Published in final edited form as:

Curr Eye Res. 2012 February ; 37(2): 132–137. doi:10.3109/02713683.2011.629071.

Outer Retinal Oxygen Consumption of Rat by Phosphorescence Lifetime Imaging

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Abstract

Purpose—Since the metabolic function of the retinal tissue is altered due to physiologic changes or disease, measurements of outer retinal oxygen consumption (Q_{OR}) may be beneficial in assessment of retinal status. The purpose of this study was to report measurements of Q_{OR} in rats using a phosphorescence lifetime imaging technique.

Methods—Phosphorescence lifetime imaging was performed and retinal PO_2 maps were generated in 10 rats under a light-adapted condition. Depth-resolved retinal PO_2 profiles were derived from the PO_2 maps. From the profiles, the maximum outer retina PO_2 ($P_{max}O_2$) was obtained and Q_{OR} was calculated using a one-dimensional oxygen diffusion model. Repeatability, inter-location variability, and inter-subject variability of $P_{max}O_2$ and Q_{OR} measurements were established.

Results—Intraclass correlation coefficients of repeated measurements of $P_{max}O_2$ and Q_{OR} were 0.89 and 0.70, respectively ($P < 0.001$). Inter-location variability of $P_{max}O_2$ and Q_{OR} measurements at superior to inferior contiguous locations on the retina were on average 9 mmHg and 0.22 ml O_2 /100g-tissue-min, respectively. Mean and standard deviation of $P_{max}O_2$ and Q_{OR} measurements averaged over all rats were 60 ± 16 mmHg and 0.73 ± 0.28 ml O_2 /100g-tissue-min, respectively. Inter-subject variability of $P_{max}O_2$ and Q_{OR} measurements were on average 2.3 and 1.5 times inter-location variability, respectively.

Conclusions—Measurements of outer retinal oxygen consumption can be made by phosphorescence lifetime imaging and may be of potential value for detecting changes in retinal oxygen metabolic activity due to altered physiological and pathological conditions over multiple locations and time points.

Keywords

Oxygen Tension; Oxygen Consumption; Retina; Rat

Introduction

Oxygen is supplied to the retina by the retinal and choroidal vasculature and is consumed by the retinal tissue according to the metabolic demand. Irregularities in oxygen supply or metabolism are believed to play a significant role in common retinal diseases such as diabetic retinopathy, retinopathy of prematurity, vascular occlusion, and age-related macular degeneration.^{1–7} Therefore, methods to measure the metabolic activity of the retina are

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Declaration of Interest: All authors have no conflict of interest in the subject matter of this manuscript.

The authors do not have any commercial relationships in the form of financial support or personal financial interest.

desired to monitor retinal health and broaden knowledge of disease physiology. Measurements of retinal tissue oxygen tension (PO_2) as a function of retinal depth and oxygen consumption (Q) by fitting a model of oxygen diffusion^{8, 9} have been reported using the oxygen sensitive microelectrode method.^{10–23} While the method has provided valuable information on retinal oxygen metabolism, it is limited to measurements obtained in a few retinal locations during a single experiment. We have previously reported a phosphorescence lifetime imaging technique for generating retinal tissue PO_2 maps and depth resolved retinal PO_2 profiles.²⁴ In the current study, we report measurements of outer retina Q at multiple locations simultaneously from retinal PO_2 maps in rats under a light-adapted condition.

Methods

Animals

Ten male Long Evans pigmented rats (450 – 650 g) were used for this study. The animals were treated in compliance with the ARVO Statement for the Use of Animals in Ophthalmic and Vision Research. Imaging was performed in one eye. The rats were anesthetized using Ketamine (85 mg/kg IP) and Xylazine (3.5 mg/kg IP). Anesthesia was maintained during imaging with periodic injections of ketamine (20 mg/kg) and xylazine (1 mg/kg). An oxygen-sensitive molecular probe, Oxyphor R2^{25, 26} (Oxygen Enterprises, Ltd. Philadelphia, PA), was dissolved in saline and 3 μ l (0.5 mM) was injected intravitreally. To allow adequate time for the probe to diffuse into the retinal tissue, the animals were imaged 24 hours post injection. Systemic arterial PO_2 (P_aO_2), PCO_2 (P_aCO_2), and pH of blood samples collected from a catheter placed in the femoral artery were measured using a blood gas analyzer (Radiometer, Westlake, OH). Blood samples were collected within several minutes of imaging. Blood pressure and heart rate were monitored using a pressure transducer attached to the catheter and linked to a data acquisition system (Biopac Systems Inc., Goleta, CA). The rats were placed on a heated animal holder to maintain a normal body temperature. A gas mixture containing 30% oxygen was administered 10 minutes before and continuously during imaging using a high-flow face mask system to maintain P_aO_2 near normal values. Before imaging, the pupils were dilated with 2.5% phenylephrine and 1% tropicamide. Hydroxypropyl methylcellulose was applied to the cornea and a glass cover slip was placed on the cornea in order to eliminate its refractive power and to prevent corneal dehydration. Since light is used to generate phosphorescence images, measurements in rats were obtained under a light-adapted condition.

Instrumentation

The optical section phosphorescence lifetime imaging system has been described previously.²⁴ Briefly, a laser at a wavelength of 532 nm was focused to a vertical line on the retina and phosphorescence from the molecular probe was imaged. Since the laser was not coaxial with the imaging axis, structures at different depths of the retina were displaced laterally in the optical section phosphorescence image. An intensified charge-coupled device attached to a slit-lamp biomicroscope at 4X magnification was used to acquire a depth-resolved phosphorescence retinal image. Phosphorescence lifetime was measured using a frequency-domain approach by incrementally varying the phase between the modulated laser light and camera sensitivity.^{27–29} From a set of phase-delayed optical section images, phosphorescence lifetime was calculated for every pixel in the image and converted to PO_2 values using the Stern-Volmer expression.²⁴ Thus, a retinal PO_2 map was generated displaying PO_2 values across the retinal depth, along a vertical length of approximately 825 μ m on the retina. Three repeated retinal PO_2 maps were generated at the same location in each animal. The retinal PO_2 maps were located approximately 3 disk diameters nasal or temporal to the optic nerve head, and the PO_2 map centers were approximately at the same

horizontal level as the center of the optic disk. From each retinal PO₂ map, 11 depth resolved PO₂ profiles from superior to inferior contiguous retinal locations were obtained, as previously described.²⁴ An example of a retinal phosphorescence image and a corresponding PO₂ map depicting superior to inferior contiguous retinal locations is shown in Fig 1. Each retinal PO₂ profile displayed the PO₂ distribution across the retinal depth (approximately 200 μm) and was derived by averaging retinal PO₂ values over a vertical length of 75 μm. The retinal boundaries were identified from retinal reflectance images as previously described.²⁴ The distance between the boundaries was approximately 45 pixels, corresponding to a previously published rat retinal thickness of 200 microns.³⁰ From each retinal PO₂ profile, the maximum outer retina PO₂ (P_{max}O₂) was recorded.

Outer Retina Oxygen Consumption Measurement

Outer retina oxygen consumption (Q_{OR}) was computed by fitting a three layer steady-state model of oxygen diffusion to PO₂ profiles, as previously described.^{9, 16} The model assumed oxygen diffusion was confined to one dimension (from the choroid toward the inner retina),

according to the following equation: $PO_{2j}(x) = \frac{1}{2}(Q_j/Dk)x^2 + A_jx + B_j$ where Q_j is the oxygen consumption of layer j, D is the diffusion coefficient, k is the oxygen solubility, x is the distance along the retinal depth measured from the choroid toward the vitreous, and A_j and B_j are constants determined by boundary conditions that require both PO₂ and flux (d(PO₂)/dx) to be continuous at the junctions between adjacent layers. From the equation, it can be deduced that the 2nd derivative of the PO₂ function (curvature of the profile) is directly proportional to Q.

The outer retina was divided into 3 layers approximately corresponding to the photoreceptor outer segments (OS) (layer 1), photoreceptor inner segments (IS) (layer 2), and outer nuclear layer (ONL) (layer 3). In contrast to previous microelectrode studies^{9, 16, 31} in which layer lengths were determined by non-linear least squares fitting, a fixed length was set for each layer based on retinal anatomy and previous mathematical modeling in cat retina^{9, 16} to improve reliability of curve fitting. Layers 1, 2 and 3 extended between 100% to 85% (0 – 30 μm), 85% to 75% (30 – 50 μm), and 75% to 50% (50 – 100 μm) of the retinal depth, respectively. According to previous microelectrode studies,^{11, 16} PO₂ was expected to vary linearly in layers 1 and 3 (Q₁ = 0, Q₃ = 0) and quadratically in layer 2 (Q₂ > 0) as a function of distance along the retinal depth.

The location of the center of layer 2 was identified as the depth position of the PO₂ profile with the maximum curvature, which was found by fitting quadratic functions to segments (67 μm, 15 data points) of the profile, incremented by 1 data point. This segment length was determined empirically to be best for identifying the location of the maximum curvature of the profile. The profile segment with the largest 2nd derivative of the quadratic function was set as the position of maximum profile curvature and assigned to 80% of the total retinal depth. The oxygen diffusion model was fitted to the outer retina portion of the PO₂ profile using a non-linear least squares algorithm and the coefficient of the quadratic term (Q₂/Dk) was derived. Q₂ was calculated from the product of Q₂/Dk, the diffusion coefficient D and oxygen solubility k, the latter two of which were assumed to be 1.97 X 10⁻⁵ cm²/sec³² and 2.4 ml O₂/(ml retina mmHg),³³ respectively. Q_{OR} was determined by weighting Q₂ according to the length of layer 2 relative to the length of the outer retina: Q_{OR} = 20μm*Q₂/100 μm. This process was repeated for all 11 PO₂ profiles derived from each retinal PO₂ map, providing measurement of Q_{OR} at 11 superior to inferior contiguous locations on the retina.

Statistical Analysis

Repeatability was assessed from 3 repeated measurements obtained at 11 retinal locations in each rat. The consistency in repeated measurements was quantified by a common metric known as the intraclass correlation coefficient (ICC)³⁴, which was calculated using a commercially available statistical software package (SPSS Software, Armonk, New York). Interlocation variability in each rat was determined as the standard deviation (SD) of measurements at 11 superior to inferior contiguous retinal locations. Inter-subject variability was determined as the SD of measurements in 10 rats. Linear regression analysis was performed to determine the correlation between Q_{OR} and $P_{max}O_2$.

Results

The systemic physiological condition of the rats was monitored during imaging. The P_aO_2 measurements were 104 ± 15 mmHg (mean $\pm$ SD; N=10). The P_aCO_2 was 56 ± 5 mmHg and the pH was 7.29 ± 0.02 . The mean blood pressure was 121 ± 26 mmHg and the mean heart rate was 248 ± 29 beats per minute.

An example of a PO_2 profile is shown in Fig 2. The maximum of the PO_2 profile was near the choroid and PO_2 decreased toward the inner retina. In the inner retina, PO_2 was relatively constant, but displayed variations due to the oxygen supply from the retinal vascular network. The curvature parameter (Q_2/Dk) derived from the fitted three-layer diffusion model to the outer retinal portion of the profile was 0.0169 mmHg/ μm^2 . Multiplying this parameter by the diffusion coefficient D and oxygen solubility k , oxygen consumption in layer 2 (Q_2) was calculated to be 4.8 ml O_2 /100g-tissue-min and Q_{OR} was 0.96 ml O_2 /100g-tissue-min.

ICC values for $P_{max}O_2$ and Q_{OR} from 3 repeated measurements obtained in all light-adapted rats were 0.89 and 0.70, respectively, and statistically significant ($p < 0.001$; N=110). $P_{max}O_2$ measurement variability along superior to inferior retinal locations was between 4 and 21 mmHg and on average was 9 mmHg. Q_{OR} measurement variability along superior to inferior retinal locations was between 0.10 and 0.36 ml O_2 /100g-tissue-min and on average was 0.22 ml O_2 /100g-tissue-min.

Histograms displaying the distribution of $P_{max}O_2$ and Q_{OR} measurements for all locations and rats are shown in Fig 3. The range of $P_{max}O_2$ and Q_{OR} measurements were 35 – 114 mmHg and 0.14 – 1.85 ml O_2 /100g-tissue-min, respectively. Mean $P_{max}O_2$ and Q_{OR} measurements averaged over all rats were 60 ± 16 mmHg (mean $\pm$ SD; N = 10) and 0.73 ± 0.28 ml O_2 /100g-tissue-min, respectively. Median of $P_{max}O_2$ and Q_{OR} measurements were 55 mmHg and 0.71 ml O_2 /100g-tissue-min, respectively. Inter-subject variability of $P_{max}O_2$ and Q_{OR} were on average 2.3 and 1.5 times higher than inter-location variability, respectively. Q_{OR} was correlated with $P_{max}O_2$ ($r = 0.68$, $p = 0.03$, N = 10), as shown in Fig 4.

Discussion

Previous studies have reported measurements of outer retinal oxygen consumption by applying a one dimensional, steady-state diffusion model to oxygen profiles from single retinal locations obtained using the oxygen microelectrode technique. In the current study, we report outer retinal oxygen consumption measured simultaneously at multiple superior to inferior contiguous locations by applying the same model to oxygen profiles derived from PO_2 maps generated with the phosphorescence lifetime imaging technique.

Oxygen microelectrodes studies in rat under normoxia have shown the maximum outer retina PO_2 to be approximately 45 mmHg.^{19, 21} The slightly higher $P_{max}O_2$ measurements in our study were likely due to the higher systemic P_aO_2 and P_aCO_2 levels, which have been shown to elevate choroidal PO_2 in rat,²¹ cat,¹⁷ and pig.³⁵ $P_{max}O_2$ in the outer retina was found to be approximately 60% of systemic P_aO_2 , similar to previous studies in rat, cat and pig under normoxia.^{16, 19, 35} Q_{OR} measurements obtained in the current study were lower than values reported in light-adapted rats (1.3 ml O_2 /100g-tissue-min)¹³ and cats (1.4 – 2.7 ml O_2 /100g-tissue-min),^{9–11} which may be attributed to technical differences between phosphorescence lifetime imaging and microelectrode methods. Due to a lower depth resolution as compared to the microelectrode technique, phosphorescence emission and scattered light from different retinal depths were likely averaged, thereby reducing the profile curvature and the measured Q_{OR} . In order to quantitatively estimate the effect of scatter, the spread of light in retinal depth was determined by the width of a laser line focused on the rat retina. A simulated PO_2 profile of known curvature was then convolved with a Gaussian function with the same width. The curvature of the convolved profile was found to be reduced by a factor of 2. Correcting for this factor, the average Q_{OR} would be approximately 1.5 ml O_2 /100g-tissue-min, a value in general agreement with microelectrode measurements of outer retinal oxygen consumption in light-adapted rats¹³ and cats.^{9–11} Q_{OR} was correlated with $P_{max}O_2$, though this result was driven by data in one animal due to the small sample size. By removing this data point, the correlation was no longer statistically significant.

Future studies aimed at improving the depth resolution by increasing the angle between the incident laser beam and imaging path, and reducing light scatter by using a probe formulation with a longer absorption wavelength will likely enhance the performance of our imaging system. Additionally, by implementing our previously reported methodology,³⁶ three dimensional retinal PO_2 maps can be generated, allowing assessment of outer retina oxygen consumption over substantial retinal areas. Furthermore, retinal oxygen consumption can be evaluated over days to weeks for detecting progression or regression of experimentally induced pathological conditions. Overall, quantitative mapping of retinal PO_2 and oxygen consumption is of potential value for assessing variations in retinal oxygen metabolism that occur due to altered physiological or pathological conditions over time and at multiple retinal locations simultaneously.

Acknowledgments

This study was supported by the National Eye Institute, Bethesda, MD, EY017918 (MS) and EY01792 (UIC), and Research to Prevent Blindness, New York, NY, senior scientific investigator award (MS) and an unrestricted departmental award.

References

1. Stefansson E. Oxygen and diabetic eye disease. *Graefes Arch Clin Exp Ophthalmol*. 1990; 228:120–123. [PubMed: 2186971]
2. Yoneya S, Saito T, Nishiyama Y, et al. Retinal oxygen saturation levels in patients with central retinal vein occlusion. *Ophthalmology*. 2002; 109:1521–1526. [PubMed: 12153805]
3. Zhang W, Ito Y, Berlin E, Roberts R, Berkowitz BA. Role of hypoxia during normal retinal vessel development and in experimental retinopathy of prematurity. *Invest Ophthalmol Vis Sci*. 2003; 44:3119–3123. [PubMed: 12824260]
4. Pournaras CJ. Retinal oxygen distribution. Its role in the physiopathology of vasoproliferative microangiopathies. *Retina*. 1995; 15:332–347. [PubMed: 8545580]
5. Arjamaa O, Nikinmaa M. Oxygen-dependent diseases in the retina: role of hypoxia-inducible factors. *Exp Eye Res*. 2006; 83:473–483. [PubMed: 16750526]

6. Cringle SJ, Yu DY. Oxygen supply and consumption in the retina: implications for studies of retinopathy of prematurity. *Doc Ophthalmol.* 2010; 120:99–109. [PubMed: 19830466]
7. Wangsa-Wirawan ND, Linsenmeier RA. Retinal oxygen: fundamental and clinical aspects. *Arch Ophthalmol.* 2003; 121:547–557. [PubMed: 12695252]
8. Cringle S, Yu DY, Alder V, Su EN, Yu P. Modelling oxygen consumption across an avascular retina. *Aust N Z J Ophthalmol.* 1996; 24:70–72. [PubMed: 8811251]
9. Haugh LM, Linsenmeier RA, Goldstick TK. Mathematical models of the spatial distribution of retinal oxygen tension and consumption, including changes upon illumination. *Ann Biomed Eng.* 1990; 18:19–36. [PubMed: 2306030]
10. Braun RD, Linsenmeier RA. Retinal oxygen tension and the electroretinogram during arterial occlusion in the cat. *Invest Ophthalmol Vis Sci.* 1995; 36:523–541. [PubMed: 7890484]
11. Braun RD, Linsenmeier RA, Goldstick TK. Oxygen consumption in the inner and outer retina of the cat. *Invest Ophthalmol Vis Sci.* 1995; 36:542–554. [PubMed: 7890485]
12. Cringle SJ, Yu DY, Alder VA. Intraretinal oxygen tension in the rat eye. *Graefes Arch Clin Exp Ophthalmol.* 1991; 229:574–577. [PubMed: 1765301]
13. Cringle SJ, Yu DY, Yu PK, Su EN. Intraretinal oxygen consumption in the rat in vivo. *Invest Ophthalmol Vis Sci.* 2002; 43:1922–1927. [PubMed: 12037000]
14. Cringle SJ, Yu PK, Su EN, Yu DY. Oxygen distribution and consumption in the developing rat retina. *Invest Ophthalmol Vis Sci.* 2006; 47:4072–4076. [PubMed: 16936126]
15. Linsenmeier RA. Effects of light and darkness on oxygen distribution and consumption in the cat retina. *J Gen Physiol.* 1986; 88:521–542. [PubMed: 3783124]
16. Linsenmeier RA, Braun RD. Oxygen distribution and consumption in the cat retina during normoxia and hypoxemia. *J Gen Physiol.* 1992; 99:177–197. [PubMed: 1613482]
17. Linsenmeier RA, Yancey CM. Effects of hyperoxia on the oxygen distribution in the intact cat retina. *Invest Ophthalmol Vis Sci.* 1989; 30:612–618. [PubMed: 2703302]
18. Yancey CM, Linsenmeier RA. Oxygen distribution and consumption in the cat retina at increased intraocular pressure. *Invest Ophthalmol Vis Sci.* 1989; 30:600–611. [PubMed: 2703301]
19. Yu DY, Cringle SJ. Oxygen distribution and consumption within the retina in vascularised and avascular retinas and in animal models of retinal disease. *Prog Retin Eye Res.* 2001; 20:175–208. [PubMed: 11173251]
20. Yu DY, Cringle SJ. Oxygen distribution in the mouse retina. *Invest Ophthalmol Vis Sci.* 2006; 47:1109–1112. [PubMed: 16505048]
21. Yu DY, Cringle SJ, Alder V, Su EN. Intraretinal oxygen distribution in the rat with graded systemic hyperoxia and hypercapnia. *Invest Ophthalmol Vis Sci.* 1999; 40:2082–2087. [PubMed: 10440264]
22. Yu DY, Cringle SJ, Alder VA, Su EN. Intraretinal oxygen distribution in rats as a function of systemic blood pressure. *Am J Physiol.* 1994; 267:H2498–2507. [PubMed: 7810746]
23. Yu DY, Cringle SJ, Yu PK, Su EN. Intraretinal oxygen distribution and consumption during retinal artery occlusion and graded hyperoxic ventilation in the rat. *Invest Ophthalmol Vis Sci.* 2007; 48:2290–2296. [PubMed: 17460293]
24. Shahidi M, Wanek J, Blair NP, Little DM, Wu T. Retinal tissue oxygen tension imaging in the rat. *Invest Ophthalmol Vis Sci.* 2010; 51:4766–4770. [PubMed: 20375336]
25. Dunphy I, Vinogradov SA, Wilson DF. Oxyphor R2 and G2: phosphors for measuring oxygen by oxygen-dependent quenching of phosphorescence. *Anal Biochem.* 2002; 310:191–198. [PubMed: 12423638]
26. Vinogradov SA, Lo LW, Wilson DF. Dendritic polyglutamic porphyrins: Probing porphyrin protection by oxygen-dependent quenching of phosphorescence. *Chem-Eur J.* 1999; 5:1338–1347.
27. Lakowicz JR, Laczkó G, Cherek H, Gratton E, Limkeman M. Analysis of Fluorescence Decay Kinetics from Variable-Frequency Phase-Shift and Modulation Data. *Biophysical Journal.* 1984; 46:463–477. [PubMed: 6498264]
28. Lakowicz JR, Szmajnski H, Nowaczyk K, Berndt KW, Johnson M. Fluorescence Lifetime Imaging. *Analytical Biochemistry.* 1992; 202:316–330. [PubMed: 1519759]

29. Shonat RD, Kight AC. Oxygen tension imaging in the mouse retina. *Ann Biomed Eng.* 2003; 31:1084–1096. [PubMed: 14582611]
30. Ruggeri M, Wehbe H, Jiao S, et al. In vivo three-dimensional high-resolution imaging of rodent retina with spectral-domain optical coherence tomography. *Invest Ophthalmol Vis Sci.* 2007; 48:1808–1814. [PubMed: 17389515]
31. Cringle SJ, Yu DY. A multi-layer model of retinal oxygen supply and consumption helps explain the muted rise in inner retinal Po₂ during systemic hyperoxia. *Comp Biochem Phys A.* 2002; 132:61–66.
32. Roh HD, Goldstick TK, Linsenmeier RA. Spatial variation of the local tissue oxygen diffusion coefficient measured in situ in the cat retina and cornea. *Adv Exp Med Biol.* 1990; 277:127–136. [PubMed: 2096618]
33. Goldstick TK, Ciuryla VT, Zuckerman L. Diffusion of oxygen in plasma and blood. *Adv Exp Med Biol.* 1976; 75:183–190. [PubMed: 1015403]
34. Weir JP. Quantifying test-retest reliability using the intraclass correlation coefficient and the SEM. *J Strength Cond Res.* 2005; 19:231–240. [PubMed: 15705040]
35. Pournaras CJ, Riva CE, Tsacopoulos M, Strommer K. Diffusion of O₂ in the Retina of Anesthetized Miniature Pigs in Normoxia and Hyperoxia. *Experimental Eye Research.* 1989; 49:347–360. [PubMed: 2792232]
36. Shahidi M, Wanek J, Blair NP, Mori M. Three-dimensional mapping of chorioretinal vascular oxygen tension in the rat. *Invest Ophthalmol Vis Sci.* 2009; 50:820–825. [PubMed: 18824736]

Figure 1. An example of a phosphorescence image (left) and the corresponding retinal PO₂ map (right) obtained in one rat. PO₂ profiles were derived from superior to inferior contiguous retinal locations, indicated with black rectangles.

Figure 2.

An example of a retinal PO_2 profile obtained in one rat. Error bars indicate standard errors of the means. The outer retinal portion of the profile was fitted with a 3-layer one-dimensional diffusion model, as shown by the solid line. Layers 1, 2, and 3 correspond to the photoreceptor outer segments, photoreceptor inner segments, and outer nuclear layer, respectively.

Figure 3. Distribution of maximum outer retina oxygen tension (left) and outer retina oxygen consumption (right) measurements obtained at 11 retinal locations in each of 10 rats.

Figure 4. Relationship between outer retinal oxygen consumption (Q_{OR}) and maximum retinal oxygen tension ($P_{\max}O_2$). Each point represents data in one rat.