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**PRESYNAPTIC INPUTS OF BIPOLAR CELLS ONTO
RETINAL GANGLION CELLS**

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ABSTRACT: Vision loss is debilitating to those that experience it, and a primary cause of incurable vision loss is glaucoma. Glaucoma, which causes damage to the optic nerve and eventually the death of neurons in the retina, disrupts the retinal circuitry and affects about 60 million people worldwide. To help those impaired, investigation of the retinal synaptic pathways is essential to produce new therapies that address optic nerve damage. The way the synaptic circuitry works in the retina is by bipolar cells (BCs) that process and transmit visual information from photoreceptors to retinal ganglion cells (RGC). We developed a novel technique to label the presynaptic inputs of RGCs, which allowed us to identify the unique pattern of BC input of specific RGC types. The function-specific RGCs that we analyzed were junctional adhesion molecule B (JAMB) and bistratified dendrite (BD). BCs found in the RGCs were divided into subtypes based on whether their primary input was from a rod or cone photoreceptor. Additionally, they were divided into their primary synaptic output; either the ON or OFF layer of the inner plexiform layer. The classification was produced from ten subtypes of cone BCs and one rod BC. We found that JAMB RGCs primarily receive input from OFF BCs, whereas BD RGCs receive input from both ON and OFF BCs. Further, BC inputs onto RGCs in CD3 ζ mice were analyzed. CD3 ζ , the key component of a Major Histocompatibility Complex Class I receptor, is expressed in RGCs. Elimination of CD3 ζ causes axon and dendritic morphology changes which results in presynaptic changes. We found that the elimination of CD3 ζ in BD and JAMB RGCs significantly disrupted their normal presynaptic circuitry. Identifying these presynaptic changes aid in identifying the molecular pathways that underlie retinal synaptic circuitry. This result may lead to innovative therapies to restore vision to those persons who suffer from vision loss.