The Chauncey D. Leake Memorial Award Fund

The Chauncey D. Leake Memorial Award Fund was established November 3, 1978, by the Board of Trustees of The Ohio State University with gifts to The Ohio State University Development Fund from friends and associates of Chauncey D. Leake, D.Sc. (Honorary), 1975, Professor Emeritus and former Assistant Dean of the College of Medicine.

All gifts are to be invested in the University’s Permanent Endowment Fund, under the rules and regulations adopted by the Board of Trustees of The Ohio State University, with the right to invest and reinvest as occasion dictates.

The annual income is to provide monies for “The Chauncey D. Leake Award for Excellence in Pharmacology” to an outstanding graduate student in Pharmacology in the College of Medicine or the College of Pharmacy or the College of Veterinary Medicine. The selection of the recipient(s) is to be made on the basis of the most outstanding research paper(s) reporting original research of scientific merit, prepared during the attainment of an advanced degree at The Ohio State University, and submitted in form acceptable for publication. Eligible candidates must be registered as doctoral students in Pharmacology in the Graduate School during the academic year in which they enter the competition. Each research paper is to be accompanied by a biographical sketch of the author’s academic preparation, research background, experience, and future professional goals.

The selection is to be made by a committee composed of five persons, four tenured faculty, including one representative from the Pharmacology programs of each of the three designated colleges and chairman, a tenured faculty member from another college of The Ohio State University or an administrator in the Graduate School, the fifth member is to be a former recipient of the Chauncey D. Leake Award.

Announcement of the Award competition is to be made by the Deans of the above Colleges in January. Papers are to be submitted in April to the offices of the Deans. Selection of the awardee(s) is to be made by the committee in May with announcement of the Award(s) in May or June. The awardee(s) is to make an oral presentation of the research paper at a special seminar or event at a time selected at the discretion of the Dean of the college in which the student is enrolled. It is the responsibility of the Deans of the colleges to arrange for Award Certificates of the appropriate significance. Each Award Certificate is to be signed by the three Deans. The Award Certificate and monetary award are to be presented by the Dean of the college in which the student is enrolled at the special seminar or event selected.
The Chauncey D. Leake Memorial Award Fund (contd)

Should the papers in any given year not be of superior quality, the award(s) will not be made until the next year. This is to be the official award in Pharmacology in the name of Dr. Chauncey D. Leake at The Ohio State University.

Should the need for this award cease to exist or so diminish as to provide unused income, then the income may be used for any purpose whatsoever as determined by the Board of Trustees with preference being given to recommendations from the appropriate administrative official(s) of the University who are then directly responsible for education of students in Pharmacology.

CERTIFICATE

This is to certify that the foregoing is a true and accurate excerpt from the minutes of the Board of Trustees meeting held November 3, 1978.

[Signature]
Edward Q. Morten
Secretary
Three Ohio State University colleges will honor a former faculty member and present an award to a graduate student at a campus program scheduled for 4 p.m. Tuesday (5/27) at the Medicine Administration Center, 370 W. Ninth Ave.

Winner of the 1980 Chauncey D. Leake Award for Excellence in Pharmacology will be announced at the program and will present a paper based on research.

The Colleges of Medicine, Veterinary Medicine and Pharmacy are sponsors of the program, which will memorialize the late Dr. Leake, an internationally known scientist. He served at Ohio State as assistant dean of medicine and chairman of pharmacology from 1955-62. At the time of his death in 1978 he was professor of pharmacology and history of medicine at the University of California Medical School.

Dean Jules B. LaPidus of Ohio State's Graduate School will welcome those attending, and Dr. Marie E. Brittin of the department of communication and award committee chairman will pay tribute to Dr. Leake and lecture on "Humanity in the Health Professions."

Award plaques will be presented also to Dr. Henry G. Cramblett, dean of the College of Medicine and acting vice president for medical affairs; Dean Albert H. Soloway, College of Pharmacy, and, Dean C. Roger Smith, Veterinary Medicine.

-wfr-
ELECTROPHARMACOLOGICAL AND HISTOFLUORESCENT EVIDENCE
FAVORING THE PRESENCE OF TWO DISTINCT MUSCARINIC
RECEPTORS IN SYMPATHETIC GANGLIA

Estelle J. Tsevdos,
Albert C. Humbertson, Jr.
and
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1Supported By: The OSU Development Fund #533768
The OSU Studbaker Fund
And NIDR Grant DE 00291
Running title: Two Types of Muscarinic Receptors

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ABSTRACT

Estelle J. Tsevdos, Albert O. Humbertson, Jr. and Robert W. Gardier:
Electropharmacological and histofluorescent evidence favoring the presence of two distinct muscarinic receptors in sympathetic ganglia.

In the cat superior cervical ganglion, reduction of the muscarinic mediated s-IPSP by pancuronium or gallamine, without affecting the s-EPSP, suggested a selective blockade of muscarinic receptors on the dopaminergic interneuron, labelled $M_1$ (Gardier et al., 1978a). That this action probably was postjunctional followed from the demonstration that the methacholine-induced s-IPSP also was susceptible to gallamine (Gardier et al., 1978b). Continued investigations into the differential postsynaptic nature of the ganglionic muscarinic blockade appear to confirm these early studies. The skeletal muscle relaxants produced a greater inhibition of the s-IPSP generated by electrical stimulation of the partially sectioned versus the intact cervical sympathetic nerve; inhibited the sole development of the s-IPSP, similarly originated, in the presence of pilocarpine; and discriminantly prevented the decrease in fluorescence from small intensely fluorescent (SIF) cells in ganglia treated in vitro with bethanechol. Such results provide further evidence for the concept of pharmacologically distinct muscarinic receptors in sympathetic ganglia.
Koppanyi (1932) demonstrated that pilocarpine injected into the circulation of cat superior cervical ganglion caused contraction of the nictitating membrane. This was the earliest suggestion for the existence of atropine-sensitive ganglionic responses. Extending this work, other investigators were able to demonstrate by surface potential recordings that the introduction of muscarinic agents into the circulation of sympathetic ganglia elicited changes in synaptic events (Jones, 1963; Roszkowski, 1961; Trendelenburg, 1954, 1955). Takeshige and Volle (1964) stated that "the feasibility of finding a blocking agent that differentiates between the atropine-sensitive sites for the hyperpolarization and late-polarization potentials in sympathetic ganglia is evidenced by the failure of pilocarpine to evoke ganglionic hyperpolarization."

Studies published by Gardier et al. (1978a) indicated that certain competitive neuromuscular blocking agents, gallamine and pancuronium, enhanced the muscarinic contraction of the cat nictitating membrane resulting from preganglionic nerve stimulation. Recordings of the superior cervical ganglion surface potential revealed that coincident with the altered nictitating membrane response, the hyperpolarization (slow inhibitory postsynaptic potential (s-IPSP)) was reduced or abolished without any change in the slow depolarization (slow excitatory postsynaptic potential (s-EPSP)). Since the s-IPSP supposedly is mediated through muscarinic stimulation of a dopaminergic interneuron (Eccles and Libet, 1961), the dopamine antagonist, haloperidol, was studied and found to inhibit the muscarinic contraction of the nictitating membrane and enhance the s-IPSP (Gardier et al., 1978a).
Based upon these results, the ganglionic model first suggested by Eccles and Libet (1961) was modified to reflect not only two distinct muscarinic receptors, $M_6$ on the ganglion cell and $M_4$ on the interneuron, but the evident prejunctional (Carlsson and Lindqvist, 1963; Carlsson, 1974) dopamine receptors (Gardier, et al., 1978a). Halcperidol is suggested to have a pre- eminent blocking action on prejunctional dopamine receptors thereby antagonizing the autoinhibitory activity of released dopamine. The net result is an exaggerated s-IPSP.

The ability of competitive skeletal muscle relaxants to effect a reduction in the release of acetylcholine from somatic nerves has been demonstrated in several laboratories (Riker and Okamoto, 1969; Gergis et al., 1972). In order to confirm a postjunctional ganglionic site of action by gallamine, its effect on the response to direct intraarterial injections of methacholine was recorded. In these experiments as in those involving electrical nerve stimulation, gallamine inhibited the s-IPSP without altering the generation of the s-EPSP (Gardier et al, 1978b). To further establish the selective postjunctional nature of the muscarinic blockade by pancuronium or gallamine, studies involving their ganglionic effect in the presence of subtotal section of the cervical sympathetic nerve and on the s-IPSP in the presence of pilocarpine were completed. These results plus those from investigations into their property to protect against bethanechol-induced abolition of ganglionic histofluorescence are the subject of the present report.
METHODS AND MATERIALS

The general animal preparation for in vivo electrical recordings of ganglionic surface potentials followed that previously reported (Gardier et al., 1978a). Briefly, once the superior cervical ganglion and postganglionic fibers were exposed in the cat anesthetized with chloralose and urethane (37.5 mg/kg and 250 mg/kg, respectively), the postganglionic trunk was crushed and suspended on a unipolar platinum electrode. The second platinum electrode was positioned on the desheathed ventral surface of the ganglion with a third electrode inserted into the body wall and used as ground. The first two electrodes were fed into a Tektronix 26A2 DC differential amplifier with $10^6$ ohm input resistance and then into a DC V20 amplifier of an Electronics for Medicine Simultrace V-6 recorder. Ganglionic depolarization was indicated by an upward deflection of the recording beam.

Frequency-Voltage Response Experiments

Experiments with varied frequency and voltage were performed in order to observe any preferential development of the s-IPSP or s-EPSP with electrical stimulation and to establish optimum parameters. The experimental preparation was similar to that described above with tetanic stimulation for 0.5 second at a pulse duration of 0.1 msec. Once threshold values for voltage were obtained (2-4 V), that setting was tested throughout a series of frequencies beginning with 2 Hz. The frequency then was doubled progressively until an upper limit of 128 Hz was reached. The entire procedure was repeated at 4, 6 and 8 V (supramaximal).

Partially-Sectioned Cervical Sympathetic Nerve Experiments

Following the recordings of baseline surface potentials, the cervical
sympathetic trunk was partially sectioned distal to the stimulating electrodes using microdissection techniques. The electrodes were left in place during the surgical procedure in order to avoid any alteration in response due to positional change. At the end of each experiment, the ganglion and connected cervical sympathetic nerve were dissected free, removed, and preserved in 10% formalin. Histological examination of the tissue revealed the extent of surgical sectioning.

Direct Drug Injections

Pilocarpine HCl was injected in a total dose of 10 μM in 0.1 ml volume into the common carotid artery through a fine catheter positioned at the base of the thyroid artery. Supramaximal preganglionic electrical stimuli of 6 V at 0.1 msec pulse duration and frequency of 16 or 32 Hz for 0.5 second were delivered to the cervical sympathetic nerve while the ganglion was under the influence of pilocarpine.

Histofluorescence

Wistar rats of either sex weighing between 250 and 350 g were anesthetized with pentobarbital, 100 mg/kg i.p. With the animal restrained in a supine position, a midline incision from the edge of the mandible to the upper border of the sternum exposed the surgical area. A tracheal cannula was inserted, and further dissection exposed the cervical sympathetic trunk. The nerve was traced cranially to reveal the left superior cervical ganglion located near the bifurcation of the common carotid artery. The ganglion with 2-4 mm of attached postganglionic nerve and 8-10 mm of cervical sympathetic nerve was dissected free and placed in cold oxygenated (95% O2/5% CO2) Krebs-Ringer solution. Using a dissecting microscope, the ganglionic connective tissue capsule was carefully removed from the ganglion and attached neuronal fibers.
The desheathed preparation was placed in 37°C oxygenated Krebs-Ringer bath solution for 30 minutes. Subsequently, the right superior cervical ganglion was removed and treated identically with the exception that the bath also contained 0.5 mMolar bethanechol Cl (Libet and Owman, 1974). Both procedures were repeated in the presence of either pancuronium or gallamine. In the case of pancuronium, the rats were pretreated with a dose of 0.1 mg/kg i.v., while with gallamine pretreatment with 2 mg/kg i.v. was followed by additional in vitro exposure of the ganglia (24 µMolar) concomitant with bethanechol.

After 30 minutes of incubation, the tissue was removed from the bath and placed in isopentane submerged in liquid nitrogen. Each ganglion was dehydrated in a lyophillizer for 5-7 days followed by exposure to formaldehyde vapor and subsequently embedded in paraplast and sectioned at 14 microns. Upon sectioning, each slide, which contained 4-6 sections, was observed under a Leitz Wetzlar #573576 microscope utilizing ultra-violet light from HBO200 W/2 mercury lamp and an oil immersion lens (Leitz Wetzlar #B26469). Sections were visually counted and photographs were taken to insure lack of bias by the observer.

Paired data were analyzed by Student's t test. A P value of 0.05 or less was considered significant (Dixon and Massey, 1969).
RESULTS

Frequency-Voltage Response Experiments

Threshold values for voltage ranged from 2 to 4 V. Once threshold was obtained, it was held constant throughout a series of test frequencies beginning with 2 Hz progressively doubling the values until supramaximal results (128 Hz) were guaranteed. The voltage systematically was increased by adding 2 V, and the entire sequence of frequencies was tested again. Supramaximal voltage was usually 6 V and 8 V always. Figure 1 presents the voltage-frequency-effect curves for the development of the s-IPSP to geometrically progressive increases in stimulation parameters from threshold to supramaximal values.

Partial Section of Cervical Sympathetic Nerve

In this series of experiments, attempts were made to section about half of the fibers in the cervical sympathetic trunk. The amount of transmitter released from brief tetanic stimulation to the preganglionic trunk thus could be markedly reduced and provide some indication as to the competition between acetylcholine and pancuronium for the M₁ receptors. The percentage decrease in s-IPSP produced by pancuronium was significantly greater for sectioned versus intact nerve preparations (Table 1). The s-EPSP, on the other hand, was not affected (Table 2).

Injection of Pilocarpine

When pilocarpine was administered i.a. to the superior cervical ganglion, it caused a long-acting slow depolarization as described by Haefely (1974). Stimulation of the preganglionic nerve during this event resulted in a s-IPSP, and a s-EPSP, that was statistically equivalent to zero potential. The s-IPSP was susceptible to inhibition by gallamine (Figure 2).
Histofluorescence

In vitro exposure of rat superior cervical ganglion to bethanechol reduced fluorescence both in ganglionic neurons and small intensely fluorescent (SIF) interneuronal cells (Figures 3 and 4). Exposure of ganglia to gallamine or pancuronium alone had no effect on the fluorescence both in ganglionic neurons and SIF cells (Figure 5). However, pretreatment of the superior cervical ganglion with gallamine or pancuronium prevented the decrease in SIF cell fluorescence ($p < 0.05$), but not the ganglionic fluorescence resulting from in vitro exposure to bethanechol (Figures 6 and 7).
DISCUSSION

Frequency-Voltage Response Experiments

Frequency-voltage response experiments performed on the ganglion surface potential indicated that there was no preferential development of either of the muscarinic waveforms. In addition, the development of both the s-IPSP and the s-EPSP followed a similar pattern, i.e., there was a sharp observable increase in amplitude at all voltages with increasing frequency until a plateau was reached at approximately 32 Hz. Voltages found to be maximal varied between 4-6 V, with 6 V usually and 8 V always being supramaximal.

The Question of Prejunctonal versus Postjunctonal Effect

The possibility of the muscle relaxants inhibiting the s-IPSP by decreasing acetylcholine release was made more remote by the experiments in which the cervical sympathetic nerve was partially sectioned. In the intact nerve, an optimum dose of pancuronium reduced the s-IPSP by approximately 50%. In comparison, the percent decrease in partially-sectioned preparations with the same dose of pancuronium approximated 75%. Theoretically, if the action of pancuronium were prejunctonal, its percent occupation of prejunctonal sites would be the same, and therefore, the same percentage reduction would be expected in both preparations. The fact that the inhibition was greater in the partially-sectioned nerve indicates a lower acetylcholine titer competing with pancuronium for postjunctonal sites, thus resulting in a greater magnitude of blockade.

In the presence of nicotinic blockade, pilocarpine caused a prolonged slow depolarization comparable in amplitude to the electrically-induced s-EPSP. Preganglionic stimulation during this event produced a
hyperpolarization, essentially devoid of the attendant subsequent s-EPSP. Such was anticipated on the basis of pilocarpine selectively occupying most of the M_e receptors. This not only limited further depolarization, but was expected to exaggerate the hyperpolarization display, and the resultant increase in neurohumor available to the M_l receptors. The fact that gallamine inhibited these "pure" hyperpolarizations contributed another dimension of evidence favoring a selective blockade of the cholinergic receptor on the interneuron.

**Histofluorescence**

Exposure of rabbit superior cervical ganglia to oxygenated buffered 0.5 mMolar bethanechol solution at 37°C indiscriminantly depletes catecholamines from both interneuronal (SIF) and ganglionic neuronal cells evidenced by our own histochemical examination as well as that by earlier workers (Libet and Owman, 1974). In comparison with paired controls, the total number of observable SIF cells decreased upon in vitro treatment with bethanechol. When gallamine or pancuronium was administered prior to and/or simultaneously with bethanechol, the ganglionic cell fluorescence resembled tissues treated with bethanechol, but the number of observable SIF cells was equivalent to the controls. Thus, pancuronium or gallamine protected the SIF cell against catecholamine depletion by blocking the M_l receptors on these interneurons to muscarinic stimulation. Conversely, as expected from the electropharmacological experiments, the amine content of the ganglion cells was depleted, since no blockade was occurring at their muscarinic (M_e) receptors. Ganglia receiving pancuronium or gallamine in the absence of bethanechol displayed a fluorescence qualitatively and quantitatively similar to the controls.
REFERENCES


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<td></td>
<td>64</td>
<td>57.7%</td>
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mean      | 55.6% ± 1.27*** | 10.37** | 78.6% ± 1.16*** |

* n = 3  
** p < 0.05  
*** ± S.E.M.
### TABLE 2

**s-EPSP PERCENTAGE CHANGE IN WHOLE AND SPLIT NERVE EXPERIMENTS FOLLOWING PANCURONIUM (0.1 mg/kg)**

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<td>8 V</td>
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<td>2.26</td>
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* n = 3
** ± S.E.M.
LEGENDS

Figure 1: Frequency-voltage response curve plotted in three separate experiments indicating maximum response between 32-64 Hz. Optimal voltage was registered with 6 V and 8 V always being supramaximal.

Figure 2: Superior cervical ganglionic surface potentials elicited from brief tetanic stimuli (6 V, 0.1 msec duration, 16 or 32 Hz for 0.5 sec) to the cervical sympathetic nerve (control response following nicotinic blockade with chlorisondamine, 2 mg/kg). Following this, pilocarpine (10 μM) was injected i.a. Pre-ganglionic stimulation was repeated during the pilocarpine response alone and coincident with gallamine (2 mg/kg). The data were collected from three separate experiments, averaged, and plotted every 0.2 sec for the first second and less frequently thereafter. I = S.E.M.

Figure 3: Section from an experiment in which excised rat superior cervical ganglion was exposed to Krebs-Ringer solution alone (control). Two types of fluorescence are observable: the general ganglionic neuronal fluorescence and that of the interneuronal (SIF) cell type. The arrow points to a cluster of SIF cells, the diamond-shaped symbol marks a ganglion cell. Filter = K530

Magnification = 240x

Figure 4: In vitro exposure to bethanechol (0.5 mM). The fluorescence both in ganglionic neurons and interneuronal (SIF) cells was significantly depressed. Paired ganglia were used as a comparison between control tissues and bethanechol-treated. Filter = K530

Magnification = 240x
Figure 5: Exposure of superior cervical ganglia of rats to gallamine (2 mg/kg) or pancuronium (0.1 mg/kg) resulted in no change in fluorescent pattern from control. The arrow marks a group of SIF cells, and the symbol marks ganglionic cells.  
Filter = K530  Magnification = 240x

Figure 6: Section of a ganglia treated with gallamine plus bethanechol. Gallamine prevented the decrease in SIF cell fluorescence, but not the ganglionic fluorescence resulting from in vitro exposure to bethanechol.  Filter = K530  Magnification = 240x

Figure 7: Summary of results of histofluorescent studies. Paired data were used throughout. "Drug" in the third column from the left = pancuronium – n = 2, gallamine – n = 4. "Drug" in the fourth column from the left was gallamine. I = S.E.M., * P < 0.05.
CHLORISONDAMINE (2-6 mg/kg)

Amplitude (µV) "p" wave

8.0 Volts
6.0 Volts
4.0 Volts
2.0 Volts
0.8 Volt

n=3

Frequency (Hz.)

0 20 40 60 80 100 120

8 16 32 64 128

Figure 1
CHAUNCEY D. LEAKE MEMORIAL COMMITTEE


LEAKE LECTURERS

Marie E. Brittin, Ph.D. "Humanity in the Health Professions" 1980.
William B. Bean, M.D. "Self as Subject" 1982.

CONTRIBUTIONS

Contributions are to be made to:
Chauncey D. Leake Memorial Fund No. 074063
The Ohio State University Development Fund
Fawcett Center for Tomorrow
2400 Olentangy River Rd.
Columbus, Ohio 43210

Contributions will support future Chauncey D. Leake Awards for Excellence in Pharmacology as well as Leake Lectures and Award events.

THE CHAUNCEY D. LEAKE AWARD FOR EXCELLENCE IN PHARMACOLOGY

Chauncey D. Leake
1896-1978

The Colleges of Medicine, Veterinary Medicine and Pharmacy
Honor
Chauncey D. Leake
Chauncey D. Leake, internationally known pharmacologist and medical historian, was recognized as a scientist, scholar, and humanist. Dr. Leake served as Associate Dean of the College of Medicine and Chair of the Department of Pharmacology at The Ohio State University (1955-1962). He was elected "Man of the Year" in 1962. Dr. Leake received an honorary Doctorate of Humanities Letters from The Ohio State University in 1975.

A pioneer in the discovery of the relationship between chemical structure and biological activity, Dr. Leake’s insights contributed to the current explosion in biotechnology. He urged preventive health care and cautioned young physicians, referring to Hippocratic medicine “…first do not harm.” Dr. Leake inspired students with the thought, “Science is a great adventure.” Concerned about ethics, he wrote, “Lacking directive ethics we cannot apply our scientific knowledge.” He stated, “One of the first lessons to be well-learned in medicine is to respect always the dignity and worth of the individual.” Dr. Leake urged scientists to express themselves well in both writing and speaking. He was president of numerous prestigious organizations including the American Association for Advancement of Science and the American Society for Pharmacology and Experimental Therapeutics.

Dr. Leake inspires us to continue his work with hope, “…how advantageous in every way it would be for health professionals to work together for the benefit of all people.”

Chauncey D. Leake Award for Excellence in Pharmacology

History of the Award

The Leake Award was initiated by Dr. Chauncey D. Leake himself with the support of Dr. Barnard Marks and the Pharmacology faculty in the College of Medicine in 1965. Dr. Joel M. Weaver was the last recipient to whom Dr. Leake presented the Award in 1975. The Award would have ceased, but for the dedicated efforts of Dr. Marie E. Brittin and professional friends in Pharmacology and Medicine. Loyalty and appreciation for Dr. Leake united these professionals to develop the Memorial Award Fund in 1978 which supports the Chauncey D. Leake Award for Excellence in Pharmacology. The Award was expanded to include all graduate students in Pharmacology at The Ohio State University in the College of Medicine, the College of Veterinary Medicine, and the College of Pharmacy.

It is appropriate that Dr. Leake be so honored and that his achievements encourage young researchers in Pharmacology. Dr. Leake expressed one of his goals in stating, “When I die, I want to deserve a respectful salute from the young men who must take my place.”

AWARDEES

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<td>No Awards</td>
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<tr>
<td>1985</td>
<td>Robert L. Stephens</td>
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<tr>
<td>1986</td>
<td>George W. Cox</td>
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<td>1987</td>
<td>Elizabeth M. Kudlacz</td>
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<tr>
<td>1988</td>
<td>Jason Y. Chang</td>
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CHAUNCEY D. LEAKE MEMORIAL COMMITTEE

Members of the original Committee initiating the Chauncey D. Leake Memorial Fund in 1978 were Marie E. Brittin, Allan M. Burkman, Henry G. Crumblett, Robert W. Gardier, Thomas E. Powers, C. Roger Smith, Albert H. Soloway, and Donald F. Westra.

LEAKE LECTURES

The Chauncey D. Leake Lectures are planned to honor Dr. Leake by sharing his goals, and to bring to The Ohio State University mature scholars of outstanding accomplishment to share their expertise with present, and future leaders of scientific endeavors. These lecturers, several of whom knew Dr. Leake, contribute their knowledge, humanity, and vision. As Dr. Leake believed, educational experiences lift the eyes of graduates to horizons less instructed individuals lack the privilege to envision.

CONTRIBUTIONS

Contributions will support future Chauncey D. Leake Awards for Excellence in Pharmacology as well as Leake Lectures and award events. The Leake Memorial Fund must continue to be increased by contributions to assure that Leake Awards will be significant monetarily as well as in prestige and that Leake Lecturers are appropriately compensated.

Contributions are to be made to:
Chauncey D. Leake Memorial Fund No. 074063
The Ohio State University Development Fund
Fawcett Center for Tomorrow
2400 Olentangy River Road
Columbus, Ohio 43210

THE CHAUNCEY D. LEAKE AWARD FOR EXCELLENCE IN PHARMACOLOGY

Chauncey D. Leake
1896-1978

The Colleges of Medicine, Veterinary Medicine and Pharmacy
HONOR
Chauncey D. Leake
Chauncey D. Leake
Biographical Notes

Chauncey D. Leake, internationally known pharmacologist and medical historian, was recognized as a scientist, scholar, and humanist. Dr. Leake served as Assistant Dean of the College of Medicine and Chair of the Department of Pharmacology at The Ohio State University (1955-1962). He was elected "Man of the Year" in 1962. Dr. Leake received an honorary Doctorate of Humane Letters from The Ohio State University in 1975.

A pioneer in the discovery of the relationship between chemical structure and biological activity, Dr. Leake contributed many insights to the current explosion in biotechnology. He urged preventive health care and cautioned young physicians, referring to Hippocratic medicine "...first do not harm." Dr. Leake inspired students with the thought, "Science is a great adventure." Concerned about ethics, he wrote, "Lacking directional ethics we cannot apply our scientific knowledge." He stated, "One of the first lessons to be well-learned in medicine is to respect always the dignity and worth of the individual."

Dr. Leake chaired the committee that established the National Library of Medicine. Renowned as a leader, Dr. Leake was President of the prestigious American Association for the Advancement of Science, as well as President of the American Society of Pharmacology and Experimental Therapeutics, The American Association of History of Medicine, and the Society of Experimental Biology and Medicine.

Dr. Leake developed and chaired the Department of Pharmacology and was Acting Dean at the University of California Medical School in San Francisco. He was Dean of the University of Texas Medical School in Galveston, and helped establish the M.D. Anderson Hospital in Houston. Dr. Leake lectured extensively and served on numerous boards. After serving at The Ohio State University Medical School, he returned to the University of California Medical School as Lecturer in Pharmacology, Lecturer in History and Philosophy of Medicine, and Director of the Research Training Program for medical students.

Prepared by Dr. Marie E. Brittin

CHAUNCEY D. LEAKE AWARD FOR EXCELLENCE IN PHARMACOLOGY
HISTORY OF THE AWARD

The Leake Award was initiated by Dr. Chauncey D. Leake, himself, with the support of Dr. Bernard H. Marks and the Pharmacology faculty in the College of Medicine in 1965. Dr. Joel M. Weaver was the last recipient to whom Dr. Leake presented the Award in 1975. The Award would have ceased, but for the dedicated efforts of Dr. Marie E. Brittin and professional friends in Pharmacology and Medicine. Loyalty and appreciation for Dr. Leake united these professionals to develop the Memorial Award Fund in 1973 which supports the Chauncey D. Leake Award for Excellence in Pharmacology. The Award was expanded to include all graduate students in Pharmacology at The Ohio State University in the College of Medicine, the College of Veterinary Medicine, and the College of Pharmacy.

The Leake Award was developed to encourage doctoral students in Pharmacology to prepare papers of acceptable quality for publication and presentation at national professional meetings. The oral presentation of the Award paper(s) fulfills Dr. Leake’s concept that scientists should express themselves well both in writing and speaking. He believed scientists should build understanding, share their research, be challenged, keep an open mind, and keep learning.

Always encouraging, Dr. Leake was desirous of helping young researchers establish themselves early in their careers as significant contributors to their profession. He urged scientists to work with hope, saying, "...how advantageous in every way it would be for health professionals to work together for the benefit of all people."

It is appropriate that Dr. Leake be honored and that his achievements encourage young researchers. Dr. Leake modestly stated, "When I die, I want to deserve a respectful salute from the young scientists who must take my place."
AWARDEES

Dale P. Svendsen 1965  No Award 1979
Kendall A. Smith 1966  Thomas J. Rimele 1980
Roy A. Filly 1968  M. Michael Snider 1982
Terrence A. Clark 1969  No Award 1983
James A. Thomas 1970  No Award 1984
Albert A. Langley 1971  Robert L. Stephens 1985
David R. Schneider 1972  George W. Cox 1986
James H. Zavec 1973  Elizabeth M. Kudlacz 1987
Joel M. Weaver, II 1975  Timothy A. Esbenshade 1989
Barbara M. Bayer 1976  No Award 1990
Robert G. Meeks 1977  Isabel Lopez 1991
Mary M. Piascik 1978  Asad D. Dalila 1993

LEAKE LECTURERS

Marie E. Brittin, Ph.D. “Humanity in the Health Professions: A Tribute to Dr. Leake” 1980.
William B. Bean, M.D. “Self as Subject” 1982.
Francisco Guerra, M.D., Ph.D. “Pharmacology in Medical History” 1992.

CHAUNCEY D. LEAKE MEMORIAL COMMITTEE

Ronald L. St. Pierre (Chairperson), Marie E. Brittin, Jean D. Dickerscheid, Dennis R. Feller, Popat N. Patil, Jean D. Powers, S. Mark Strauch, Sarah A. Tjoe, and Joel M. Weaver, II.

CHAUNCEY D. LEAKE AWARD
FOR EXCELLENCE COMMITTEE

This committee is responsible for selecting the recipients of the Leake Award. The committee is composed of a former Leake Awardee, a pharmacology professor from another university, a biostatistician, and a senior faculty representative from each college not involved in the direction of the research.