Rod Monochromacy		Blue Cone Monochromacy
Severe aversion to light.	**Hemeralopia** **Day Blindness**	More variable- some have severe aversion while some show much less aversion to light.
Complete loss of color vision is the expected with only rod receptors functioning. Some RM patients reports they see some colors. This could the the result of judgements based on brightness not color. Possibly some minimal cone function might still be working in some patients. The Berson Color Vision Test may help differentiate RM patients from BCM.	**Color Vision**	All have incomplete loss of color vision. They have both rod receptors and blue cone receptors functioning. Typically they have some color detection along the yellow- blue axis. The Berson Color Vision Test may help differentiate RM patients from BCM.
Nystagmus may be first sign of problems often starting about three to six months after birth. It usually lessens or disappears by adulthood which may improve vision.	**Nystagmus**	Nystagmus may be first sign of problem often starting about three to six months after birth. It usually lessens or disappears by adulthood which may improve vision.
Typical cases show 20/120 to 20/200. This is a variable range of visual acuities and may be worse in young children.	**Visual acuity**	Typical cases 20/60 to 20/200 This is a variable range of visual acuities and may be worse in young children. A significant portion of Blue Cone Monochromats have better visual acuity than RMs. However, some BCMs are just as impaired as RMs.
First signs common about three months after birth.	**Onset of Condition**	First signs common about three months after birth.

Rod Monochromacy		Blue Cone Monochromacy
Autosomal recessive: Each parent provides one gene. Requires two genes to express the condition.	**Inheritance**	X-linked: from the mother to the son. Requires only one gene. Because women have two X chromosomes and one good gene is enough to prevent the condition.
1, 2, 8 and 10 chromosones	**Chromosones**	X chromosone
Currently these four genes have been documented, but there are still genetic causes not yet identified. CNGB3, CNGA3, GNAT2 and PDE6C	**Genes**	Currently these two genetic processes have been well documented, but other genes may exist. Direct mutation of the red and green opsin genes: OPN1LW OPN1MW on Xq28 Indirect affect OPN1LW OPN1MW by mutation at the LCR, Locus of Control Region, also on Xq28. This controls the opsin genes. Thus both methods affect the red green opsin areas on Xq28 but in different manners.
Typically one child and/ or some siblings only. Usually no other cases.	**Family Presentation**	Grandfather to carrier daughter to male child Some male cousins are usually affected.

Rod Monochromacy		Blue Cone Monochromacy
All children of an RM parent will be carriers of the condition due to getting one of the two RM genes from the affected parent. It would require the unaffected parent to be a carrier for any children to also have RM. This is a rare disease so the chance is extremely rare for the unaffected parent to be a carrier. The chance of a child of an RM parent being born is "like hitting the lottery twice in a row". It could be possible, but extremely unlikely. However if both parents had RM, all children would have RM.	**Status of Offspring**	All daughters will be carriers, but will not have BCM. All sons of a BCM parent will not have the condition. All daughters have a 50% chance of their male children having BCM. Thus the grandsons of males with BCM have a 50 % chance of having BCM.
Males and Females	**Gender**	Males There is a theoretical model as to how a female might develop BCM, but the odd have been suggested as 1 in 6 billion.
More than 25% of clinical RMs do not present with the established genetic findings on routine clinical genetic testing. Thus, we still may not have identified all the genetic causes.	**Cases of Genetic Uncertainty**	Some studies suggest the as many as 25% of those with the clinical BCM presentation do not have the established genetic defects at Xq28 for BCM. Thus we still may not have identified all causes.

Rod Monochromacy		Blue Cone Monochromacy
Incidence studies suggest about 1 in 30,000 births.	**Incidence**	Most studies estimate 1 in 100,000. Some suggest a maximum 1 in 50,000 births
Usually stable over lifetime.	**Stability**	Usually stable over the lifetime. However, cases of Cone Dystrophy 5 which may mimic BCM can progress. This dystrophy is linked to xq26.1-qter, but also affects the opsin genes at xq28.
Some are able to become bioptic drivers.	**Driving**	Because of the milder hemeralopia (day blindness) and better visual acuity in some, more BCM patients are able to drive and many can do it without bioptics.