^{13}C NMR (75.4 MHz, $CDCl_3$) δ: 26.5, 118.9, 121.7, 127.5, 128.0, 128.2, 128.3, 129.1, 129.8, 129.9, 131.1, 131.3, 131.6, 134.3, 136.0, 138.1, 140.2, 150.0, 153.3, 169.2. MS *m*/*z* (relative intensity) EI: 407 (M^++2, 6), 405 (M^+, 18), 349 (35), 347 (100), 303 (8), 268 (9), 222 (27), 204 (36), 194 (51), 167 (28), 151 (21), 125 (16), 90 (18), 63 (9). Anal. Calcd for $C_{22}H_{16}ClN_3O_3$: C, 65.10; H, 3.95; N, 10.36. Found: C, 64.88; H, 4.11; N, 10.56.

*5-[2-(2′-(Methylcarbamoyl)phenyl)phenyl]-3-(4-bromophenyl)-1,3,4-oxadiazol-2(3H)-one (**4e**).* (368 mg, 82%)*. Mp: 60−62 °C. IR (KBr) ν/cm^{-1}: 3414, 3060, 2962, 1784, 1653, 1540, 1492, 1262, 1096, 1035, 803. ^{1}H NMR (300 MHz, $CDCl_3$) δ: 2.57 (d, 3H, *J* = 5.0 Hz), 5.62 (bs, 1H), 7.10−7.18 (m, 1H), 7.30−7.42 (m, 7H), 7.42−7.56 (m, 2H), 7.56−7.64 (m, 1H), 7.83 (dd, 1H, J_1 = 7.6 Hz, J_2 = 1.5 Hz). ^{13}C NMR (75.4 MHz, $CDCl_3$) δ: 26.6, 119.2, 119.3, 121.8, 127.6, 128.1, 128.3, 128.4, 129.9, 130.0, 131.2, 131.7, 132.2, 134.9, 136.1, 138.2, 140.3, 149.9, 153.5, 169.3. MS *m*/*z* (relative intensity) EI: 451 (M^++2, 19), 449 (M^+, 18), 394 (22), 393 (90), 392 (22), 391 (90), 350 (9), 348 (9), 296 (12), 268 (26), 239 (12), 222 (66), 204 (72), 194 (100), 167 (50), 151 (51), 90 (49), 63 (36). Anal. Calcd for $C_{22}H_{16}BrN_3O_3$: C, 58.67; H, 3.56; N, 9.33. Found: C, 58.72; H, 3.81; N, 9.07.

*5-[2-(2′-(Methylcarbamoyl)phenyl)phenyl]-3-(4-acetylphenyl)-1,3,4-oxadiazol-2(3H)-one (**4f**).* (342 mg, 83%)*. Mp: 61−63 °C. IR (KBr) ν/cm^{-1}: 3419, 2923, 1786, 1643, 1602, 1377, 1266, 933, 841, 770. ^{1}H NMR (300 MHz, $CDCl_3$) δ: 2.61 (s, 3H), 2.67 (d, 3H, *J* = 4.9 Hz), 5.7 (bs, 1H), 7.44 (d, 1H, *J* = 7.4 Hz), 7.48−7.68 (m, 7H), 7.68−7.74 (m, 1H), 7.94−8.0 (m, 3H). ^{13}C NMR (75.4 MHz, $CDCl_3$) δ: 26.5, 26.6, 117.2, 121.7, 127.6, 128.1, 128.2, 128.3, 129.6, 129.9, 130.0, 131.1, 131.8, 133.2, 136.0, 138.2, 139.5, 148.8, 149.8, 171.8, 196.7. MS *m*/*z* (relative intensity) EI: 414 (M^++1, 4), 413 (M^+, 13), 356 (24), 355 (100), 312 (4), 297 (17), 269 (9), 222 (11), 204 (18), 194 (33), 166 (17), 151 (12). Anal. Calcd for $C_{24}H_{19}N_3O_4$: C, 69.73; H, 4.60; N, 10.17. Found: C, 70.07; H, 4.91; N, 9.89.

*5-[2-(3-(Methylcarbamoyl)-pyridin-2-yl)pyridin-3-yl]-3-phenyl-1,3,4-oxadiazol-2(3H)-one (**5a**).* (238 mg, 64%)*. Mp: 161−162 °C. IR (KBr) ν/cm^{-1}: 3415, 3056, 2925, 1773, 1663, 1578, 1378, 1043, 735. ^{1}H NMR (300 MHz, $CDCl_3$) δ: 2.72 (d, 3H, *J* = 5.0 Hz), 6.4 (bs, 1H), 7.15 (t, 1H, *J* = 8.0 Hz), 7.3 (t, 2H, *J* = 8.0 Hz), 7.4−7.54 (m, 4H), 8.06 (dd, 1H, J_1 = 7.9 Hz, J_2 = 1.6 Hz), 8.28 (dd, 1H, J_1 = 8.2 Hz, J_2 = 1.6 Hz), 8.64 (dd, 1H, J_1 = 4.6 Hz, J_2 = 1.6 Hz), 8.72 (dd, 1H, J_1 = 4.6 Hz, J_2 = 1.6 Hz). ^{13}C NMR (75.4 MHz, $CDCl_3$) δ: 26.7, 117.9, 119.6, 123.3, 123.5, 126.1, 129.0, 136.0, 136.3, 136.6, 150.3, 150.6, 151.8, 155.0, 156.4, 167.4. MS *m*/*z* (relative intensity) CI: 414 (M^++41, 6), 402 (M^++29, 19), 374 (M^++1, 40), 343 (100), 329 (21), 269 (11), 207 (14). Anal. Calcd for $C_{20}H_{15}N_5O_3$: C, 64.34; H, 4.02; N, 18.77. Found: C, 63.98; H, 4.23; N, 18.91.

*N-Methyldibenzo[c,e]azepine-5-one-7-(ylidene-2-phenylhydrazine) (**6a**).* (43 mg, 13%)**. Mp: 74−75 °C. IR (KBr) ν/cm^{-1}: 3276, 2923, 1642, 1602, 1445, 1372, 1252, 1120, 752, 736, 696. ^{1}H NMR (300 MHz, $CDCl_3$) δ: 2.0 (bs, 1H), 3.24 (s, 3H), 6.82 (t, 1H, *J* = 7.3 Hz), 7.0 (d, 1H, *J* = 7.6 Hz), 7.16 (d, 1H, *J* = 7.6 Hz), 7.3−7.55 (m, 5H), 7.57 (d, 1H, *J* = 7.6 Hz), 7.67 (t, 1H, *J* = 7.9 Hz), 7.86 (d, 1H, *J* = 7.6 Hz), 7.97 (d, 1H, *J* = 7.9 Hz), 8.14 (d, 1H, *J* = 7.9 Hz). ^{13}C NMR (75.4 MHz, $CDCl_3$) δ: 31.8, 113.6, 121.2, 128.0, 128.1, 128.3, 128.5, 129.3, 129.4, 129.8, 130.7, 131.5, 132.2, 132.9, 135.8, 137.1, 137.3, 143.7, 169.0. MS *m*/*z* (relative intensity) EI: 328 (M^++1, 22), 327 (M^+, 89), 269 (17), 222 (100), 204 (54), 195 (28), 178 (17), 165 (35), 151 (15), 91 (3), 77 (5). Anal. Calcd for $C_{21}H_{17}N_3O$: C, 77.06; H, 5.20; N, 12.84. Found: C, 77.21; H, 5.01; N, 12.66.

*N-Methyldibenzo[c,e]azepine-5-one-7-(ylidene-2-(4-methoxyphenyl)hydrazine) (**6b**).* (220 mg, 62%)**. Mp: 213−215 °C. IR (KBr) ν/cm^{-1}: 3264, 3058, 2926, 1643, 1514, 1444, 1366, 1236, 1035, 826, 740. ^{1}H NMR (300 MHz, $CDCl_3$) δ: 3.27 (s, 3H), 3.73 (s, 3H), 6.8 (d, 2H, *J* = 9.1 Hz), 7.0 (d, 2H, *J* = 9.1 Hz), 7.4−7.55 (m, 6H), 7.6 (d, 1H, *J* = 7.0 Hz), 7.9 (d, 1H, *J* = 7.9 Hz). ^{13}C NMR (75.4 MHz, $CDCl_3$) δ: 31.6, 55.7, 114.5, 114.6, 125.9, 127.9, 128.0, 128.2, 128.4, 129.3, 129.6, 129.9, 130.6, 130.8, 131.2, 131.4, 137.0, 137.7, 154.3, 168.9. MS *m*/*z* (relative intensity) EI: 358 (M^++1, 19), 357 (M^+, 72), 300 (3), 222 (100), 204 (44), 195 (25), 178 (19), 165 (32), 151 (22), 122 (41), 92 (13), 77 (8), 65 (5). Anal. Calcd for $C_{22}H_{19}N_3O_2$: C, 73.95; H, 5.32; N, 11.76. Found: C, 74.22; H, 5.60; N, 11.49.

*N-Methyldibenzo[c,e]azepine-5-one-7-(ylidene-2-(4-chlorophenyl)hydrazine) (**6d**).* (255 mg, 71%)**. Mp: 226−228 °C. IR (KBr) ν/cm^{-1}: 3276, 3058, 2923, 1637, 1616, 1506, 1492, 1370, 1250, 1087, 824, 737. ^{1}H NMR (300 MHz, $CDCl_3$) δ: 3.22 (s, 3H), 6.94 (d, 2H, *J* = 8.9 Hz), 7.12 (d, 2H, *J* = 8.9 Hz), 7.3−7.55 (m, 7H), 7.58 (d, 1H, *J* = 7.8 Hz), 7.82 (d, 1H, *J* = 7.8 Hz). ^{13}C NMR (75.4 MHz, $CDCl_3$) δ: 31.9, 114.0, 114.3, 128.0, 128.3, 128.6, 129.2, 129.4, 129.9, 130.7, 131.6, 132.7, 136.2, 136.7, 137.0, 137.2, 142.4, 169.2. MS *m*/*z* (relative intensity) EI: 363 (M^++2, 21), 361 (M^+, 60), 304 (12), 269 (7), 222 (100), 204 (58), 195 (31), 178 (18), 167 (31), 165 (36), 151 (23), 125 (10), 111 (12), 90 (10), 75 (8). Anal. Calcd for $C_{21}H_{16}ClN_3O$: C, 69.71; H, 4.43; N, 11.62. Found: C, 69.50; H, 4.61; N, 11.53.

*N-Methyldibenzo[c,e]azepine-5-one-7-(ylidene-2-(4-bromophenyl)hydrazine) (**6e**).* (357 mg, 88%)**. Mp: 232−234 °C. IR (KBr) ν/cm^{-1}: 3280, 3058, 2931, 1636, 1593, 1497, 1487, 1368, 1250, 1140, 1121, 1069, 819, 762, 737. ^{1}H NMR (300 MHz, $CDCl_3$)

δ: 3.14 (s, 3H), 6.81 (d, 2H, J = 8.9 Hz), 7.1 (d, 2H, J = 8.9 Hz), 7.2−7.5 (m, 7H), 7.62 (d, 1H, J = 7.6 Hz), 7.84 (s, 1H). ^{13}C NMR (75.4 MHz, $CDCl_3$) δ: 32.0, 112.9, 115.0, 127.9, 128.0, 128.3, 128.5, 129.4, 129.8, 130.7, 131.5, 131.9, 132.6, 135.8, 136.8, 137.0, 137.2, 142.9, 168.7. MS m/z (relative intensity) EI: 407 (M^++2, 17), 405 (M^+, 17), 350 (4), 348 (4), 269 (6), 222 (100), 204 (52), 195 (28), 178 (19), 165 (35), 151 (21), 91 (6), 76 (4), 63 (8). Anal. Calcd for $C_{21}H_{16}BrN_3O$: C, 62.07; H, 3.94; N, 10.34. Found: C, 62.21; H, 4.13; N, 10.24.

*N-Methyldibenzo[c,e]azepine-5-one-7-(ylidene-2-(4-acetylphenyl)hydrazine) (**6f**).* (310 mg, 84%)**. Mp: 138−140 °C. IR (KBr) ν/cm^{-1}: 3256, 3059, 2928, 1654, 1598, 1522, 1444, 1359, 1261, 1108, 835, 740. ^{1}H NMR (300 MHz, $CDCl_3$) δ: 2.5 (s, 3H), 3.3 (s, 3H), 7.1 (d, 2H, J = 8.5 Hz), 7.27−7.67 (m, 7H), 7.85 (d, 3H, J = 8.9 Hz), 8.0 (s, 1H). ^{13}C NMR (75.4 MHz, $CDCl_3$) δ: 26.3, 32.2, 112.7, 127.9, 128.0, 128.4, 128.6, 129.4, 130.0, 130.3, 130.4, 130.7, 131.6, 132.6, 136.5, 136.9, 137.1, 137.6, 147.5, 168.7, 196.5. MS m/z (relative intensity) EI: 370 (M^++1, 23), 369 (M^+, 98), 312 (7), 297 (16), 269 (7), 222 (100), 204 (71), 195 (39), 178 (21), 165 (42), 151 (22), 91 (8), 77 (6). Anal. Calcd for $C_{23}H_{19}N_3O_2$: C, 74.80; H, 5.15; N, 11.38. Found: C, 74.65; H, 5.33; N, 11.46.

■ ASSOCIATED CONTENT

Supporting Information

IR, MS, and NMR (^{1}H and ^{13}C) spectra of **4**−**6** and ORTEP drawings and CIFs for **4a**, **5a**, and **6b**. This material is available free of charge via the Internet at http://pubs.acs.org.

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Notes

The authors declare no competing financial interest.

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