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RETINITIS PIGMENTOSA: CLINICAL PICTURE AND DIAGNOSTIC INSIGHTS

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Abstract: Retinitis Pigmentosa (RP) is an inherited retinal degenerative disorder primarily affecting rod photoreceptors, followed by secondary cone cell loss. The disease manifests clinically with progressive night blindness, peripheral vision constriction, and eventual tunnel vision. As degeneration advances, central vision and color perception are also impaired. Modern diagnostic techniques such as full-field electroretinography (ffERG), spectral-domain optical coherence tomography (SD-OCT), and fundus autofluorescence (FAF) imaging play crucial roles in assessing disease severity and progression. This article summarizes the key clinical manifestations of RP, emphasizing the correlation between photoreceptor degeneration and functional visual loss, and reviews multimodal diagnostic approaches essential for early detection and management.
Key words: Retinitis Pigmentosa; clinical symptoms; night blindness; tunnel vision; photoreceptor degeneration; visual field loss; electroretinography; optical coherence tomography; fundus autofluorescence; retinal imaging.

Introduction: Retinitis Pigmentosa (RP) encompasses a diverse group of inherited retinal disorders characterized by the progressive loss of photoreceptor cells, resulting in gradual visual impairment [1]. The disease typically begins with dysfunction of rod photoreceptors, leading to night blindness and reduced peripheral vision, followed by cone degeneration that compromises central vision and color perception [2]. The prevalence of RP is estimated at approximately 1 in 4,000 individuals worldwide, with over 80 genes identified as causative [18]. Early diagnosis through electrophysiological and imaging modalities allows for better monitoring of disease progression and patient counseling [3]. Understanding the clinical spectrum of RP is critical for differentiating it from other retinal dystrophies and for guiding patients toward emerging therapeutic interventions [4].

Aim of study: is to analyze the characteristic clinical features of Retinitis Pigmentosa and their relationship with the underlying photoreceptor degeneration.

The paper seeks to describe the sequence of visual impairment, including nyctalopia, progressive peripheral vision loss, and central visual decline, while reviewing the diagnostic value of electrophysiological testing and multimodal imaging in evaluating disease progression.

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Clinical Symptoms: RP involves the primary degeneration of rods, followed by the secondary degeneration of cones [1]. As each photoreceptor type plays a specific role in the establishment of vision, there is a classic order in which the clinical symptoms of RP manifest. Due to the initial loss of rod photoreceptors, which are primarily used for vision in dim light conditions and peripheral visual functions, patients experience difficulty or inability to see in dark or dimly lit environments, which is commonly known as 'night blindness' or nyctalopia [5]. The second symptom found in RP is a progressive loss of peripheral visual fields, although this may be unnoticed in the initial stages of disease due to compensating mechanisms [6].

When the degeneration of photoreceptors further expands, so do the visual field defects. Constriction of visual fields progresses over time, eventually reaching the central part of the visual field. In advanced stages of RP, only a small residual central island of visual field may remain—with or without peripheral remnants—which results in severely constricted vision known clinically as 'tunnel vision' (Fig. 1) [7,8]. Because of visual field loss, one of the major perceived difficulties in patients with RP is mobility, which requires input from both central and peripheral vision [9].

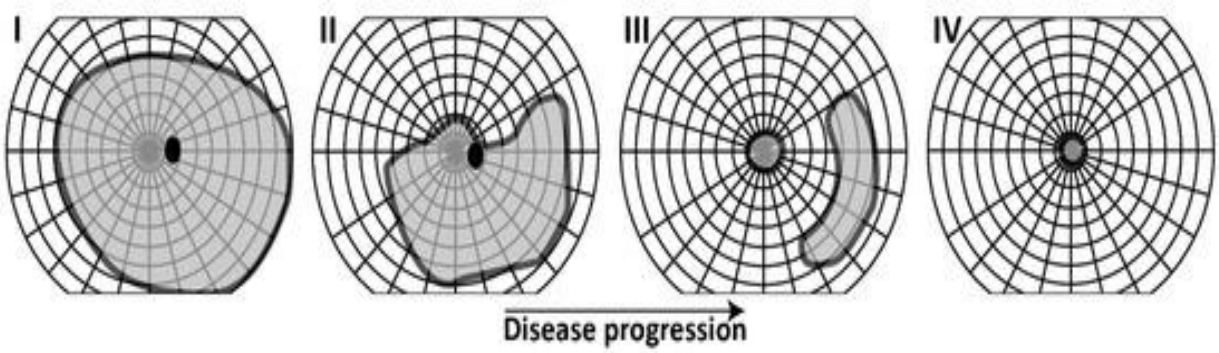

Fig. 1. Illustrative example of typical visual field progression in a patient with retinitis pigmentosa using kinetic perimetry.

Visual fields can be within normal limits in early stages of disease (**I**), although visual field defects may already be present but not detectable within the used target stimulus. With time, constriction of the visual fields occurs, with defects typically being symmetric and expanding more rapidly outwards and slower inwards (**II**,**III**). Ultimately, a small central remnant of visual field may remain in end-stage retinitis pigmentosa, which is commonly experienced and known as 'tunnel vision' (**IV**). Note that the clinical course of visual field loss varies between individuals and may follow a progression pattern that is different from this illustration.

Cone photoreceptors, which are densely packed in the macula, are responsible for visual acuity and color vision [10]. Gene variants that target specifically rods but not cones (e.g., disease-associated variants in the *RHO* gene affecting rhodopsin, a rod-specific protein) can still cause death of cone photoreceptors. It remains unclear how cone degeneration in these specific circumstances occurs. Several theoretical concepts have been suggested for the secondary degeneration of cones, including the lack of trophic factors, such as rod-derived cone viability factor, nutrient shortages, oxidative stress and microglial activation, which are induced following rod photoreceptor apoptosis [10-13]. Loss of cone photoreceptors leads to a gradual loss of central vision once sufficient cones in the macula are compromised. This

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process can ultimately lead to severe visual impairment or even functional blindness based on criteria established by the World Health Organization [14]. Importantly, most patients with RP in advanced stages of their disease will likely retain some degree of residual vision, and total blindness, i.e., no light perception, is uncommon [15]. Previous studies reported that 7–8% of patients with generalized RP end up with a vision of counting fingers or worse in their fourth or fifth decade of life, while less than 1% of RP patients progress to no light perception [15-16]. In addition to central vision loss, patients may lose color vision, and they may have increased sensitivity to light (i.e., photophobia) [5, 17]. Photopsia, i.e., seeing light flashes or static noise when no light enters the eye, is very common in RP, possibly due to reduced afferent nerve impulses or spontaneous signaling from the inner retina [18].

Clinical Testing and Evaluation. (Diagnostic insights) Clinical evaluation of patients with presumed RP consists of a comprehensive ophthalmic examination that includes best-corrected visual acuity (BCVA), intraocular pressure, slit-lamp, fundus, perimetric, retinal imaging, and electrophysiological evaluation.

Electrophysiological Testing Electrophysiological testing plays a major role in the diagnosis and follow-up of RP, as well as the differentiation of RP from other diagnoses. Among all electrophysiological tools, full-field electroretinography (ffERG) is the most common technique used for diagnosing RP, which follows the guidelines established by the International Society for Clinical Electrophysiology of Vision (ISCEV) [19]. In brief, the ffERG evaluates the retinal function in response to light stimulus. A dim white single flash in a dark-adapted eye (i.e., scotopic test conditions) invokes a rod response, whereas a flickering white light (30-Hz) in a light-adapted eye elicits a cone response [19]. When RP becomes detectable in ffERG, i.e., when the retina is sufficiently affected, scotopic responses demonstrate a significant reduction in amplitudes of both a- and b-waves, which are responses mostly derived from photoreceptor and bipolar cells, respectively (Figure 2). Ultimately, both scotopic and photopic responses can be fully extinguished and are non-recordable in end-stage disease [5]. Other diagnostic tools that measure retinal function include multifocal ERG (mfERG), which assesses macular function, and dark adaptometry, which measures the time it takes for photoreceptors to retain maximal sensitivity following photoreceptor bleaching [20-22]. These other electrophysiological testing tools play a smaller role in the initial diagnosis of RP, and are instead sometimes used to complement ffERG/clinical findings and to rule out other potential diagnoses.

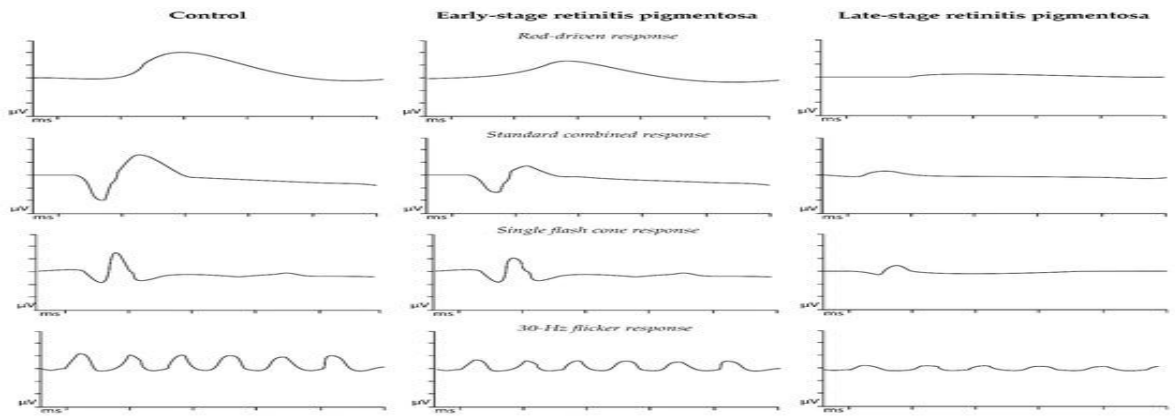

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Fig. 2. Example full-field electroretinography recordings in a healthy patient and in patients with different disease stages of RP.

Different stimuli are used to establish the diagnosis of retinitis pigmentosa, which is based on the guidelines of the International Society for Clinical Electrophysiology of Vision (ISCEV). In patients with advanced stages of diseases, rod-driven responses are severely diminished or even absent, whereas residual cone-driven responses may still remain.

Multimodal imaging, including widefield fundus imaging, spectral-domain optical coherence tomography (SD-OCT), and fundus autofluorescence (FAF) imaging, is used to visualize the extent of retinal degeneration in patients with RP. Widefield fundus imaging yields a comprehensive overview of the retina, which can be used to monitor progression in RP. Multiple studies have used structural markers on SD-OCT, such as the central retinal thickness and/or ellipsoid zone (EZ) band width, as another means of tracking disease progression [23,24,25,26,27,28,29]. In addition, SD-OCT yields the detection of secondary complications associated with RP, such as the presence of CME and epiretinal membrane. FAF is a non-invasive imaging technique that measures the level of autofluorescent lipofuscin components in the photoreceptors and RPE. A hyperautofluorescent macular ring can typically be observed in earlier disease stages of RP and indicates the transition zones between healthy and degenerating retina, which are often accompanied by progressive thinning of the EZ, external limiting membrane (ELM) and outer nuclear layer (ONL) on SD-OCT (Figure 3) [30]. It is important to note that hyperfluorescent rings are not specific to RP and can also be seen in other retinal diseases such as cone–rod dystrophies. Gradual constriction of hyperautofluorescent rings towards the central retina occurs in RP, whereas gradual expansion of the ring is observed in cone–rod dystrophies due to differences in order of photoreceptor degeneration. In advanced stages of RP, when extensive photoreceptor and RPE degeneration has occurred, resulting in the depletion of lipofuscin levels in the retina and RPE, extensive hypo-autofluorescent areas are seen on FAF (Fig. 3)

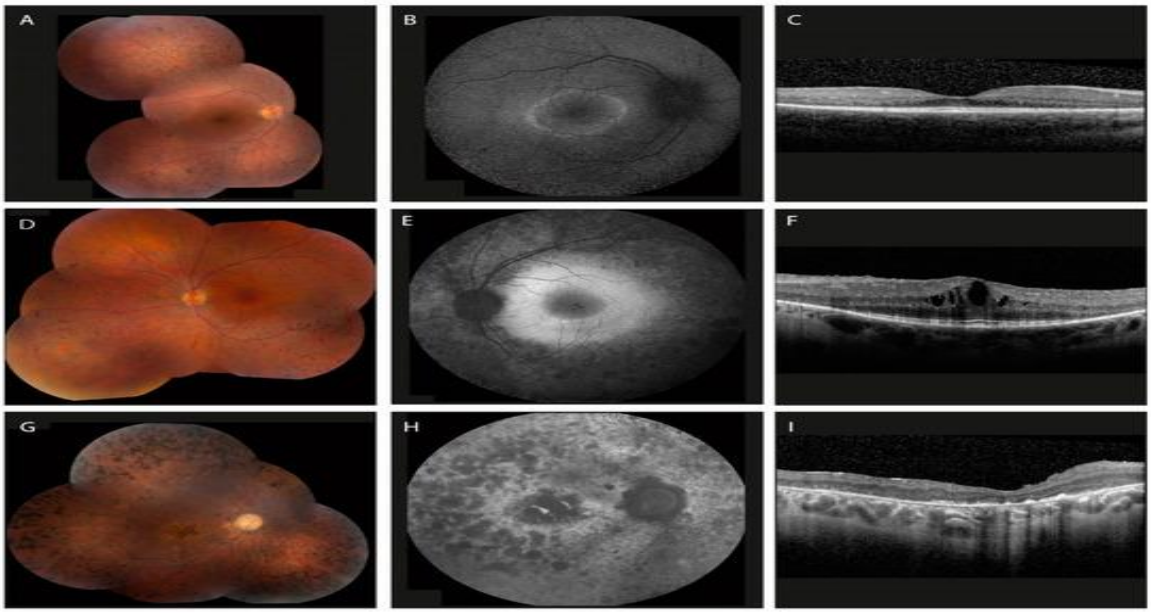

Fig. 3. Multimodal imaging in three patients with retinitis pigmentosa (RP). (**A**–**C**):

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Multimodal imaging in a patient with RP caused by a variant in the *RHO* gene showing the clinical hallmarks of RP, including attenuated vessels and bone-spicule-like hyperpigmentation in the (mid)peripheral retina (**A**). On autofluorescence imaging, a small hyperfluorescent ring is observed in the macula (**B**). Spectral-domain optical coherence imaging shows a relatively intact central retina with loss of the outer retinal layers (i.e., ellipsoid zone and external limiting membrane) outside this area (**C**). (**D**–**F**): Multimodal imaging in a different patient with *RHO*-associated RP reveals hypo-autofluorescent areas in the midperipheral retina and around the vascular arcades, with a broad hyperautofluorescent ring-like region in the macula (**E**). The foveal area shows hypo-autofluorescence some petaloid, likely due to the presence of cystoid macular edema that masks underlying autofluorescence (**F**). SD-OCT confirms the presence of CME, along with the perifoveal loss of the outer retinal layers. (**G**–**I**): More extensive bone-spicule-like hyperpigmentation is observed in this patient with advanced *RPGR*-associated RP, showing not only hyperpigmentation in the midperipheral retina, but also in the fovea (**G**). Autofluorescence imaging (**H**) shows some residual regions of normal or increased autofluorescence, together with regions of mottled hypo-autofluorescence that also include the fovea. As expected, there is clear outer retinal and retinal pigment epithelium on optical coherence tomography (**I**).

Conclusion: Retinitis Pigmentosa represents a heterogeneous group of hereditary retinal dystrophies with well-defined yet variable clinical manifestations. The hallmark features—night blindness, progressive peripheral vision loss, and eventual tunnel vision—reflect the sequential degeneration of rod and cone photoreceptors. Modern multimodal imaging and electrophysiological tests have greatly enhanced the understanding and monitoring of RP progression. Although no curative treatment currently exists, advances in gene therapy, retinal prosthetics, and neuroprotective strategies offer hope for slowing degeneration and improving visual outcomes. Comprehensive clinical evaluation and early intervention remain essential for optimizing patient quality of life and for the successful application of future therapeutic innovations.

References:

1. Hartong, D.T.; Berson, E.L.; Dryja, T.P. Retinitis pigmentosa. *Lancet* **2006**, *368*, 1795–1809. [Google Scholar] [CrossRef] [PubMed]
2. Ferrari, S., Di Iorio, E., Barbaro, V., Ponzin, D., Sorrentino, F. S., & Parmeggiani, F. (2011). *Retinitis pigmentosa: Genes and disease mechanisms*. Current Genomics, 12(4), 238–249.
3. Broadhead, G. K., Grigg, J. R., & McCluskey, P. (2019). *Autoimmune retinopathy: A review of current concepts and practices*. Clinical & Experimental Ophthalmology, 47(4), 512–524.
4. Sahel, J. A., & Roska, B. (2013). *Gene therapy for blindness*. Annual Review of Neuroscience, 36, 467–488.
5. Hamel, C. Retinitis pigmentosa. *Orphanet J. Rare Dis.* **2006**, *1*, 40. [Google Scholar] [CrossRef] [PubMed]
6. Weleber, R.G.; Gregory-Evans, K. Chapter 17-Retinitis Pigmentosa and Allied Disorders. In *Retina*, 4th ed.; Ryan, S.J., Hinton, D.R., Schachat, A.P., Wilkinson, C.P., Eds.; Mosby: Edinburgh, UK, 2006; pp. 395–498. [Google Scholar] [CrossRef]

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https://westerneuropeanstudies.com/index.php/2
ISSN (E): 2942-190X Open Access| Peer Reviewed
This article/work is licensed under CC Attribution-Non-Commercial 4.0

7. Grover, S.; Fishman, G.A.; Brown, J. Patterns of visual field progression in patients with retinitis pigmentosa. *Ophthalmology* **1998**, *105*, 1069–1075. [Google Scholar] [CrossRef]
8. Timmis, M.A.; Allsop, J.; Baranian, M.; Baker, J.; Basevitch, I.; Latham, K.; Pardhan, S.; Van Paridon, K.N. Visual Search Behavior in Individuals With Retinitis Pigmentosa During Level Walking and Obstacle Crossing. *Investig. Opthalmol. Vis. Sci.* **2017**, *58*, 4737–4746. [Google Scholar] [CrossRef]
9. Campochiaro, P.A.; Mir, T.A. The mechanism of cone cell death in Retinitis Pigmentosa. *Prog. Retin. Eye Res.* **2018**, *62*, 24–37. [Google Scholar] [CrossRef] [PubMed]
10. Georgiou, M.; Grewal, P.S.; Narayan, A.; Alser, M.; Ali, N.; Fujinami, K.; Webster, A.R.; Michaelides, M. Sector Retinitis Pigmentosa: Extending the Molecular Genetics Basis and Elucidating the Natural History. *Am. J. Ophthalmol.* **2021**, *221*, 299–310. [Google Scholar] [CrossRef] [PubMed]
11. Aït-Ali, N.; Fridlich, R.; Millet-Puel, G.; Clérin, E.; Delalande, F.; Jaillard, C.; Blond, F.; Perrocheau, L.; Reichman, S.; Byrne, L.C.; et al. Rod-Derived Cone Viability Factor Promotes Cone Survival by Stimulating Aerobic Glycolysis. *Cell* **2015**, *161*, 817–832. [Google Scholar] [CrossRef] [PubMed]
12. Léveillard, T.; Sahel, J.-A. Rod-Derived Cone Viability Factor for Treating Blinding Diseases: From Clinic to Redox Signaling. *Sci. Transl. Med.* **2010**, *2*, 26ps16. [Google Scholar] [CrossRef] [PubMed]
13. World Health Organization. International Classification of Diseases 11th Revision. Available online: https://icd.who.int/en (accessed on 25 December 2020).
14. Vezinaw, C.M.; Fishman, G.A.; McAnany, J.J. Visual impairment in retinitis pigmentosa. *Retina* **2020**, *40*, 1630–1633. [Google Scholar] [CrossRef]
15. Fishman, G.A. Retinitis Pigmentosa. *Arch. Ophthalmol.* **1978**, *96*, 1185–1188. [Google Scholar] [CrossRef]
16. A Fishman, G.; Young, R.S.; Vasquez, V.; Lourenço, P. Color vision defects in retinitis pigmentosa. *Ann. Ophthalmol.* **1981**, *13*, 609–618. [Google Scholar] [PubMed]
17. Bittner, A.K.; Diener-West, M.; Dagnelie, G. A survey of photopsias in self-reported retinitis pigmentosa. *Retina* **2009**, *29*, 1513–1521. [Google Scholar] [CrossRef] [PubMed]
18. Verbakel, S.K.; van Huet, R.A.C.; Boon, C.J.F.; den Hollander, A.I.; Collin, R.W.J.; Klaver, C.C.W.; Hoyng, C.B.; Roepman, R.; Klevering, B.J. Non-syndromic retinitis pigmentosa. *Prog. Retin. Eye Res.* **2018**, *66*, 157–186. [Google Scholar] [CrossRef] [PubMed]
19. McCulloch, D.L.; Marmor, M.F.; Brigell, M.G.; Hamilton, R.; Holder, G.E.; Tzekov, R.; Bach, M. ISCEV Standard for full-field clinical electroretinography (2015 update). *Doc. Ophthalmol.* **2015**, *130*, 1–12. [Google Scholar] [CrossRef] [PubMed]
20. Hood, D.C.; Odel, J.G.; Chen, C.S.; Winn, B.J. The Multifocal Electroretinogram. *J. Neuro-Ophthalmol.* **2003**, *23*, 225–235. [Google Scholar] [CrossRef]
21. Alexander, K.R.; A Fishman, G. Prolonged rod dark adaptation in retinitis pigmentosa. *Br. J. Ophthalmol.* **1984**, *68*, 561–569. [Google Scholar] [CrossRef]
22. Stavrou, P.; A Good, P.; Broadhurst, E.J.; Bundey, S.; Fielder, A.R.; Crews, S.J. ERG and EOG abnormalities in carriers of X-linked retinitis pigmentosa. *Eye* **1996**, *10*, 581–589. [Google Scholar] [CrossRef]

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https://westerneuropeanstudies.com/index.php/2
ISSN (E): 2942-190X Open Access| Peer Reviewed
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23. Cabral, T.; Sengillo, J.D.; Duong, J.K.; Justus, S.; Boudreault, K.; Schuerch, K.; Belfort Jr, R.B.; Mahajan, V.B.; Sparrow, J.R.; Tsang, S.H. Retrospective Analysis of Structural Disease Progression in Retinitis Pigmentosa Utilizing Multimodal Imaging. *Sci. Rep.* **2017**, *7*, 10347. [Google Scholar] [CrossRef]
24. Cai, C.X.; Locke, K.G.; Ramachandran, R.; Birch, D.G.; Hood, D.C. A Comparison of Progressive Loss of the Ellipsoid Zone (EZ) Band in Autosomal Dominant and X-Linked Retinitis Pigmentosa. *Investig. Opthalmol. Vis. Sci.* **2014**, *55*, 7417–7422. [Google Scholar] [CrossRef] [PubMed]
25. Hariri, A.H.; Zhang, H.Y.; Ho, A.; Francis, P.; Weleber, R.G.; Birch, D.G.; Ferris, F.; Sadda, S.R. Trial of Oral Valproic Acid for Retinitis Pigmentosa Group Quantification of Ellipsoid Zone Changes in Retinitis Pigmentosa Using en Face Spectral Domain-Optical Coherence Tomography. *JAMA Ophthalmol.* **2016**, *134*, 628–635. [Google Scholar] [CrossRef]
26. Liu, G.; Li, H.; Liu, X.; Xu, D.; Wang, F. Structural analysis of retinal photoreceptor ellipsoid zone and postreceptor retinal layer associated with visual acuity in patients with retinitis pigmentosa by ganglion cell analysis combined with OCT imaging. *Medicine* **2016**, *95*, e5785. [Google Scholar] [CrossRef]
27. Phadikar, P.; Saxena, S.; Ruia, S.; Lai, T.Y.Y.; Meyer, C.H.; Eliott, D. The potential of spectral domain optical coherence tomography imaging based retinal biomarkers. *Int. J. Retin. Vitr.* **2017**, *3*, 1. [Google Scholar] [CrossRef] [PubMed]
28. Schuerch, K.; Marsiglia, M.; Lee, W.; Tsang, S.H.; Sparrow, J.R. Multimodal imaging of disease-associated pigmentary changes in retinitis pigmentosa. *Retina* **2016**, *36*, S147–S158. [Google Scholar] [CrossRef] [PubMed]
29. Xue, K.; Oldani, M.; Jolly, J.; Edwards, T.; Groppe, M.; Downes, S.M.; MacLaren, R. Correlation of Optical Coherence Tomography and Autofluorescence in the Outer Retina and Choroid of Patients With Choroideremia. *Investig. Opthalmol. Vis. Sci.* **2016**, *57*, 3674–3684. [Google Scholar] [CrossRef]
30. Tee, J.J.; Yang, Y.; Kalitzeos, A.; Webster, A.; Bainbridge, J.; Michaelides, M. Natural History Study of etinal Structure, Progression, and Symmetry Using Ellipzoid Zone Metrics in RPGR-Associated Retinopathy. *Am. J. Ophthalmol.* **2019**, *198*, 111–123. [Google Scholar] [CrossRef] [PubMed]
31. Элмуротова Д.Б., Арзикулов Ф., Олимов А. Параметры и характеристики импульсной техники // Open Herald: Periodical of Methodical Research, V.3, Issue 2, February-2025 ISSN (E): 2810-6385, C.33-37, Chile, Website: https://academiaone.org/index.php/6
32. Nuritdinov I, Eshbekov A.A., Yusupov Q.X, Mussaeva M.A., Elmurotova D.B. Study of luminescent characteristics of chromium-doped crystals // Web of scientist: Int. scientific research journal, ISSN:2776-0976, V.6, Issue 4, April-2025, P.46-57, Indonesia. https://wos.academiascience.org/index.php/wos/article/view/5342/5118
33. Elmurotova D.B., Kattaxodjayeva D.U., Jaxongirova Sh.U., Yusupova M.B. Physics of remote gamma therapy // Web of Discoveries: Journal of Analysis and Inventions, V.3, Issue 4, ISSN(E): 2938-3773, P.50-54, April – 2025, Испания, https://webofjournals.com/index.php/3/article/view/3880
34. Elmurotova D.B., Shodiev A.A., Mussaeva M.A. Impact of electron radiation on resistivity in YBCO and GdBCO high-temperature superconducting tapes // Web of scientist: international scientific research journal, ISSN: 2776-0979, V.6, Issue 5, may-

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2025, P.161-173, Indonesia, https://wos.academiascience.org/index.php/wos/article/view/2672

35. Elmurotova D., Fayziyeva N.A., Bozorov E.H. History of the discovery of radioactivity and x-rays, nuclear explosions explanation of the phenomenon research using interactive methods // Web of Discoveries: Journal of Analysis and Inventions, V.3, Issue 5, ISSN(E): 2938-3773, P.61-65, May-2025. Испания https://webofjournals.com/index.php/3/article/view/4233
36. Elmurotova D.B., Ro‘zimatova Sh.Sh., Umarova F.S. Insonning estetik tafakkuri // Лучшие интеллектуальные исследования, ISSN:3030-3680, Ч.45, Т.1, С.130-135, май-2025, Россия. scientific-jl.com/luch/.
37. Elmurotova D.B., Farmonova Sh.Sh., Umarova F.S. Borliq va bo‘shliq: mavjudlik chegaralari haqida tafakkur // Лучшие интеллектуальные исследования, ISSN:3030-3680, Ч.44, Т.5, С.411-416, май-2025, Россия. scientific-jl.com/luch/.
38. Elmurotova D.B., Jo’rayeva R.A, Umarova F.S. “Bilimning chegarasi va rad etilishi”: eskeptitsizm va bilimga bo‘lgan ishonchsizlik muammosi // Лучшие интеллектуальные исследования, ISSN:3030-3680, Ч.44, Т.5, С.417-423, май-2025, Россия. scientific-jl.com/luch/.
39. Elmurotova D.B., Umarova F.S., G’uzorova O.U. Hayot va o‘lim chegarasida: bioetikaning zamonaviy tibbiyotdagi o‘rni // Лучшие интеллектуальные исследования, ISSN:3030-3680, Ч.44, Т.4, С.261-266, май-2025, Россия. scientific-jl.com/luch/.