Immunohistochemical Classification and Functional Morphology of Human Choroidal Ganglion Cells

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PURPOSE. To characterize human choroidal ganglion cells (CGCs) further, regarding their immunohistochemical and ultrastructural appearance and their pre- and postsynaptic connections.

METHODS. Choroidal wholemounts and serial sections of human donor eyes were stained with antibodies against neuronal nitric oxide synthase (nNOS), vasoactive intestinal peptide (VIP), tyrosine hydroxylase (TH), vesicular monoamine transporter (VMAT)-2, vesicular acetylcholine transporter (VACHT), neuropeptide Y (NPY), substance P (SP), calcitonin gene-related peptide (CGRP), calretinin, galanin, synaptophysin, and α-smooth muscle actin. Ultrathin sections of glutaraldehyde-fixed eyes were studied with an electron microscope.

RESULTS. All CGCs stained for nNOS, most for VIP, approximately 45% for calretinin, and only single neurons for NPY and galanin. Ultrastructurally, the CGCs showed an incomplete glial sheath and, in places, showed close contact to surrounding collagen fibrils. The CGCs were in close contact with numerous boutons staining for the different neuronal markers including synaptophysin, nNOS, VIP, NPY, TH, VMAT-2, VACHT, calretinin, and NPY.

CONCLUSIONS. The data indicate a complex integrative function of CGCs. The immunohistochemical and ultrastructural characteristics also indicate that the CGCs may have mechanosensory properties. The complex synaptic information points to a specific regulatory CGC function in parallel with ciliary muscle contraction (accommodation). Axons originating from CGCs mainly supply the choroidal vasculature, thus implicating the CGCs as vasodilative neurons, but single CGCs may also innervate other structures such as nonvascular choroidal smooth muscle cells. (Invest Ophtalmol Vis Sci. 2004;45:361–367) DOI:10.1167/iovs.03-0624

It is well established that the uvea in most species is innervated by two parasympathetic pathways, namely by nerves deriving from the ciliary ganglion and by those deriving from the sphenopalatine ganglion.

Within the uvea of human eyes, an additional group of nitric oxide synthase (NOS) and vasoactive intestinal peptide (VIP)-immunoreactive (IR) neurons has recently been discovered.1–5 Approximately, 2000 of these neurons are located in the inner portion of the ciliary muscle and presumably are involved in fine regulation of the accommodation.3 In addition, approximately 2000 neurons are present in the choroid. Most of the postganglionic nerve fibers of these choroidal ganglion cells (CGCs) join the perivascular nerve fiber plexus that supports the vasodilative innervation of the choroidal vasculature.1–2 A small number of these CGCs is located within the ciliary nerves. The postganglionic nerve fibers of this group of neurons do not enter the choroid but join the nerve fiber plexus that innervates the outflow system.4

When different mammalian eyes were compared, substantial numbers of CGCs in the posterior choroid were found only in higher primates and human eyes containing a fovea centralis.2 CGCs are also present in a number of birds with a specialized accommodative system and one or more foveae.6,7 Further characterization of the CGCs in the duck revealed that, in addition to NO and VIP, the CGCs express galanin. The postganglionic fibers of these cells innervate not only the choroidal vasculature, but also the numerous non-vascular smooth muscle cells (NVSMCs) present in the avian choroidal stroma.6 Duck CGCs are surrounded by tyrosine hydroxylase (TH)/dopamine-β-hydrolase (DBH)-immunoreactive fiber nerves forming synaptic contacts with the CGCs.7 Evidence has shown that these fibers derive from the superior cervical ganglion. The CGCs in the duck also receive calcitonin gene-related peptide (CGRP)-positive efferent collaterals of trigeminal afferents that may indicate precentral reflex arcs.10 Innervation of CGCs by postsynaptic sympathetic fibers in the avian eye, however, indicates a more complex integrative function of the ganglion cell plexus, similar to that in the enteric nervous system.9

It is not yet known whether CGCs in primate eyes have a similar complex function. In the present study we investigated human CGCs to clarify which neurotransmitters are expressed by these cells, to characterize their presynaptic input, and to define their target tissue.

MATERIAL AND METHODS

The studies were performed in 50 human eyes with no previous clinical history of ocular disease, the donors ranging in age between 12 and 95 years. The eyes were obtained during autopsy 4 to 36 hours after death. All donors had given permission to use their tissues for research. Tissue observation was in accordance with the Declaration of Helsinki and the local regulations. Eyes with obvious signs of ocular disease (e.g., macular degeneration, loss of retinal pigmented epithelium) or ocular treatment other than artificial lens implantation (e.g., laser-treatment of the retina) were excluded from the study. A further histologic screening for ocular disease was not performed.

Immunohistochemistry

Thirty-seven eyes of human donors ranging in age between 12 and 95 years were observed 11 to 36 hours after death. All eyes were incised equatorially and fixed either for 4 hours in neutral buffered 4% para-
formaldehyde (PFA) or for 12 hours in Zamboni fixative, containing 4% PFA and 0.01% picric acid. The tissue was then rinsed in phosphate-buffered saline (PBS, pH 7.4) several times. In 27 eyes, the posterior segments were divided into four quadrants and wholemounts of the choroid and sclera were performed. From some of the wholemounts and from the remaining eyes, serial 14- to 16-μm-thick sagittal and tangential sections were cut through the choroid and sclera with a cryostat (Leica, Bensheim, Germany) and mounted on poly-L-lysine-coated glass slides.

Incubation with the primary antibody was performed overnight at room temperature, with the antibody diluted in PBS containing 1% bovine serum albumin and 0.1% Triton X. Control experiments were performed by incubating the sections only with the dilution solution. The primary antibodies used are listed in Table 1. The sections and wholemounts were then rinsed in PBS and incubated for 1 hour with an appropriate fluorescent-dye-conjugated secondary antibody, diluted in PBS (Dianova, Hamburg, Germany). In the double-staining procedure, the steps were the same, but the incubation time of the primary antibody was reduced to 4 to 6 hours.

Most of the antibodies showed sufficient staining in all specimens obtained, regardless of the donor age, time since death, or fixation. For some antibodies, certain special conditions must exist in the tissue to promote sufficient staining. Therefore, the number of tissue specimens was limited for these markers. The antibody against VIP showed sufficient staining of CGCs only in tissue fixed for 24 hours in Zamboni fixative and with a time since death of less than 24 hours. PFA-fixed specimens showed axonal VIP staining but no staining of the CGCs. Staining with the antibody against neuropeptide Y (NPY) was only possible by using cryosections of both PFA- and Zamboni-fixed specimens. Wholemounts did not show sufficient staining of either neurons or nerve fibers.

All sections were mounted with Kaiser glycerol jelly and viewed with a fluorescence microscope (Aristoplan; Leica) or with a confocal laser scanning system (MRC 1000; Bio-Rad, Munich, Germany) attached to an inverted microscope (Diaphot 300; Nikon, Tokyo, Japan). In estimating the size of single neurons, a diameter was defined as the shortest distance between opposing cell boundaries, when passing through the center of the cell.

**Electron Microscopy**

The ultrastructural morphology and the surrounding tissue of the CGCs was investigated in 13 eyes of donors ranging in age between 56 and 91 years. The eyes were obtained 4 to 24 hours after death, incised equatorially, and fixed in 4% PFA and 1% glutaraldehyde for at least 24 hours.

Small samples of the choroid and sclera were prepared and embedded in Epon. Sagittal and tangential semithin serial sections (1 μm thick) were performed and stained with toluidine blue. From localized CGCs, ultrathin sections were performed, stained with lead citrate and uranyl acetate, and viewed with a transmission electron microscope (model EM 902; Carl Zeiss Meditec, Oberkochen, Germany).

**RESULTS**

**Choroidal Ganglion Cells**

In human choroidal sections and wholemounts incubated with antibodies against neuronal NOS (nNOS), all 1820 visualized CGCs showed positive staining (Fig. 1A). No additional CGCs, either identified by their content of autofluorescent lipofuscin or by a nonspecific light fluorescent background in the neuronal cytoplasm, were detected in the nNOS-stained specimens. The nNOS staining was independent of the size (10–37 μm in

![Image](http://iovs.arvojournals.org/pdfaccess.ashx?url=/data/journals/iovs/933227/ on 06/18/2018)
human Choroidal Ganglion Cells

FIGURE 2. Ultrastructural appearance of human CGCs. (A) The nucleus (ZK) contained light-appearing chromatin, and the cytoplasm contained stacks of rough endoplasmic reticulum (rER) and mitochondria (M). (B) Numerous vesicle-filled boutons (arrows) were present around the CNC, forming synaptic contacts (S) with the neuron. (C) In places, the basement membrane of the neurons directly contacted the adjacent collagen fibrils (#). (D) Those CGCs arranged in doublets or clusters of even more cells were partly apposed to each other without being separated by glial cell processes (arrows). Original magnifications: (A) ×3,600; (B) ×26,000; (C) ×17,000; (D) ×25,000.

diameter) and the location of the neurons. Numerous axons within the suprachoroidal nerve plexus and around choroidal vessels were also nNOS IR.

Staining with antibodies against VIP revealed that most of the CGCs (554/362 CGCs evaluated) were VIP IR. Colocalization was documented by double staining with antibodies against nNOS and VIP (Fig. 1B). It appeared that the few VIP-negative CGCs, visualized by autofluorescent lipofuscin or by a nonspecific light fluorescent background, were of smaller size (12–18 μm in diameter).

Less frequent IR in CGCs was seen in choroidal tissue stained with antibodies against calretinin. Wholemounts revealed that 82 of the 146 CGCs evaluated were calretinin IR (Fig. 1C). In some cases, these CGCs were preferentially of larger size (24–36 μm in diameter), but there were also other cases showing mainly small calretinin-positive cells (17–21 μm in diameter).

Only few small CGCs (14–18 μm in diameter) were NPY IR (7/152 evaluated CGCs). Single larger CGCs (25–34 μm in diameter) revealed faint staining with antibodies against galanin (3/189 evaluated CGCs). Most of the neurons in NPY- and galanin-stained sections, however, were unstained. There was no staining of CGCs with the antibodies against TH, DBH, vesicular monoaminergic transporter (VMAT)-2, vesicular acetylcholine transporter (VACHT), substance P (SP), and CGRP (total number of evaluated CGCs: 1340).

Ultrastructural investigations showed, that the CGCs appeared as typical neurons with a large nucleus containing light-appearing chromatin and a clear nucleolus and a cytoplasm containing ribosomes, stacks of rough endoplasmic reticulum, and mitochondria (Fig. 2A). In CGCs of aged donor eyes, lipofuscin granules were frequently seen within the cytoplasm of the neurons. Numerous vesicle-filled boutons were present around the CGC, forming synaptic contacts with the neuron (Fig. 2B). The surface of the CGCs toward the surrounding connective tissue space was only incompletely covered by glial cell processes. In places, the basement membrane of the neurons directly contacted the adjacent collagen fibrils (Fig. 2C) without forming specialized contacts, such as with hemidesmosomes. Those CGCs arranged in doublets or clusters of even more cells were partly apposed to each other without being completely separated by glial cell processes (Fig. 2D). Neither single cells nor groups of CGCs were supplied by or adjacent to capillaries. All CGCs were embedded in a nerve fiber plexus.

Neuronal Boutons Apposed to CGCs

Staining of the choroid with antibodies against synaptophysin revealed the presence of synaptic contacts around each CGC, independent of size (cell diameter) and localization (choroidal versus suprachoroidal; in the choroidal stroma versus in the ciliary nerves). However, not all boutons around the CGCs revealed the same neurochemical composition (Table 2).

All CGCs showed appositions of nNOS and VIP IR boutons (Fig. 3A). Single boutons also stained for NPY (Fig. 3B). Calretinin IR boutons were seen only at CGCs that were not calretinin IR themselves (Fig. 3C). However, only 42 of the 64 calretinin-negative CGCs investigated had close calretinin-positive contacts. The calretinin-negative CGCs that showed no

<table>
<thead>
<tr>
<th>Antibody</th>
<th>CGC (n)</th>
<th>% CGC</th>
<th>Boutons</th>
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<tbody>
<tr>
<td>Neuronal nitric oxide synthase</td>
<td>1820/1820</td>
<td>100</td>
<td>++</td>
</tr>
<tr>
<td>Vasointestinal peptide</td>
<td>554/362</td>
<td>97.8</td>
<td>+ to ++</td>
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<tr>
<td>Calretinin</td>
<td>82/146</td>
<td>56.2</td>
<td>+ (29%)</td>
</tr>
<tr>
<td>Neuropeptide Y</td>
<td>7/152</td>
<td>4.6</td>
<td>+</td>
</tr>
<tr>
<td>Galanin</td>
<td>5/185</td>
<td>1.6</td>
<td>0</td>
</tr>
<tr>
<td>Tyrosine hydroxylase</td>
<td>0/275</td>
<td>0</td>
<td>+ (50%)</td>
</tr>
<tr>
<td>Dopamine-beta-hydroxylase</td>
<td>0/78</td>
<td>0</td>
<td>+ (50%)</td>
</tr>
<tr>
<td>Vesicular monoaminergic transporter 2</td>
<td>0/234</td>
<td>0</td>
<td>+ to ++</td>
</tr>
<tr>
<td>Vesicular acetylcholine transporter</td>
<td>0/348</td>
<td>0</td>
<td>+ to ++</td>
</tr>
<tr>
<td>Substance P</td>
<td>0/215</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Calcitonin gene-related peptide</td>
<td>0/190</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Synaptophysin</td>
<td>0/215</td>
<td>0</td>
<td>+</td>
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*The number of positive CGCs in comparison to all CGCs in the sections evaluated and the percentages of positive CGC are given. The number of surrounding boutons was semiquantified: 0, no boutons; +, single boutons; ++, numerous boutons. If all CGCs did not have positive boutons apposed, the percentage of CGC with positive boutons is added in brackets.*
calretinin IR boutons were always next to calretinin-positive CGCs (Fig. 3D). No colocalization occurred with antibodies against calretinin and VMAT-2 (Fig. 4A), whereas some but not all boutons showed colocalization with antibodies against calretinin and VAChT (Fig. 4B).

Approximately half of the CGCs showed an intense inner-ervation of TH-positive nerve fibers. One third of the TH-IR boutons around the CGCs were colocalized with antibodies against VMAT-2, and others showed colocalization with anti-bodies against VACHT. No staining of boutons in close apposi-
tion to the CGCs was seen using antibodies against galanin, SP, and CGRP.

I nnervation of the Choroidal Vasculature

A dense perivascular network of nerve fibers was seen around the arteries, arterioles, and veins when using antibodies against VACHT, nNOS, or VIP as the marker for parasympathetic, and TH, DBH, VMAT-2, or NPY as the marker for sympathetic nerve fibers. The larger arteries were also accompanied by CGRP-positive nerve fibers. SP-positive nerve fibers formed a fine network in the outer choroid without showing close affiliation to the vessels but rather contacting cells in the choroidal stroma. In the inner choroid, only single TH/VMAT-2/NPY-positive nerve fibers reached the choriocapillary layer, forming a fine network at the level of the precapillary arterioles.

I nnervation of NVSMCs

NVSMCs were identified by their general content of α-smooth muscle actin. A subgroup of these cells (15%) also stained for smooth muscle myosin, as described previously.11

The general presence of nerve fibers in close proximity to NVSMCs was detected by double staining of choroidal sections and wholemounts with antibodies against α-smooth muscle actin and PGP 9.5. However, not every muscle cell was inner-
nerated separately. Approximately 20% of all NVSMCs showed close contact to nerve fibers. As the choroid is densely inner-
nerated and many NVSMCs also run parallel with nerve fiber bundles, the number of innervated cells could only be esti-
mated. Double staining with antibodies against smooth muscle myosin and PGP 9.5 showed that close contact of the NVSMCs with nerve fibers was not exclusively characteristic of the myosin-containing NVSMCs.

Specific neuronal markers that stained nerve fibers with boutons in close contact to the NVSMCs included NPY and VACHT. Choroidal wholemounts double stained for α-smooth muscle actin and NPY (Fig. 5) or α-smooth muscle actin and VACHT and investigated by confocal microscopy revealed that the same estimated number of NVSMCs as for PGP 9.5 received input from NPY- and VACHT-positive nerve fibers. All other
antibodies tested, including nNOS, TH, VMAT-2, VIP, SP, and CGRP, showed no nerve fibers in close relation to the NVSMCs.

**DISCUSSION**

In the present study we showed that numerous CGCs stain not only for nNOS and VIP, but also show immunoreactivity for antibodies against calretinin. Single CGCs were NPY IR. Ultrastructurally, the CGCs showed an incomplete glial sheath and, in places, close contact to surrounding collagen fibrils. The CGCs were in close contact with numerous boutons staining for different neuronal markers, including synaptophysin, nNOS, VIP, NPY, TH, VMAT-2, VACHT, calretinin, and NPY. Because we observed NPY-positive nerve fibers supplying the NVSMCs, it is tempting to speculate that the NPY-positive CGCs may regulate the NVSMCs.

In mammals without a fovea centralis, the vasodilative innervation of the choroid derives from the sphenopalatine ganglion, similar to mammals with foveas, including humans. To some extent, the CGCs in the human choroid also show similarities to the neurons of the sphenopalatine ganglion. There, most of the neurons stain for nNOS and VIP, and single neurons stain for NPY. A colocalization of NPY and VIP is also reported for some neurons in the human sphenopalatine ganglion. However, in contrast to the mammalian sphenopalatine ganglion, where numerous neurons also contain acetylcholinesterase, choline acetyltransferase and acetylcholine, there is no staining for VACHT or choline acetyltransferase present in human CGCs. The CGCs therefore differ from sphenopalatine ganglion neurons in regard to their cholinergic immunohistochemical staining. Whether this failure of staining in human tissue is due to the sensitivity of the transmitters or to a true functional diversity between CGCs and sphenopalatine ganglion neurons remains to be determined.

It is unknown, why primates have neurons in the choroid. In this context it is interesting to note that the CGCs reveal glia-free areas, covered only by a basement membrane, that show contact to the surrounding connective tissue. An incomplete glial sheath is typical for enteric neurons in the gut and gallbladder, but not for other autonomic ganglia. Direct exposure of enteric neurons to periganglionic matrix has been discussed in the context of a possible mechanosensory function. There exist electrophysiological data on direct mechanosensitivity of enteric neurons, and intrinsic primary afferent neurons in enteric ganglia have been characterized recently by both morphologic and electrophysiological methods. Furthermore, vagal afferent terminals in the gastrointestinal tract are typically associated with enteric ganglia where they are located immediately beneath the basement membrane facing the periganglionic collagen matrix. Their low threshold mechanosensory function has been elegantly demonstrated by recent electrophysiological studies. Thus, it is tempting to speculate that, in addition to motor neurons, choroid ganglia harbor sensory neurons, as do enteric ganglia, and that their intimate relationship to the periganglionic matrix hints of a mechanosensory subpopulation of CGCs.

Many of the processes issued by CGCs are targeted to choroidal arteries where they form a dense perivascular plexus. Although hitherto interpreted in favor of a vasomotor function, some of them may represent mechanosensory terminals similar to carotid and aortic baroreceptors of the IXth and Xth cranial nerves, respectively. In conjunction with indications for intrinsic CGC-CGC connections in both primates (this study, discussed below) and birds, this could be interpreted in terms of an intrinsic neuronal mechanism for regulation of choroidal blood flow.

Another hint of the possible existence of a mechanosensory subpopulation of CGGs is calretinin immunoreactivity in some of them. This calcium-binding protein is typically contained in low-threshold spinal mechanosensors (e.g., muscle spindles) in both rat and chicken, in vagal mechanoreceptors in the rat esophagus, and in intrinsic primary afferent type II neurons in the human small intestine. However, the predictive value of immunocytochemical neuronal markers for neuronal function should be discussed with great care because of significant species differences.

The second major finding of this study was the characterization of the numerous synaptic contacts on the CGGs. Besides calretinin, several neurotransmitters were found in the synaptic boutons in close contact with the CGGs. Calretinin, VIP, and nNOS-positive boutons probably demonstrate an inter-CGCs connection, although they could also derive from the sphenopalatine ganglion. The distribution of cholinergic boutons differed markedly from that seen for VIP and nNOS. Because virtually all sphenopalatine ganglion neurons express one of these markers, we concluded that the source of the cholinergic boutons is most likely not only the sphenopalatine ganglion, nNOS-positive neurons also exist in the trigeminal ganglion. but they do not express VIP and most of the trigeminal neurons express SP and CGRP. As these markers do not stain CGCs synaptic boutons, the trigeminal ganglion seems to play no role in regulation of human CGCs. Most of the superior cervical ganglion neurons stain positively for NPY, noradrenalin, TH, and DBH. Most of the TH/VMAT-2 positive endings around the CGCs derive from this source, similar to the duck. These contacts point to a more complex regulation of the CGCs that is generally not common to other intrinsic neurons beside the enteric nervous system. Presumably these contacts are formed to restrain the vasoconstrictive effect of the fibers.

Within the ciliary ganglion, various neurons exist that also show great species differences. In the human, approximately 23% stain positively for TH, whereas only single cells stain for nNOS. All ciliary ganglion neurons are thought to be cholinergic. Therefore, the VACHT-positive-NOS-negative synaptic contacts on CGCs could also derive from this ganglion. As the ciliary ganglion mainly supplies the ciliary muscle, one can speculate that, during ciliary muscle contraction for accommodation, the CGCs receive a copy of this information and therefore can modulate the volume of the choroid to keep the fovea centralis in place. This could explain, why choroidal ganglion cells occur only in species with a well-developed accommodative system and a fovea centralis. In support of this evidence, one can add that the cholinergic nerve fibers are in close contact with the NVSMCs. In contrast to paraffin-embedded sections described in the literature, our careful examination of frozen serial sections revealed that the NVSMCs in the human choroid do not receive input from nNOS- or VIP-positive nerve fibers. Rather, they are innervated by NPY-VACHT-positive axons. However, CGGs may not represent the main source of these axons. This differs from the avian eye, where a clear innervation of NVSMCs deriving from the choroidal ganglion cells could be demonstrated. Human CGGs also do not have CGRP-positive endings being present in the avian eye and leading to the postulation of a local precentral reflex arc. Regarding these differences, the function of the CGGs in birds may be somewhat different from that in the human eye.

In conclusion, we found a rather homogenous staining pattern of CGGs in the human choroid that showed similarities to the pterygopalatine ganglion. In addition, ultrastructural findings and immunofluorescence for calretinin point to a possible mechanoreceptor-like function of the CGGs them-
several, thus explaining the location of these neurons in the choroid proper. The target of the CGCs in the human eye is mainly the choroidal vasculature. This could recently be confirmed by intracellular staining of the processes of human CGCs with neurobiotin. The supply of the CGCs to the choroidal vasculature is regarded as supplementary to a direct vasodilative innervation of the sphenopalatine ganglion. Because of their complex synaptic input the CGCs seem to form a highly regulated intrinsic system that reacts not only to parasympathetic and sympathetic information, but as an integrated local vasoregulatory system for foveal vision during accommodation. The importance of the choroid for proper vision is also known during recovery from form-deprivation accommodation. The choroid for proper vision is also known during recovery from form-deprivation accommodation. The importance of the choroid for proper vision during recovery from form-deprivation accommodation. The importance of the choroid for proper vision during recovery from form-deprivation accommodation. The importance of the choroid for proper vision during recovery from form-deprivation accommodation. The importance of the choroid for proper vision during recovery from form-deprivation accommodation. The importance of the choroid for proper vision during recovery from form-deprivation accommodation. The importance of the choroid for proper vision during recovery from form-deprivation accommodation. The importance of the choroid for proper vision during recovery from form-deprivation accommodation. The importance of the choroid for proper vision during recovery from form-deprivation accommodation. The importance of the choroid for proper vision during recovery from form-deprivation accommodation. The importance of the choroid for proper vision during recovery from form-deprivation accommodation. The importance of the choroid for proper vision during recovery from form-deprivation accommodation. The importance of the choroid for proper vision during recovery from form-deprivation accommodation. The importance of the choroid for proper vision during recovery from form-deprivation accommodation. The importance of the choroid for proper vision during recovery from form-deprivation accommodation. The importance of the choroid for proper vision during recovery from form-deprivation accommodation. The importance of the choroid for proper vision during recovery from form-deprivation accommodation. The importance of the choroid for proper vision during recovery from form-deprivation accommodation. The importance of the choroid for proper vision during recovery from form-deprivation accommodation. The importance of the choroid for proper vision during recovery from form-deprivation accommodation. The importance of the choroid for proper vision during recovery from form-deprivation accommodation. The importance of the choroid for proper vision during recovery from form-deprivation accommodation. The importance of the choroid for proper vision during recovery from form-deprivation accommodation. The importance of the choroid for proper vision during recovery from form-deprivation accommodation. The importance of the choroid for proper vision during recovery from form-deprivation accommodation. The importance of the choroid for proper vision during recovery from form-deprivation accommodation. The importance of the choroid for proper vision during recovery from form-deprivation accommodation. The importance of the choroid for proper vision during recovery from form-deprivation accommo...

**ERRATUM**


It is erroneously mentioned at the end of our general introduction on page 5235 that “immunoglobulins are composed of heavy (H) and light (L) chains, both encoded by rearranged genes assembled from sets of variable (V), diversity (D), joining (J), and constant (C) germline gene segments.” It should be noted that D segments are found only in the heavy chain locus.