# REVIEW ARTICLE

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## NEUROTRANSMITTER SYSTEMS IN THE VERTEBRATE RETINA

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**Abstract**

*Retina putative neurotransmitters, namely, acetylcholine (Ach), gamma-aminobutyric acid (GABA), dopamine (DA), and several amino acid putative neurotransmitters, concerning their anatomical distribution, their synthesis, storage, release, action, termination of activity, and their electrophysiological properties in the retina are reviewed. In addition, specific roles of these neurotransmitters in certain synaptic layers of the retina are also included.*

Most known putative neurotransmitters found in the central nervous system can also be isolated from the retina. In the last fifteen years considerable data on the neurotransmitter functions in the retina have been greatly accumulated. Many putative neurotransmitters in the retina have been identified and their roles in neurotransmission have been partially elucidated. Some neurotransmitter systems in the retina are reviewed here.

### The Cholinergic System in the Retina.

In most vertebrate retinas, acetylcholine meets the criteria for a neurotransmitter as summarized by Phillis (1).

Acetylcholine, its synthetic enzyme (choline acetyltransferase, ChAc), and its catabolic enzyme (acetylcholinesterase, AchE) are present in every vertebrate retina studied so far (for review, see ref. 2). The distribution of ChAc activity among retinal layers is similar for all vertebrate species investigated. The highest activity for ChAc has been found in the inner plexiform layer (garfish, goldfish, frog, turtle, pigeon,

mouse, rat, cat, rabbit and monkey)[3]. Choline, a substrate for ACh synthesis and a product of ACh breakdown, is taken up by retina with a system which possesses characteristics similar to those of the brain (rat and rabbit[4]; chicken[5]). Most AChE is located in the inner plexiform layer in amacrine and in horizontal cells (chicken[6]; rabbit[7]). There are also high concentrations of ACh receptors (cat[8]), primarily nicotinic in nature, in both the inner and outer plexiform layers (rabbit and chicken[9]). Muscarinic cholinergic receptors are also present in the retina (bovine[10]), but in the chicken retina, at least, they are largely concentrated in the inner plexiform layer[11]. $Ca^{++}$ dependent release of ACh *in vitro* occurs through light stimulation (rabbit[12]), and electrophysiology has shown that a variety of cholinergic agents, (e.g., ACh, physostigmine, mecamylamine, scopolamine, dihydro-β-erythroidine) can modify ganglion cell activity (rabbit[13,14]).

**The GABAergic System in the Retina.**

Gamma-aminobutyric acid (GABA) is also a well studied putative neurotransmitter in vertebrate retina. GABA, glutamic acid decarboxylase (GAD), a synthetic enzyme for GABA, and GABA-transaminase (GABA-T), a degradative enzyme for GABA are present in every vertebrate retina studied (for review, see ref. 2). GABAergic systems are present in the inner plexiform layer, in some amacrine and ganglion cells, and in horizontal cells (guinea pig, goat, cat, rabbit[15]; goldfish[16]; rat[17]; guinea pig, rat, cat, monkey and human[18]; rabbit[19,20]).

In addition, a high affinity uptake system for GABA in retina has been relatively well characterized (chicken[21]; frog[22]; rabbit[23]; rat[24]). GABA uptake sites, located by autoradiographic techniques, vary according to species. GABA is largely taken up by glial elements, namely Müller cells, and by some amacrine cells in the rat[17]. In the rabbit, it is also taken up by Müller and amacrine cells, as well as by the inner plexiform layer[19,25]. In most other species, it is taken up by amacrine cells, or in some cases by horizontal cells (pigeon, chicken[26]; goldfish[27]).

Radioactive GABA, which has been taken up by retina, can be released by direct electrical stimulation, by exposure to depolarizing levels of potassium (frog[28]; rat[22]), or by flashing light stimulation (rabbit[29]). In 1977, Redburn[30] showed that both $^{14}C$-GABA uptake and $Ca^{++}$ dependent release sites were more pronounced in the retinal $P_2$ fraction (which contains inner plexiform layer synaptosomes) than in the retinal $P_1$ fraction (which contains outer plexiform layer synaptosomes). GABA or its antagonists (e.g., picrotoxin) can modify the activities of several kinds of ganglion cell receptive fields in the retina[14,31-34]. Enna and Snyder[35] have shown specific binding of $^{3}H$-GABA to GABA receptors in the pig, cow and sheep retina.

**Dopamine (DA)**

Dopamine is one of the most extensively studied putative neurotransmitters in the retina. Its characteristics comply with most criteria used for neurotransmitters, and its presence in the retina has been demonstrated with chemical[36], histochemical and fluorescence microscopic techniques[37-39]. Dopaminergic cell bodies generally lie in the inner part of the inner nuclear layer (the amacrine cell layer) and synapse in the inner plexiform layer. DA cells represent approximately 10% of the cells within the amacrine cell layer in rabbit retina. In other species dopamine-containing cells in the inner nuclear layer send out processes which connect the two plexiform layers. The processes of these interplexiform cells are sparsely spread in both plexiform layers in fish and in some New World monkeys. There are no dopaminergic interplexiform cells in the rabbit retina[40].

Kramer[41] has shown that DA has met most biochemical criteria for its establishment as a neurotransmitter in rabbit retina: (a) DA is synthesized in the retina; (b) its degradative enzymes are present; (c) DA has an inhibitory effect on the electrical activity of the retina, and (d) it is released in increasing amounts as flash frequency increase when the retina is stimulated by light flashes. It has also been shown that radioactive DA is taken up by retinal cells whose locations appear to be the same as those which show fluorescence for endogenous DA[42].

In addition, iontophoretically applied dopamine has an inhibitory effect on retinal ganglion cell activity (cat[31]), and when isolated retina is perfused with dopamine, different retinal ganglion cells are excited or inhibited[14]. Retina has been shown to have a dopamine-sensitive adenylate cyclase whose activity can be blocked by haloperidol, chlorpromazine and fluphenazine (calf[43]). Thomas and coworkers[44] showed that in rabbit retina the activity of DA sensitive adenylate cyclase was higher in the $P_2$ retinal synaptosomal fraction than in the $P_1$ fraction. They also showed that DA released from $P_2$ was $Ca^{++}$ dependent, whereas that from $P_1$ was not. Monoamine oxidase (MAO) the degradative enzyme for biogenic amines, has also been found in the retina[45].

**Other Amino Acids**

In the last decade, several putative amino-acid neurotransmitters have been studied in the retina. Starr[46] has chemically demonstrated the presence of taurine, glycine, aspartate and β-alanine in the retina of vertebrates. Uptake systems for these compounds have also been demonstrated, both biochemically and autoradiographically[25,46-49]. These particular amino acids have electrophysiological effects on retinal neurons[14,50-52]. When chicken retina is stimulated by high intensity light flashes, a $Ca^{++}$ dependent release of labeled taurine has been found[52]. A light stimulated release of glycine has been shown in rabbit retina[53], and the spontaneous and light evoked release of glutamate

has been demonstrated[54]. Glutamate and aspartate are taken up by both retinal synaptosomal fractions from rabbit retina[55]. The uptake rates for aspartate and glutamate were higher in the $P_2$ retinal synaptosomal fraction than in $P_1$. Both aspartate and glutamate are competitive inhibitors of each other's high affinity uptake systems in the rabbit retina[55].

In summary, most putative neurotransmitter systems which have been identified in the brain have also been demonstrated in the retina. However, the specific functions of the putative neurotransmitters in particular neuron types are yet to be determined.

**Neurotransmission in the Outer Plexiform Layer (OPL) of the Retina**

The anatomy of photoreceptor synapses which shown numerous synaptic vesicles in presynaptic terminals and a prominent gap between junctional elements, suggests that synaptic transmission between vertebrate photoreceptors and second-order neurons (horizontal and biopolar cells) is chemically mediated. Furthermore, intracellular recordings from photoreceptor, bipolar and horizontal cells show a significant synaptic delay between pre- and post-synaptic responses (mudpuppy[56]), which is also characteristic of chemical transmission. There is, in addition, the finding that hyperpolarizing electrical stimulation of horizontal cells will change the polarity of the cells in the same direction as will light stimulation (tortoise[57]; *Necturus* and *Gekko*[58]). Photoreceptors are partially depolarized in the dark by a steady inward flow of $Na^+$ at the outer segments. Light stimulation, however, decreases $Na^+$ conductance in the outer segment and causes hyperpolarization of the photoreceptor. Divalent ions (e.g., $Co^{++}$, $Mg^{++}$ and low $Ca^{++}$ concentrations) caused horizontal cells of the retina to hyperpolarize (skate[59]; mudpuppy[60]; turtle[61]). The effects of these ions on reducing the release of putative neurotransmitters in other systems, lends support to the hypothesis that $Mg^{++}$, $Co^{++}$ and low $Ca^+$ should also decrease the release of excitatory neurotransmitter(s) from photoreceptor terminals and thereby cause hyperpolarization of horizontal cells. Schacher and coworkers[62] showed that in the dark there is an increase in the uptake of horseradish peroxidase by frog photoreceptor terminals. This reflects an increase in the release of neurotransmitter(s) at these particular synpases. Cobalt ions which usually block the release of neurotransmitters reduce the horseradish peroxidase uptake into rod terminals (frog[63]). These data seem to verify the findings of Schacher and coworkers[62].

It is still not clear which specific neurotransmitters are endogenous to the photoreceptors. It has recently been shown that certain amino acids, such as glutamate and aspartate, can powerfully depolarize the horizontal cells of the skate retina[64]. They are, therefore, likely candidates for excitatory neurotransmitters. Gerschenfeld and Piccolino[65] demonstrated that acetylcholine may also be an excitatory putative neurotransmitter at the photoreceptor nerve terminals. Their studies showed that the

hyperpolarizing light responses of horizontal cells are reduced by a large number of cholinergic antagonists. This action results from an increase in the membrane resting potential (hyperpolarization) in the dark, and indicates that in the dark ACh may be tonically released from photoreceptors in order to keep the horizontal cells depolarized. There are several other lines of evidence which support this suggestion; some photoreceptors of turtle retina, for example, can synthesize ACh but not GABA, DA, or norepinephrine (goldfish and turtle[66]). Vogel and Nirenberg[9] showed that both the outer and inner plexiform layers in chicken and rabbit retinas have a high density of nicotinic cholinergic receptors. There are indications that some horizontal cells may be postsynaptic components of cholinergic synapses in the outer plexiform layer (OPL). Acetylcholinesterase (AChE), for instance, has been demonstrated in the rough endoplasmic reticulum of some horizontal cells[67].

Most known putative neurotransmitters are absent in horizontal and bipolar cells. The retinas of both cold and warm blooded vertebrates which have an abundance of cones also seem to have a great number of horizontal cells which take up $^3$H-GABA (goldfish[68]; frog[69];pigeon and chicken[26]). Lam and coworkers[70] have recently demonstrated GABAergic horizontal cells in the retinas of catfish. Further studies by Marc and coworkers[27] have shown, autoradiographically, an uptake mechanism for $^3$H-GABA which is specifically localized in red cone horizontal cells of goldfish. There horizontal cell neurites are postsynaptic components of the outer plexiform layer. The possible putative neurotransmitters in other kinds of horizontal cells are unknown. Glycine, dopamine, glutamic acid, aspartic acid and taurine are not taken up by many horizontal cells in goldfish[27]. Ehinger[20] failed to show the presence of any catecholamines or indolamines in horizontal cells. Lam[71] has demonstrated that isolated horizontal cells cannot synthesize catecholamines, indolamines or ACh from precursors. These later findings lessen the probability that they are neurotransmitters in horizontal cells.

**Neurotransmission in the Inner Plexiform Layer (IPL) of the Retina.**

Ultrastructural studies of the inner plexiform layer show synaptic vesicles in the presynaptic elements of bipolar and amacrine cell contacts (rabbit[72]; cat[73]; mudpuppy[74]; frog and primates[75]). These data indicate the presence of chemical synaptic transmission in the IPL. Most data on neurotransmission in this layer has been obtained indirectly from studies on the neuronal activities of amacrine or bipolar cells which are presynaptic neurons of the inner plexiform layer, and of ganglion cells which are postsynaptic components of the IPL[76]. At present, no known putative neurotransmitters have been identified in either bipolar or ganglion cells. Most known putative neurotransmitter systems, however, have been found in amacrine cells which are presynaptic neurons of the inner plexiform layer.

Amacrine cells have been shown to take up labeled choline for acetylcholine (ACh) synthesis (rabbit[77]; chicken[5]). There is also a high activity rate for acetylcholinesterase (AChE) in amacrine cells (rabbit[7]; pigeon, ground squirrel, rabbit, rat and cat[78]). In addition, the activity of retinal choline acetyltransferase (ChAc) is highest in the inner plexiform layer (cat, monkey, mouse, rabbit, frog, turtle, garfish and goldfish[3]). And it has also been demonstrated that the inner plexiform layer of rabbit, chicken and rat retinas have a very high density of cholinergic receptors[9].

Amacrine cells also show an uptake of GABA (chicken[26]; rabbit[19]; goldfish and skate[70]). Furthermore, the activity of glutamic acid decarboxylase (GAD) and GABA-transaminase (GABA-T) are very high in the inner plexiform layer (rabbit[79]). Glycine has been shown to be taken up primarily by amacrine cells and by the inner plexiform layer (rabbit[48]). Amacrine cells also show an uptake of dopamine (DA) (rabbit[38]; cat[42]). Fluorescent microscopic techniques have demonstrated an abundance of dopamine in amacrine cells and in their processes in inner plexiform layer (rabbit[39]; rat, guinea pig, rabbit[80]).

It can be seen that amacrine cells are the main sources of known putative neurotransmitters in the inner plexiform layer. There is also considerable evidence that putative neurotransmitters of the inner plexiform layer are involved in a variety of ganglion cell receptive fields. Masland and Ames[13], for example, have demonstrated that cholinergic amacrine synapses in the inner plexiform layer may mediate on-center or directionally selective fields of ganglion cells. They were also able to show that these receptive fields could be modified by cholinergic agonists, antagonists and anticholinesterases.

In 1969, Ames and Pollen[14] had already demonstrated the modification of on-off and off ganglion receptive fields by cholinergic, adrenergic and serotoninergic agents, as well as by amino acids. They hypothesized that amacrine cells which possessed these suspected neurotransmitters might in some ways be involved with the receptive fields.

Picrotoxin and strychnine (antagonists of GABA and glycine respectively) can both modify the activities of direction-sensitive ganglion cells in the retina (rabbit[32]). These drugs can also modify inhibitory and excitatory postsynaptic potentials (IPSP and EPSP) in on-off ganglion cells[76]. In addition, picrotoxin and strychnine modify activities in a variety of other complex receptive fields[34]. These findings further support the hypothesis that GABAergic and glycinergic amacrine cells may be involved in the neurotransmission processes of these receptive fields. The involvement may take place in the inner plexiform layer with amacrine cells contributing GABA, ACh, DA and glycine as putative neurotransmitters.

Ultrastructural data indicate that retinas may possess a large number of synaptosynaptic synapses in both the outer and inner plexiform layers[72-75]. The number

of extensive biochemical studies of the synaptosynaptic interactions involving specific putative neurotransmitter is limited so far.

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## บทคัดย่อ

ได้รวบรวมสรุปรายงานการวิจัยในระยะ 15 ปี ที่แล้วมาเกี่ยวกับสารเคมีที่ทำหน้าที่เป็นนิวโรทรานสมิตเตอร์ในเรตินาของลูกตา ตัวอย่างเช่น อะซีทิลโคลีน, แกมม่าอะมิโนบูทีริกแอซิด, ดอปปามีน, และอะมิโนแอซิดหลายตัวที่เป็นนิวโรทรานสมิตเตอร์. ได้สรุปเกี่ยวกับตำแหน่งที่พบ, การสังเคราะห์, การเก็บสะสม, การหลั่งสารเหล่านี้จากปลายประสาท, การทำงาน, การกำจัดสารเหล่านี้, และได้พูดถึงคุณสมบัติของสารเหล่านั้นในการดัดแปลงสรีรวิทยาทางไฟฟ้าของเซลล์ต่าง ๆ ในเรตินา. และได้สรุปถึงหน้าที่การทำงานในปลายประสาทในชั้นต่าง ๆ ของเรตินาของลูกตาเท่าที่รู้ในปัจจุบัน.