

Texas Society for Electron Microscopy

NEWSLETTER
VOLUME 10, NUMBER 3
SUMMER, 1979
ISSN 0191-3360

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Summer, 1979

ISSN 0191 -3360

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ON THE COVER

Photo courtesy of: Margaret Ann Goldstein, Ph.D., Department of Medicine, Baylor College of Medicine. High voltage electron micrograph of cross section (half-micron thick) of adult rat soleus muscle taken at Boulder, Colorado in the HVEM Laboratory in the Department of Molecular, Cellular and Developmental Biology at the University of Colorado at Boulder. Note the reinforcement of superposition profiles in this highly oriented specimen. A tilt of as little as 3° in these thicker sections results in elliptical profiles of the myofilaments rather than the polka dot pattern shown here. Bundles of thick filaments only, thin filaments only and thick and thin filaments overlapping are seen, due to the registration pattern typical of this skeletal muscle. X 56,000.

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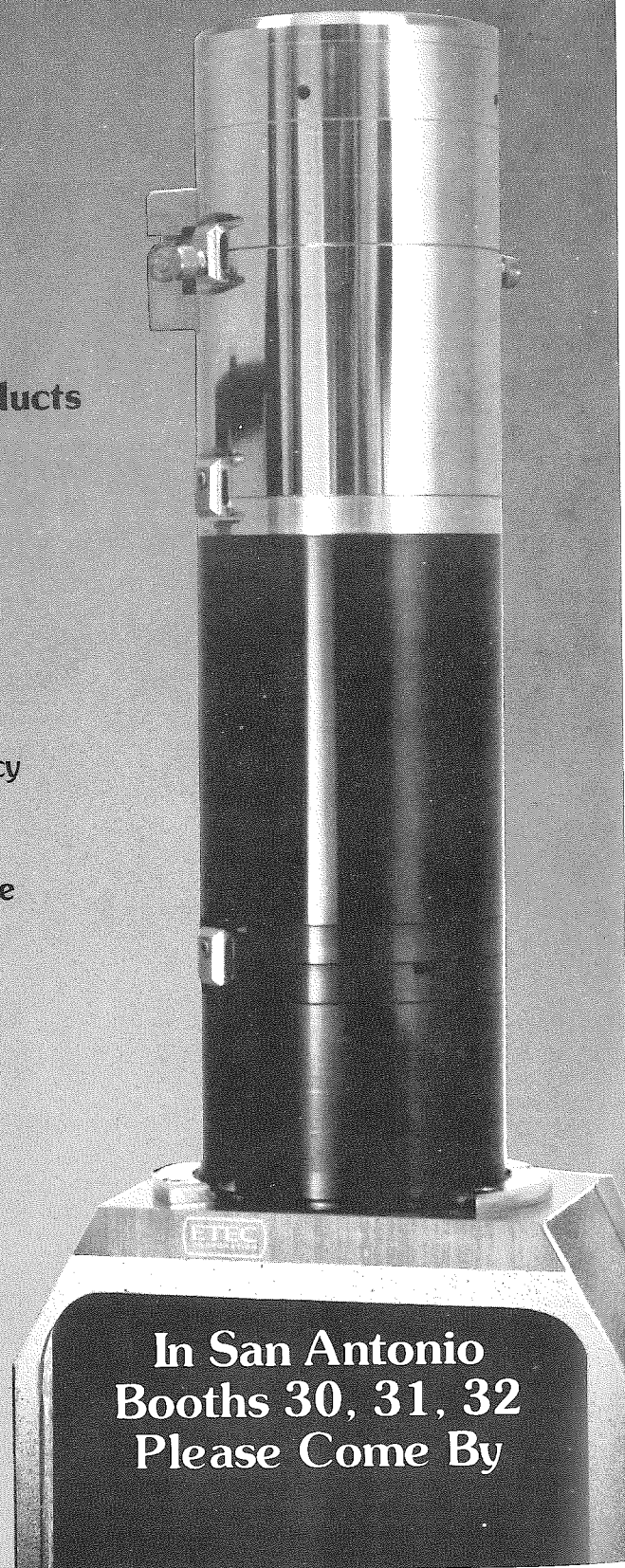


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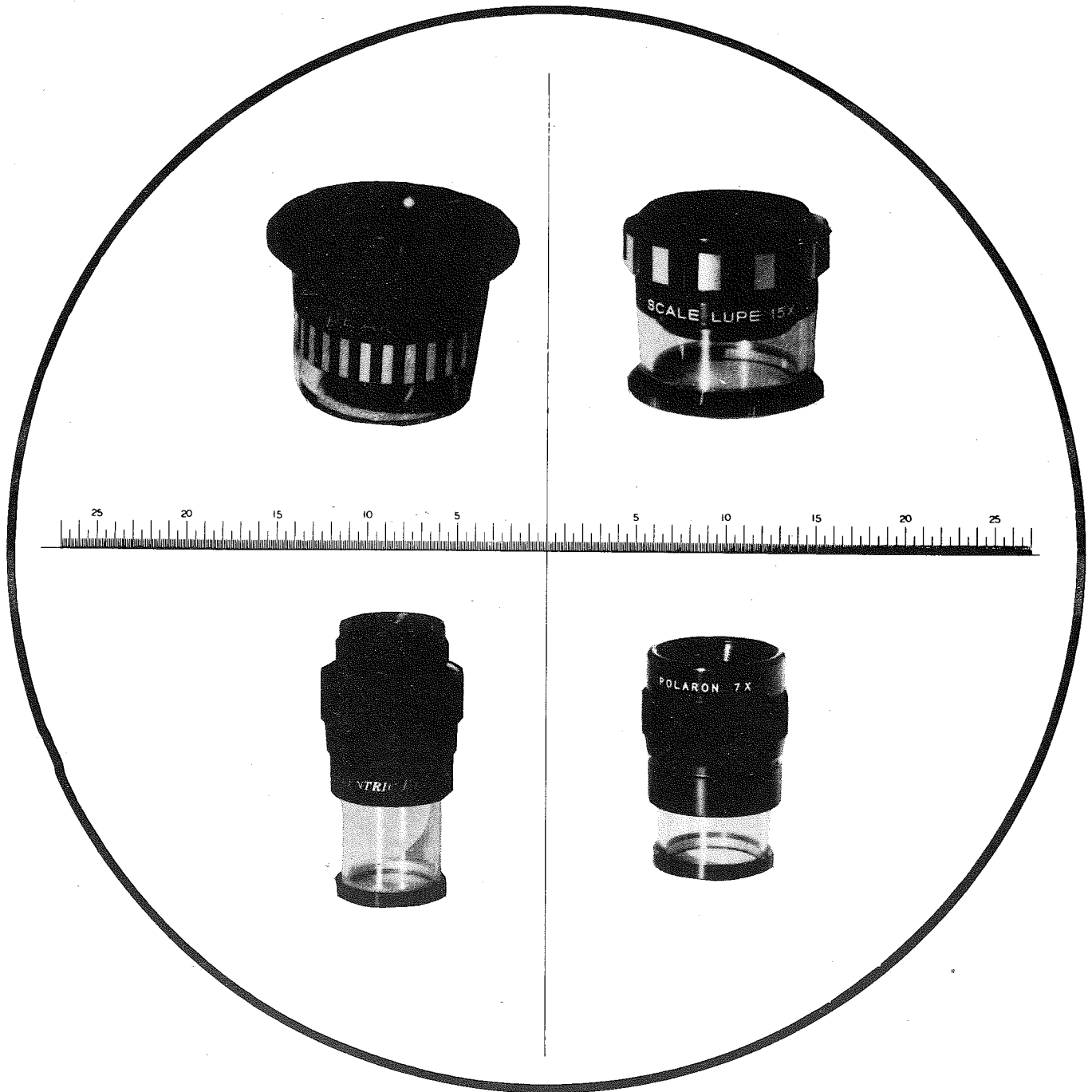
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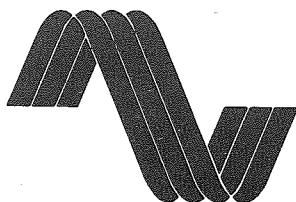


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President's Message

The EMSA Meeting is finally here. Many don't know the history and effort put forth by many TSEM members to get such a meeting held "way out here" in Texas. It all began when Bob Turner promoted the Texas image and got the TSEM council to agree to consider Texas as a possible site. The president of TSEM at that time (1975) was Ward Kischer. Ward corresponded with the EMSA council and issued a formal invitation. It was later confirmed that the 1979 EMSA meeting would be held in San Antonio, Texas. In 1976, President Larry Thurston appointed me as EMSA Local Arrangements Chairman. Presidents Jerry Berlin (1977) and Ivan Cameron (1978) provided the support necessary to insure that TSEM be one of the best hosts ever. Such a meeting will focus a lot of national and world wide recognition on our local society. Recognition of this type is most beneficial and we will reap the benefits for many years. I would personally like to thank these past TSEM Presidents and Councils for their unselfish efforts to insure a successful meeting.

I have learned a lot being Local Arrangements Chairman over the last three years. It takes tremendous effort and time by many people to produce a successful meeting. A national meeting of this size will cost approximately \$120,000. Over half

of this will be paid for by the commercial exhibitors. So again, we owe a lot to these commercial companies which continue to provide us support.

I would also like to take this opportunity to thank the Local Arrangements Committee which was composed of familiar TSEM Members. Paul Baur served as Treasurer; Jerry Berlin, Chairman of Scientific Exhibits; Elaine McCoy, Spouse Entertainment; and Bob Turner, EMSA Executive Council Entertainment. All of these have served in other TSEM capacities in addition to these added responsibilities.

I hope you will all enjoy the annual EMSA meeting. Remember that each of you as TSEM Members will be serving as host for this meeting, so extend a welcome and show that good old southern hospitality to our many guests from all over the world.

See you in San Antonio.

Sincerely,

William B. McCombs, III
President, TSEM

Editor's Comments

Special thanks go to our corporate members and other advertisers who have purchased ads for all three issues of Volume 10. Without them the quality you now expect would simply be impossible. Our special thanks go to Jeol and Zeiss, our corporate members who helped to make the May meeting in Dallas so pleasant.

Our feature articles for this issue range from paraneurons to paraquats. We can still take one more feature article for the next issue which will coincide with the joint meeting of TSEM and LSEM in Houston in 1980. We have not heard from some of you in a long time. When you are reviewing or summarizing for grant-writing, you will find that this is a good time to put together a short article for TSEM. If you do not meet the December 15, 1979 deadline, you can send it in early for the Spring or Fall issues. If you tell me you do not have time to do this when you are writing a grant, I say perhaps you have not allowed yourself enough time to write a good grant.

The response to the reader questionnaire has not been as big as we had hoped. The following answers have been gleaned from 20 questionnaires: most people read everything, including advertisements; most people like the abstracts and feature articles best; most people like the regional news and would like

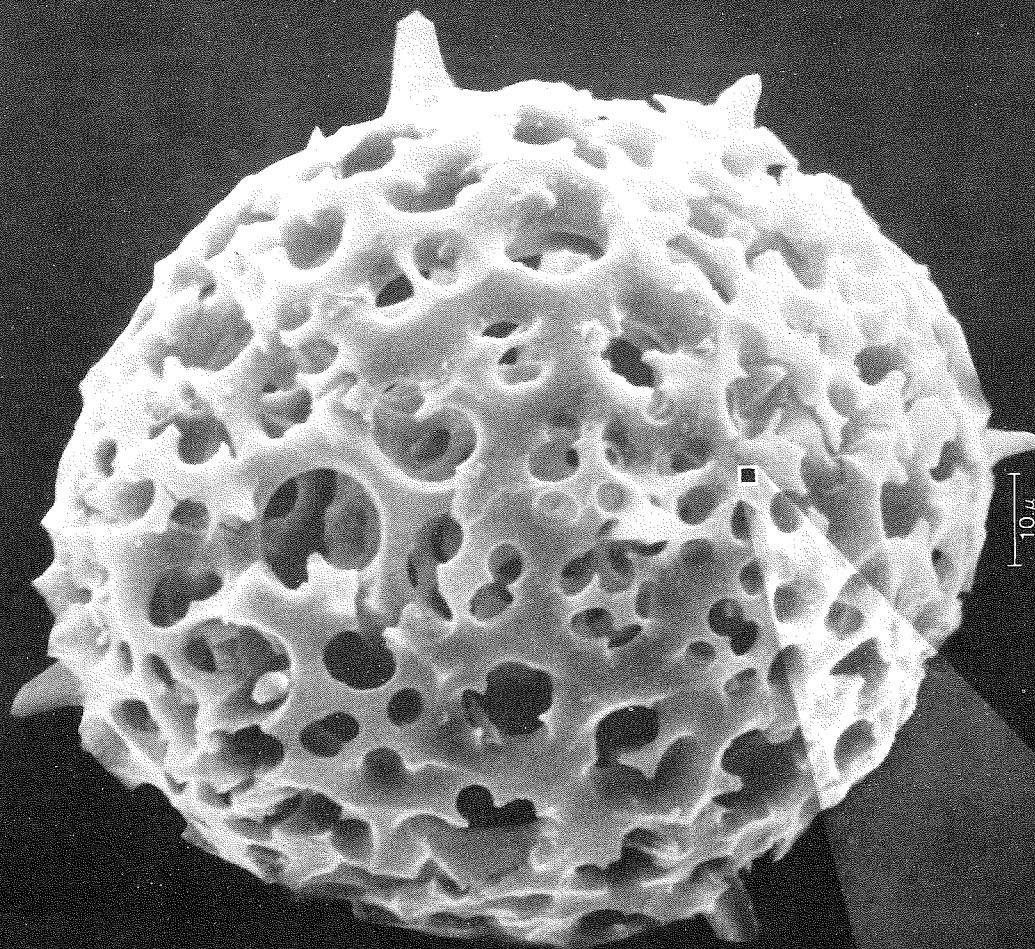
to see this continued but without entries such as "birthdays, gall bladder operations etc.;" most people would like to change the name from TSEM newsletter, but there is no agreement on what name is best; most people would like to see a feature column on tips and procedures that are useful to electron microscopists; most people dislike poorly written letters to the editor.

We are no longer publishing the questionnaire, but you can look back to Vol. 10, No. 1 or 2 if you want to send me one. I appreciate the kind words and the good new ideas, but most of all I appreciate the contributions of articles, news and features. Remember, this is your newsletter.

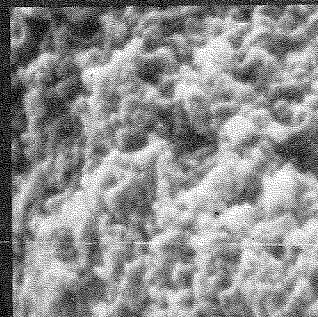
One of our readers has suggested a prize for the best feature article in each volume; i.e. one from each year chosen from the 6 or 7 published. What do you think?

Remember our fall TSEM business meeting will be held in San Antonio on Wednesday, August 15, 1979 from 6:00 - 8:00 P.M. in the La Reina Rooms at the Hilton Palacio Del Rio. There will be chips and beer. We will discuss the newsletter. See you there.

Ann Goldstein
Newsletter Editor



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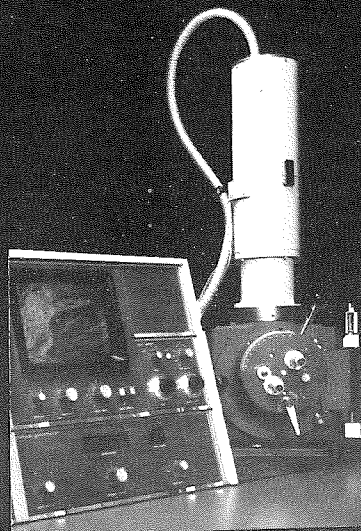
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A History of the Texas Society for Electron Microscopy

By JERRY D. BERLIN
Department of Biological Sciences
Texas Tech University
Lubbock, Texas 79409

In the evening of May 4, 1965, twenty-six electron microscopists attended a meeting on the Rice University campus for the expressed purpose of establishing the Texas Society for Electron Microscopy (TSEM). The purpose of the Society founded that evening, as related by the Bylaws approved at the meeting, was to increase and diffuse the knowledge of electron microscopes and related instruments and results obtained through their use in whatever fields they may be found to be applicable, and to promote fellowship and free exchange of ideas among the members.

The groundwork for this meeting was laid by Lee Rudee and Charles Philpott, both of Rice University. In fact, Rudee had earlier (December 7, 1964) written a letter of inquiry about initiating a chapter of EMSA in Houston to George Cocks, then Executive Secretary of EMSA. From those efforts some 14 years ago evolved the current TSEM, one of the strongest local affiliates of EMSA. Today TSEM has over 500 individual members, approximately 25 corporate members, holds three meetings per year and published three issues of a Newsletter annually.

A brief history of "The First Decade of the Texas Society for Electron Microscopy," anonymously written by Ward Kischer, was published for the 10th Anniversary Meeting held in Houston. This work contains a number of details about the early meetings, policies, scientific contributions, etc. Further, Dr. Donald Duncan (1972) has provided a candid summary of the early days of electron microscopy in Texas. It is not my intent to duplicate the information available in these earlier efforts. Rather, I will concern myself with the more mechanistic events related to TSEM's history. Specifically, I will provide a list of the meetings and the principal speakers at those meetings, a history of the Newsletter including a list of all issues to date, a review of the membership, and, finally, a history of the Bylaws.

Meetings

Table I lists the dates, location and principal speakers for the 43 TSEM meetings to date. Several salient points are apparent. For example, TSEM has heard many of the best electron microscopists in the world. The guest speakers, as well

as (not given) meeting themes, special topics, symposia, workshops, etc., have reflected the advances in the technical aspects of electron microscopy and its application toward extending our knowledge in various disciplines.

The location of our meetings closely parallels the membership rolls in that most of our members and meetings share a close proximity to the Houston area. The one notable exception being our out-of-state treks to New Orleans every other year for the joint meetings with the Louisiana Society for Electron Microscopy (LSEM). The first joint TSEM-LSEM Symposium was in February, 1972 in Ft. Worth. Thereafter, our Winter meetings have alternated between New Orleans and Texas and the 8th Annual Joint Meeting is scheduled for Houston in February, 1980.

Newsletter

A compilation of the various Newsletters is shown in Table II. One reason for listing the volumes and issues is to validate some of the minor problems in the numbering system. Carl Tessmer was instrumental in establishing the Newsletter and was its first Editor. The early issues, i.e., volumes 1-3, were mimeographed and by and large contained meeting announcements and related information. Corporate members play a large role in sustaining many of TSEM's activities. The first acknowledgement of this support was given in volume 1, issue 2 with a listing of 15 corporate members.

The Presidential Message is perhaps the most consistent feature of the Newsletter; a tradition that was started in volume 1, issue 3. This same issue also contained the first of many technique reports in an article entitled "Addenda to Reports on E.M. Techniques from Surgical Pathology Laboratories, Baylor University Medical Center, Dallas, Texas" written by J. A. Lynn. The first electron micrographs were published in volume 2, issue 1. Mitochondrial variability was shown in a full-page plate of 25 figures contributed by various Society members. The first cover electron micrograph was provided by Franklin Bailey and Ward Kischer and showed a Y modulation scan of human hypertrophic scar.

In recent years the Newsletter has contained featured articles and has nearly assumed full-blown journal status. As a result of Ann Goldstein's endeavors the Newsletter has an International Standard Serial Number (ISSN 1191-3360). The importance of this number is that, for the first time, librarians know how to catalogue the Newsletter. Thus, it should start appearing in library shelves across the country.

Membership

That the founding fathers were clairvoyant in recognizing the need for a society such as TSEM is obvious from the early growth in membership. From the 26 hearty souls who convened the first TSEM meeting (May, 1965) the membership grew to 38 within one year and to 221 by May, 1967. During the early 1970's the membership fluctuated around 300. Ward Kischer, the 1975-76 President, initiated a membership drive and the Society membership rolls jumped to approximately 500 where it has stabilized. Our current membership is approximately 510 individual members and 25 corporate members.

The Society has two honorary members: Donald Duncan and Robert Turner. Dr. Duncan, an early pioneer of electron microscopy in Texas, has made many contributions to the field including, as previously noted, a historical treatment of electron microscopy in Texas (Duncan, 1972). Bob Turner's contributions to the Society are numerous. He has held nearly every office in the Society and he alone has the distinctive record of attending every TSEM meeting to date. Bob is currently an EMSA director.

Bylaws

TSEM's Bylaws were originally approved at the organizational meeting May 4, 1965. The original Bylaws stood

the test of nearly a decade before they were revised. In the early 1970's it became obvious that the Bylaws were in need of change to keep pace with the Society and the first revision was undertaken by a committee composed of Bill Brinkley, Bob Turner, and Joe Wood. Their effort was ratified by the membership and published in the Fall, 1974 Newsletter. The major effect of that revision was to increase the number of elected officers and to recognize four types of memberships, viz., regular, student, corporate and honorary. In the summer of 1975, then President Ward Kischer charged a Bylaws committee (consisting of Ann Goldstein, Terry Hoage, Carl Tessmer and Jerry Berlin as Chairman) to revise the Bylaws to more nearly reflect the Society's activities. The committee's endeavor was ratified by the membership and the current Bylaws were published in the Fall, 1977 TSEM Newsletter (volume 8, issue 4). The major thrust of the last revision was to clarify the membership application and admission procedures, provide for an audit of the Society's finances, provide mechanisms to fill any officer vacancy, and included necessary verbiage to enhance the Society's tax-free application to the Internal Revenue Service.

In summary, the Society appears to be accomplishing the original goals set forth some 14 years ago.

Literature Cited

Anonymous. 1975. The first decade of the Texas Society for Electron Microscopy. Texas Society for Electron Microscopy publication, pages not numbered.

Duncan, D. 1972. A short history of development of electron microscopy in the great state of Texas. TSEM Newsletter 3(2): Suppl. 1. (Reprinted 1976. TSEM Newsletter 7(1).)

TABLE I

Date, Location, Speaker and Comments.

May 14, 1965; Rice University, Houston; Organization meeting, approved by-laws.
 October 15, 1965; Statler Hotel, Dallas; Ralph W. G. Wyckoff.
 February, 1966; Hamman Hall, Rice University.
 May 14, 1966; Flagship Hotel, Galveston; A. C. Van Dorsten.
 October 21, 1966; Sheraton Hotel, Dallas; Edwin B. Bradford and John H. Luft.
 March 18, 1967; Holiday Inn, College Station; John Reisner (this meeting was held with the Texas Academy of Science).
 May 27, 1967; Flagship Hotel, Galveston; Gunter Bahr and Garth Thomas.
 October 20-21, 1967; Holiday Inn, Houston; F. S. Sjostrand and A. C. Van Dorsten.
 January 19-20, 1968; Royal Coach Inn, Dallas; John Watson, A. P. Wilska and Kenneth M. Smith.
 May 17-19, 1968; Holiday Inn, San Antonio; M. Moses and A. Crewe.
 October 26, 1968; Hamman Hall, Rice University, Houston.
 January 24-25, 1969; Villa Capri Motel, Austin; Peter Marsh and E. C. Ketler; a workshop meeting with 19 demonstrations.
 May 16-17, 1969; Flagship and Gamboa Cay, Galveston; R. Barnett.
 October 17-18, 1969; Stagecoach Inn, Salado; J. McAlear and A. Crew.
 February 6-7, 1970; Nassau Bay Hotel, NASA; R. L. Steere, G. Cocks, L. L. Ross and G. Thomas.
 April 24-25, 1970; Palacio Del Rio, San Antonio; Dan Friend and Don Fawcett.
 October 5-9, 1970; Shamrock Hilton, Houston; With EMSA.
 February 13, 1971; Flagship Hotel, Galveston; M. Brightman and G. Pappas.
 April 30-May 1, 1971; Six Flags Inn, Arlington; D. Scarpelli.
 October 29-30, 1971; Astroworld Hotel, Houston; D. Pease, S. J. Singer, N. Feder; A. Seligman.
 February 4-5, 1972; Sheraton, Fort Worth; K. Porter and R. Fisher; First joint TSEM-LSEM meeting.

May 19-20, 1972; Holiday Inn, Huntsville; J. P. Revel.
 October 27-28, 1972; Palacio Del Rio, San Antonio; H. Fernancez-Moran and John Watson.
 February 8-9, 1973; Chateau Le Moyne Hotel, New Orleans; Fernandez-Moran, W. J. Humphreys, K. R. Lawless and B. M. Siegel.
 May 25-27, 1973; Jack Tar Hotel, Galveston; R. Barnett.
 September 28-29, 1973; Mayan Dude Ranch, Bandera.
 February 7-9, 1974; Menger Hotel, San Antonio; W. Humphreys, R. Barnett.
 May 24-25, 1974; Texas A&M, College Station; J. Russ.
 October 4-5, 1974; Waterwood, J. H. Brown.
 February 20-22, 1975; Delta Towers Hotel, New Orleans; G. Thomas, J. Watson, and G. Simon.
 May 2-3, 1975; Hyatt Regency, Houston; A. B. El-Kareh.
 October 10-11, 1975; Mayan Dude Ranch, Bandera; B. Stewart and D. Wherry.
 February 5-7, 1976; Menger Hotel, San Antonio; E. de Harven and G. Thomas.
 April 30-May 1, 1976; Cibola Inn, Arlington; H. Ris.
 September 30-October 2, 1972; Ponderosa Motel, Temple; H. D. Sybers.
 February 3-5, 1977; Monteleone Hotel, New Orleans; J. D. Robertson.
 May 5-7, 1977; Villa Capri Motel, Austin; C. Hackenbrock and D. Harling.
 September 23-24, 1977; Rodeway Inn, Arlington; R. Bolender, H. Smith, and E. Underwood; Stereology Workshop.
 February 9-11, 1978; St. Anthony Hotel, San Antonio; M. Ledbetter and Jean-Paul Revel.
 May 5-6, 1978; Southpark Inn, Lubbock; M. Brown.
 September 22-23, 1978; Fredonia Inn, Nachodochus; M. Fuller.
 February 8-10, 1979; Monteleone Hotel, New Orleans; P. Nakane and H. Ris.
 May 4-5, 1979; Marriott Hotel, Dallas; P. Sterling.

TABLE II

Volume (Issue), Date, Comments

*0 (1), Fall, 1967; Carl Tessmer's "first step in our development of a newsletter, one which we hope will serve a function useful to the membership."

- 1 (1), February 22, 1967; Carl F. Tessmer, editor.
- 1 (2), May 3, 1967.
- 1 (3), October 4, 1967.
- 1 (4), December 15, 1967.
- 1 (5), April 1, 1968.
- 1 (6), April 25, 1968.
- 1 (7) +, October, 1968.
- 1 (8), December 16, 1968.
- 1 (9), April 22, 1969.
- 1 (10), June 6, 1969; C. Ward Kischer, editor.
- 1 (11), September 26, 1969; first cover used.
- 1 (12), February, 1970.
- 1 (13), 1 (13) Suppl. 1, March 5, 1970; supplement was report from Leon Dmochowski.
- 2 (1), Summer, 1970; First semi-hard paper cover; first Plate Page; societies symbol first appeared on cover.
- 2 (2), Winter, 1970.
- 2 (3), 2 (3) Suppl. 1, Spring, 1970; Suppl. Current Electron Microscope Installations in Texas.
- 2 (4), Fall, 1971.
- 3 (1), Winter, 1971.
- 3 (2), Spring, 1972; only two issues of volume 3 published.
- 3 (2) Suppl. 1, Spring, 1972; "A short history of development of electron microscopy in the great state of Texas" by Donald Duncan.

4 (1), Fall, 1972.

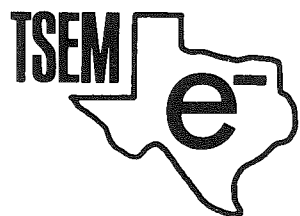
4 (2), Spring, 1973; A Winter, 1972 issue was not published; this would correspond to the joint TSEM-LSEM meeting held in New Orleans that year and at this time the publication of the Newsletter coincided with meetings.

4 (3), Fall, 1973; Ivan Cameron, editor; William A. Pavlat, managing editor.

- 5 (1), Winter, 1974.
- 5 (2), Spring, 1974.
- 5 (3), Fall, 1974.
- 6 (1), Winter, 1975.
- 6 (1), Spring, 1975; Note two 6 (1)'s were published.
- 6 (2), Fall, 1975; Ron Gruener, editor.
- 6 (3), Winter, 1976.
- 7 (1), Spring, 1976.
- 8 (1), Fall, 1976; Robert A. Turner, editor.
- 8 (2), Winter, 1977.
- 8 (3), Spring, 1977.
- 8 (4), Fall, 1977.
- 9 (1), Winter, 1977.
- 9 (2), Spring, 1978.
- 9 (3), Fall, 1978; Ann Goldstein, editor.
- 10 (1), Winter, 1979.
- 10 (2), Spring, 1979.

*0 (1) was not originally numbered; volume 1 does not appear on the originals, only the issue number was used until the start of volume 2.

+ (7) was not numbered on the original.



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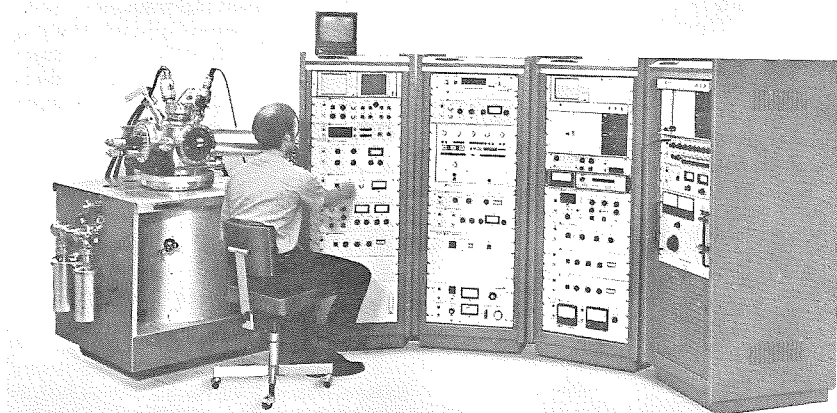
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X-ray Photoelectron Spectroscopy (XPS)

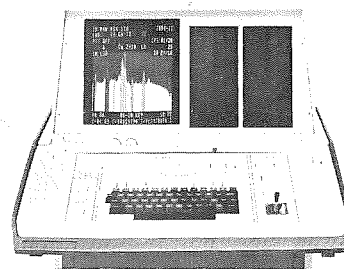
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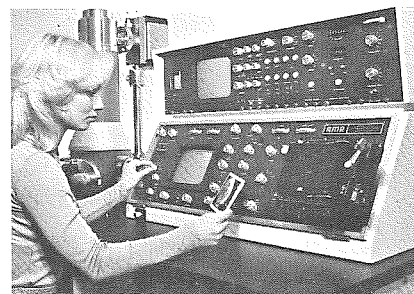
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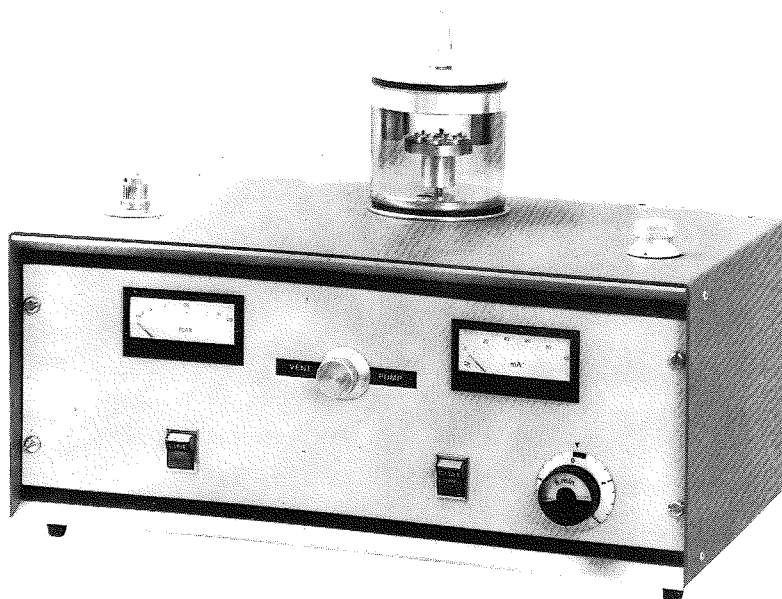


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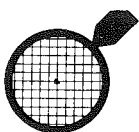
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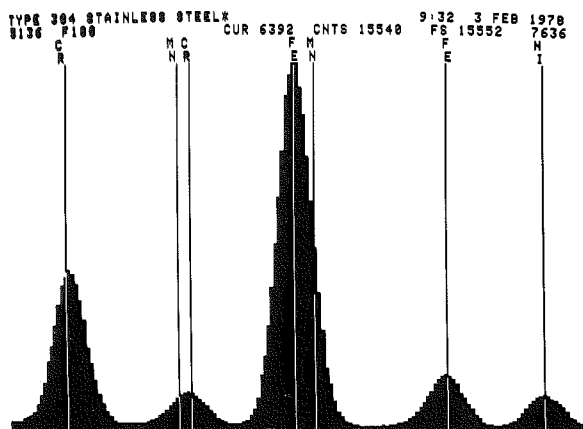


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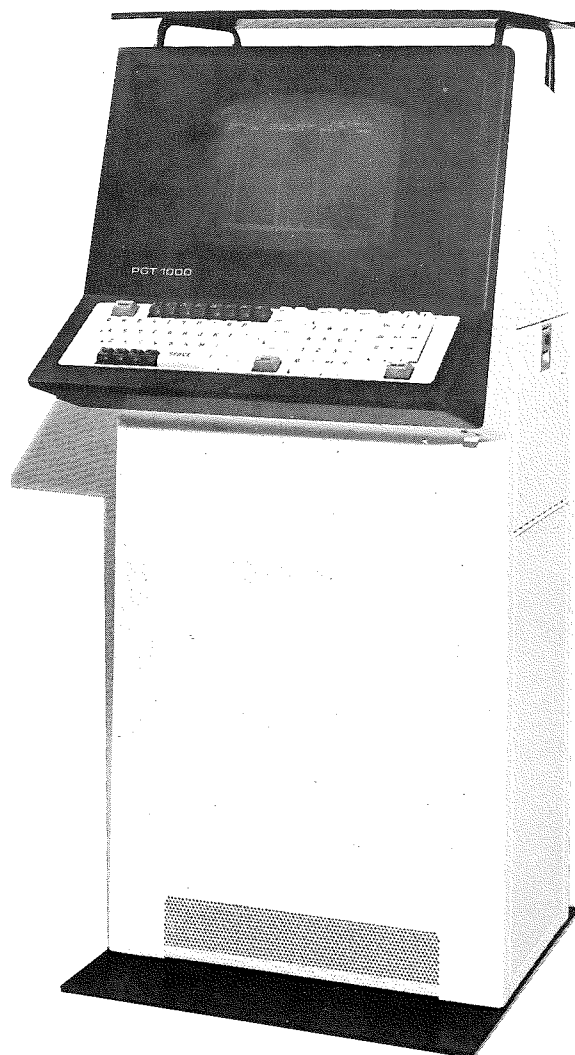
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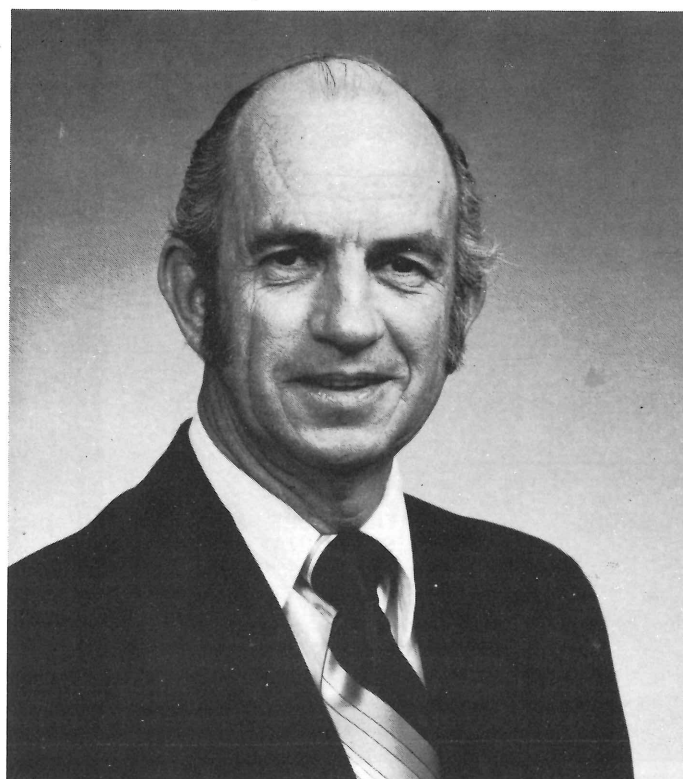
Date: February 8, 1979

Subject: Honorary Membership in TSEM

I propose that Robert A. Turner be offered an "Honorary Membership" in TSEM for the following reasons:

1. His attendance at all TSEM meetings in the past.
2. His constant, dependable, and honorable support of the society during his tenure as: Past-President, Secretary, Newsletter Editor, Director of EMSA, Local Arrangements Committees, etc.
3. His willingness to serve the society by raising funds for past meetings, publications, etc.
4. His continued interest in Electron Microscopy.
5. The respect he has engendered from his fellow society members as evidenced by his recent election to an Electron Microscopy Society of America Directorship.

Paul S. Baur
Paul S. Baur, Ph.D.



ROBERT A. TURNER

Robert A. Turner

Honorary Member

Texas Society for Electron Microscopy

Native Texan. Born in Ennis, Texas.

Graduated from San Jacinto High School, Houston, Texas, 1943.

Served four years in Naval Seabees during World War II.
Honorable discharge in Spring 1946.

Graduated 1950 with B.S. degree from Texas Tech.

Graduated 1953 with M.S. degree from Sam Houston State Teachers College

Taught High School and College Science Courses 1953 through 1958.

Enter EM Field (1958-60) M.D. Anderson Virology Department.

Accepted position as Technical Director of E.M. Lab in Pathology at U.T.M.B. Galveston, Texas 1960-1970.

Accepted Director of EM Section in Pathology, Scott and White Clinic, 1970 to the present.

Charter member of TSEM, May 1965.

Secretary, TSEM, 1968-71.

President-Elect, TSEM, 1972-73.

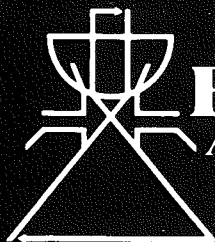
President, TSEM, 1973-74.

Newsletter Editor, TSEM, 1976-78.

Local Society Director on EMSA Council, 1975-78.

Local Arrangements Chairman for American Society for Cell Biology 33rd Annual Meeting, San Antonio, Texas, November, 1978.

Elected Biological Director on EMSA Council, 1979-1981.

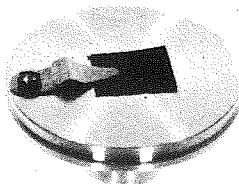


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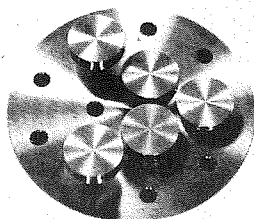
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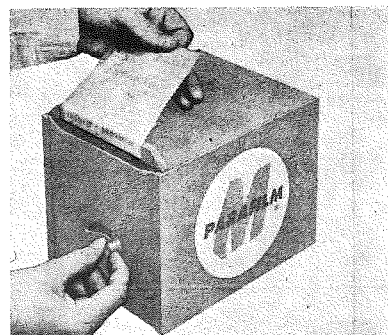
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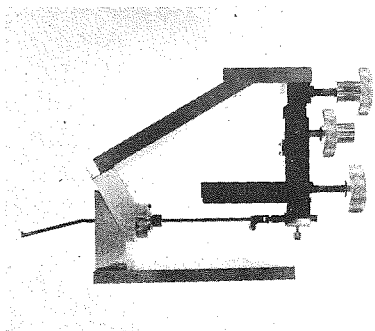
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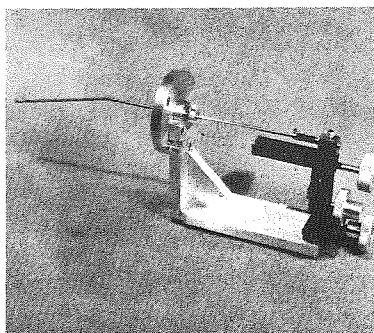
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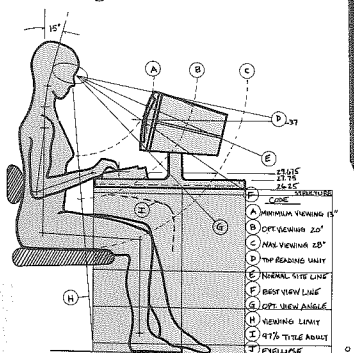
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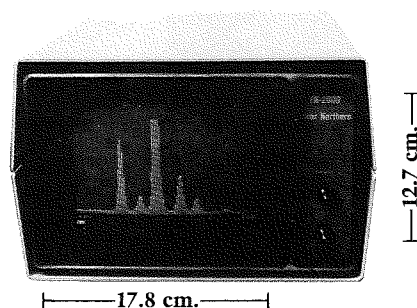
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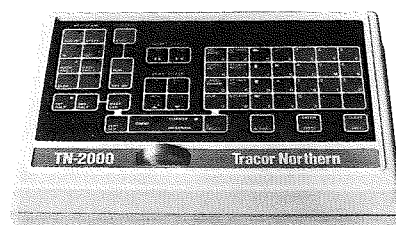


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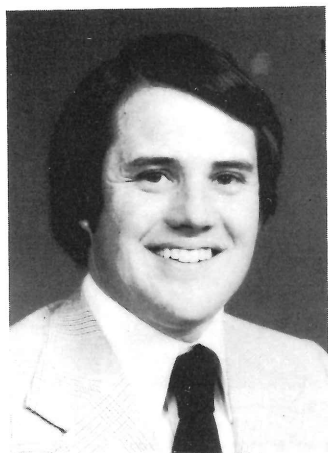
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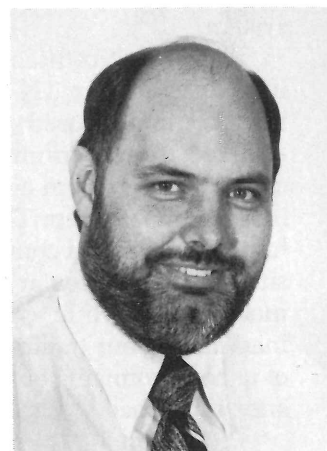


PRESIDENT

WILLIAM B. McCOMBS, Ph.D., is currently Chief of the Microbiology Department at Scott and White Clinic and Hospital in Temple, Texas. A native of New Mexico, Dr. McCombs received his Doctor of Philosophy degree from Texas Tech University in 1975. Dr. McCombs is certified as a Specialist in Microbiology by the American Society of Clinical Pathologists and a Specialist in Public Health and Medical Microbiology by the American Academy of Microbiology. He has served as Local Arrangements, Secretary, and President-Elect of TSEM and is presently Chairman of Local Arrangements for the 1979 EMSA Meeting being held in San Antonio.

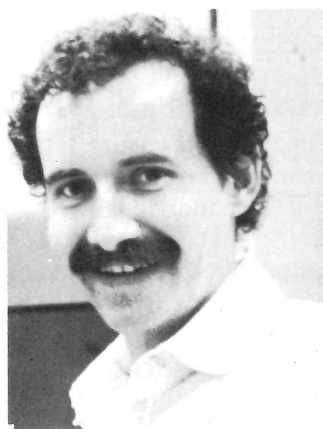
PRESIDENT ELECT

PAUL SCHUHBAUR, JR., Ph.D., is an Assistant Professor of Cell Biology, Department of Human Biological Chemistry and Genetics, U.T.M.B., and Chief, Division of Cell Biology, Shriners Burns Institute, Galveston. Born November 22, 1938, he was raised in Cairo, Illinois. He received his B.S. and Ph.D. degrees (Plant Pathology) from Texas A&M University. He is married and has three children. Research interests are histology, cytology (TEM-STEM), topology (SEM), and microchemistry (EDS) of wound healing tissues and EM procedural techniques. Member of TSEM since 1965. Hobbies include woodworking, art metals, motorcycles, hunting and fishing.



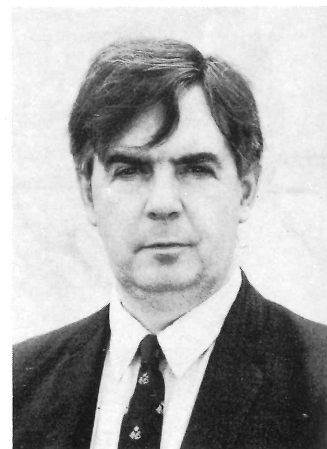
SECRETARY

JOHN T. HANSEN, Ph.D., is an Assistant Professor, Department of Anatomy, The University of Texas Health Science Center at San Antonio. He received his Ph.D. at Tulane University School of Medicine under the direction of Dr. Robert D. Yates. Current research interests include the function and ultrastructure of the arterial chemoreceptors and other peripheral paraneurons. Techniques employed include electron microscopy, fluorescence histochemistry, freeze-fracture and scanning electron microscopy. Teaching responsibilities include medical gross anatomy and graduate cell biology.



TREASURER

BRUCE MACKAY, Ph.D., is an Associate Pathologist and Chief of E.M. Service at M. D. Anderson Hospital. Graduated in medicine, University of Edinburgh, Scotland, 1956. Ph.D., anatomy, University of Edinburgh, 1961. Assistant professor, anatomy, University of Iowa, 1961-1963. Residencies in surgery and pathology, Vancouver General Hospital, Vancouver, Canada, and University of Washington, Seattle. Came to Houston in 1969. Major interests include surgical pathology, tumor pathology, and electron microscopy in oncology. Fellow College of Pathologists. Member EMSA Education and Program Committees. Ex-program chairman TSEM. Married to Joy Rosslyn Mackay, M. D. Catriona is five and Christopher is two. Kirstie joins us in September. U.S. citizen. Presbyterian. Temperate.





NEWSLETTER EDITOR

MARGARET ANN GOLDSTEIN, Ph.D., is Associate Professor of Experimental Medicine in the Department of Medicine at Baylor College of Medicine, Houston, Texas, and has a joint appointment in the Department of Cell Biology. Ann is a native of Texas and received a B. A. in Biology (1965) and a Ph.D. in Cell Biology (1969) from Rice University. Married to Alexander Goldstein, Jr., M.D.; has one son. Her major research interest is structural analysis of muscle proteins. Current research deals with optical diffraction and reconstruction of mammalian Z bands and with microtubules and components of the cytoskeleton in cardiac and skeletal muscle. Charter member of TSEM.

PROGRAM CHAIRMAN

CHARLES W. MIMS, Ph.D., is an Associate Professor of Biology at Stephen F. Austin State University in Nacogdoches. Ph.D. degree in Botany-Mycology received in 1969 from the University of Texas at Austin. In addition to serving as Program Chairman from TSEM, he has also been active in the Mycological Society of America. Dr. Mims served one term on the governing board of the Society and has also served as Chairman of the Committee on Research Grants and Publications, as well as Chairman of the Teaching Committee. His research interests relate primarily to the ultrastructure of plant pathogenic fungi. Along with Dr. C. J. Alexopoulos, he is co-author of the Third Edition of *Introductory Mycology* to be published in September of 1979 by John Wiley and Sons.

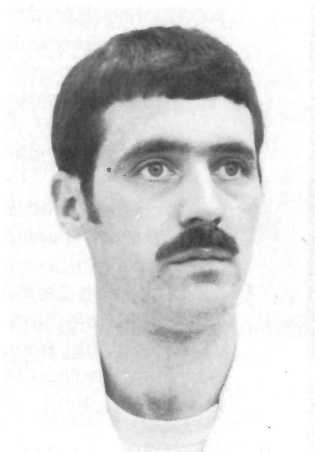


PROGRAM CHAIRMAN-ELECT

LEON MCGRAW, JR., Ph.D., is Professor of Biology at Lamar University in Beaumont, Texas, where he has been employed since 1967. He is in charge of the cellular biology program and electron microscopy laboratory. Leon received his M.S. and Ph.D. from Texas A&M University in 1962 and 1967. He has been honored at Lamar with a "Regents' Merit Award" in 1972 and as Beta Beta Beta "Science Teacher of the Year" in 1977. He served as Historian for TSEM in 1977 and 1978.

GRADUATE STUDENT REPRESENTATIVE

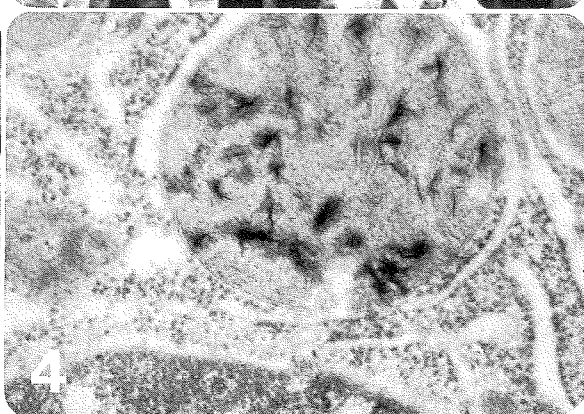
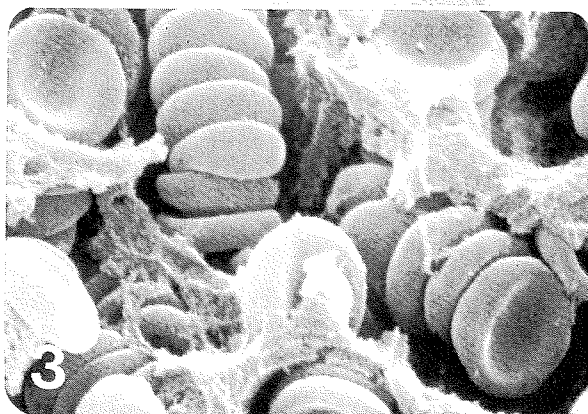
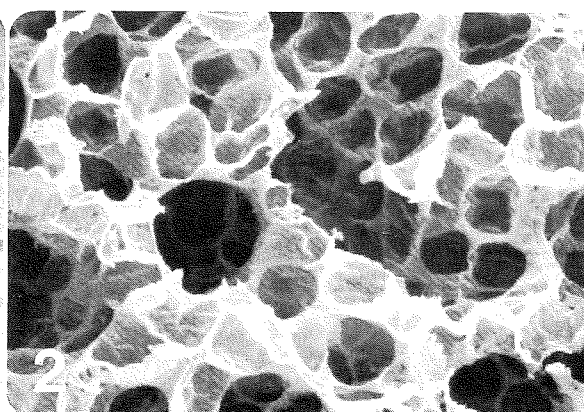
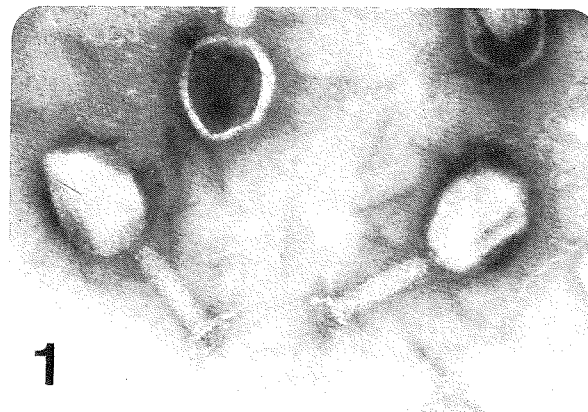
THOMAS M. DREIER is a Ph.D. student under the direction of Dr. E. L. Thurston at the Electron Microscopy Center Department of Cell Biology at Texas A&M University, College Station, Texas. Tom was born and raised in Minnesota, receiving a B.A. from Mankato State College in 1972. He served as a research assistant for two years at the U.S. Army Virology and Immunology Laboratory, Ft. Baker, California. He entered Texas A&M University in 1975 and received a Master of Science in 1977. Population dynamics and community structure of aquatic bacteria are his primary research interests.



IMMEDIATE PAST PRESIDENT

IVAN L. CAMERON, Ph.D., is Professor in the Department of Anatomy, The University of Texas Health Science Center at San Antonio. He received his Ph.D. in Anatomy from U.C.L.A. in 1962. His major research interest is in cell reproduction using morphological and chemical approaches. Current research deals with intracellular concentration of sodium and other elements as related to mitogenesis and to oncogenesis using X-ray microanalysis. He has been a member of TSEM since 1969. He has been newsletter editor and program chairman in TSEM.

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Optical Diffraction and Reconstruction of Electron Images

by

M. A. Goldstein, J. P. Schroeter* and R. L. Sass°

Department of Medicine and Cell Biology,

Baylor College of Medicine

Departments of *Chemical Engineering and °Biology

Rice University, Houston, Texas

Optical diffraction is an interaction between coherent light and ordered structures. In this article we will deal specifically with optical diffraction patterns obtained with a helium-neon laser and electron micrographs or photographs in a device called an optical diffractometer. The purpose of this paper is to illustrate with examples from muscle the principles and uses of optical diffraction techniques.

Optical diffraction techniques (masking, optical transforms and Fourier analysis) have been available since the 1940's. As early as 1938, Sir Lawrence Bragg suggested the use of optical analogues to aid in the interpretation of x-ray diffraction pictures (19). With the development of better x-ray devices and computer programs for simulating diffraction patterns, the use of optical transforms in the materials sciences has declined.

There has been increasing interest in optical diffraction of electron micrographs of biological material. A number of very good articles and reviews are now available on diffractometers (1,9,11,14), practical aspects of optical diffraction of electron micrographs (10,11,15), and image filtering and reconstruction (3,4,5,13,15). In the 1950's, mercury vapor lamps were used to generate coherent light for optical diffraction patterns. Long exposure times were required for biological materials such as viruses. When lasers became commercially available, Klug and co-workers made use of a helium-neon laser as a source of intense coherent light for optical diffraction of electron micrographs (12). Complete diffractometers are now available from several manufacturers.

OPTICAL DIFFRACTION IN MUSCLE RESEARCH

In studying muscle structure we go from the whole muscle constructed of organized cell arrays to the light microscope level of sarcomeres, to the electron microscope level of macromolecular complexes of filament lattices to the arrangement of individual component proteins and finally, to the substructure of the pure proteins themselves. Diffraction techniques have been used at all of these levels. The earliest x-ray diffraction patterns from living muscle led to the concept that the whole muscle can

be viewed as a paracrystalline array. There has been tremendous interest recently in analyzing sarcomere lengths of contracting muscle by laser diffraction of single fibers, small bundles of muscle, and small whole muscles. The use of electron micrographs of muscle makes possible study of real dimensions on the order of 5-50 nm. Values obtained by x-ray diffraction for living intact structures can be compared with optical diffraction patterns of fixed and sectioned material. Both techniques can be used on isolated pure crystals and paracrystals of muscle proteins under a variety of experimental conditions. The packing arrangement of the molecules can give clues about the substructure of the pure protein molecules.

Optical diffraction techniques have been applied to muscle protein structure by Caspar and co-workers (2), Hanson and co-workers (8) and Ohtsuki and Wakabayashi (17). Since a pure crystal has a unique diffraction pattern, the optical diffraction pattern is a "fingerprint" for this molecular structure. Caspar, Cohen and Longley (2) compared optical diffraction patterns from electron micrographs of fixed, embedded and sectioned tropomyosin crystals. They noted that shrinkage along a particular axis occurred during the dehydration of tropomyosin crystals in preparation for electron microscopy. Tropomyosin, like a number of muscle proteins can also form paracrystals (ordered arrays of tropomyosin filaments). Caspar and co-workers compared negatively stained paracrystals with sectioned material and determined the relationship between the various net structures, collapsed nets and the "striped" paracrystals.

O'Brien, et al. (16) and Ohtsuki (17) showed that *in vitro* actin paracrystals differ in structure when varying amounts of troponin-tropomyosin complex are co-crystallized with actin. Hanson and co-workers (8) showed the relationship between periodicities along the I band filaments observed in electron micrographs and the structure of actin-tropomyosin-troponin paracrystals. Thus in muscle we have been able to go from pure crystals to crystalline mixtures, from *in vitro* structures back to the intact muscle and in some cases on back to the living muscle.

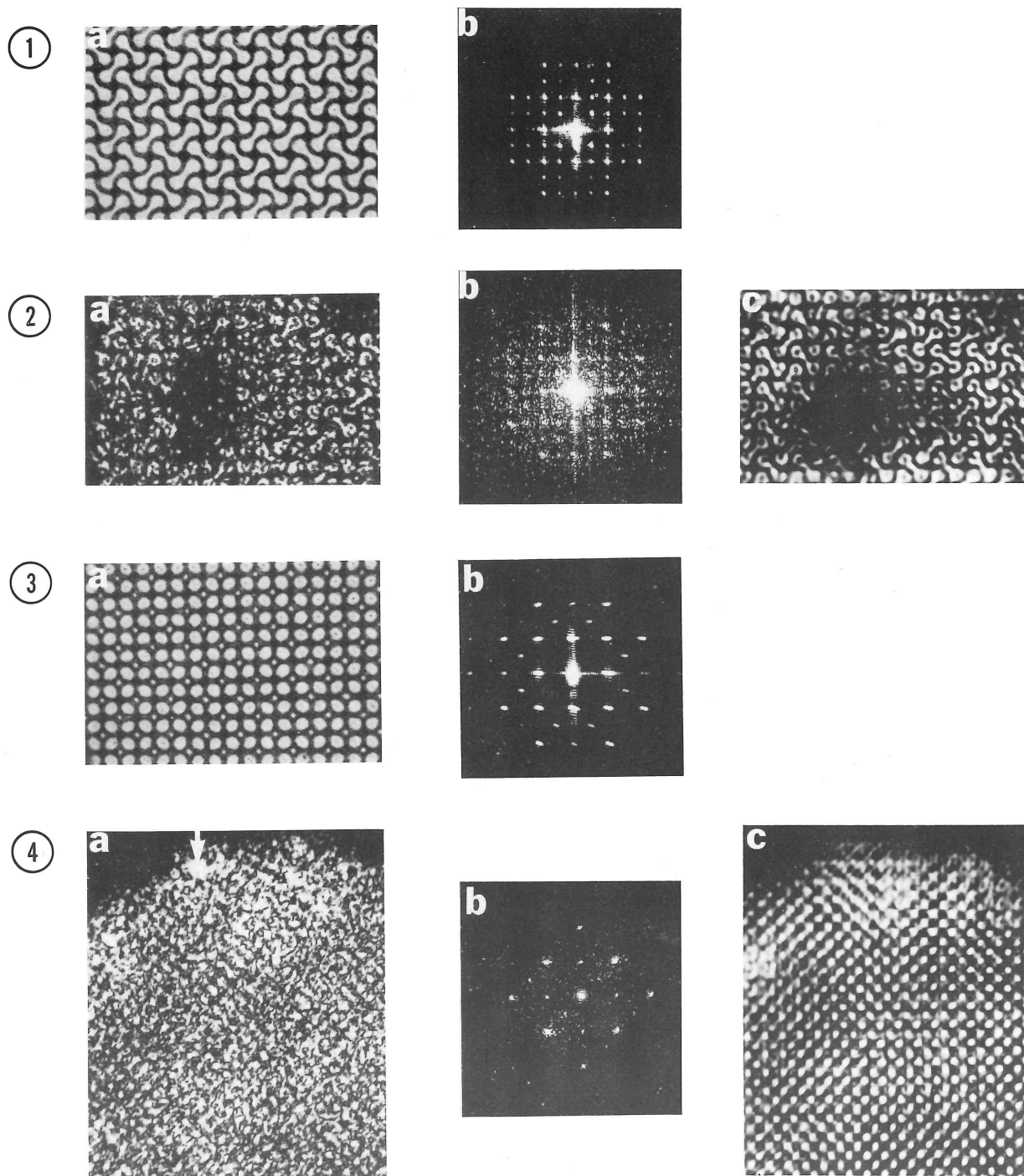


FIGURE 1 — (a) A reconstructed image of a drawing of a regularly repeating basket weave pattern associated with cross sections of Z bands of cardiac and skeletal muscle. This image was recorded on Polaroid film from a transparency of the drawing mounted in the optical system operating in the reconstruction mode. (b) An optical diffraction pattern generated by the model drawing used in Fig. 1(a). This pattern was photographed at the transform plane of the optical system.

FIGURE 2 — (a) A reconstructed image obtained from a drawing similar to that used in Fig. 1 but which has been obscured by an overlay of irregularly distributed ink blots. (b) The optical diffraction pattern of this drawing. Note that it is composed of a regular lattice of diffraction spots comparable to those of Fig. 1(b) plus a randomly distributed background pattern forming the optical transform of the irregular overlay. (c) An optically filtered, reconstructed image of the object pattern used to generate Fig. 2(a). Filtering was accomplished by masking at the optical transform plane. Only the regular lattice seen in Fig. 2(b) was allowed to pass through the mask to form this image. Note that the regular basket weave pattern is now clearly visible except in one area of the image which was originally very heavily obscured by the overlay.

FIGURE 3 — (a) A reconstructed image of a model drawing of the small square pattern associated with cross sections of Z bands of skeletal muscle. (b) An optical diffraction pattern generated by this model drawing.

FIGURE 4 — (a) An unfiltered reconstructed image of the area under the arrows shown in the electron micrograph in Fig. 5. In this image the muscle protein material is dark. The background appears as a semi-regular pattern of small white areas of varying density and size. (b) The optical diffraction pattern associated with this image. Note the regular lattice pattern indicative of an ordered structure as well as the general "noise" pattern due to amorphous scattering matter. The regular features of this pattern compare well with the model lattice pattern in Fig. 3(b). (c) This figure is a filtered image of the same area of the micrograph used to form the image in Fig. 4(a). Filtering at the optical transform plane was done in a manner similar to that used in the model experiment outlined in Fig. 2. Only the regular lattice pattern of the optical transform was passed through the filter mask. Note the enhanced appearance of the ordered structure when compared with either the unfiltered image in Fig. 4(a) or the original micrograph in Fig. 5.

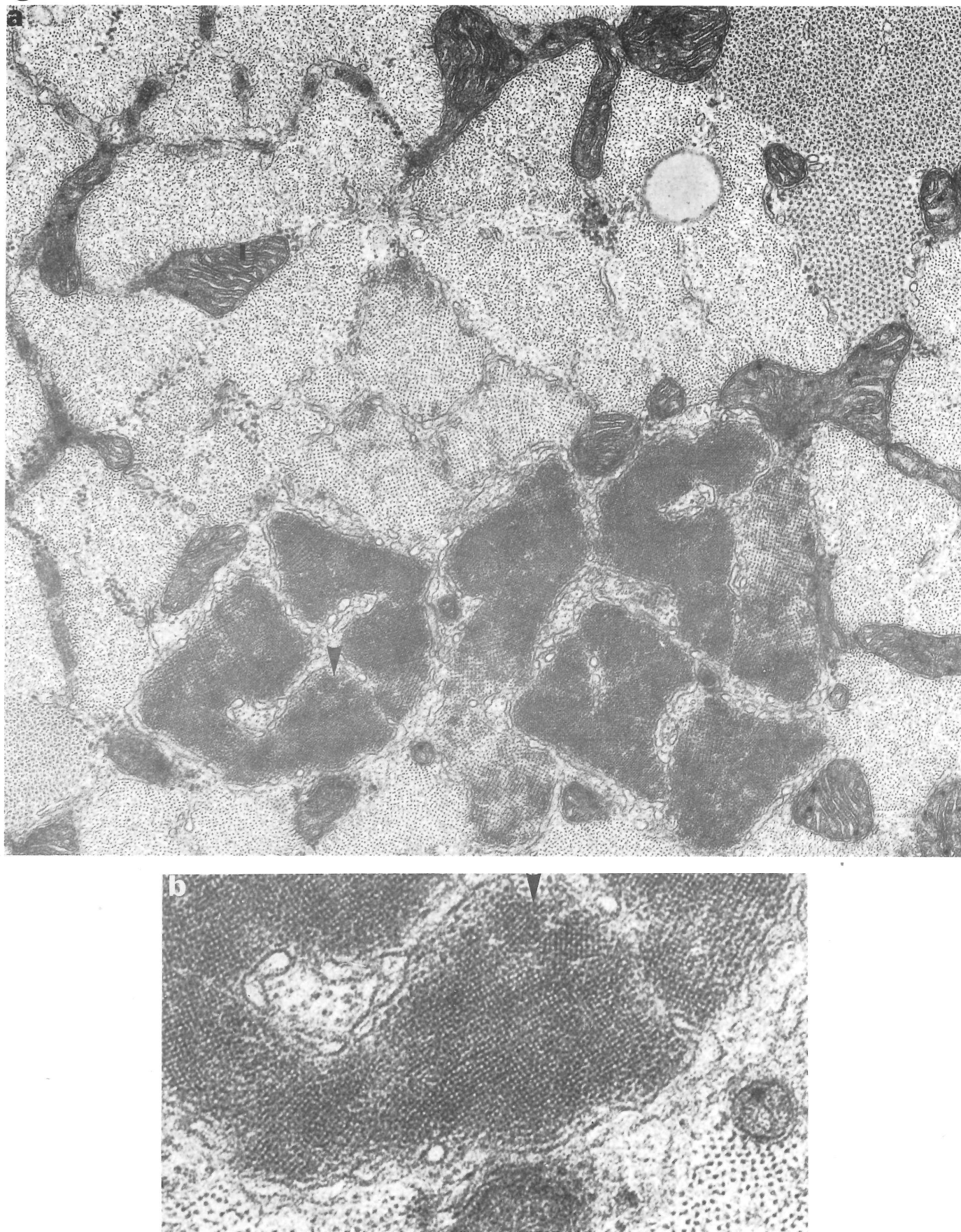


FIGURE 5 — (a) A cross section of adult rat skeletal muscle (soleus) showing a number of Z bands. X 35,000. (b) The region used for the optical diffraction pattern shown in Fig. 4(b) is enlarged to show the details of the Z lattice. X 94,000.

INSTRUMENTATION

The principles of diffraction are identical for all types of radiation. Formation of the diffraction pattern and instrumentation may vary for electron diffraction, x-ray diffraction and optical diffraction, but the usual components are a source of coherent energy, the ordered structure (specimen) to be studied, and a detector. In electron diffraction and optical diffraction additional components are a lens system for illuminating the specimen, and a lens system for observing the diffraction pattern. For optical reconstruction an additional lens system is added to retransform to the original image with or without modification (called filtering or masking).

Diagram 1 shows the principal components of our optical diffractometer in the diffraction mode. The exact position of the components is not diagrammed to scale. We use a magnetized flat table and a series of magnetic bases to mount the various components. The beam from a 35 mW helium-neon laser is directed through a collimating network to the relevant area of the electron micrograph and the resulting diffraction pattern is focused on a ground glass screen. The spatial filter is an objective lens and pinhole device which focuses the beam on a 25 micron pinhole to eliminate undesirable noise in the laser beam. The collimating lens produces a parallel beam about 1.5 cm in diameter. The adjustable aperture reduces the beam size to correspond to the area of interest on the micrograph. The diffraction pattern is magnified by the first and second focusing lenses. All the lenses are high quality simple lenses. Their exact alignment in the diffractometer is crucial.

The relationship between the ordered repeat distance, d , of the micrograph and the distance between the resultant diffraction spot and the central spot is: $D = \frac{LT}{2md}$ ($D < L$), where T is the wavelength of the incident light, L is the camera length of the system, D is the diffraction spot distance and m is the magnification of the micrograph. In actual practice the system is calibrated with a grid of known spacing, d , and the conversion factor is obtained by measuring the value of D on the diffraction pattern for this grid. We align all lenses and calibrate the diffractometer each time we scan a series of micrographs. After a pilot study, the best approach is to scan a few micrographs, with high resolution, high contrast, optimal orientation and minimal background noise due to stain graininess, selected from several hundred micrographs. Positives from electron micrographs on contrast lantern slides are used to improve the contrast of the original EM negative and to spare the precious negative. Care must be taken to insure adequate exposure of these plates or valuable information will be lost. The plates are mounted between optical flats and immersed in xylene, which has a refractive index similar to the emulsion and the glass plate. This step minimizes diffraction artifacts due to emulsion irregularities. The diffraction patterns are recorded on PN/55 film and are measured using a Vernier diffraction reader. The distance between two corresponding diffraction spots located on either side of the central spot is measured. This procedure eliminates the error introduced in measuring the distance from a diffraction spot to the diffuse central spot. Information about the ordering distances, the angular positions of the diffraction spots and

the relative intensities of the spots makes it possible to make inferences about the structure of the material in the corresponding micrograph. An advantage of working with electron micrographs is that one can return to images of the structures in question.

OPTICAL RECONSTRUCTION AND FILTERING

When operating in the image reconstruction mode, we record the optical diffraction pattern on Polaroid P/N 105 film closer to the second lens so that an optical diffraction pattern of a convenient size is obtained. The Polaroid positive is trimmed to fit a cardboard mount for a 35 mm slide. The diffraction pattern is then placed in a Nikon particle projector to obtain an enlarged image of the pattern. A thin brass plate mounted on a microscope stage is moved with each corresponding change in position of the cross-hairs on the enlarged image in the Nikon projector. Thus, holes can be drilled through the plate to correspond to the positions of the central laser beam and all the diffraction spots recorded from the original periodic structure.

The size of the mask hole is important (3). A hole smaller than the actual diffraction spot size imposes a regularity that may not be justified. On the other hand, too large a hole increases the local averaging distance for the filtered reconstructed image. For example, in our recent study of the cardiac Z band (7) the hole sizes for the filtering masks were adjusted to be larger than corresponding spot diameters. The large diffraction spots were about 0.25 mm in diameter, each hole size was 0.63 mm in diameter except for the main beam hole which was twice this size. For a unit cell of 24 nm and a corresponding distance between mask holes of 1.3 mm, the local averaging distance is calculated to be 0.63 mm/1.3 mm ($1/24 \text{ nm}^{-1} = 1/48 \text{ nm}^{-1}$ or the inverse of $2 \times 24 \text{ nm}$. Thus averaging occurs over two unit cells.

The filtering mask (i.e. the thin blackened brass plate with holes) is placed at the Fourier transform plane (the plane of the diffraction pattern). A mirror is used to deflect temporarily the resulting optical diffraction pattern to check registration between the mask and the diffraction pattern. Precise alignment of the mask is crucial. Usually a different mask has to be used for each corresponding image of the lattice structure. Enhancement of the regular structure occurs when only light rays due principally to the regular portion of the structure are allowed to pass through the retransformation lenses. This reconstructed image can now be viewed on a screen or recorded on film. Focusing of the reconstructed image requires precise adjustment of the second retransformation lens. A comparison of the optically filtered image to the original EM image and to the unfiltered reconstructed image (no brass plate at the Fourier transform plane) shows how the regular features of the structure can be enhanced by this technique. Figures 1-5 illustrate applications of these techniques.

HOW TO GET INTERESTING RESULTS

Any regularly repeating structure in electron micrographs is a potential diffraction subject. First the appropriate magnification for studying the average repeat

distance is determined (we used a distance of approximately .1–.2 mm). Two methods of approach are commonly used. In both cases a micrograph of the lattice parallel to the lattice planes is obtained. This electron micrograph, a projected image of a tissue section with finite thickness, becomes the diffraction subject. One method is to take randomly oriented sections and tilt a given specimen at known orientations to the electron beam (18). The transition from detectable order in the diffraction patterns from complex patterns containing 4 or more orders to a single pair of diffraction spots, to no detectable pattern and back again to patterns of increasing complexity. There is a tilting range for the visibility of a lattice plane in a tissue section and the range depends on the section thickness. If a crystal is tilted with an angle θ , the effective lattice plane spacing is $d \cos \theta$, where d is the actual lattice plane spacing. Diffraction patterns of a tilting series represent a set of inclined sections through the reciprocal lattice. The three dimensional structure of the ordered structure is obtained from the construction of the reciprocal lattice.

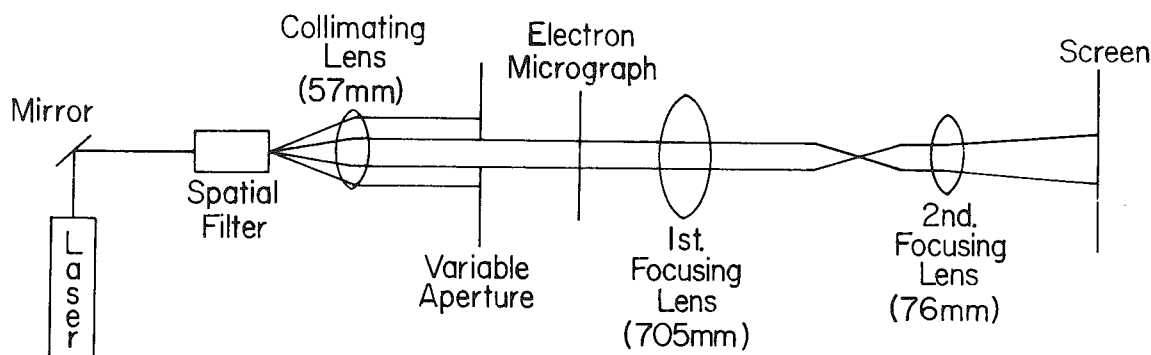
The other method, which works quite well in a highly oriented structure such as muscle, is to take specific sections of known orientation (e.g. transverse to the myofibril axis as in Figure 5) (6). The main advantage here is that one is working in real space. The diffraction patterns confirm or deny the suggested orientation (6). They reveal additional structural features that are seen on a second inspection of the micrograph (6). Three dimensional models can then be constructed in real space and their two dimensional projection used as diffraction subjects (7).

Optical diffraction is a powerful tool for analyzing and comparing, quantitatively, periodic structures in electron micrographs. Periodicities which are difficult or impossible to solve by direct viewing can be studied. Optical diffraction bridges a gap between x-ray diffraction and electron microscopy. Further use of the optical transforms involves masks for optical reconstruction, two dimensional drawings, and three dimensional models. In using optical diffraction techniques one is forced to go from three dimensional real structures to two dimensional projections as in EM images, to two dimensional drawings, as in visual presentations for teaching. This process of going from one visual representation to another stimulates

thought and aids in the development of intuition so vital for understanding complex biological structures.

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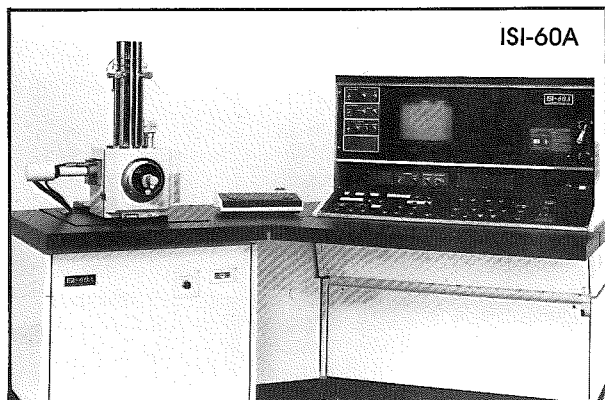
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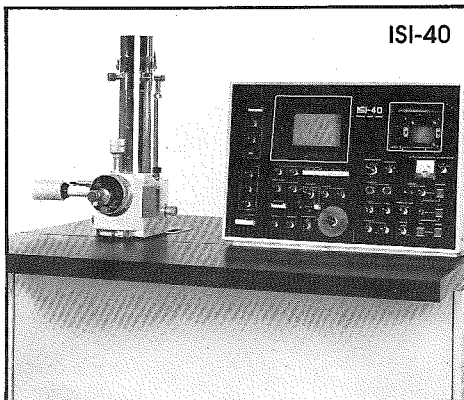
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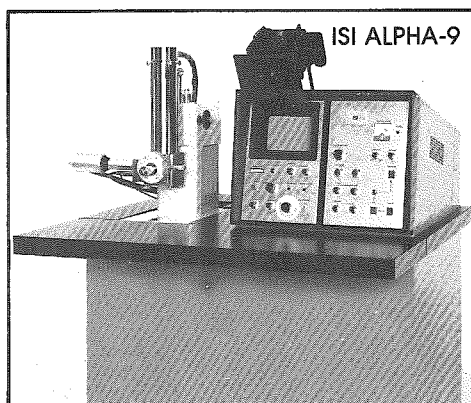
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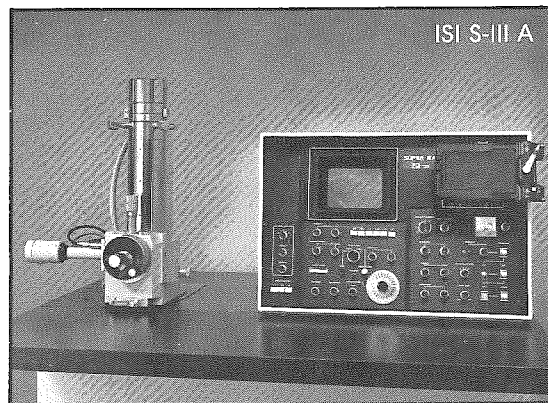
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The Paraneuron Concept

by

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"However, it . . . became apparent that both the nervous system and the endocrine system were not entirely distinct and that each utilized 'methods' of the other."

M. X. Zarrow, 1964

INTRODUCTION

The distinction between endocrine and nervous tissues is no longer as clear-cut as once envisioned by the light microscopists. With the advent of specific histochemical techniques and the electron microscope, it has become increasingly more difficult to classify cells into purely endocrine or nervous categories. Early histologists first encountered this dilemma during their studies of the adrenal chromaffin cells which are classically endocrine in nature yet derivatives of the neural crest. These cells release epinephrine and norepinephrine into the circulation as hormones while similarly derived postganglionic sympathetic neurons release identical substances as neurotransmitters. Originally, light microscopists could distinguish neurons from endocrine cells by the specific staining characteristics of the nervous elements. However, further studies with the electron microscope confused the issue by demonstrating that the morphological differences between some endocrine cells and neurons perceived at the light microscopic level are less than obvious at the ultrastructural level. For example, neurofilaments and neurotubules, although associated by prefix with the nervous system, are found in many cell types. Likewise, Nissl bodies, diagnostic of neuronal cell bodies, are merely stacks of cisternae of rough endoplasmic reticulum and free polysomes, and are common organelles in most cells. Similar confusion arose over the secretory products of endocrine cells and neurons. As alluded to above, are epinephrine and norepinephrine hormones or neurotransmitters? Without restricting one's self to rigid definitions or classifications, it is clear that they can be both, depending upon their site of action and/or mode of transport.

THE PARANEURON CONCEPT

The "paraneuron" concept originally evolved as an attempt to achieve a better classification of a large number

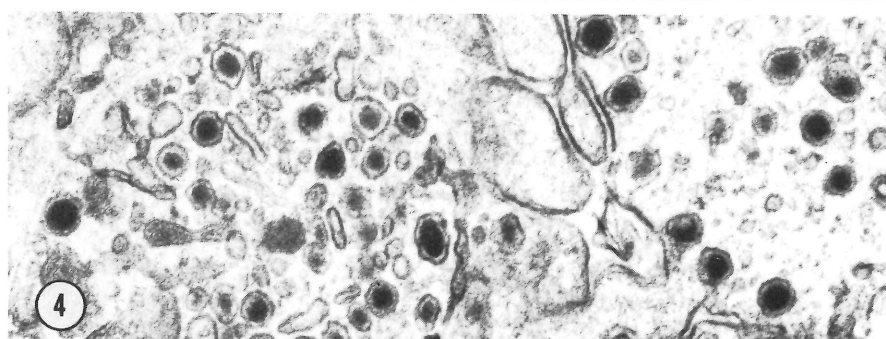
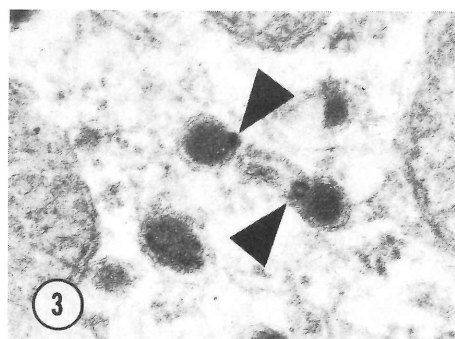
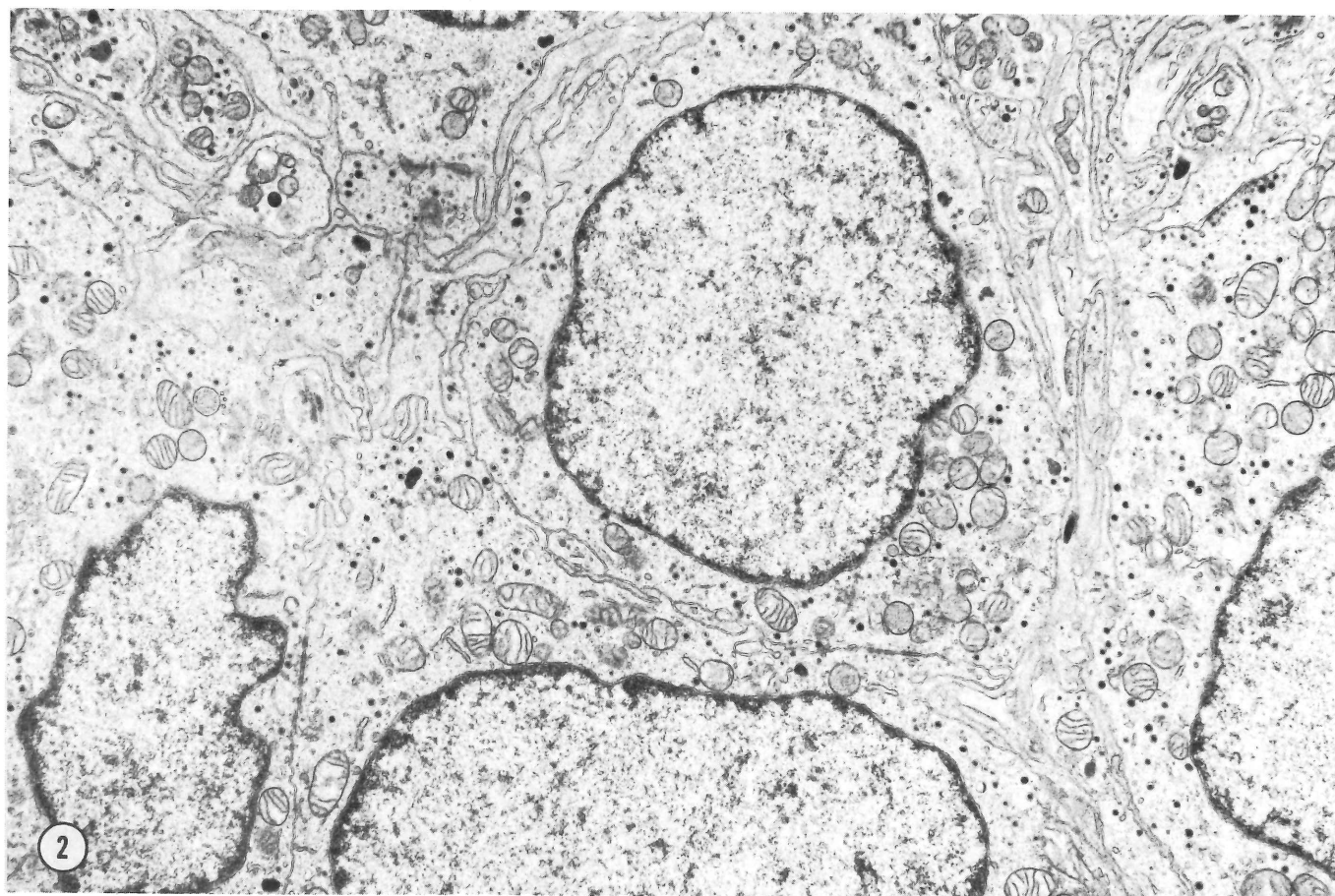
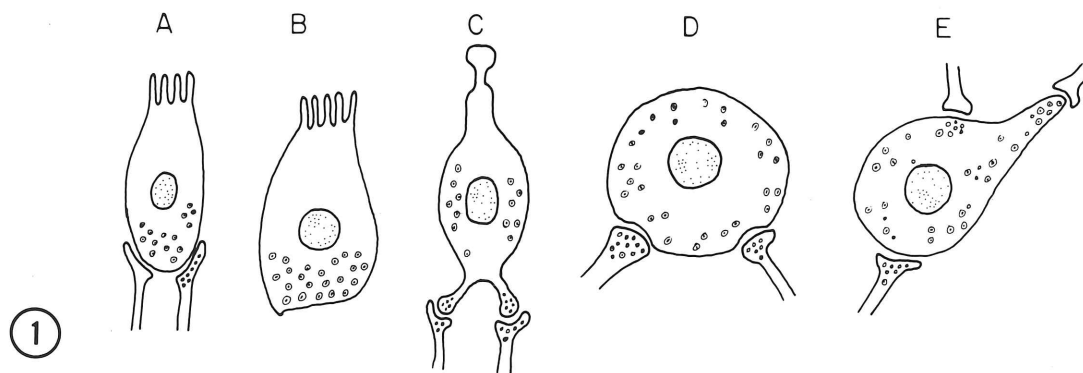
of specialized cells which share similar ultrastructural, metabolic and/or functional characteristics with endocrine cells and neurons. In 1975, on the occasion of the International Symposium on "Chromaffin, Enterochromaffin and Related Cells" held in Gifu, Japan, Tsuneo Fujita and Shigeru Kobayashi proposed that paraneurons are cells which meet the following criteria:

- (1) A cell which is able to produce (a) substance(s) identical with or related to neurotransmitters or suspected transmitters, and (b) protein/polypeptide substance(s) which may possess hormonal actions.
- (2) A cell which possesses synaptic vesicle-like and/or neurosecretion-like granules.
- (3) A cell which is recepto-secretory in function.
- (4) A cell whose origin is common with neurons, i.e. neuroectodermal origin.

Recently, Fujita and Kobayashi (1979) have eliminated the last criterion since only a few paraneurons have been shown conclusively to originate from neuroectoderm. As presently conceived, the paraneurons appear to be of multiple origin and their ontogenic differentiation may be either of a divergent variety from a single stem cell or of a convergent variety from different stem cell lines.

TYPICAL PARANEURONS

Members of the paraneuron family are listed in Table 1. Paraneurons may be of several different functional varieties (Figure 1). Some paraneurons are primarily sensory in nature and structurally are similar to the gustatory cell of taste buds and the basal-granulated cell of the bronchial epithelium (Figure 1). Other sensory paraneurons are mechanoreceptors such as the hair cells of the inner ear or Merkel cells of the skin. The retinal photoreceptor cells, also sensory paraneurons, are



FEATURE 1 — Diagram of several typical paraneurons: (A) basal-granulated cell of the bronchial epithelium; (B) gastroenteric endocrine cell; (C) avian pinealocyte retaining its ancestral photoreceptor feature; (D) adrenal chromaffin cell; and (E) carotid and aortic body chief cell.

FIGURE 2 — Electron micrograph of several carotid body chief cells. Note large nucleus and dense-core vesicles. X 20,200.

FIGURE 3 — The demonstration of calcium in carotid body vesicles as an eccentrically located electron dense particle (arrowheads). Tissue unstained. X 87,500.

FIGURE 4 — Aortic body chief cell vesicles. Note the presence of small electron-lucent vesicles and larger vesicles which exhibit an electron dense core. X 61,200.

phylogenetically homologous with the pinealocyte, although mammalian pinealocytes are similar structurally to adrenal medullary cells.

Other paraneurons may be internuncial in nature possessing both an efferent and afferent innervation (Figure 1). Typical examples of internuncial paraneurons are the small intensely fluorescent (SIF) cells of various autonomic ganglia and, perhaps, the carotid body chief cells.

The last category of paraneurons are endocrine in nature. Typical examples include the gastroenteric endocrine or basal-granulated cells, which open directly to the gut lumen with specialized microvilli (Figure 1). Other endocrine paraneurons such as some mammalian pancreatic endocrine cells and the adrenal chromaffin cells possess an efferent innervation.

ULTRASTRUCTURAL CHARACTERISTICS

All paraneurons have several basic ultrastructural features in common. Paraneurons typically are characterized by a large central nucleus, well developed Golgi and endoplasmic reticulum, cytoplasmic vesicles or secretion granules, and a receptor site which often takes the form of cellular processes, cilia or microvilli (Figure 2). The secretion vesicles or granules are believed to be the storage sites of the neurotransmitter-like or polypeptide substances secreted by the paraneuron. ATP (and other adenine nucleotides) and calcium probably are contained in the secretion vesicles as well. In fact, calcium can be

demonstrated in the vesicles of the carotid and aortic body chief cells as an electron dense particle after appropriate fixation procedures (Figure 3) (Hansen and Smith, 1979). Secretion of paraneuronic vesicular contents probably occurs by the energy and calcium dependent process of exocytosis (Nagasawa, 1977). Paraneurons also can possess more than one variety of cytoplasmic vesicles. For example, some paraneurons, and many neurons, possess both small 40-60 nm electron-lucent vesicles and larger 90-200 nm vesicles, some exhibiting an electron-dense core (Figure 4). Some paraneurons clearly contain more than one transmitter or polypeptide substance within a single cell.

PATHOLOGICAL CONSIDERATIONS

As pointed out by Fujita and Kobayashi (1979), the paraneurons concept is based upon the fact that there is no clear boundary between many nervous and non-nervous tissues. "Neurons and paraneurons are continuous, like the colors of the rainbow." Furthermore, neoplasms of the paraneurons (paraneuromas, paraneuroblastomas) are represented by all varieties between endocrine cells and neurons, particularly in light of the fact that many of the same transmitters and hormones are shared by both neuronal and endocrine neoplasms.

ACKNOWLEDGEMENTS

Portions of the work reviewed were supported by a Grant-in-Aid from the American Heart Association (77 630) and with funds contributed in part by the Texas Affiliate.

TABLE I
REPRESENTATIVE PARANEURONS

PARANEURON	PRODUCT	REFERENCE
Adrenomedullary Cells	E,NE	Kobayashi, '77
Paraganglia	E,NE	Mascorro, '75
SIF Cells	DA	Williams and Palay, '69
Carotid Body	DA,NE	McDonald and Mitchell, '75
Aortic Body Chief Cells	DA,NE	Hansen and Yates, '75
Parafollicular Cell	5-HT,DA,C.	Kameda, '77
Adenohypophyseal Cells	5-HT,ACTH	Dahlstrom and Fuxe, '66
Pinealocyte	Melatonin,5-HT	Ueck and Wake, '77
Pancreatic Islet Cells	5-HT,Glucagon, Insulin,GH	Pearse and Polak, '71
Gastro-Enteric Endocrine Cells	5-HT,DA,H Substance P Gastrin, Secretion	Fujita and Kobayashi, '73
Bronchial Endocrine Cells	5-HT,DA,	Wasano, '77
Gustatory Cell	DA,5-HT	Nada and Hirata, '75
Merkle Cell	H,5-HT	Iggo and Muir, '69

Abbreviations: C, Calcitonin; E, epinephrine; DA, dopamine; GH, growth hormone; H, histamine; NE, norepinephrine; 5-HT, serotonin.

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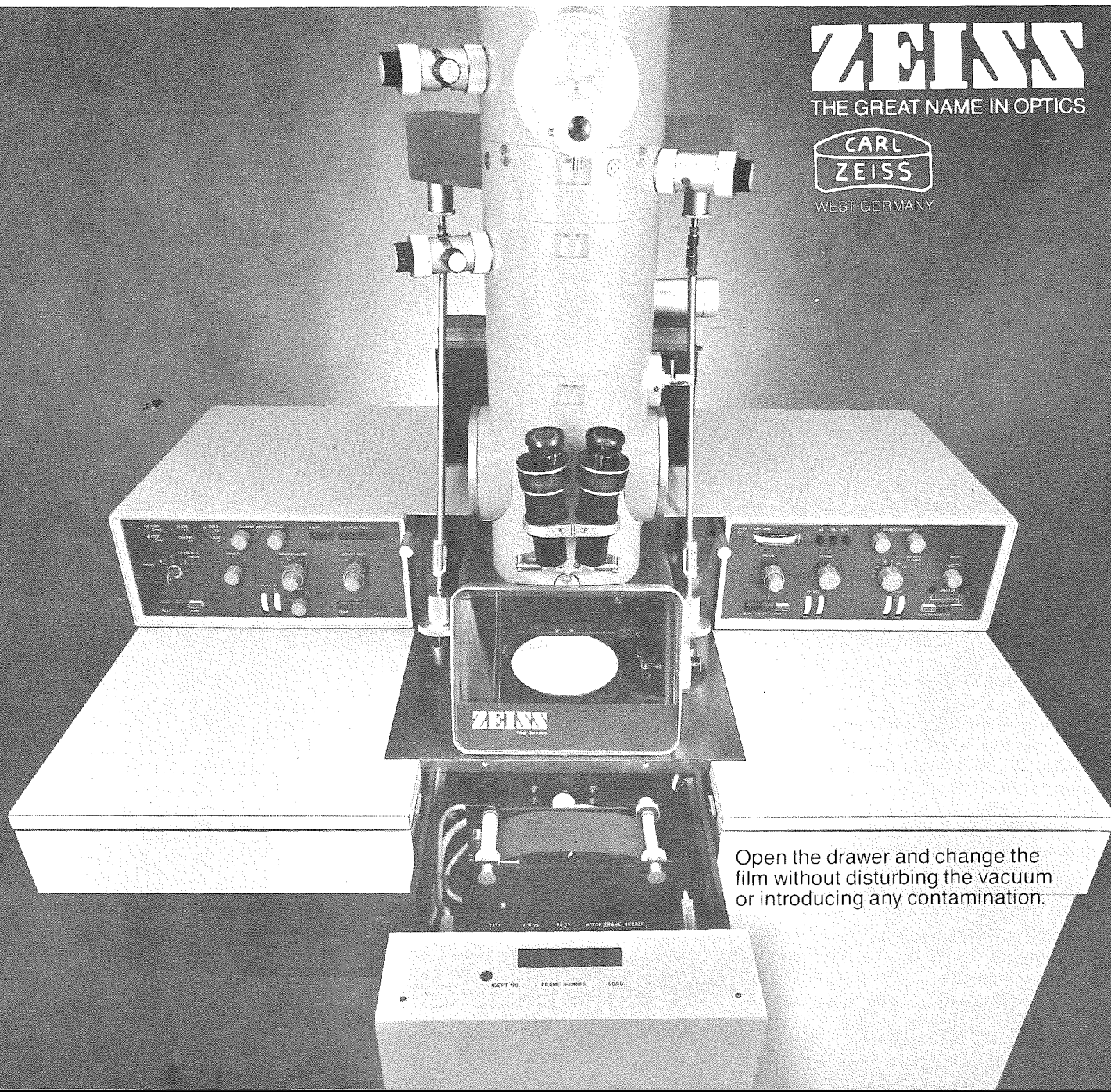
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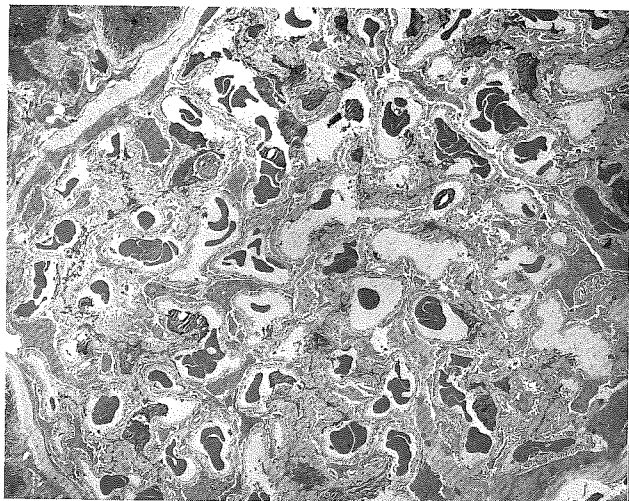
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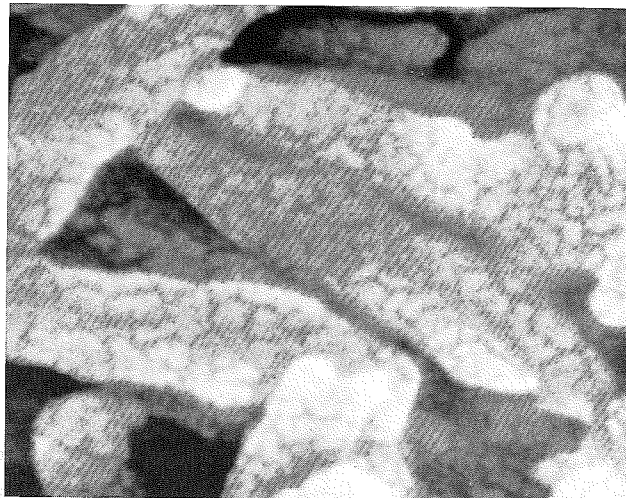


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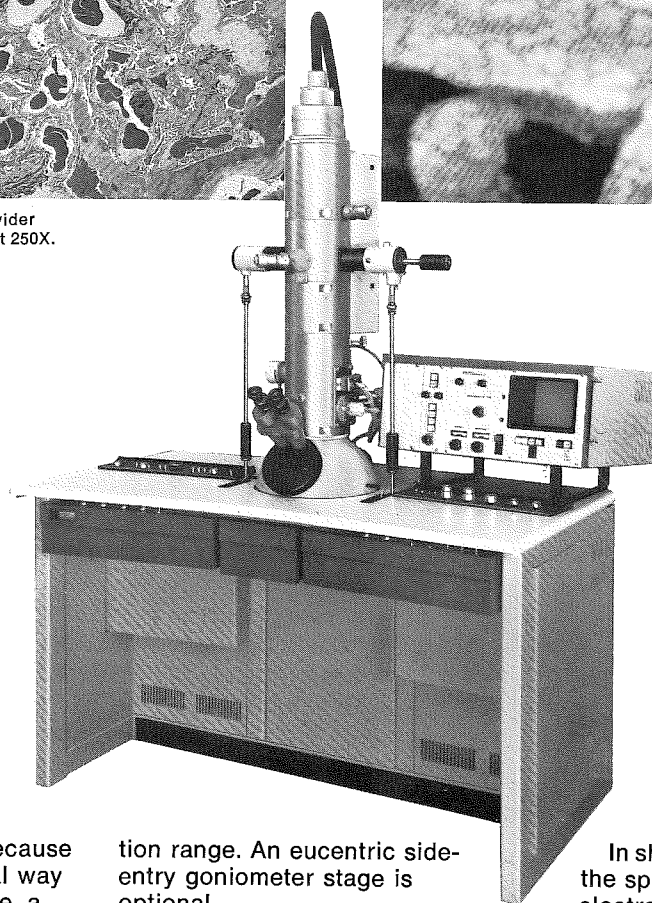
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Cellular Response to Paraquat Toxicity A Review

by
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INTRODUCTION

The herbicide Paraquat has gained considerable public attention during the last three years. Paraquat has been used in Mexico to defoliate marijuana fields (See Smith, 1978). When contaminated marijuana is smoked, paraquat and its derivatives can produce a variety of respiratory side effects (Dasta, 1978, and Zavala and Rhodes, 1978). Paraquat requires two to three days to exert full herbicidal effect on a plant as evidenced by leaf drop. Mexican farmers would harvest the marijuana quickly after government planes had sprayed the fields. This action resulted in residual paraquat on the harvested plant. Considerable public outcry to these practices called attention to paraquat and its potential human health hazard.

Paraquat, however, can produce more severe health problems. By 1977 over 600 deaths due to paraquat ingestion have occurred world wide (Dearden et al., 1978). Some poisonings resulted when individuals drank the herbicide from temporary storage containers such as soft drink bottles. Smith and Heath (1976) reviewed some of the bizarre circumstances connected with accidental ingestion of paraquat. As many as two-thirds of paraquat poisonings are suicides; and it has been reported that paraquat ingestion is a common form of suicide in

Malaysia (Smith and Heath, 1976).

Treatment of paraquat poisoning has stirred considerable interest in the medical profession. Ingestion of 15 ml of the undiluted herbicide or exposure to as little as 4 mg/kg can lead to death (Dasta, 1978). There is no known antidote to paraquat poisoning. The mortality rate is from between 33 to 50% of the poisoning victims. Research into the toxic effects of paraquat spurred by the human deaths has proved very interesting. This report will serve as a brief review of toxic effects of paraquat and discuss some future applications.

PARAQUAT, the herbicide

Paraquat is a **para** substituted **quat**ernary bipyridyl. Its common chemical form is 1,1'-dimethyl-4,4'-bipyridylium dichloride. Paraquat was first synthesized in the 1930's and was named methyl viologen. It was used as an indicator dye in oxidation-reduction reactions. In the 1950's the herbicidal qualities of bipyridyls were discovered. The Imperial Chemical Industries of England commercially introduced paraquat and a related compound, diquat, as herbicides in 1962. (See Smith and Heath, 1976, for a review.) This water soluble chemical is distributed in the United States by Chevron as Ortho Paraquat Chloride (a

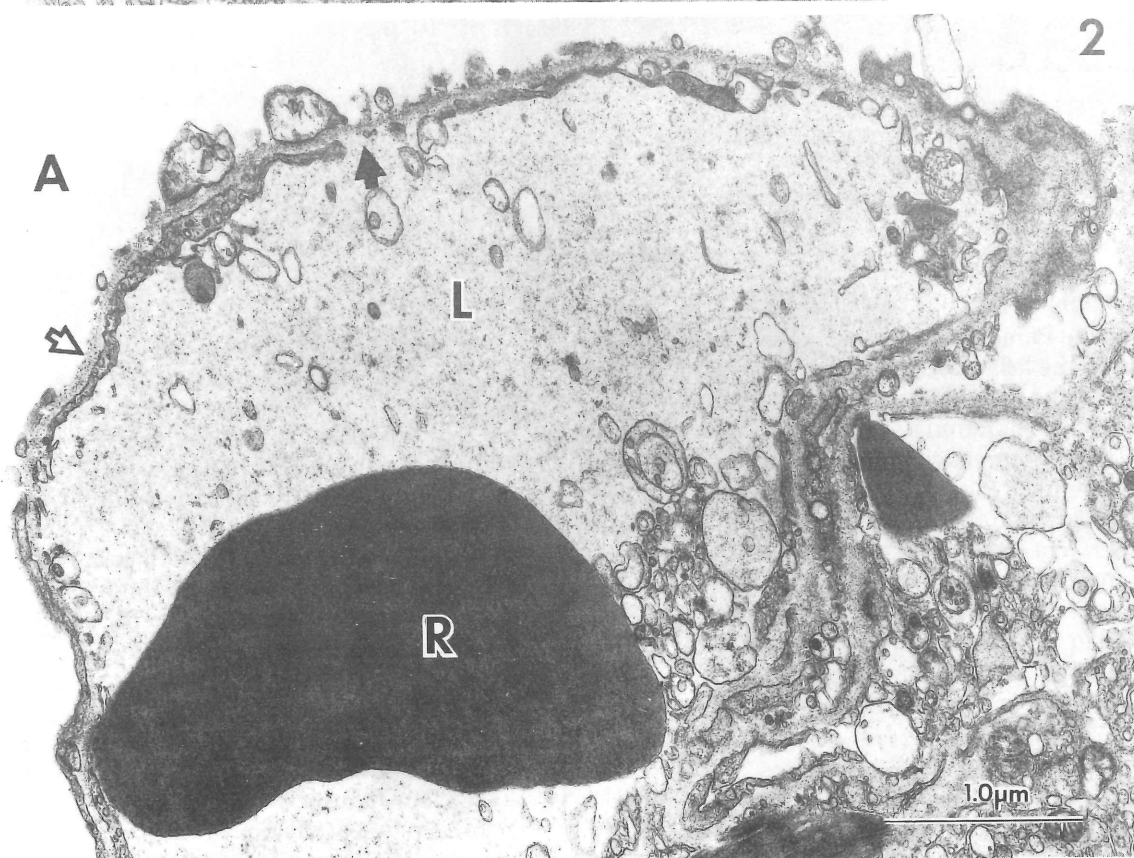
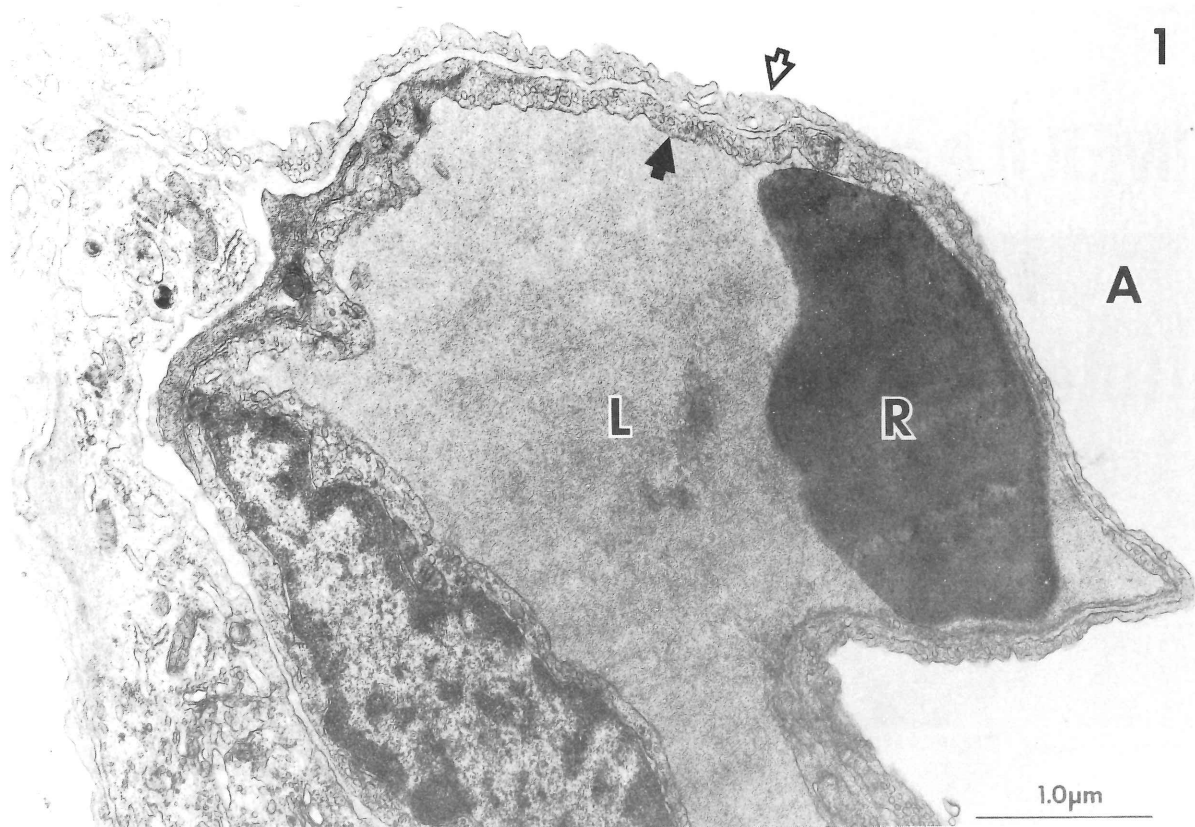


FIGURE LEGEND

A - Alveolar space
L - Capillary lumen

R - Red blood cell
F - Fibrotic material

X - Tubular myelin
* - Tubular myelin

FIGURE 1 — Control rat lung showing capillary with closely applied type I cell. The dark arrow indicates the endothelium of the capillary and the open arrow indicates the alveolar type I cell. Note the basal lamina between the endothelial cell and alveolar cell. Also note the relatively homogeneous material in capillary lumen.

FIGURE 2 — Lung of rat treated with 25 mg/kg paraquat and sacrificed four days post-injection. The open arrow indicates the highly disrupted alveolar type I cell. The dark arrow indicates gaps in the capillary endothelial lining. Note the contents of capillary lumen.

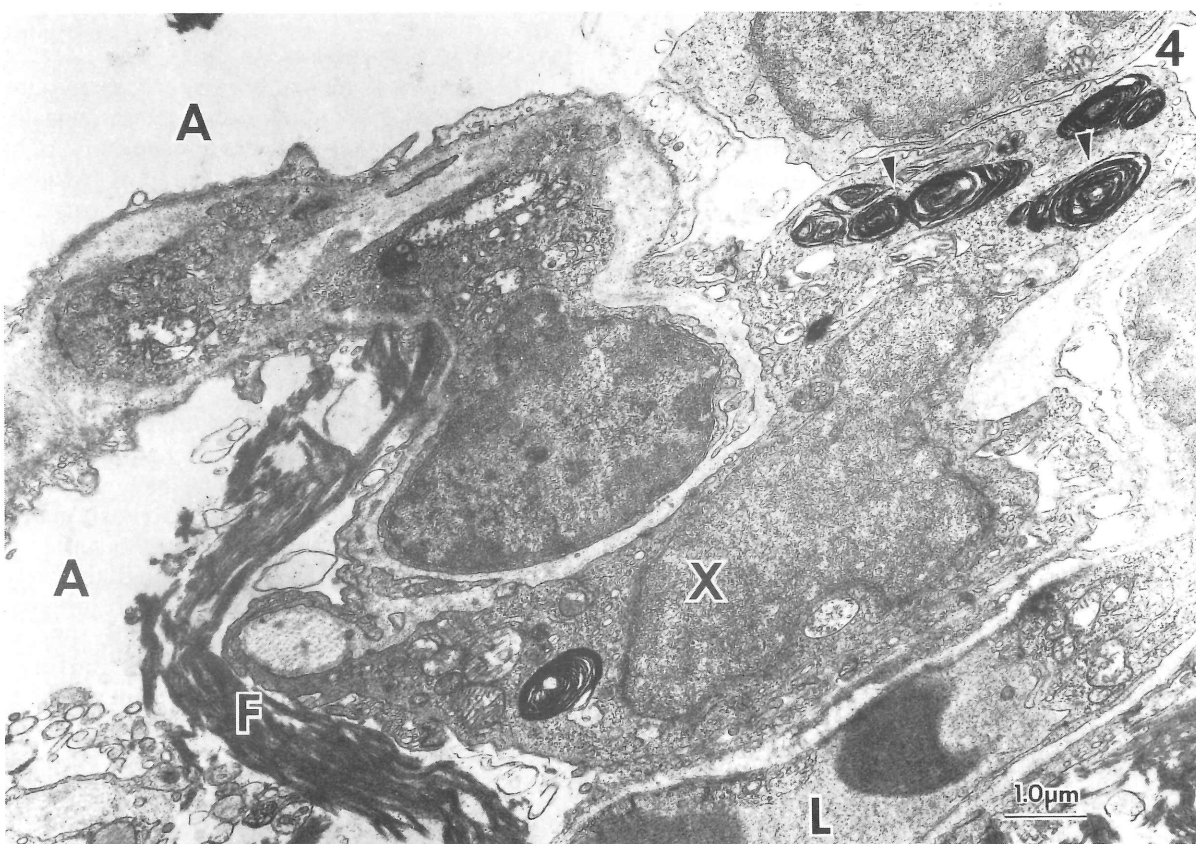
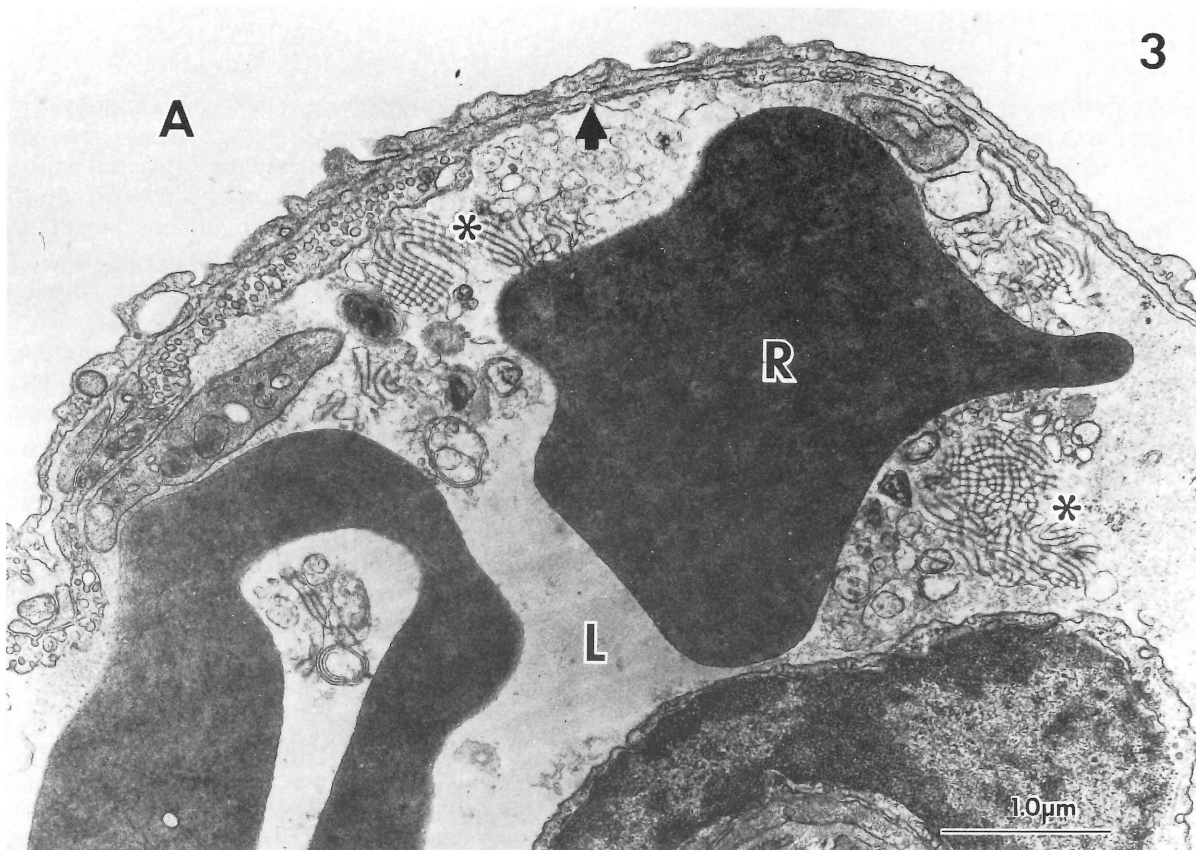


FIGURE 3 — Lung of rat treated with 15 mg/kg paraquat and sacrificed four days post injection. The capillary endothelial wall (dark arrow) is less regular than the control. Of considerable interest is the tubular myelin within the capillary lumen.

FIGURE 4 — Lung of rat treated with 25 mg/kg paraquat and sacrificed four days post injection. Fibrotic material may be seen in the alveolar lumen. The alveolar type II cell demonstrates numerous multi-lamellar bodies (at arrows). The type II cell appears less compact than usual.

24.6% aqueous solution), Ortho Grammoxone Chloride, and Ortho Spot Weed and Grass Killer (a 0.2% aerosol foam). (For additional paraquat trade names see Fletcher, 1975, p. 89.) Tens of thousands of kilograms of paraquat are manufactured and sprayed each year.

Paraquat, unlike many other herbicides, is reported to be environmentally harmless once it moves into the soil. Currently the Environmental Protection Agency lists 41 herbicidal applications for the chemical (EPA, 1975). A principal use is for weed control in orchards and with numerous food crops. Paraquat is also used to defoliate leaves from plants such as cotton just before mechanical harvesting. Paraquat is highly effective in agricultural weed control and allows for minimum of tillage of the soil.

Paraquat exerts its herbicidal qualities in the following ways. The chemical may be reduced either by NADPH oxidation or by Photosystem I. (See Dodge, 1971; Witschi et al., 1977, and Boger and Kunert, 1978, for details.) Reduced paraquat is a stable free radical which then may react with oxygen produced by the chloroplasts. Subsequent reactions may produce a variety of phytotoxic molecules such as hydrogen peroxide, superoxide anion, hydroxyl radical and singlet oxygen (Boger and Kunert, 1978). The superoxides and peroxide ions produced are then in a position to react with the unsaturated lipids in the chloroplast membrane and plasmalemma (Dodge 1971). Biochemically this event is marked by the formation of malonaldehyde (Heath and Packer, 1968). Morphologically, Baur et al. (1969) showed breakdown of the plasmalemma and thylakoids in mesquite mesophyll cells after treatment with paraquat. Most sources indicate an enhancement of the phytotoxic effect with sunlight and subsequent oxygen liberation (Smith and Heath, 1976).

PARAQUAT, the poison

In 1966, Bullivant was first to report human death due to exposure to paraquat. The course of paraquat poisoning is dose dependent. Human ingestion of over 30 mg/kg usually results in death within a few days. Consumption of between about 5 to 30 mg/kg of paraquat most likely results in death within one to three weeks of ingestion. With doses of less than 5 mg/kg, recovery is probable. Dermal exposure is by far the more likely event in human contact with paraquat. Smith and Heath reviewed the reported cases of dermal exposure as of 1976. Undiluted agricultural strength paraquat (about 24.6%) is reported to produce blisters and soreness on dermal contact. Careless mixing of the concentrate or continued exposure can result in fingernail damage. Workers who used diluted paraquat daily on a Malayan rubber plantation were reported to have frequent cases of skin irritation but these exposures were considered of minor consequence. Dermal absorption of the Malayan workers was calculated to be an average of 0.04 ppm as measured in the urine. Using this data, Howard (1978) calculated that a spray operator following all directions and working an eight hour day should be

dermally exposed to no more than 0.4 mg/kg of paraquat per day. He estimated that this exposure rate would be about 0.5% of the toxic dose per day based on the estimated human dermal LD₅₀. Two reports in 1978 indicated dermal exposure can lead to death in humans. Jaros (1978) reported the death of a farm worker after a four hour dermal exposure to a 5% paraquat spray. Newhouse et al. (1978) reported the death of a farm woman who regularly mixed paraquat for a fruit orchard. The paraquat was absorbed through scratches on the skin and that action led to her death. Limited data exists as to the consequences of long term, subclinical exposure to paraquat.

The clinical course of paraquat poisoning demonstrates two defined stages. (See Smith and Heath, 1976, for a review.) The first state is referred to as the early or destructive stage which lasts from two days to seven days. This stage may be marked by gastro-intestinal tract inflammation, uremia, renal failure, and hepatic involvement. The lungs may become edematous. Death usually results during the first stage period with doses of 30 mg/kg or higher. With lower dose ingestions, victims survive into the late or proliferative stage of poisoning. The proliferative stage is marked by a loss of pulmonary function. This second stage lasts from one to three weeks after exposure. Ingestion of less than 5 mg/kg may lead to recovery.

PARAQUAT, movement in the body

The course of clinical treatment for paraquat poisoning is dependent upon the amount ingested. Little can be done for victims of massive ingestion of bipyridyl. (See Raffin, et al., 1978, for a case report.) More clinically challenging are the moderate exposure cases. (See Dasta, 1978, for treatment protocol.) Irregardless of the mode of administration of paraquat (intraperitoneal, intravenous, oral, respired, dermal), the most sensitive tissue of the body to toxic effects of paraquat is the lung. Lung involvement in paraquat poisoning is so typical that the term "paraquat lung" has been descriptively coined (Smith and Heath, 1974, 1976). Zavala and Rhodes (1978) instilled intrabronchially as little as one picogram of paraquat and produced pulmonary lesions. The lesions consisted of diluted congested capillaries, intraalveolar macrophages, and moderate thickening of the alveolar septa.

The movement of paraquat through the body has proved puzzling. Ingested paraquat is absorbed over the gastrointestinal wall at a rate of from 1 to 10% in humans (Dasta, 1978; Walters and Dugard, 1978; Raffin et al., 1978). A substantial amount of paraquat is lost via the feces and urine (Smith and Heath, 1976). Acute renal failure may occur with paraquat poisoning (Vaziri et al., 1979). Paraquat can be detected in the urine up to 31 days after exposure. Plasma concentrations drop dramatically within hours after initial exposure (Davies et al., 1975). The paraquat concentration in the lungs show a steady linear increase to where the concentration may be as much as 80 times higher than the plasma. (Smith and Heath, 1976).

Rose et al. (1974) reported that paraquat accumulation in the lung was an energy dependent phenomenon. Paraquat, therefore, moves into the tissues, especially the lung, and gradually comes out again as detected by trace amounts in the urine.

An interesting analogy would be a radioactive compound selectively concentrating in the lung with a half-life of 10 to 20 days. The paraquat is capable of producing oxygen free radicals which in turn may (1) disrupt cell membranes, (2) oxidize enzyme -SH groups and (3) break down nucleic acids (Raffin et al., 1978). Based on the selective uptake by the body of paraquat, the lung would have exposure to the paraquat and its action over a prolonged period of time. The remainder of this report will deal with the action of paraquat on the lung.

PARAQUAT, action in the lung

The laboratory rat has been extensively studied with paraquat doses near the LD₅₀. (See Clark et al., 1966, for LD₅₀ information.) This dosage would put the animal in the second stage of paraquat poisoning. Pulmonary fibrosis is most characteristic of this stage. The fibrosis is said to consist of fibroblasts within a network of ground substance and collagen (Smith et al., 1974). Clark et al. (1966) and Vijeyaratnam and Corrin (1971) have described the pulmonary fibrosis as an interstitial type. Smith and Heath (1976) argue that the fibrosis is of an intra-alveolar type not involving the alveolar walls. The fibrosis severely affects the function of the lung as alveolar collapse is common. The type of fibrosis produced seems to be dose dependent. Brooks (1971) and Kimbrough and Linder (1973) administered oral doses of 500 ppm or less of paraquat over a period of 4 to 16 weeks. Both these studies resulted in interstitial pulmonary fibrosis with open alveoli. The doses used were one-fifth or less of the estimated LD₅₀ for rats.

The alveolar type I cell is affected by paraquat. Ultrastructural analysis reveals that this cell type is destroyed (see Modee et al., 1972, and Robertson, et al., 1976). A comparison of Figure 1 and 2 clearly represents this phenomenon. Figure 1 illustrates a capillary covered by an intact type I cell. Figure 2 is from a rat given 25 mg/kg I. P. of paraquat four days prior to sacrifice. Both the type I cell and the capillary endothelium have broken down. Figure 3 is from an animal given 15 mg/kg paraquat. Both cell types are still intact at four days post ingestion. Type II cells respond to type I cell destruction by increasing in number and attempting to spread over denuded capillary surfaces (Kimbrough and Linder, 1973). Figure 4 illustrates a distended Type II cell at the 25 mg/kg dose rate. This micrograph also shows fibrotic material within the alveolar space.

Figure 3 depicts a capillary-alveolar region subjected to 15 mg/kg of paraquat. The lumen of the capillary of this rat demonstrates tubular myelin figures. (For tubular myelin references see Georke, 1974; Ryan et al., 1975; and Williams, 1978.) Multilamellar bodies, tubular myelin and

alveolar surfactant are thought to represent a progression of stages in the development of the surface tension qualities of the alveolar surface. Manktelow (1967) was first to report that paraquat increased the surface tension of the lung and speculated that there was a loss of pulmonary surfactant. Manktelow further suggested that paraquat induced pulmonary lesions might be due in part to surfactant loss. Paraquat lung is said to resemble idiopathic respiratory distress syndrome (Smith and Heath, 1976). The knowledge of the role of surfactant and the attendant alveolar surface tension changes after paraquat introduction is important. However, there is no agreement in the literature as to what is happening with regard to surfactant and paraquat lung. Manktelow's speculation as to the loss of alveolar surfactant in paraquat exposure has not been substantiated (Smith and Heath, 1976).

PARAQUAT, biochemistry

Moderators of paraquat toxicity have been actively sought. Various antioxidants have been suggested. The most studied of the moderators has been the enzyme superoxide dismutase (SOD). Bus et al. (1974) found that paraquat produced lipid peroxidation *in vitro* in the presence of NADPH and NADPH-cytochrome C reductase. They measured peroxidation in rat liver microsomes by the formation of malondialdehyde. The peroxidation process was inhibited by superoxide dismutase and 1,3 - diphenylisobenzofuran. Autor (1974) reported her findings with SOD *in vivo* in rats. Paraquat treated rats kept in an oxygen enriched atmosphere died sooner than air maintained animals. Animals treated with SOD survived longer in both environments. Bus, et al. (1976) in further work determined that paraquat in mice decreased the antioxidant, glutathione, in liver and lipid soluble antioxidants (tocopherols) in the lung. Rats treated at chronic paraquat levels showed elevations in glucose-6-phosphate dehydrogenase and glutathione reductase. In oxygen tolerant animals the G-6-P- dehydrogenase and SOD levels are higher. Bus et al. (1976) then showed that oxygen tolerant rats were more resistant to paraquat toxicity. (See Frank and Massaro, 1979, for a review of lung and oxygen toxicity.) Bus, Cagen et al. (1976) proposed an *in vivo* mechanism of paraquat toxicity and lipid peroxidation based on their earlier reports. Goldstein et al. (1979) used diethyldithiocarbamate (DDC) to inhibit the action of superoxide dismutase. Mice treated with DDC and then paraquat died sooner. Wasserman and Block (1978) reported that rats given acute doses of paraquat (50 mg/kg I. P.) responded well to 2-20 mg/kg/d of SOD therapy. The lipid peroxidation concepts and SOD antioxidant theories have gained wide acceptance as they relate to paraquat toxicity.

Shu et al., 1979, and Talcott et al., 1979, have called for a re-examination of paraquat mediated lipid peroxidation data. Shu et al. (1979) indicated that paraquat and ferric pyrophosphate react at the membrane-buffer interface (in

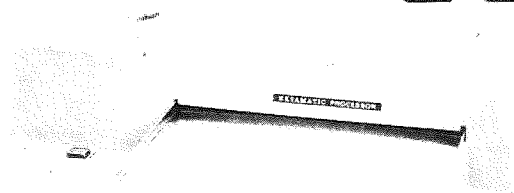
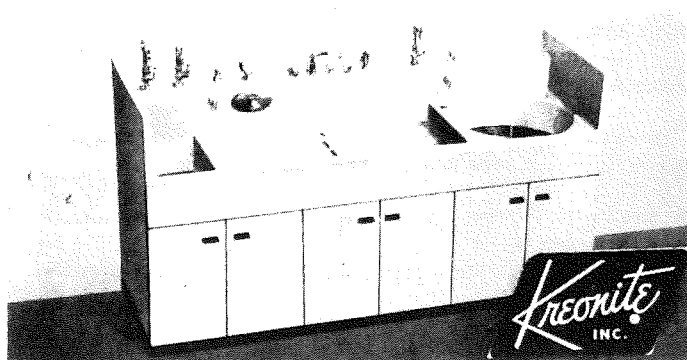
vitro) with target fatty acids. They then speculate that if the superoxide anion and hydrogen peroxide react in the water soluble area then catalase and superoxide dismutase should operate. They continued by reporting that superoxide dismutase inhibition of microsomal lipid peroxidation was not demonstrated. They also speculate that superoxide anion is generated in the hydrophobic region of the membrane and is inaccessible to SOD for a period of time.

PARAQUAT, a summary

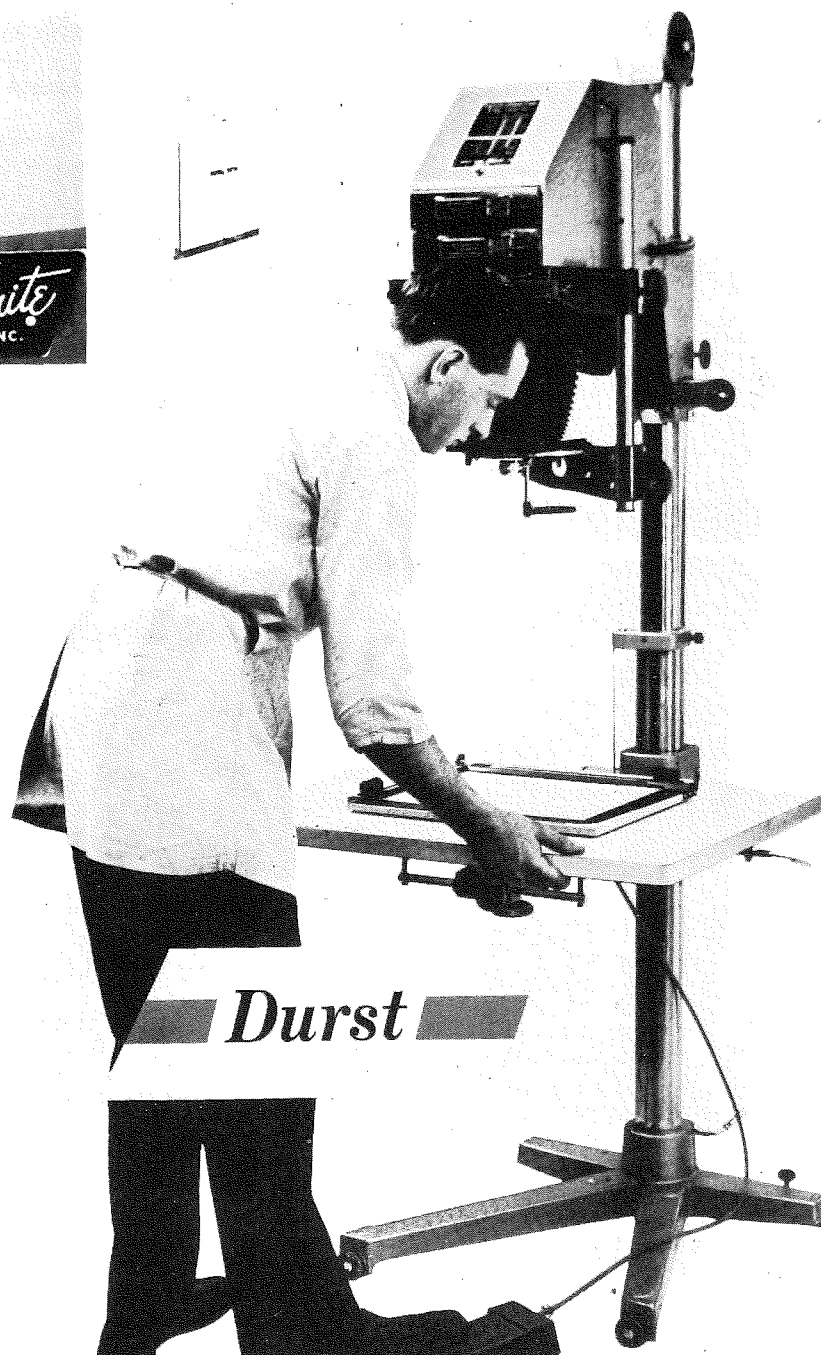
Paraquat is an effective herbicide. Its use has increased agricultural productivity and economy. This herbicide can kill animals and humans. Evidence of dermal toxicity to humans is beginning to mount. Very little data as to the effects of chronic exposure or subclinical cases of paraquat toxicity are available. The toxic effects are most noted in the lung and presumably involve lipid peroxidation. The roles of surfactant, lipid peroxidation and superoxide dismutase are unclear in paraquat poisoning and sometimes seemingly in conflict. Although paraquat poisoning occupies center stage, the uses of paraquat in new research approaches such as tumorigenesis are appearing (Bojan et al., 1978). Our lab at Trinity is exploring the chronic and subclinical manifestations of paraquat toxicity, especially as it relates to surfactant production and release.

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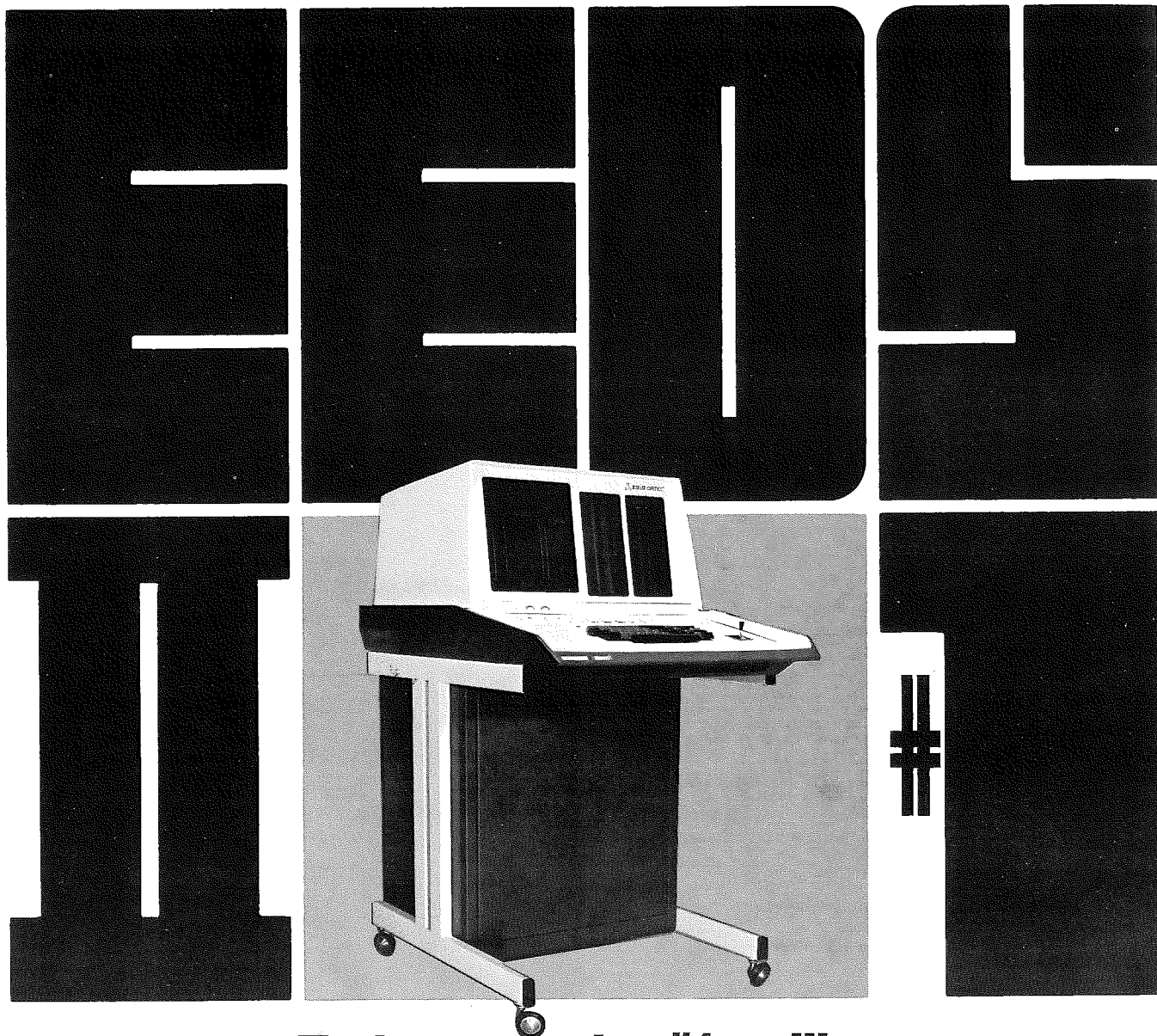
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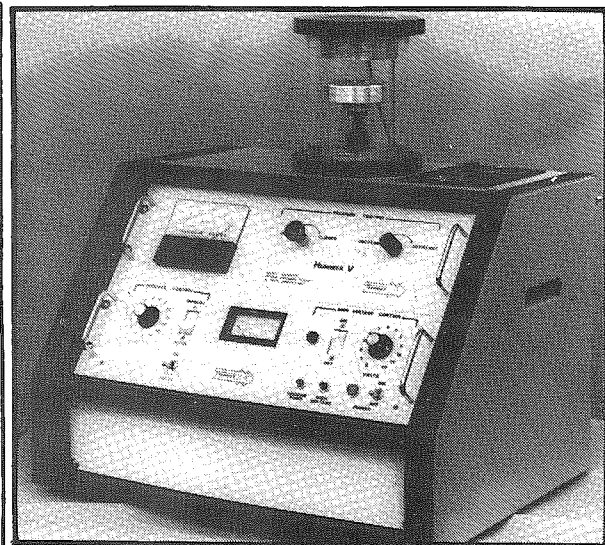
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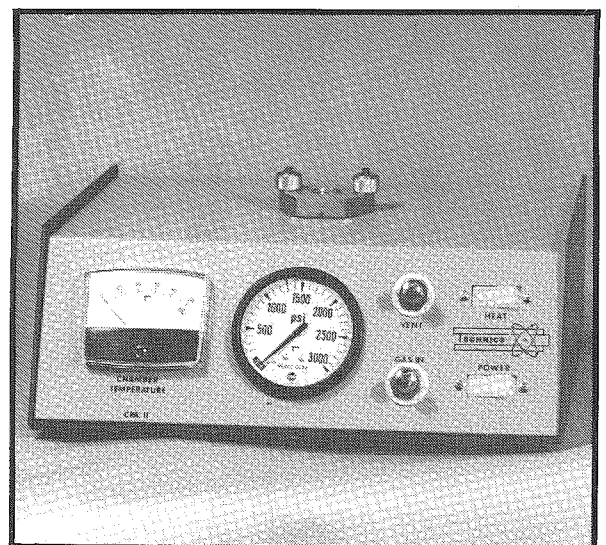
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Regional News

HOUSTON

BAYLOR COLLEGE OF MEDICINE DEPARTMENT OF MEDICINE SECTION OF CARDIOVASCULAR SCIENCES SEMINARS

Dr. Ann Goldstein presented a paper "A morphometric analysis of ischemic myocardium" in Florence, Italy at the Florence International Meeting on Myocardial Infarction on May 11, 1979.

Dr. Goldstein presented a talk on "The Z lattice in cardiac muscle" at the University of Birmingham Medical School in Birmingham, England on May 29, 1979.

DEPARTMENT OF MICROBIOLOGY SEMINARS

In May, 1979, Dr. H. D. Mayor presented a talk at NIH, Bethesda, Maryland, on parvoviruses.

DEPARTMENT OF NEUROBIOLOGY AND ANATOMY, THE UNIVERSITY OF TEXAS MEDICAL SCHOOL AT HOUSTON

GRANTS AWARDED

Nachum Dafny, Ph.D., Professor, has received a grant from the National Institute on Drug Abuse entitled "Mechanisms of Narcotic Addiction" for a period of three years. The amount awarded is \$137,000.

Nachum Dafny, Ph.D., has been awarded a Fogarty Fellowship by the National Institutes of Health to study the "Neurobiology of Obesity and Satiety" at Tel Aviv University in Israel. Time for Dr. Dafny's sabbatical has not yet been determined.

Jon DeFrance, Ph.D., Associate Professor, received a grant from the National Institutes of Mental Health for his project titled "Nucleus Accumbens: Action of Dopamine and Acetylcholine." The grant is in the amount of \$60,000 for two years.

John Haycock, Ph.D., Instructor, received a Biomedical Research Support Grant titled "An In Vitro Approach to Synaptic Plasticity." Effective March, 1979, the grant period is for one year.

Louise Cope Moorhead, M.D., Research Scientist, received an Academic Investigators Award from the National Eye Institute for her project titled "Vitrectomy: Effect on Retinal Function and Structure." For two years the amount awarded was \$57,399.

Michael Oberdorfer, Ph.D., Assistant Professor, was awarded a National Eye Institute grant for three years in the amount of \$51,803, and supplemental funding of \$9,633. The grant is titled "Eye development and Abnormal Visual Pathways."

GENERAL NEWS

Dianna A. Redburn, Ph.D. has been promoted to Associate Professor with tenure in recent action of the Faculty Appointments, Promotions and Tenure Committee.

LECTURES AND MEETINGS

John DeFrance, Ph.D., Associate Professor and Gerald Kozlowski, Ph.D., Associate Professor, attended the Winter Conference on Brain Research in Sun Valley, Idaho 1/20-27. Dr.

DeFrance presented a symposium titled "Septal Nuclei: Prolegomenon to Neuroendocrinology," and Dr. Kozlowski presented a paper titled "Septal Nucleus and Neuroendocrine Function."

Zehava Gottesfeld, Ph.D., Associate Professor, traveled to San Diego in February to attend and participate in discussion sections at the "Conference on the Molecular Basis of Cell-Cell Interaction."

John Haycock, Ph.D., Instructor, traveled to Colorado Springs, Colorado in January to attend the Western Pharmacology Society meetings.

Dianna Redburn, Ph.D., Associate Professor, Richard Wiggins, Ph.D., Assistant Professor, Margaret Bell, graduate student, and John Ferkany, graduate student in Dr. S. J. Enna's lab, attended the 10th Annual Meeting of the American Society for Neurochemistry in Charleston, SC, March 11-16. Dr. Redburn presented a paper titled "GABA Receptors in Bovine Retina;" Margaret Bell presented a paper titled "Double and Single Isotope Studies of Sciatic Nerve Wallerian Degeneration" which she co-authored with Dr. Wiggins; and John Ferkany presented a paper titled "Measurement of Drug-Induced Changes in Brain, CSF and Blood GABA Content."

Joe G. Wood, Ph.D., Department Chairman, David McCandless, Ph.D., Assistant Professor, JoAnn McConnell, Ph.D., Assistant Professor, and Gayle Hostetter, Ph.D., Research Scientist, represented the Department at the 92nd Annual Meeting of the American Association of Anatomists in Hollywood-by-the-Sea, Florida, April 1-6. Dr. McCandless presented a paper titled "Effect of Unconjugated Bilirubin on Energy Metabolism in Cerebellar Layers;" Dr. McConnell presented a poster titled "EM Analysis of the Adrenergic and Possible Peptidergic Innervation of the Human Vas Deferens and Corpus Cavernosum;" and Dr. Hostetter presented a paper titled "Distribution of a Brain ACTH Neurosecretory System."

Attending the 63rd Annual Meeting of the Federation of American Societies for Experimental Biology from the Department were: Nachum Dafny, Ph.D., Professor, Richard Yeoman, Ph.D., Postdoctoral Fellow, Eve Andersen, graduate student, S. J. Enna, Ph.D., Associate Professor, Adrianna Maggi, Ph.D., Teaching Associate, John Ferkany, graduate student, and Elaine Mann, Research Assistant. Dr. Dafny presented abstracts titled "Opiate Discrimination Sensory Input Recorded from Several Brain Sites" and "Effect of Morphine on Unit Activity Patterns Recorded from Reticular Formation, Caudate Nucleus and Medial Thalamus." From Dr. Dafny's lab, Dr. Yeoman presented a paper titled "Enflurane Effects on Reticular Formation Sensory Evoked Potentials in Rats," Eve Andersen presented a paper titled "5HT Involvement in Sensory Input to Basal Ganglia of Freely Behaving Rats," and a paper by Ricardo Pardo, medical student, titled "Dopamine Modulates Sensory Input to Caudate Nucleus and Globus Pallidus" was presented. From Dr. Enna's lab, John Ferkany presented a paper titled "Neurochemical Alterations Following Chronic Inhibition of GABA Transaminase."

Gerald Kozlowski, Ph.D., Associate Professor, and John Linner, Ph.D., Teaching Associate, travelled to Colorado in April. Dr. Kozlowski conducted an Immunocytochemistry workshop in Keystone (4/7-4/13). They then went to Boulder to use the high voltage electron microscope at the University of Colorado, examining rat brain tissue.

Dianna A. Redburn, Ph.D., Associate Professor, and

members of her laboratory attended the Spring meeting of The Association for Research in Vision and Ophthalmology, Inc. in Sarasota, FL (4/30-5/4). Reports presented were: "Localization of Kainic Acid-Sensitive Cells in Rabbit Retina" by Cynthia A. Keiller and Dr. Redburn, "Distribution of ³H-Spiroperidol Binding within Synaptosomal Fractions of Retina" by Dr. Redburn and Cheryl K. Mitchell, and "Localization of Serotonergic Neurons in Bovine Retina" by Thomas N. Thomas and Dr. Redburn.

Michael D. Oberdorfer, Ph.D., Assistant Professor, also attended the ARVO meeting in Sarasota and presented a paper entitled "Gap Junction Vesicles in Developing Mammalian Retina."

S. J. Enna, Ph.D., Associate Professor, travelled to Europe in May. He presented seminars to research staff at Synthelabo Pharmaceutical Co. on May 8 in Paris, at Merrill International Pharmaceutical Co. on May 9 in Strasbourg, France, and on May 10 at F. Hoffmann-LaRoche and Co. in Basel, Switzerland. Dr. Enna also presented a symposium lecture at the First International Colloquium on Receptors in Capri, Italy (5/13-14).

JoAnn McConnell, Ph.D., Assistant Professor, was in New York City to give a talk during the Urodynamics Society workshop held in conjunction with the annual meeting of the American Urological Association (5/11-14).

Gayle Hostetter, Ph.D., Research Scientist, presented a seminar entitled "Neuropeptides: Immunocytochemistry and Behavior" at Tulane University in New Orleans on May 3rd. On May 9th, Dr. Hostetter presented a seminar entitled "Anatomical and Behavioral Correlates of Neuropeptides" to the Department of Anatomy at the University of Oregon in Portland.

S. J. Enna, Ph.D. presented a lecture entitled "Recent Advances in the Pharmacology of GABA" in the Neuroscience Conference at Baylor Medical School, May 25th. On June 1st, Dr. Enna chaired a session entitled "Receptors Unlimited" at the Texas Pharmacology Society Meeting in San Antonio. And, in Washington, D.C. on June 14th, Dr. Enna presented a seminar entitled "Biochemical Properties of GABA Receptors" at the National Institute of Mental Health.

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GRANTS AWARDED

Markwald, R. R.: grant renewal, Mucopolysaccharide Metabolism in Developing Cardiac Anomalies, National Institutes of Health, \$262,000 for 5 years.

Markwald, R. R.: grant, Renewal Research Matrical Macromolecular Effects on Cardiogenesis, National Heart, Lung, Blood Institute, \$32,400.

Seliger, W. G.: grant continuation, Matrix Mediated Expression in Cranial Facial Embryology, National Institutes of Health, \$31,099 for 1 year.

LECTURES

Bolender, D. L., Markwald, R. R.: presentation and paper, Epithelial-Mesenchymal Transformation in Chick Atrioventricular Cushion Morphogenesis, Scanning Electron Microscopy, 1979. Washington, D.C.

Coates, P. W., Davis, S. L.: presentation and paper, The Sheep Third Ventricle: Correlated Scanning Electron Microscopy and Transmission Electron Microscopy, and Plasma Luteinizing Hormone in Wethers and Testosterone Propionate Treated Wethers, Scanning Electron Microscopy, 1979, Washington, D.D.

Roberts, L. A., Dalley, B. K.: abstract, Ultrastructural changes accompanying Hyper-osmolar induced Contracture of Isolated Atria, Federation Proceedings 38(3):1387, 1979.

Fitzharris, T. P., Markwald, R. R.: presentation and paper, Modification of Matrix Ordering During In Vivo Cell Movement, The Cytoskeleton: Membranes and Movement, Cold Spring Harbor, New York, May, 1979.

Stocco, D. M., Hutson, J. C.: abstract, Cytochrome-Oxidase Activity and the Ultrastructure of Mitochondria Isolated Regionally From Normal Liver and Novikoff Hepatomas, Federation Proceedings 38(3):840, 1979.

Hutson, J. C.: invited presentation and workshop, Immunocytochemical Localization of an FSH-like Molecule in the Testis: Are Immunological Controls Sufficient?, International Histochemical Society Meeting, Immunocytochemical symposium, Keystone, Colorado.

Karkos, K. R.: presentation, Response of Cerebellar Cells to Clicks of Varied Intensity, American Association of Anatomists 92nd Annual Session, Miami, April, 1979.

Menchaca, J. A., Lefkowitz, S. S., Trying, S. Bartholomew, B., Markwald, R.: abstract, Effects of Immunostimulation with Levamisole (Lev) on Murine Lupus Nephritis, Pediatric Research 13(4):451, 1979.

Markwald, R. R.: invited guest lecturer and seminar speaker, Oral Roberts University School of Medicine, Tulsa, Oklahoma, March, 1979.

Markwald, R. R., Bernanke, D. H., Krook, J. M.: presentation, Cell Mediated Reordering of Extracellular Macromolecules in Atrioventricular (AV) Morphogenesis, American Association of Anatomists, 92nd Annual Session, Miami, April, 1979.

Markwald, R. R., Fitzharris, T. P., Bernanke, D. H.: invited presentation and workshop, Structural Identification of Complex Carbohydrates, International Histochemical Society Meeting, Complex Carbohydrate Symposium, Keystone, Colorado.

Young, H. E., Dalley, B. K.: presentation, Regional Distribution of Matrical Components During the Early Stages of Limb Regeneration in the Adult Salamander, Ambystoma Annulatum, American Association of Anatomists, 92nd Annual Session, Miami, April, 1979.

PUBLICATIONS

Markwald, R. R., Fitzharris, T. P., Bolender, D. L., Bernanke, D. H.: article, Structural-Analysis of Cell-Matrix Association During the Morphogenesis of Atrioventricular Cushion Tissue, Development Biology 69(2):634-654, 1979.

Peluso, J. J., Bolender, D. L., Perri, A.: article Temporal Changes Associated with the Degeneration of the Rat Oocyte, Biology of Reproduction 20(3):423-430, 1979.

Yee, John A. 1979. Response to periodontal ligament cells to orthodontic force: ultrastructural identification of proliferating fibroblasts. Anat. Rec. in press.

Pang, P. K. T. and J. A. Yee. 1979. Hormonal control of calcium metabolism in lower vertebrates. In: The Proceedings of the International Symposium on Hormones and Evolution, in press.

BIOLOGY

Dr. S. Kosmidou-Dimitropoulou from the Hellenic Cotton Board in Athens, Greece visited the Department of Biology at Texas Tech University and worked in Dr. Jerry Berlin's laboratory for 6 weeks to learn some techniques in electron microscopy.

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COURSES

"Introduction to Scanning Electron Microscopy," a graduate course, is being taught by Dr. Nancy Smith during the summer session.

PRESENTATIONS

Murr, L. E., N. Chakraborti, and V. K. Berry. 1979. Observations of a natural thermophilic microorganism in the leaching of a large, experimental, copper-bearing waste body. (Paper presented at the annual meeting of the American Institute of Mining and Metallurgical Engineers, New Orleans, February, 1979.)

King, R. D., J. C. Lee, D. J. Drutz, R. W. Osgood, and V. K. Berry. 1979. Involvement of adherence in renal candidiasis. (Paper presented at the annual meeting of the American Society for Microbiology, Los Angeles, May, 1979.)

Cameron, I. L., J. J. Brokaw, S. G. Dykes, and N. K. R. Smith. 1979. Elemental concentration changes in mouse erythrocytes during postnatal development. (This paper is being presented at the Joint EMSA/MAS meeting in San Antonio this summer.)

PUBLICATIONS

Berry, V. K., and L. E. Murr. 1979. Ultrastructure of the cell envelope of an acidophilic thermophile: Comparison with *T. ferrooxidans* and a *Sulfolobus*-like microorganism. 37th Annual Proceedings Electron Microscopy Society of American, in press.

Dykes, S. G., I. L. Cameron, and N. K. R. Smith. 1979. Electron probe microanalysis of elemental composition of mouse cardiac myocytes during postnatal maturation. *J. Cell. Physiol.*, in press.

Heitman, D. W., T. B. Pool, and I. L. Cameron. 1979. Changes in proliferation and surface morphology in the rat ileum in response to total parenteral nutrition. *J. Anat.*, in press.

TSEM FINANCIAL REPORT

Period Ending July 10, 1979

Certificates of Deposit	\$ 3,000.00
Transfer from Galveston Accounts.....	2,873.00

RECEIPTS:

Dues:		
Regular Membership.....	\$ 280.00	
Student Membership.....	40.00	
Corporate Membership.....	200.00	
Registration (Dallas).....	1,202.00	
Corporate donations (Dallas)		
Jeol.....	200.00	
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Interest from Certificate of Deposit.....	17.68	
Interest from Savings Account.....	15.69	
Advance Registration, 1980 meeting.....	100.00	
Subtotal	\$ 8,128.99	(+) \$ 8,128.99

DISBURSEMENTS:

Dallas Meeting expenses	\$ 98.00	
Travel:		
Student (Dallas).....	170.00	
Council (Galveston).....	78.88	
Adding Machine	94.33	
Treasurer's Expenses	64.67	
Printing for 1980 Meeting	13.44	
Subtotal	\$ 519.32	(-) \$ 519.32

ASSETS:

Certificate of Deposit No. 1099, Univ. Bank.....	\$ 2,000.00	
Certificate of Deposit No. 17864, Fannin Bank	1,000.00	
Savings Account 10-0502435, Fannin Bank	3,115.69	
Balance in Checking Account, Fannin Bank	1,493.98	
Total Assets	\$ 7,609.67	\$ 7,609.67

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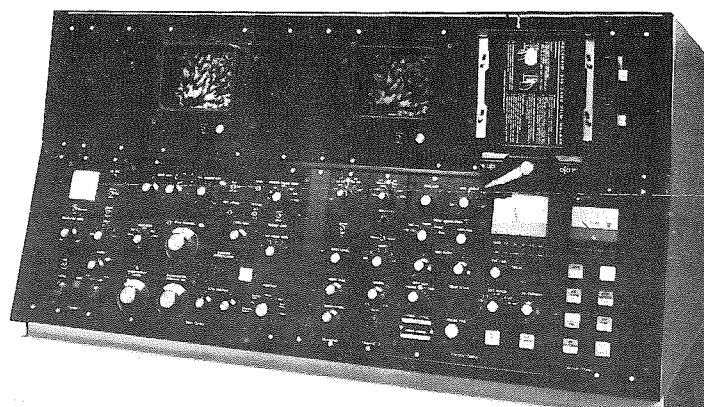
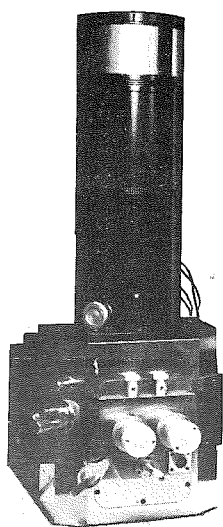
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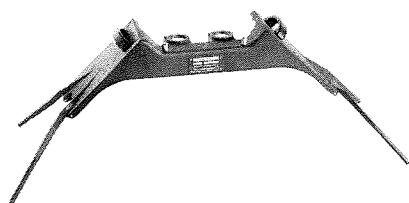
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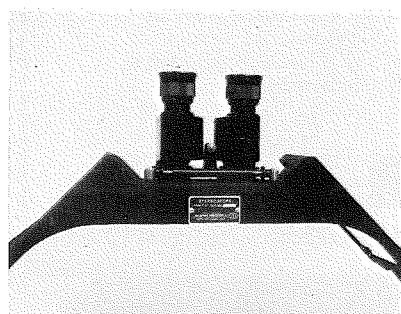


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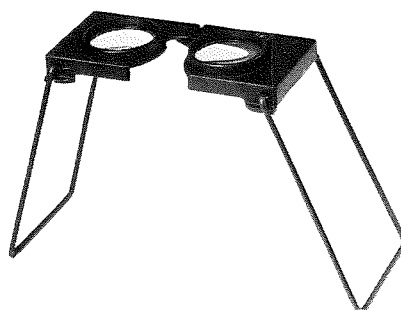
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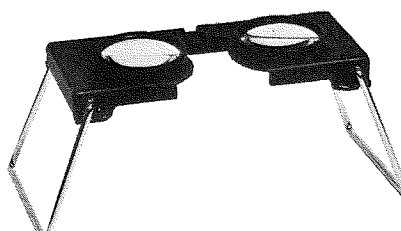
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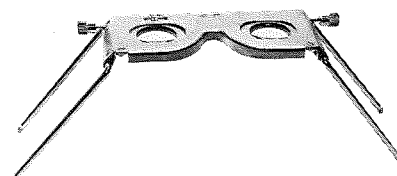
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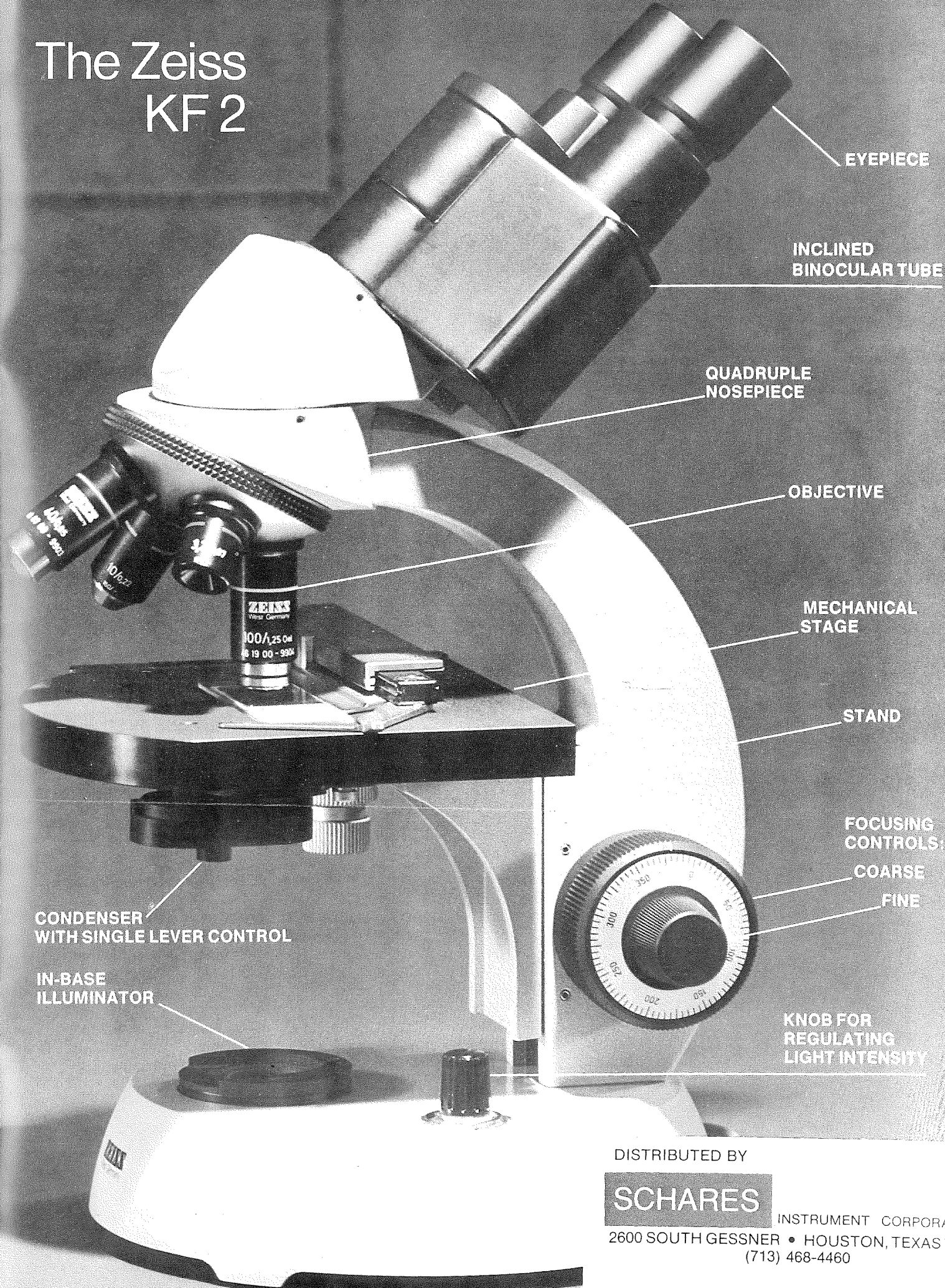
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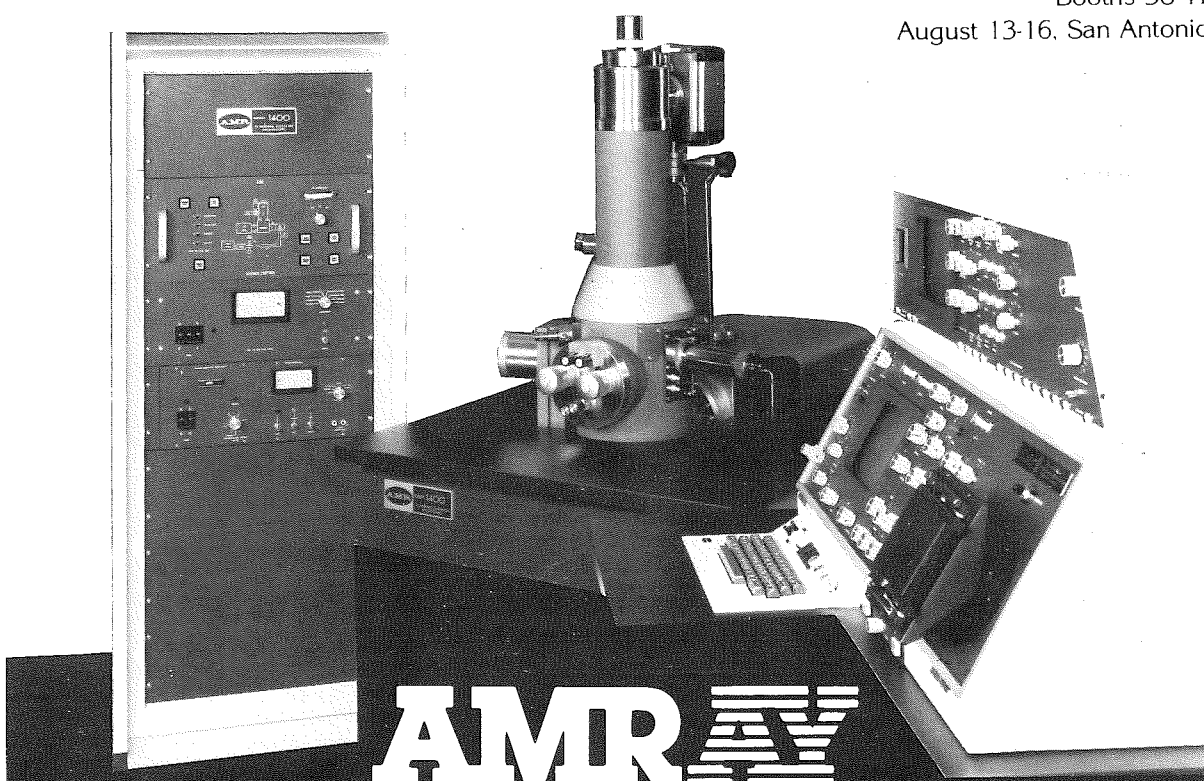
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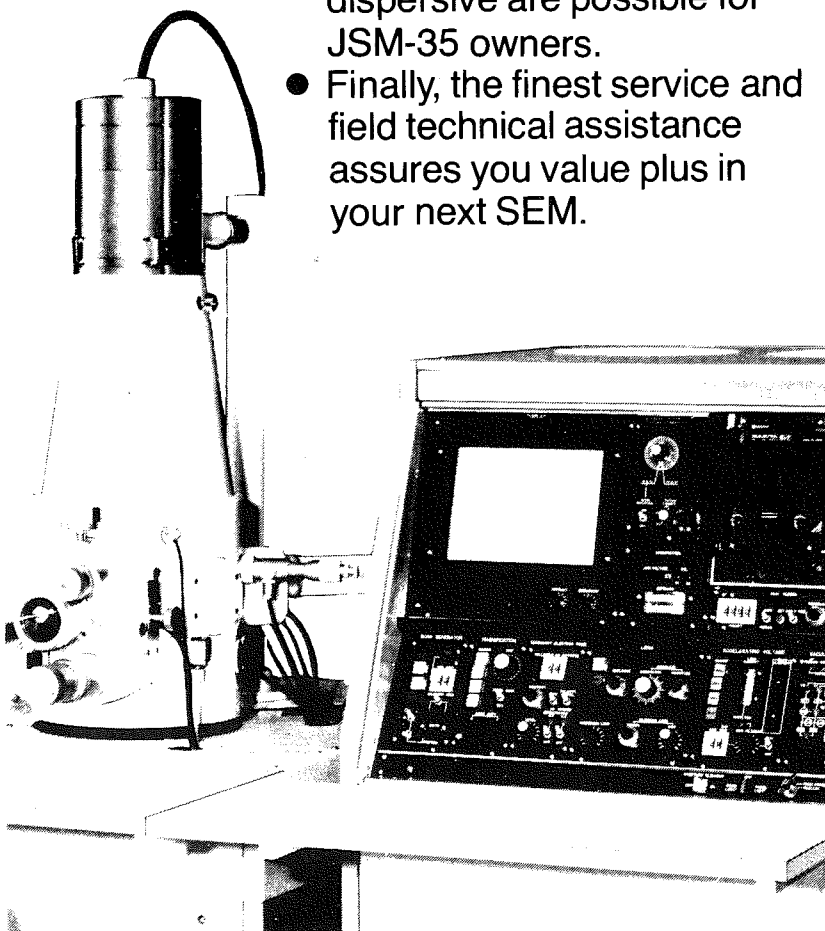
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