Depressant 1,2-Dihydroquinolines and Related Derivatives

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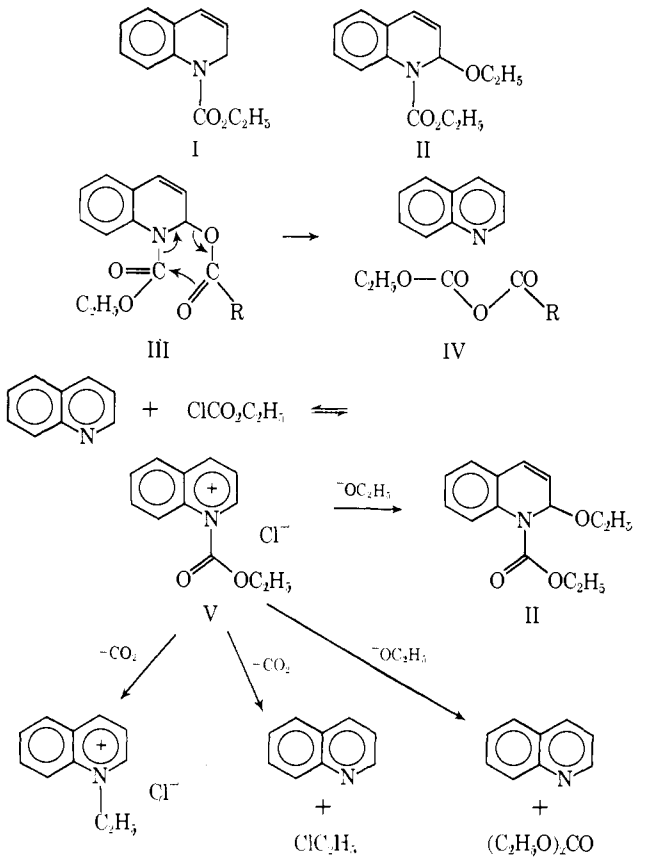
A series of 1,2-dihydroquinoline carbamates and 1,2-dihydroquinoline amides was prepared to study the relationship of structure to CNS activity. Benzothiazole, benzoxazole, and phenanthridine derivatives did not share the depressant activity of their quinoline counterparts.

Belleau, *et al.*,[1] and Martel[2] have reported that carbamate I, at low doses, depresses behavior and irreversibly inhibits α-adrenergic receptors. *In vitro*, this compound appears to be oxidized to a biological equivalent of II, which is probably the active metabolite.[1]



Ethoxy derivative II induces rapid formation of peptide linkages,[3] possibly by a mechanism involving reversible alkylation of the carboxyl group of an acylamino acid, followed by intramolecular acylation, and formation of a mixed carbonic anhydride (IV), which functions as the activated ester in the peptide coupling step.

The outstanding peptide coupling ability of II can be attributed to chemical features, which may have important biological implications. Firstly, II is an alkylating agent reminiscent of such α-adrenergic blockers as Dibenamine, but differing in that (a) the alkylation process is reversible because of the cation-stabilizing influence of the pseudoaromatic system and (b) II is more discriminating when selecting a nucleophilic reaction partner, *i.e.*, II is nonreactive with amines, alcohols, and mercaptans at neutral pH. Only two functional groups likely to be present at a biological receptor are alkylated by II: carboxylate and phosphate (pyrophosphate). Therefore, under physiological conditions II represents a reversible and highly selective alkylating agent. Secondly, intermediate III is ideally predisposed to undergo intramolecular acylation *via* a six-membered transition state. Quinoline aromatization provides the major driving force and insures the irreversibility of this stage. Mixed anhydride IV, presumably the "active ester" in peptide coupling, is highly sensitive to hydrolysis.

The likely relationship of biological activity to this alkylation–acylation sequence was explored. A series of acyl analogs, as well as some heterocyclic variants of I and II were prepared and tested for depressant and antiamphetamine effects in rodents.

Synthesis.—Preparation of II and its derivatives may be regarded as being composed of 3 stages: (a) quinoline acylation, (b) reaction with a nucleophile, and (c) product isolation. Since II in wet organic solvents is rapidly hydrolyzed to quinoline and EtOH, contact with H_2O during work-up was minimized.

Carbethoxyquinolinium chloride (V) reacts rapidly and exothermically with NaOEt to yield products which result from attack at C-2 (II) and at the CO group (quinoline and diethyl carbonate). Attack at the CO group may be minimized in two ways: (a) by replacement of NaOEt by EtOH and a basic, sterically hindered amine, such as diisopropylethylamine, and (b) by the use of NaSMe as the nucleophile. As expected, each modification had a significant positive effect on product yield.

When conditions for work-up and nucleophile addition had been optimized, the quinoline acylation stage was examined. Carbethoxyquinolinium chloride (V) decomposes *via* three paths: (a) attack of Cl^- at CO to yield the starting materials, (b) attack of Cl^- at the α-C of the Et group to yield EtCl, CO_2, and quinoline, and (c) intramolecular extrusion of CO_2 to yield 1-ethylquinolinium chloride. When the reaction mixture was allowed to stand for 24 hr at 25°, V decomposed primarily *via* path b. Decomposition of V was minimal when II was prepared at $-10°$ to $+10°$ in the absence of solvent. Similar reaction conditions were employed in the synthesis of acyl analogs of II, as well as benzothiazole **32** and benzoxazole **33**.

A second route to 1,2-dihydroquinoline structures is exemplified by the preparation of **17**. LAH reduction of quinoline yields *ca.* 60% 1,2-dihydroquinoline. The yield is less than quantitative because of rapid disproportionation to quinoline and tetrahydroquinoline, but acylation with BzCl, AcCl, trifluoroacetic anhydride, $MeSO_2Cl$, etc., proceeds smoothly, and the products

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(1) B. Belleau, R. Martel, G. Lacasse, M. Menard, N. L. Weinberg, and Y. G. Perron, *J. Amer. Chem. Soc.*, **90**, 823 (1968).

(2) R. Martel, *J. Pharmacol. Exp. Ther.*, **166**, 44 (1969).

(3) B. Belleau and G. Malek, *J. Amer. Chem. Soc.*, **90**, 1651 (1968).

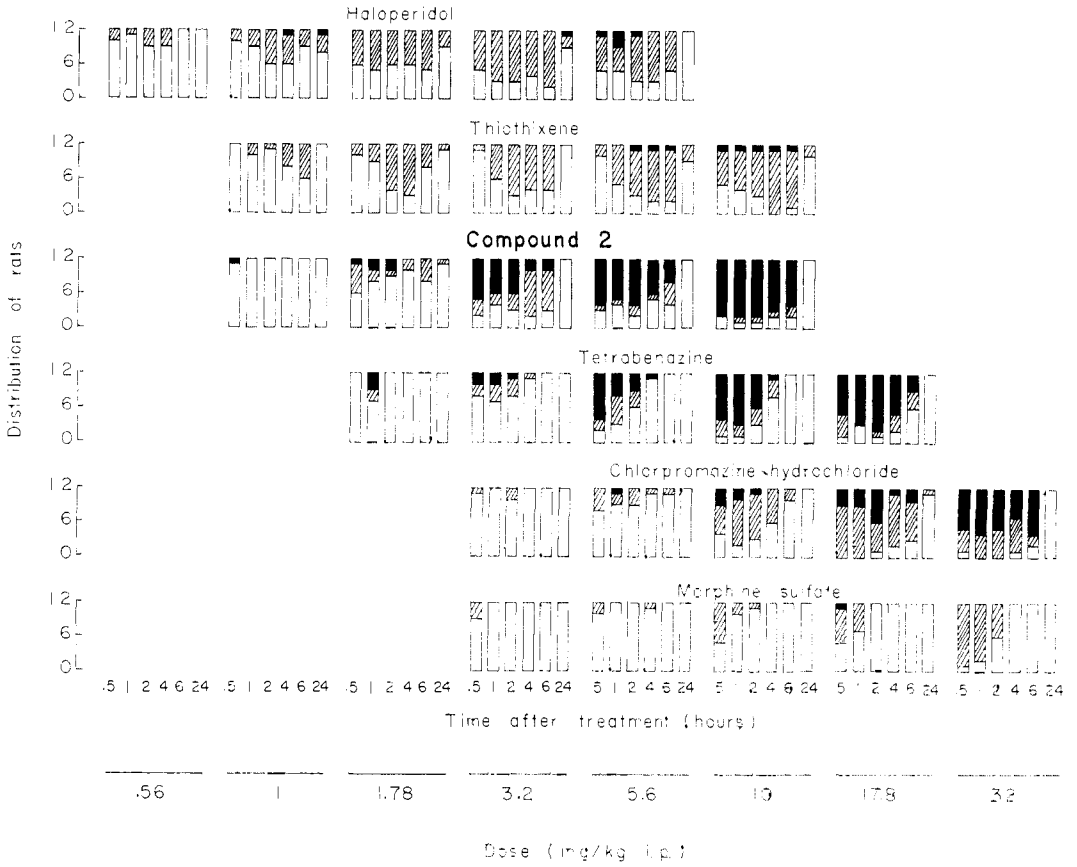


Figure 1.—Effect of **2** and standard drugs on a crossover test of discriminated avoidance behavior in rats. The procedure and experimental design used are identical to those described by Weissman (Table I, footnote *c*). Each bar shows the distribution of avoidance scores among the 12 rats exposed to that dose at the time of administration shown on the secondary abscissa. Open areas: number of rats unaffected; striped areas: number of rats exhibiting disruption of avoidance behavior but retention of escape behavior; black areas: number of rats exhibiting loss of both avoidance and escape behavior.

were isolated in fair yield. Phenanthridine **34** was prepared in the same manner.

Hydride reduction of benzothiazole is not a practical method of producing benzothiazoline; however, treatment of *o*-aminobenzenethiol with CH_2O proved to be an excellent preparative method. Acylation of benzothiazoline with ethyl chloroformate provides carbamate **31**.

Pharmacology.—Using published techniques (footnotes to Table I), compounds were screened in mice for symptomatic and rotorod-disrupting effects, and for antagonism of amphetamine mortality. They were also screened in rats for blockade of conditioned avoidance behavior and of amphetamine-induced stereotypy. Pronounced depressant and antiamphetamine effects were seen after administration of **1**, **2**, and related esters.

In mouse screening, active compounds produced such symptoms as ptosis, miosis, tremors, catalepsy, ataxia, and reduced locomotor activity. These symptoms are essentially identical with those reported by Martel[2] for **1**. The antiamphetamine potency of these compounds was pronounced, in many cases exceeding that of chlorpromazine. Suppression of conditioned avoidance in rats tended to be long-lived but nonselective, compared with known antipsychotic drugs, as illustrated in Figure 1 for **2**.

Data in Table I suggest that carbamate analogs of **1** and **2** are qualitatively similar in their depressant action, although quantitative differences are apparent. Compounds which produced potent disruptive effects on rotorod performance and conditioned avoidance behavior invariably also blocked amphetamine mortality in aggregated mice and amphetamine stereotypy in rats. Quinolone **6** and tetrahydroquinoline **13** are notable inactives.

Of the 1,2-dihydroquinoline amides (Table II), the acetyl derivative, **14**, exhibited a profile similar to that of **1** and **2**, though less potent. The benzoyl derivative, **17**, also retained activity, but was still less potent. The 2-substituted analogs of **14** and **17** (**15** and **18**), like the remaining amides in Table II, were essentially inactive. Hydrolytic instability almost surely plays a role.

Heterocyclic variants of **2** (Table III) which are structurally and electronically closely related to dihydroquinoline exhibited no appreciable activity characteristic of **2**.

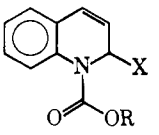
Experimental Section

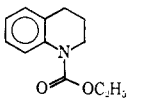
Melting points (Thomas-Hoover capillary melting point apparatus) are uncorrected. Ir spectra were measured with a Perkin-Elmer Model 21 spectrophotometer, uv spectra with a Cary 14 recording spectrophotometer, and nmr spectra with a Varian Model A-60 (Me_4Si, in $CDCl_3$, unless otherwise stated). Spectra were recorded for all compds, but the results are not reported if they were only confirmatory and as expected.

Method A. 2-Ethoxy-1(2*H*)-quinolinecarboxylic Acid, Ethyl Ester (3).—Ethyl chloroformate (10.8 g, 0.10 mole) was added dropwise to anhyd quinoline (12.9 g, 0.10 mole) under N_2. The stirred reaction mixture was maintained at 0 to 7° for 1 hr during which time a white solid sepd. A solution of diisopropylethyl-

TABLE I

LETHAL, DEPRESSANT, AND ANTIAMPHETAMINE EFFECTS OF 1,2-DIHYDROQUINOLINE CARBAMATES

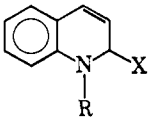


No.	R	X	LD_{50}[a] (mouse) mg/kg ip	Rotorod[b] ED_{50} (mouse) mg/kg ip	Antiavoidance ED_{50}[c] (rat) mg/kp ip	Antiamphetamine ED_{50} (mouse)[d] mg/kp ip	Antiamphetamine ED_{50} (rat)[e] mg/kg ip
1 (I)	C_2H_5	H	32–100	3.2–10	0.3–1	1–3.2	1–3.2
2	C_2H_5	OCH_3	32–100	1–3.2	1–3.2	3.2–10	1–3.2
3 (II)	C_2H_5	OC_2H_5	32–100	3.2–10	<5.6	1–3.2	<32
4	C_2H_5	SCH_3	32–100	0.3–1	<32	1–3.2	1–3.2
5	C_2H_5	$SCH_2CH{=}CH_2$	320–1000	10–32	<32	10–32	<32
6	C_2H_5	=O	320–1000	32–100	>32	100	NT
7	n-C_3H_7	OCH_3	32–100	1–3.2	<10	0.3–1	1–3.2
8	i-C_3H_7	H	32–100	0.3–1	<10	0.3–1	<10
9	i-C_3H_7	OCH_3	32–100	1–3.2	<10	0.3–1	3.2–10
10	n-C_4H_9	OCH_3	32–100	1–3.2	<10	1–3·2	1–3.2
11	$CH_2C_6H_5$	H	32–100	3.2–10	<32	0.1–0.3	<10
12	$CH_2C_6H_5$	OCH_3	100–320	3.2–10	<32	3.2–10	<10
13			320–1000	100–320	>32	32–100	>32
	Chlorpromazine·HCl[f]		165[g]	1–3.2	3.2–10	1.6[g]	6.0[g]

[a] Three mice were treated with doses of 32, 100, 320, and 1000 mg/kg ip. Lower entries are highest doses at which 0 or 1 mice died (24-hr basis); higher entries are lowest doses at which 2 or 3 mice died. [b] Inability to remain on a grooved metal cylinder of 13 mm diameter rotating at 15 rpm. [c] Blockade of conditioned jump-out avoidance: method of Weissman [A. Weissman, *Psychopharmacologia*, **12**, 142 (1968)]. [d] Blockade of aggregation toxicity: method of Weissman.[c] [e] Blockade of sniffing-licking-gnawing syndrome: method of A. Weissman, B. K. Koe, and S. S. Tenen, *J. Pharmacol. Exp. Ther.*, **151**, 339 (1966). [f] Dose calcuated as free base. [g] Previously reported by Weissman.[c]

TABLE II

LETHAL, DEPRESSANT, AND ANTIAMPHETAMINE EFFECTS OF 1,2-DIHYDROQUINOLINE AMIDES[a]



No.	R	X	LD_{50} (mouse) mg/kg ip	Rotorod ED_{50} (mouse) mg/kg ip	Antiavoidance ED_{50} (rat) mg/kg ip	Antiamphetamine ED_{50} (mouse) mg/kg ip	Antiamphetamine ED_{50} (rat) mg/kg ip
14	$COCH_3$	H	32–100	3.2–10	3.2–10	10–32	1–3.2
15	$COCH_3$	OCH_3	100–320	10–32	>10	>100	>32
16	$COC(CH_3)_3$	H	100–320	10–32	>32	32–100	10–32
17	COC_6H_5	H	100–320	10–32	10–32	10–32	10–32
18	COC_6H_5	SCH_3	NT	NT	>32	NT	>32
19	COC_6H_4-p-Cl	H	100–320	32–100	>32	>32	>32
20	COC_6H_4-p-OCH_3	H	320–1000	32–100	>32	10–32	>32
21	COC_6H_4-p-NO_2	H	>1000	100–320	>32	>100	>32
22	$COCF_3$	H	100–320	32–100	>32	>100	10–32
23	$COCH_2OCH_3$	H	100–320	10–32	NT	3.2–10	>32
24	$COCO_2C_2H_5$	H	320–1000	100–320	>32	>100	>32
25	$CONH_2$	H	NT	10–32	>32	>100	>32
26	$CONHCH_3$	H	100–320	3.2–10	>32	>100	>32
27	$CON(CH_3)_2$	H	NT	NT	>32	NT	>32
28	$CONHC_6H_5$	H	320–1000	10–32	>32	>100	>32
29	SO_2CH_3	H	32–100	0.32–1	>32	>32	>32
30	$SO_2C_6H_4$-p-CH_3	H	>1000	320–1000	>32	>100	>32

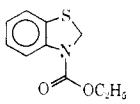
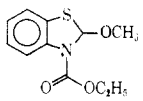
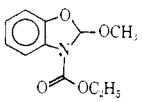
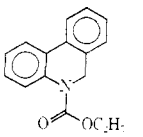
[a] For methods, see Table I.

amine (12.9 g, 0.10 mole) in 50 ml of abs EtOH was added and the solid dissolved. After warming to room temp, the volatile components were evapd under vacuum. The residue was treated with 200 ml of cyclohexane, then 200 ml of ice water added, and the organic phase dried over $MgSO_4$. Evaporation of the solvent yielded 22.5 g of a pale yellow oil. Vacuum distillation provided 16.9 g of pure **3**, a colorless liquid: bp 115–118° (0.1 mm); ir (film) 5.8 (C=O), 6.1 (C=C), 9.4 μ (CO); uv max (EtOH) 229, 261 mμ (log ϵ 4.5, 3.9); nmr δ 1.1 (t, 3 H), 1.3 (t, 3 H), 3.6 (q, 2 H), 4.3 (q, 2 H), ABX pattern at 6.1 (2 H) and 6.6 (1 H), 6.9–7.4 (m, 3 H), 7.6 (m, 1 H). The oil solidified on standing (mp 59–61°).

Method B. 2-Methylthio-1(2*H*)-quinolinecarboxylic Acid, Ethyl Ester (4).—Ethyl chloroformate (10.8 g, 0.10 mole) was added dropwise to anhyd quinoline (12.9 g, 0.10 mole) under N_2 and the reaction mixture maintained at 0 to 5° for 1 hr. A solu-

TABLE III

LETHAL, DEPRESSANT, AND ANTIAMPHETAMINE EFFECTS OF HETEROCYCLIC VARIANTS[a]

No.		LD_{50} (mouse) mg/kg ip	Rotorod ED_{50} (mouse) mg/kg ip	Antiavoidance ED_{50} (rat) mg/kg ip	Antiamphetamine ED_{50} (mouse) mg/kg ip	Antiamphetamine ED_{50} (rat) mg/kg ip
31		320–1000	32–100	>56	>100	>32
32		>1000	32–100	>32	>100	>32
33		320–1000	10–32	>32	>100	>32
34		320–1000	100–320	>56	>100	>32

[a] For methods, see Table I.

TABLE IV

No.	Preparative method	Bp, °C (mm)	Mp, °C	Formula[a]
1	C	108 (0.02)		$C_{12}H_{13}NO_2$
2	A	125–129 (0.02)[b]		$C_{13}H_{15}NO_3$
3	A	115–118 (0.1)[c]	59–61[c]	$C_{14}H_{17}NO_3$
4	B	138–140 (0.5)		$C_{13}H_{15}NO_2S$
5	B	150–152 (0.01)		$C_{15}H_{17}NO_2S$
6		135–138 (0.02)	68–70	$C_{12}H_{11}NO_3$
7	A	128–130 (0.2)		$C_{14}H_{17}NO_3$
8	C	*d*		$C_{13}H_{15}NO_2$
9	A	126–127 (0.2)		$C_{14}H_{17}NO_3$
10	A	133–134 (0.5)		$C_{15}H_{19}NO_3$
11	C	*d*		$C_{17}H_{15}NO_2$
12	A	172–173 (0.1)		$C_{18}H_{17}NO_3$
13		95–98 (0.025)		$C_{12}H_{15}NO_2$
14	C	115–117 (0.1)[e]		$C_{11}H_{11}NO$
15	A	124–125 (0.1)		$C_{12}H_{13}NO_2$
16	C	105–108 (0.1)	74–76	$C_{14}H_{17}NO$
17	C	153–155 (0.1)	85–87	$C_{16}H_{13}NO$
18	B		135–136[f]	$C_{17}H_{15}NOS$
19	C		109–111[g]	$C_{16}H_{12}ClNO$
20	C		150–152[h]	$C_{17}H_{15}NO_2$
21	C		131–134[i]	$C_{16}H_{12}N_2O_3$
22	C	90–92 (0.1)		$C_{11}H_8F_3NO$
23	C	110–111 (0.05)		$C_{12}H_{13}NO_2$
24	C	143–145 (0.1)		$C_{13}H_{13}NO_3$
25	C		149–150[j]	$C_{10}H_{10}N_2O$
26	C	140–142 (0.2)	82.5–84	$C_{11}H_{12}N_2O$
27	C	109–111 (0.2)		$C_{12}H_{14}N_2O$
28	C		123–125[h]	$C_{16}H_{14}N_2O$
29	C		87–89[f]	$C_{10}H_{11}NO_2S$
30	C		90–91[f,k]	$C_{16}H_{15}NO_2S$
31		107–110 (0.1)		$C_{10}H_{11}NO_2S$
32	A	118–120 (0.02)		$C_{11}H_{13}NO_3S$
33	A	112 (0.1)		$C_{11}H_{13}NO_4$
34		158–159 (0.01)		$C_{16}H_{15}NO_2$

[a] All compounds analyzed correctly for C, H, N. [b] N. L. Weinberg [U. S. Patent 3,389,142 (1968)] reported bp 130° (0.2 mm). [c] Reported: bp 125–128° (0.1 mm); mp 56–57°.[b] [d] Purified by silica gel chromatography. [e] E. Braude, J. Hannah, and R. Linstead [*J. Chem. Soc.*, 3254 (1960)] report bp 102° (0.8 mm). [f] Crystallized from cyclohexane. [g] Crystallized from MeOH. [h] Crystallized from *i*-PrOH. [i] Crystallized from MeOH. [j] Crystallized from EtOAc. K. Rosenmund and F. Zymalkowski [*Chem. Ber.*, **86**, 37 (1953)] reported mp 148–149°. [k] Reported mp 94°.[i]

tion of NaSMe (prepared by bubbling MeSH into a solution of 0.11 mole of NaOMe in MeOH) was added. Work-up as in method A yielded 12.5 g of **4**: bp 138–140° (0.5 mm); nmr δ 2.0 (s, SMe).

Method C. 1-Benzoyl-1,2-dihydroquinoline (17).—A suspension of 7.6 g (0.20 mole) of LAH in 300 ml of anhyd Et_2O was heated at reflux for 20 min, a solution of quinoline (12.9 g, 0.10 mole) in 100 ml of Et_2O added, and the mixture refluxed an additional 3 hr. The greenish yellow slurry was cooled to 0° and treated carefully with 8 ml of H_2O followed by 10 ml of 3 *N* aq KOH. The salts were filtered, the solvent evapd, and the white crystalline residue dissolved in 20 ml of pyridine. BzCl (14 g, 0.10 mole) in pyridine (20 ml) was added dropwise at 0°. The mixture was stirred at room temp overnight, then dild with Et_2O. The Et_2O solution was washed (3 *N* aq HCl, H_2O), dried ($MgSO_4$), and evapd to yield a viscous oil. Vacuum distillation afforded 9.7 g of material, bp 153–155° (0.10 mm), which crystallized (mp 78–80°). By nmr analysis the product consists of 85% of **17** and 15% of its 3,4-dihydro derivative; the 1-benzoyl-1,2,3,4-tetrahydroquinoline signals are at δ 3.9 (H-2), 2.0 (H-3), 2.8 (H-4) and the **17** signals are at δ 4.5 (H-2), 6.1 (H-3), 6.6 (H-4). Recrystallization from cyclohexane afforded **17**: mp 85–87°; ir (KBr) 6.1 μ.

In general the tetrahydroquinoline byproduct was difficult to remove. All other products of method C were isolated in 85–95% purity.

2-Oxo-1(2*H*)-quinolinecarboxylic Acid, Ethyl Ester (6).—A solution of carbostyril (10.9 g, 75 mmoles) in 100 ml of anhyd THF under N_2 was treated with 3.2 g (75 mmoles) of NaH (56% in oil). When H_2 evolution subsided, ethyl chloroformate (8.2 g, 75 mmoles) was added, and the reaction mixture was maintained at room temp until the concentration of starting material (tlc) reached a minimum (2 hr). The solvent was evapd *in vacuo* and the residue partitioned between CH_2Cl_2 and H_2O. Chromatography of the CH_2Cl_2 extract on silica gel (elution with 25% EtOAc in cyclohexane) removed several polar impurities. Vacuum distillation provided 11.3 g of material [bp 135–138° (0.02 mm)] consisting of **6** and its *O*-acyl isomer in a 2:1 ratio. Crystallization from cyclohexane afforded pure **6**: mp 68–70°; ir (KBr) 5.7, 6.0 μ; nmr δ 6.6 (d, J = 10 Hz, H-3) 7.7 (d, J = 10 H, H-4).

3,4-Dihydro-1(2*H*)-quinolinecarboxylic Acid, Ethyl Ester (13).—A solution of 24.0 g (180 mmoles) of 1,2,3,4-tetrahydroquinoline in 180 ml of Et_2O was treated at 5–15° with 9.7 g (90 mmoles) of ethyl chloroformate in 50 ml of Et_2O. After 2 hr sufficient H_2O was added to dissolve the pptd HCl salt, the layers sepd, and the Et_2O residue vacuum distd to yield 13.0 g of **13**: bp 95–98° (0.025 mm); ir (film) 5.8 μ.

3-Benzothiazolinecarboxylic Acid, Ethyl Ester (31).—To a solution of 17 g (0.12 mole) of benzothiazoline (prepared according to the method of Jenkins, *et al.*[4]) in 85 ml of CH_2Cl_2 and 180 ml of

(4) G. L. Jenkins, A. M. Knevel, and C. S. Davis, *J. Org. Chem.*, **25**, 2062 (1960).

1 *N* aq $KHCO_3$ was added 17 g (0.16 mole) of ethyl chloroformate with vigorous stirring. When CO_2 evolution subsided the organic phase was washed with H_2O, dried ($MgSO_4$), and evapd to an oil. Vacuum distillation provided 14.5 g of pure **31**: bp 107–110° (0.1 mm); ir (film) 5.8 μ; nmr δ 5.2 (s, 2 H).

5(6*H*)-Phenanthridinecarboxylic Acid, Ethyl Ester (34).—A solution of 9.1 g (50 mmoles) of 5,6-dihydrophenanthridine (prepared according to the method of Wooten and McKee[5]) was treated with ethyl chloroformate as in the preceding experimental procedure. Pure **34** was obtained as a colorless liquid: bp 158–159° (10 μ); ir (film) 5.8 μ; nmr δ 4.8 (s, 2 H).

(5) W. C. Wooten and R. L. McKee, *J. Amer. Chem. Soc.*, **71**, 2946 (1949).

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Notes

Synthesis and Activity of Some 1,2,4-Triazolylthiazolidones

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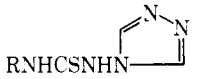
Thiazolidine derivatives exhibit sedative,[1] anesthetic,[2] anticonvulsant,[3] antituberculous,[4] amebicidal,[5] and fungicidal[6] activity. Previous publications from this laboratory[7–12] have shown that some derivatives of 5-carboxymethylthiazolidine-2,4-dione inhibit viral growth.

In continuing these investigations some new thiazolidine derivatives have been synthesized. 1,2,4-Triazolylthioureas (**1**) (Table I), obtained by condensing 4-amino-1,2,4(4*H*)-triazole with several isothiocyanates, were cyclized with maleic anhydride to the corresponding thiazolidin-4-ones (**2**) (Table II).

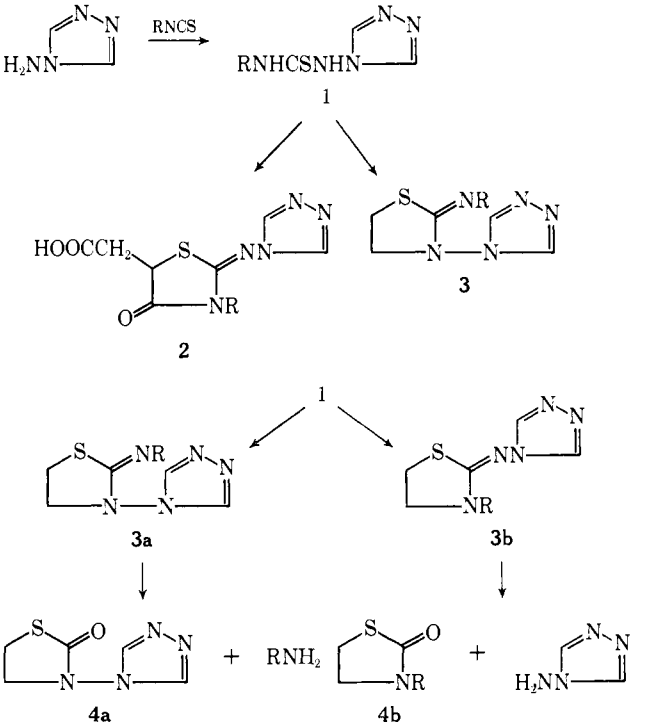
The 1,2,4-triazolylthioureas were also condensed with 1,2-dibromoethane to afford the corresponding thiazolidines (**3**). It can be envisaged that the reaction could take place to give two different monocyclic products, *i.e.*, **3a** or **3b**, or even a bicyclic product. To ascertain the structure of the products, some of these were hydrolyzed with HCl at 200°. The expected primary cleavage products would be **4a** and $PhNH_3^+Cl^-$ from **3a**, or 4-amino-1,2,4-triazole·HCl and **4b** from **3b**. From the hydrolysates of **3a** (R = C_6H_5) $PhNH_3^+Cl^-$ and an unidentified product were isolated. Similarly, when 2-phenylimino-3-phenylthiazolidine, as a model compound, was hydrolyzed under the same conditions, 3-phenylthiazolidin-2-one and $PhNH_3^+Cl^-$ were identi-

TABLE I



No.	R	Reaction time, hr	Mp, °C	Yield, %	Formula[a]
1	*p*-$C_2H_5OC_6H_4$	0.5	164	38	$C_{11}H_{13}N_5OS$
2	*p*-ClC_6H_4	0.5	176	70	$C_9H_5ClN_5S$
3	n-C_4H_9	2.0	134	6	$C_7H_{13}N_5S$
4	2,6-$Me_2C_6H_3$	6.0	168–170	8	$C_{11}H_{13}N_5S$
5	2,4-$Me_2C_6H_3$	1.0	182–184	57	$C_{11}H_{13}N_5S$
6	*o*-$CH_3OC_6H_4$	1.0	152–154	26	$C_{10}H_{11}N_5OS$
7	*p*-$CH_3OC_6H_4$	0.5	172–174	45	$C_{10}H_{11}N_5OS$

[a] All compds had analyses for C,H,N, and S within 0.4% of the theoretical values.



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