Kinetics of Ascorbate Transport by Cultured Retinal Capillary Pericytes

Inhibition by Glucose

Mahin Khatami, Weiye Li, and John H. Rockey

Accumulation of radioactive L-[carboxyl-14C]-ascorbic acid by cultured bovine retinal capillary pericytes was studied. Kinetic analysis of the transport showed a time-dependent, saturable system with an apparent $K_m$ of 76.0 µM and a $V_{max}$ of 42 pmole/µg DNA/min. A facilitated carrier diffusion process was established on the basis that the system was not sensitive to 2,4-dinitrophenol, ouabain, or reduced sodium concentration in the incubation media, and that the carrier system demonstrated stereospecificity for an ascorbate analogue, dehydroascorbate, and for sugar analogues such as 3-0-methyl-D-glucose (3-0-MG), but not for 3-0-fructose or 3-glucose. Transport of ascorbate by cultured pericytes was insulin-insensitive. 3-0-Methyl-D-glucose inhibited ascorbate transport into pericytes in a non-competitive manner with a $K_i$ of 22 mM. These results indicate that, in cultured retinal capillary pericytes, a common facilitated carrier diffusion system is involved in the transport of ascorbate and sugar analogues such as 3-0-D-glucose or 3-0-MG. Invest Ophthalmol Vis Sci 27:1665-1671, 1986

Retinal damage by chemical insults, such as hyperglycemia, or by light may be influenced by vital metabolites, such as ascorbic acid. Ascorbic acid, a carbohydrate vitamin, is an electron donor and antioxidant, and is involved in many hydroxylation reactions and biosynthetic pathways, such as dopamine $\beta$-hydroxylation and the synthesis of collagen and glycosaminoglycans.1-3 Its biochemical role in vascular disease (e.g., diabetic microangiopathy), however, is not completely understood. Alterations in the biosynthesis of capillary basement membrane components (e.g., collagen, glycosaminoglycans) may contribute to the defective function and pathological changes of microvascular basement membranes seen in diabetic microangiopathy.6,7

There is increasing evidence that insulin facilitates the transport and distribution of vitamin C in a variety of tissues similar to its effect on glucose, and that insulin deficiency and/or hyperglycemia may interfere with the transport and metabolism of vitamin C.6-14 Several ocular tissues may be independent of insulin,15,16 and the mechanism of ascorbate transport in tissues which are sensitive or non-sensitive to insulin may be different.11-14,17,18 Mann8 has suggested that species that do not synthesize ascorbic acid (e.g., human, monkey, guinea pig) and require dietary ascorbate must also have a mechanism for transporting this vitamin into cells. Hyperglycemia may interfere with vitamin C transport, and diabetic animals that require exogenous ascorbic acid may have a double disadvantage.

Extensive publications deal with the mechanism of hexose transport across the blood retinal barrier19,20 and in isolated retinal microvessels21 or pigment epithelium.22-25 Recent studies also have demonstrated the presence of a facilitated diffusion system for glucose transport in cultured bovine retinal capillary pericytes26 and endothelial cells.27 The presence of active transport systems for ascorbate in rat whole retina27 and in guinea pig ciliary body-iris28 have been reported. However, nothing is known about the mechanism(s) by which ascorbate is transported into retinal vessel cells. Whether glucose interferes with ascorbate transport and function in capillary cells also is an open question.

The present studies establish the kinetics of ascorbate transport into cultured bovine retinal microvessel pericytes. Evidence is presented for a facilitated diffusion, insulin-independent system which is inhibitable by $\alpha$-D-glucose and its analogues.
Materials and Methods

Preparation of Bovine Retinal Microvessels

Retinal microvessels from fresh bovine eyes were prepared as previously described\(^{29,30}\) with minor modifications. Retinal homogenates, prepared in Dulbecco Modified Eagle Medium (DMEM) containing 20% fetal calf serum, were applied to a 55 μm mesh size nylon screen under aseptic conditions and washed thoroughly with saline. Microvessels were collected only from the back side of the nylon mesh, and assessed for purity by phase contrast and differential interference microscopy\(^{29,30}\).

Pericyte Culture

Pericytes were isolated from microvessels as previously described\(^{29,30}\). Cells were cultured in 60 or 100 mm plastic Petri dishes at a density of approximately \(1 \times 10^6 \text{cells/cm}^2\) in DMEM supplemented with 20% fetal calf serum, 100 U/ml penicillin, and 100 μg/ml ascorbic acid (Medium). Cells at confluency were trypsinized with 0.25% trypsin in a Ca\(^{++}\)-Mg\(^{++}\)-free salt solution containing 6 mM KC\(_1\), 154 mM NaCl, 1.2 mM NaHCO\(_3\), 0.83 mM NaH\(_2\)PO\(_4\), 1 mM EDTA, pH 8.0, at 25°C for 2 min. Trypsinized cells were added to five volumes of Medium (over trypsin solution volume) and centrifuged at 1,000 rpm for 10 min at 25°C. Sedimented cells were taken up in Medium, replated in Petri dishes, and incubated at 37°C under 95% air-5% CO\(_2\). The Medium was changed every 2-3 days. Confluent cells were collected every 4-6 days and re-passaged 20-25.

Ascorbate Uptake by Pericytes

L-Ascorbic acid (sodium salt, Sigma Chemical Co., St. Louis, MO) was dissolved in balanced salt solution (BSS) composed of 150 mM KCl, 4.2 mM K\(_2\)HPO\(_4\), 1.0 mM Na phosphate, 0.7 mM MgCl\(_2\), 2.0 mM CaCl\(_2\), and 10 mM Hepes buffer, pH 7.4. Prior to the experiments, 0.5-5.0 μCi/ml of lyophilized L-[carboxyl-\(^{14}\)C]-ascorbic acid (specific activity 17 mCi/mmole, 98% purity, Amersham, Arlington Heights, IL) was dissolved in BSS and mixed with the freshly prepared unlabeled ascorbate solution (0.5-5 mM) in the presence or absence of 1-10 mM thiourea. Radioactive ascorbate solutions were either used immediately or kept frozen (\(-20^\circ\text{C}\)) under nitrogen in light-tight containers for up to 7 days. Confluent pericytes were washed with BSS (2×, 2 ml each), and the ascorbate transport was initiated by the addition of 10-1,000 μM (final concentration) radioactive ascorbate. Cells were incubated for selected periods at 37°C in a water bath with gentle automatic shaking under atmospheric oxygen. Additional dishes were incubated at 4°C. Ascorbate uptake was terminated by aspirating the media and immediately washing (5X) the cells with ice-cold BSS. The total time for washing was 2 min or less. Cells were extracted with 0.1 ml of 0.2 N NaOH, and the extract was transferred into 5 ml of Ultrafluor (National Diagnostic, Somerville, NJ) for radiometry. In selected experiments, cells were incubated in the presence of 150 mM LiCl or 150 mM choline-Cl in place of 150 mM NaCl in BSS.

Effects of Sugars and Other Compounds on Ascorbate Transport

Cultured pericytes at confluency were incubated in BSS in the presence of 0-20 mM 3-0-methyl-D-glucopyranoside (3-0-MG; Sigma Chemical Co., St. Louis, MO). Radioactive L-ascorbate at selected concentrations (50, 200, or 500 μM) was added 30 sec after the addition of 3-0-MG, and incubated as described above. In a series of similar experiments, either α-D-glucose, 5-thio-D-glucopyranoside (5-TDG), L-glucose, β-D-fructose or myo-inositol were used in place of 3-0-MG. In addition, the effect of phloretin and phlorizin, inhibitors of glucose transport; dehydroascorbate (DHA), an ascorbate analogue; 2,4-dinitrophenol (DNP), a metabolic inhibitor; ouabain, an inhibitor of Na\(^+\)-K\(^+\)-ATPase; and insulin on ascorbate uptake were studied.

Analysis of radioactive samples

Radioactive samples obtained from incubation media were analyzed in a liquid scintillation spectrometer. Radioactive components also were spotted on silica gel plates (20 × 20 cm, LK6DG linear-K, Whatman, Clifton, NJ) in the presence or absence of thiourea. Radioactive and non-radioactive ascorbate, dehydroascorbate (DHA, ICN, Nutritional Biochemical, Cleveland, OH), and D-isoascorbate (DIA; Sigma Chemical Co., St. Louis, MO) were run in parallel. Chromatography was performed in benzene:methanol:acetic acid:water (20:20:68:6) and DHA spots were identifiable after 10 min heating at 65°C. Plates were scraped (0.5 cm fractions), and the radioactivity was determined.

Cell Counts, DNA Measurements, and Determination of Cellular Water

Representative cultured pericytes at confluency were used to count viable cells with a hemocytometer in the presence of Trypan blue.\(^{30}\) DNA content per culture dish also was measured in parallel as previously described.\(^{15}\) The intracellular water space of cultured pericytes was determined as previously described using \([\text{H}]-3-0-MG.\(^{32}\)
Results

Kinetics of Ascorbate Transport by Cultured Pericytes

Confluent pericytes, incubated in the presence of 200 \(\mu\)M radioactive ascorbate, progressively accumulated ascorbate (Fig. 1). The uptake was essentially linear up to 10 min. The linearity of ascorbate uptake was similar when cells were incubated in the presence of 50 or 500 \(\mu\)M of radioactive ascorbate (data not shown).

Cells incubated in the presence of varying concentrations of radioactive ascorbate exhibited saturation kinetics (Fig. 2). From the initial velocities, it was found that the uptake followed Michaelis-Menton kinetics, assuming a steady state. The kinetic constant (\(K_m\)) for the ascorbate uptake was determined from a Lineweaver-Burk transformation of the Michaelis-Menton equation (Fig. 2, inset). From the plots of the reciprocal of the initial velocity vs the reciprocal of the ascorbate concentration, an apparent half maximal velocity was found at 76.0 \(\mu\)M of ascorbate (\(K_m\)) with a maximal velocity (\(V_{max}\)) of 42 pmole/\(\mu\)g DNA/min. In addition, a plot of the ratio of the ascorbate concentration to the corresponding velocity (\([S]/V\)) vs the ascorbate concentration was linear, and gave a \(K_m/V_{max}\) (intercept of the \([S]/V\) axis) ratio of 1.92. From this plot, \(V_{max}\) (1/slope) was 40 pmole/\(\mu\)g DNA/min.

Cells incubated at 4°C in the presence of 50 or 200 \(\mu\)M ascorbate showed a significant reduction in their ability to accumulate ascorbate when compared to control experiments carried out at 37°C (Table 1).

Analysis of Radioactive Samples

Radioactive ascorbate from the incubation media remained in the reduced form (>70%) when analyzed up to 24 hr (Fig. 3). Radioactive stock solutions of ascorbate, as well as non-radioactive samples at higher concentrations (≥100 mg%), were found to be more stable during the storage period than diluted samples (1–10 mg%). The TLC analysis showed that ascorbate in stock solutions, when kept frozen (−20°C) up to 7 days under nitrogen, remained in its reduced form (>90%). Longer storage of ascorbate, particularly in dilute solutions, produced oxidized products detected by TLC (Fig. 3). Freshly prepared radioactive ascorbate in the presence or absence of thiourea showed similar uptake by pericytes (Table 1).

Effects of Sugars and Other Compounds on Ascorbate Uptake

Confluent pericytes, incubated at 37°C in the presence of increasing concentrations of 3-O-MG, showed a progressive reduction in the uptake of ascorbate at fixed concentrations (50, 200, or 500 \(\mu\)M) present in the media. Figure 4 is a Dixon plot of the reciprocal velocities for ascorbate uptake plotted against 3-O-MG concentration. The y-axis of Figure 4 shows the reciprocal velocities in the absence of 3-O-MG. The inhibition of ascorbate uptake, at fixed concentrations, by increasing concentrations of 3-O-MG was characteristic of a non-competitive inhibition.

Alpha-D-Glucose similarly inhibited the transport of ascorbate into cultured pericytes. Figure 5 shows the extent of ascorbate uptake (50 \(\mu\)M extracellular concentration) in the presence of increasing concentrations of α-D-glucose or 3-O-MG. Confluent cells at passage 20–25 also showed a similar pattern of uptake of ascorbate and inhibition by glucose.

Phloretin (0.1 mM) or phlorizin (0.4 mM) significantly reduced the net ascorbate uptake by pericytes (Table 1). In contrast to the effect of α-D-glucose or 3-O-MG...
Table 1. Effect of various compounds on ascorbate uptake by cultured retinal capillary pericytes

<table>
<thead>
<tr>
<th>Additions</th>
<th>Concentration</th>
<th>Relative uptake</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) None</td>
<td></td>
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</tr>
<tr>
<td>Thiourea</td>
<td>10 mM</td>
<td>1.09 ± 0.10</td>
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<tr>
<td>Insulin</td>
<td>1000 μIU/ml</td>
<td>0.98 ± 0.05</td>
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<td>Dehydroascorbate</td>
<td>1 mM</td>
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<tr>
<td></td>
<td>10 mM</td>
<td>0.39 ± 0.09†</td>
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</tr>
<tr>
<td>5-Thio-D-glucose</td>
<td>5 mM</td>
<td>0.60 ± 0.10*</td>
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<td>Phloretin</td>
<td>0.1 mM</td>
<td>0.11 ± 0.06†</td>
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<td>2,4-Dinitrophenol</td>
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<td>Ouabain</td>
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<tr>
<td>None (4°C)</td>
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<td>0.13 ± 0.09†</td>
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<tr>
<td>2) None</td>
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<td>Phlorizin</td>
<td>0.4 mM</td>
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<td>myo-Inositol</td>
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<td>Dehydroascorbate</td>
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<td>2 mM</td>
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<td>Insulin</td>
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<td>0.17 ± 0.10†</td>
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* Differed significantly from control, P < 0.01. † P < 0.001. ‡ Cells were incubated with radioactive ascorbate at 4°C.

0-MG, β-fructose and L-glucose did not have a significant effect on the uptake of ascorbate (Table 1). Dehydroascorbate significantly reduced the accumulation of ascorbate, and the inhibitory effect of DHA was concentration dependent (Table 1). myo-Inositol at 10 mM did not change the level of radioactive ascorbate uptake by pericytes (Table 1). The transport of ascorbate into pericytes was energy-independent, and a reduced sodium ion concentration, or the presence of either ouabain (1 mM) or DNP at different concentrations in the incubation media, did not significantly affect ascorbate uptake (Table 1). When pericytes were incubated for up to 40 min with low concentrations of ascorbate (5 μM), the ratio of intracellular to extracellular concentration of radioactive ascorbate never ex-

![Fig. 3. Analysis of radioactive ascorbate by thin layer chromatography (TLC).](image)

![Fig. 4. Dixon Plots of inhibition of ascorbate uptake by 3-0-MG.](image)
ceeded unity, indicating that ascorbate was not accumulating against a concentration gradient.

**Discussion**

The present studies indicate that a facilitated diffusion system mediates the transport of L-ascorbate into cultured retinal capillary pericytes on the basis of the following observations: 1) the system is saturable; 2) the carrier requires physiological temperature to mediate ascorbate transport; 3) the carrier demonstrates stereospecificity for ascorbate analogues, such as dehydroascorbate, and for sugars, such as α-D-glucose and its non-metabolizable analogue, 3-O-methyl-D-glucose, but not for fructose or L-glucose; 4) the system does not require metabolic energy and is not dependent on the sodium concentration in the external media; and 5) ascorbate does not accumulate against a concentration gradient.

The time course of ascorbate transport, shown in the present studies, as well as in those reported for cultured chromaffin cells, adrenal cortex and brain cortex, and cultured retinal pigment epithelial cells, is much slower than the time course reported for transport of dehydroascorbate into red blood cells, neutrophils, and fibroblasts. The oxidized form of ascorbate, dehydroascorbate, is a non-ionized carbohydrate and may cross the plasma membrane of cells more rapidly than the reduced ascorbate. The time course for dehydroascorbate transport into several tissues is comparable to that of glucose. Reduced ascorbate is an anion and may enter the cellular barriers as a neutral species by its interaction with monovalent cations, such as Na⁺ or K⁺ ions. In the present studies, we have presented evidence that the replacement of NaCl by 150 mM LiCl or choline Cl did not affect the ascorbate transport into pericytes. This reflects the fact that the uptake does not involve an active transport system that depends on a sodium gradient established by an Na⁺-K⁺-ATPase system. However, since the ascorbate concentration in the media is in the micromolar range, the reduced extracellular sodium concentrations may not affect the interaction of the remaining Na⁺ (> 1 mM) present in the media in the form of Na phosphate, as well as Na ascorbate, which were used in the experiments. The neutral sodium (or potassium)-ascorbate species perhaps binds to the carrier, which then is internalized. Alternatively, ascorbate-free radical, semidehydroascorbate, which is formed by partial autoxidation of ascorbic acid in solutions, may electrostatically bind to specific site(s) on the carrier molecule.

D-glucose and its non-metabolizable analogues, 3-O-methyl-D-glucose and 5-thio-D-glucose, but not fructose or L-glucose, inhibited the transport of ascorbate into pericytes. The fact that the non-metabolizable analogues of glucose similarly inhibited ascorbate uptake into pericytes demonstrated that the inhibitory effect was not due to the enhanced metabolic products of glucose, but was due to a stereospecific inhibition of the ascorbate transporter by glucose. Ascorbate and D-glucose, therefore, may share a common carrier mechanism for transport into retinal microvascular pericytes. The kinetics of inhibition of ascorbate by 3-O-MG indicated that the inhibition was noncompetitive. Binding of glucose to the carrier may allosterically interfere with subsequent ascorbate binding. The $K_m$ values for the uptake of glucose and ascorbate are very different (76 μM for ascorbate and 1.53 mM for glucose). Glucose at 200 times the concentration of ascorbate was able to inhibit ascorbate uptake 50–60%. Therefore, the affinity and/or efficiency of the carrier for ascorbate appears to be greater than for glucose. Normal plasma concentrations of glucose are in the 4–6 mM range, and of ascorbate are in the 30–100 μM range.

Transport of ascorbate into cultured bovine capillary pericytes was not dependent on insulin. Similarly, as we have recently reported, insulin did not have any significant effect on the glucose transport system in cultured pericytes. This further supports the conclusion that, in pericytes, ascorbate and glucose may be taken up by the same facilitated diffusion carrier system.

We have recently reported that glucose, but not its non-metabolizable analogues, inhibited myo-inositol uptake by cultured pericytes in a non-competitive manner, and that the inhibition of myo-inositol uptake was reversed by Sorbinil, and we have concluded that glucose inhibited myo-inositol uptake through the sorbitol pathway. In the present studies, we have observed that myo-inositol at 10 mM concentration did not have any significant effect on ascorbate uptake, whereas glucose and its non-metabolizable analogues significantly inhibited ascorbate transport into peri-
cytes. These observations support the conclusion that, in pericytes, glucose and ascorbate may share the same carrier system, which is different from that for myo-inositol.

Glucose at physiological concentrations inhibited the transport of ascorbate, but a significantly higher percent inhibition of uptake was obtained when the glucose concentrations were increased to those occurring in diabetes mellitus. The ascorbate transport system, in the presence of hyperglycemic concentrations of glucose, may operate at levels below its $K_m$ value, which, perhaps, is vital to the cellular function of this vitamin.

Ascorbate supplementation has been shown to provide a degree of protection against retinal photo-damage in rats. Scorbatic guinea pigs suffered a greater light-induced retinal damage than normal animals. Light-induced oxidative changes in rod outer segments have been reported to produce hydroperoxide radicals, which may be detrimental to visual cells. Ascorbate, therefore, may protect against the potentially damaging effects of light in the retina through its role as a scavenger of free radicals. Hyperglycemia in diabetes may cause a relative ascorbate deficiency in the retina, thereby enhancing retinal photo-sensitivity and damage.

Although ascorbate is the most efficient reducing cofactor for collagen synthesis (e.g., ascorbate-dependent prolyl hydroxylation), in a variety of pathological states and experimental studies, other endogenous factors (e.g., tetrahydrofolate, glutathione, lactate) or conditions (e.g., glucose composition of the growth media, cell density) are known to regulate collagen synthesis independent of, or in the absence of, ascorbate (ascorbate-independent prolyl hydroxylation). High glucose levels in the growth media stimulated the rate of cellular collagen/protein synthesis in cultured retinal capillary pericytes and cultured retinal pigment epithelial cells (Khatami, Slysh, Landsburg, Li, and Rockey: unpublished observations). The hyperglycemia of diabetes, therefore, may induce factor(s) capable of deregulating the cells from their dependency on ascorbate for collagen synthesis. Increased collagen production and thickening of basement membranes in diabetes may be the result of such a deregulation phenomenon induced by hyperglycemia.

Hexose transport systems are subject to metabolic regulation. The number of sugar carriers of different cell types in culture was reduced as the glucose concentration in the growth media increased. Such a down-regulation of glucose carrier density as a result of hyperglycemia may additionally suppress ascorbate transport into cells (e.g., pericytes) which share the same carrier system for ascorbate and glucose transport. The impairment of ascorbate transport by hyperglycemia, therefore, may be a factor to be considered in the development of diabetic angiopathy.

**Key words:** ascorbate transport, retinal capillary pericytes, culture, glucose inhibition, diabetic retinopathy

**Acknowledgments**

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**References**

Announcements

ARVO Resolution on the Use of Animals in Research

The Visual-Science community has long recognized a scientific and ethical responsibility to provide appropriately for the welfare of animals used for research and education in biology and medicine.1

The Association for Research in Vision and Ophthalmology strongly endorses the continued conservative and humane use of animals in vision research. The vast majority of the major advances made in this field over the past several decades have come from animal studies—advances that have saved or restored the vision of millions of people. The recent development of new animal models for human disease offers hope for those now suffering from currently incurable eye problems or for the thousands who will soon encounter unpredictable and untreatable diseases.

At the same time, ARVO applauds the efforts of those who seek alternatives to animals for certain types of research. However, animal research will of necessity continue to be of vital importance in the struggle against human blindness.2

The NIH and several major biomedical research societies have been working together to insure the adequate care and humane treatment of laboratory animals. Therefore, ARVO directs its Government and Public Relations Committee to work with the National Eye Institute, other NIH components concerned with the use of animals in research, and other biomedical research societies in formulating policies and procedures in this area to assure that recognition of the essentiality of the continued humane use of animals in research is included in any federal, state, or local legislation or university edicts on this subject.

References


Association Update

The year 1986 has proven to be full of administrative changes for ARVO. In May, the membership elected a new Secretary/Treasurer and six new Trustees including, for the first time, a Trustee from the Clinical Research Section. A new Executive Director was hired and the Association Headquarters has moved from New Rochelle, New York, to Bethesda, Maryland.

Board of Trustees

Harry A. Quigley, MD, Professor of Ophthalmology and Director of the Glaucoma Section at the Wilmer Eye Institute, was elected as Secretary/Treasurer ('87-'91). A graduate from Harvard College and Johns Hopkins Medical School, Dr. Quigley was the ARVO Glaucoma Section Chairperson in 1985 and serves on the editorial board of OVS. Although Dr. Quigley's term of office does not officially begin until January 1987, he has already begun working with the Section Chairpersons and the new Executive Director in developing the program for the 1987 Annual Meeting.

Douglas R. Anderson, MD, the Glaucoma Trustee, was elected by the Board to serve as President for 1987. Dr. Anderson is Professor of Ophthalmology at the Bascom Palmer Eye Institute, Miami, Florida.

Daniel M. Albert, MD, the Anatomy Pathology Trustee, will serve as the 1987 Vice President. Dr. Albert is the David G. Cogan Professor of Ophthalmology and Director of the Eye Pathology Laboratory, Harvard Medical School Massachusetts Eye and Ear Infirmary, Boston, Massachusetts.

The newly elected Trustees are as follows:

Biochemistry—Richard N. Lolley, PhD—Chief, Laboratory of Developmental Neurology, VA Medical Center, Sepulveda, California.

Clinical Research—Barbara E. K. Klein, MD—Associate Professor of Ophthalmology at the University of Wisconsin, Madison, Wisconsin.

Cornea—Henry F. Edelhauser, PhD—Professor of Physiology and Ophthalmology at the University of Wisconsin, Madison, Wisconsin.

Electrophysiology—Daniel G. Green, PhD—Professor of Physiological Optics at the University of Michigan, Ann Arbor, Michigan.
Eye Movements—Robert D. Reinecke, MD—Professor of Ophthalmology and Director of the Ethel Brown Foerderer Center for the Study of Eye Movement Disorders in Children, Wills Eye Hospital, and Jefferson Medical College, Thomas Jefferson University, Philadelphia, Pennsylvania.

Lens—Joram Piatigorsky, PhD—Chief, Laboratory of Molecular and Developmental Biology, National Eye Institute, Bethesda, Maryland.

ARVO Headquarters Move

During the summer, ARVO moved its headquarters from New Rochelle, New York, to Bethesda, Maryland. The new office is located on the Federation of American Societies for Experimental Biology (FASEB) Campus, approximately 1/2 mile from the National Eye Institute. Kathleen C. McCasland was selected by the Search Committee, appointed by the Board of Trustees, to serve as Executive Director. Prior to accepting this position, Ms. McCasland was the Executive Director of the California Association for Medical Laboratory Technology. In addition, she was a Program Director at a Washington, DC, based consulting firm, where she wrote proposals and directed several Department of Health and Human Services and Association related projects.

The new address and telephone number are:

ARVO
9650 Rockville Pike
Bethesda, Maryland 20814
301-530-7000

Looking Ahead

Based on results of the 1986 ARVO Meeting Survey, coupled with suggestions and comments offered by many members, a few changes worth noting have already taken place:

• Poster Boards—Twenty-five new poster panels are being constructed which should allow for the acceptance of approximately 250–300 more abstracts that are judged to be of high scientific value.

• Audio Visual Requirements—As in the past, ARVO will provide dual 35 mm slide projectors in all rooms. This year the Association will also provide overhead projectors and 16 mm movie projectors, if it is requested on the abstract form. A “special requirements” box has been added to the form for authors to indicate their special A/V needs. Although ARVO will not provide video equipment, every effort will be made to facilitate cost sharing among the presenters.

• Membership Renewal Notices—Transferring Headquarters to Bethesda, Maryland, necessitated new inputting of all the membership data into the computer. The Membership Renewal Notices were designed to let the members know exactly what information is on record at the Association Office. If you have not already mailed your renewal notice, be sure and make any additions or corrections that are needed. Don’t let your membership lapse!

• Discount on Car Rentals—AVIS and Budget Rental Cars are both offering special discounted rates to attendees at ARVO’s Annual Meeting. Look for details in the Pre-Registration material that will be sent to members in December.

Fight For Sight Scientific Awards Program

The Fight For Sight Scientific Awards Program for 1987–88 has been announced by Mildred Weisenfeld, who is the founder and executive director of Fight For Sight, Inc. The awards program and peer review of applications is administered by the Association for Research in Vision and Ophthalmology.

Applications are available for the following programs in ophthalmic and vision research:

1. Grants-in-aid for research projects, stressing pilot and feasibility projects, with awards of $1,000 to $10,000;

2. Postdoctoral fellowships, with maximum awards of $12,000;

3. Student (summer) fellowships, with maximum awards of $400 per month.

The deadline for receipt of completed applications will be March 1, 1987. Starting dates are July 1 or September 1, 1987, and June 1, 1987, for student fellowships. For further information and application forms (indicate category desired) write to: Fight For Sight, Inc., Box 474, 601 N. Broadway, Baltimore, MD 21205.
Ludwig von Sallmann Prize

Gerald Westheimer, PhD, professor of physiology at the University of California in Berkeley, has won the prestigious Ludwig von Sallmann Prize for significant contributions to vision research and ophthalmology. The $30,000 international prize, awarded every two years, was presented at the International Congress for Eye Research held in September in Japan.

A Berkeley faculty member since 1960, Dr. Westheimer has shown through his research how the eye processes optical signals and how the human brain channels visual information.

The von Sallmann Prize, which is administered by the College of Physicians and Surgeons of Columbia University, is named for Ludwig von Sallmann, who was professor of ophthalmology there and one of the world’s leading authorities on the lens of the eye and its diseases, particularly the development of cataracts.

National Institutes of Health—100th Anniversary

In October of this year, the National Institutes of Health (NIH) began a year-long observance of its 100th Anniversary. All components of NIH are participating in this celebration of a century of excellence in biomedical research. It is anticipated that individual grantees, academic institutions, voluntary organizations, and groups representing physicians and other health care providers will become involved in a meaningful way. In addition, NIH hopes that medical journals, medical news publications, newsletters, and other publications prepared by and for these groups will provide information about the Centennial and the impact of NIH-supported research advances on the welfare of all humankind. Local and national coverage of Centennial events by the general news media is expected as well.

It is hoped that all members of ARVO will join NIH in commemorating its Centennial Year. For example, one could make announcements about the Centennial at meetings, seminars, and workshops. One might be able to arrange for organizations to formally dedicate their next annual meeting or portion of it to NIH and the many research advances it has supported. A message about the Centennial also might be incorporated into the printed programs for these meetings. And of course it is always appropriate to mention the source of support for research when the results are published, or presented at meetings, news conferences, and hospital rounds.

The National Eye Institute’s Office of Scientific Reporting is assembling a “How to participate in the NIH Centennial” kit for friends of the NEI. The kit will contain background information on NIH and the NEI; Centennial Year objectives; a list of available photographs, slides, and film segments; brochures for laymen containing information on various eye diseases and relevant research findings; and a description of an NEI exhibit for laymen that will be available for display at appropriate locations. If you would like to receive any of these materials, please contact Ms. Marsha Corbett, Chief, Office of Scientific Reporting, National Eye Institute, Building 31, Room 6A32, Bethesda, MD 20892 or call (301) 496-5248.

The NEI’s kit will also contain a timetable for Centennial events. In the timetable established by NIH, all Institutes have been assigned specific months to provide a focal point for their Centennial participation. The NEI’s month is January 1987.

ISCEV Annual Symposium

The International Society for the Electrophysiology of Vision (ISCEV) announces its XXV annual symposium to be held April 26-30, 1987, at the Lido Beach Holiday Inn, Sarasota, Florida. For more information contact Dr. William Biersdorf, Department of Ophthalmology, University of South Florida and JAH Veterans Hospital, 13000 N. 30th Street, Tampa, FL 33618 or Dr. William W. Dawson, Department of Ophthalmology, University of Florida, Box J-284 JHMHC, Gainesville, FL 32610.