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Modeling Transient Pupillary Light Reflex Induced by a Short Light Flash

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Abstract

A pupillary light reflex (PLR) model was proposed in this paper by considering the iris muscle mechanical properties and modulation inputs from both parasympathetic and sympathetic systems. The model can describe very well the experimental PLR responses induced by a short light flash of various intensities. In addition, an inverse method was developed to fit numerically this model to experimental PLR data. The model was tested in experimental human PLR data to extract separately the parasympathetic and sympathetic modulations during PLR. The results indicated a higher parasympathetic and a lower sympathetic activity in females than in males, which was consistent with previous findings in cardiovascular studies. This new model may help improve our understanding of the PLR process and could be applied to analyze autonomic nervous interaction during pupillary responses.

Index Terms

Biological system modeling; biomedical engineering; nervous system

I. Introduction

The Objective of this study is to develop a mathematical model that can describe short-light-flash-induced transient pupillary light reflex (PLR) and extract autonomic nervous system (ANS) inputs from measured pupillograms.

The size of the pupil is regulated by two antagonistic muscles, the constrictor and dilator, which are innervated by the parasympathetic and sympathetic nervous system, respectively [1]. PLR refers to the pupil size changes in response to illuminance changes. The pupil constriction is dominantly mediated by the parasympathetic pathway, which involves the Edinger–Westphal nucleus, the ciliary ganglion, and the short ciliary nerves. Abnormal PLR can be observed when one or more parts of the PLR pathway are dysfunctional. Because of its easy accessibility and effectiveness, PLR has been explored in many ophthalmic and

neurological applications, such as visual perimetry for glaucomatous defects [2], optic neuritis detection [3], Alzheimer's disease [4], and autism spectral disorders [5]. It was suggested that pupillary constriction and/or dilation is more sensitive than other autonomic measures [6].

Modeling the PLR responses is helpful for understanding the mechanism of the pupil system. In the pioneering work of Stark *et al.* [7]–[10], pupil was treated as a servocontrol system and analyzed using linear servoanalytic approaches. Similar control-theory-based approaches were also used to model the nonlinearities of the pupillary responses [11]–[13]. Although these control models provided valuable insight into the potential neural circuit governing PLR, the associated transfer functions are difficult to be converted into closed-form equations and have limited applications in interpreting pupil behaviors.

Longtin and Milton [14], [15] proposed a nonlinear first-order delay differential equation to generate the autonomous oscillations under “high-gain” negative-feedback conditions. However, the PLR responses generated from this model were not consistent with the observed data. Bressloff *et al.* [16] improved this model by considering the membrane potential of the retinal ganglion cells to improve the calculated pupillary responses to sinusoidal inputs. Recently, Pamplona *et al.* [17] further modified the model of Longtin and Milton by incorporating an empirical function describing the dependency of average pupil size on ambient illuminance. However, no first-order differentialequation-based model could produce realistic transient PLR profiles induced by short-pulse optical stimulation.

Usui and Hirata [18] proposed an entirely new PLR model based on a homeomorphic biomechanical model of the pupil muscle plant. The constrictor and dilator were modeled using the same basic mechanic units including an elastic element, a viscous element, and an active contractile element. The model generated consistent results with the observed data and was used to monitor autonomic nervous activity [19]. However, this model was overly complicated with a total of 19 equations. Even with all dependent equations being combined, the model still consisted of three independent second-order delay differential equations. Therefore, it was impractical to design a robust inverse algorithm to obtain model parameters from experimental data.

In this study, a physiologically based PLR model was proposed by considering the viscoelastic properties of the iris muscle and the ANS input induced by a transient light stimulation. This model can describe very well experimental PLR responses induced by a short light flash of various intensities. Since this PLR model was composed of only one second-order differential equation, using optimization algorithms to fit this model to experimental data becomes feasible. We demonstrated that this model can be used to reliably extract separately the parasympathetic and sympathetic inputs by fitting experimental PLR data.

II. Method

A. Model Construction

Since the two iris muscles play essential roles in controlling the pupil size, their dynamic properties must be considered in the PLR model. In this study, the constrictor and dilator muscles were modeled as viscoelastic materials using Kelvin–Voigt model in a circular and linear shape, respectively. The movement of the iris muscles is governed by three forces: passive muscle elastic force, viscous resistance, and the active forces generated from the ANS modulation. The constrictor is modulated by the parasympathetic system, whereas the dilator is modulated by the sympathetic system.

Let the radius of constrictor be r_c , and the length of dilator muscle be r_d . Assuming the constrictor has a circular shape with a perimeter of $2\pi r_c$, the dynamic equations can be constructed for constrictor and dilator as

$$m_c \frac{d^2 r_c}{dt^2} = -2\pi P_{ec} + P_{vc} - \frac{F_p(t)}{2\pi} \quad (1-1)$$

$$m_d \frac{d^2 r_d}{dt^2} = -P_{ed} + P_{vd} - F_s(t) \quad (1-2)$$

where m_c (in mg) and m_d (in mg) are the mass for constrictor and dilator, respectively. P_{ec} and P_{ed} are the elastic forces from the constrictor and dilator, respectively. The passive iris muscle elasticity was modeled as a second-order equation of muscle length based on experimental observation [20]

$$P_{ec} = \begin{cases} k_c(r_c - l_{0c})^2, & (r_c \geq l_{0c}) \\ 0, & (r_c < l_{0c}) \end{cases} \quad (2-1)$$

$$P_{ed} = \begin{cases} k_d(r_d - l_{0d})^2, & (r_d \geq l_{0d}) \\ 0, & (r_d < l_{0d}) \end{cases} \quad (2-2)$$

where k_c and k_d are elastic constants, and l_{0c} is the radius of the constrictor at the rest, while l_{0d} is the dilator muscle resting length.

In (1), P_{vc} and P_{vd} are the viscous resistance in the constrictor and dilator, respectively. They were modeled as a linear function of velocity [18]

$$P_{vc} = -D_c \frac{dr_c}{dt} \quad (3-1)$$

$$P_{vd} = -D_d \frac{dr_d}{dt} \quad (3-2)$$

where D_c and D_d are viscous constants.

The $F_p(t)$ and $F_s(t)$ in (1) are the muscle forces originated from the parasympathetic and sympathetic system induced by light stimulus, respectively. These are effective forces that could be affected by both the afferent and efferent pupillary pathway. For short-pulse stimulus, the forces originated from the parasympathetic and sympathetic system were assumed to be square-wave pulses with different delays, τ_p and τ_s , and durations, t_p and t_s

$$F_p(t) = \begin{cases} f_p + f_{p0}, & (\tau_p \leq t \leq \tau_p + \Delta t_p) \\ f_{p0}, & (t < \tau_p, t > \tau_p + \Delta t_p) \end{cases} \quad (4-1)$$

$$F_s(t) = \begin{cases} f_s + f_{s0}, & (\tau_s \leq t \leq \tau_s + \Delta t_s) \\ f_{s0}, & (t < \tau_s, t > \tau_s + \Delta t_s) \end{cases} \quad (4-2)$$

where f_{p0} and f_{s0} indicate the muscle forces originated from the parasympathetic and sympathetic systems at the resting condition. Considering the pupil radius $r = r_c$, (1-1) becomes

$$m_c \frac{d^2 r}{dt^2} = -2\pi k_c (l_{0c} - r)^2 - D_c \frac{dr}{dt} - \frac{F_p(t)}{2\pi}. \quad (5)$$

Since the summation of pupil radius (r) and the dilator length (r_d) is equal to the radius of the whole iris $r_0 = r + r_d$, (1-2) can be changed to

$$m_d \frac{d^2 r}{dt^2} = k_d (l_{0d} - r)^2 - D_d \frac{dr}{dt} - F_s(t) \quad (6)$$

where $L_{0d} = r_0 - l_{0d}$. Adding (5) and (6), and we have

$$m \frac{d^2 r}{dt^2} = -2\pi k_c (l_{0c} - r)^2 + k_d (l_{0d} - r)^2 - (D_c + D_d) \frac{dr}{dt} - \frac{F_p(t)}{2\pi} + F_s(t) \quad (7)$$

where m is the total iris muscle mass $m = m_c + m_d$. Equation (7) can be simplified by normalizing the iris muscle mass

$$\frac{d^2 r}{dt^2} = -K_c (l_{0c} - r)^2 + K_d (L_{0d} - r)^2 - D \frac{dr}{dt} - f'_p(t) + f_s(t) + P_0 \quad (8)$$

where $K_c = 2\pi k_c/m$, $K_d = k_d/m$, $D = (D_c + D_d)/m$, and $f'_p = f_p/2\pi$. The constant $P_0 = f_{s0} - f_{p0}/2\pi$ represents the static force that controls the resting pupil size.

B. Inverse Model to Fit Experimental Data

The aforementioned model (8) was applied to fit experimental data of pupillary responses to short-flash lights. Experimental data were acquired using a customized pupillogram recording system that has been described in detail earlier [21], [22]. The detailed test procedure has been reported elsewhere [22]. Briefly, the optical stimulation was a short 100-ms flash at 530 nm and was presented 1 s after starting the acquisition of pupil images. PLR data were acquired under light-adapted (LA) condition (30 cd/m²) using three stimulus

intensities ($10^{10.4}$, $10^{11.4}$, and $10^{12.5}$ photons/cm²/s), and dark-adapted (DA) condition (<0.01 cd/m²) using five stimulus intensities ($10^{8.2}$, $10^{9.3}$, $10^{10.4}$, $10^{11.4}$, and $10^{12.5}$ photons/cm²/s). Five repeated measurements were obtained at each condition by stimulating the left eye, and the pupil responses of both direct eye (stimulated eye) and consensual eye (nonstimulated eye) were recorded.

A pattern search algorithm [23] was applied in MATLAB (Version 7.1, The Mathworks, Inc., Natick, MA) to fit the experimental data. To ensure a consistent convergence of the algorithm, a two-step fitting procedure was adopted. First, the model fitting was applied to the PLR data acquired at DA $10^{12.5}$ photons/cm²/s without applying constraints. Since the SNR ratio at this condition was the highest among all the conditions, the corresponding fitting parameters were the most reliable. Second, the stimulus-independent muscle parameters (L_{0d} , l_{0c} , K_d , K_c , and D) inherited from the first step were specified within a narrow range ($\pm 5\%$), and model fitting was then performed on other stimulus conditions. The durations of the parasympathetic and sympathetic modulation were set to the same as the flash stimulus duration.

After the model fitting, two dimensionless indicators, the rootmean-squared percentage error (RMSPE) and Willmott's index-of-agreement (d) were calculated to quantitatively evaluate the goodness of fitting [24]. The RMSPE was defined as

$$\text{RMSPE} = \sqrt{\frac{1}{N} \sum_{i=1}^N \left(\frac{X_i - Y_i}{X_i} \right)^2} \quad (9)$$

where X represents the experimental measurements and Y is the model-fitting result. The Willmott's index-of-agreement was calculated as

$$d = 1 - \frac{\sum_{i=1}^n (X_i - Y_i)^2}{\sum_{i=1}^n (|X_i - \bar{X}| + |Y_i - \bar{X}|)^2} \quad (10)$$

where $\bar{X}$ represents the mean value of experimental data.

III. Results

A. Model Evaluation

The PLR profiles produced from (8) showed excellent agreement with experimental results. Fig. 1 shows an example comparison between model calculation and experimental data at different stimulus intensities in both LA and DA condition. The input parameters in the PLR model were obtained using the fitting algorithm described in Section II-B.

As shown in Fig. 1, the PLR model described all experimental results quite well. Quantitatively, the corresponding RMSPEs were very small: 0.25%, 0.54%, and 0.47% for the three intensities from low to high at LA condition, and 0.29%, 0.24%, 0.20%, 0.41%, and 0.29% at DA condition, respectively. The Willmott's index of agreement was larger than 0.99 at all conditions, further confirming a good agreement between model prediction and experimental data.

The fitted iris muscle parameters were consistent at different stimulation conditions (see Table I). As shown in the model equation (8), all mechanical and force parameters were normalized by the iris muscle mass. The elastic constant of the dilator was much larger than that of the constrictor, which was in agreement with the experimental data in rabbit iris [20]. As expected, the parasympathetic modulation f'_p increased with the stimulus intensity. The negative sign for P_0 indicated the static force makes the pupil to constrict at the resting condition. The absolute value of P_0 was much larger at LA condition than DA condition, confirming that the pupil constricted much less at dark. The fitted delay time for the muscle force originated from system τ_s was 100–200 ms longer than the maximal pupil constriction time. In another word, the force originated from sympathetic system appeared to affect only the pupillary redilation phase.

The fitted parasympathetic delay τ_p coincided with the pupillary constriction latency calculated by using a different method described by [25]. The constriction latency was defined as the time delay from the stimulus onset to the beginning of pupil constriction. This PLR latency was directly retrieved from the experimental data. A Savitzky–Golay filter was first applied to the experimental data to remove the spike noises, and a cubic spline function was used to interpolate the experimental data to 300 Hz. The starting point of pupil constriction was determined as the one with the minimal second derivative. The difference between PLR latency and parasympathetic delay was smaller than 3%.

To further study the stability of fitted parameters, we applied the model to fit binocular experimental data of pupillary responses to flash light at three DA stimulus intensities, $10^{10.4}$, $10^{11.4}$, and $10^{12.5}$ photons/cm²/s (see Fig. 2). The fitted parameters (see Table II) were similar for both eyes as the papillary responses were almost identical in the two eyes. It was noticed that the resting pupil size and constriction amplitude of the left eye was slightly larger than that of the right eye at $10^{10.4}$ and $10^{11.4}$ photons/cm²/s. Therefore, the fitted P_0 and f'_p for the left eye were also slightly larger. At $10^{12.5}$ photons/cm²/s, however, as there was no difference in resting pupil sizes and constriction between the two eyes, the fitted P_0 and f'_p were also closed. The fitted f_s of the two eyes appeared similar at all the three intensities, since the constriction phase was mainly mediated by the parasympathetic activities.

To verify whether the fitting algorithm can converge at a truly optimized solution (global minimal), we examined the dependency of the fitted model parameters on various initial input values. Fig. 3 shows a typical example of the converging trajectories of the viscoelastic parameters, K_d , K_c , and D , for data acquired at DA $10^{12.5}$ photons/cm²/s. Although the fitting algorithm was run with starting values over a wide range, the same set of output parameters were always reached upon convergence. Similar good convergence was observed for PLR results measured at other conditions.

B. Extracting Autonomic Nervous Inputs From PLR Data

As an application example, we used this model to extract individual contribution from parasympathetic and sympathetic modulation in human PLR data. The raw PLR data [22] was obtained in 13 female and 12 male participants (age 18–22, 20.0 ± 1.3 years) using a 100-ms optical flash at various stimulus conditions. In this example, the data acquired at LA

and DA $10^{12.5}$ photons/cm²/s was used for model fitting. The parameters indicating the autonomic nervous activity, e.g., the amplitude of the forces originated from parasympathetic and sympathetic systems f'_p and f_s , and the static force P_0 , were analyzed using ANOVA for the effects of gender, measured eye (direct or consensual), and retinal adaptation level (LA or DA). *Post hoc t*-tests with Bonferroni correction were then applied for pairwise comparison for significant factors revealed in ANOVA. A *p*-value smaller than 0.05 was considered to be significant.

The ANOVA indicated that the force originated from parasympathetic system [see Fig. 4(a)] f'_p varied with the retinal adaptation. The muscle force from parasympathetic input was significantly smaller at LA $10^{12.5}$ photons/cm²/s than at DA $10^{12.5}$ photons/cm²/s ($p < 0.0001$). The effect of gender was also revealed significant ($p = 0.0003$). The force originated from parasympathetic system of the female participants was significantly larger at both LA and DA $10^{12.5}$ photons/cm²/s when *post hoc t*-tests were applied for pairwise comparisons ($p < 0.05$).

For sympathetic activity [see Fig. 4(b)], the ANOVA revealed a significant effect of retinal adaptation. The force originated from sympathetic system obtained at LA $10^{12.5}$ photons/cm²/s was significantly larger than that obtained at DA $10^{12.5}$ photons/cm²/s ($p < 0.0001$). A significant interaction of gender by adaptation was also observed ($p = 0.0026$), and the gender effect was marginally significant ($p = 0.056$). The *post hoc t*-tests indicated that pupils of female participants received significantly smaller sympathetic modulation at DA $10^{12.5}$ photons/cm²/s ($p = 0.001$), but not at LA $10^{12.5}$ photons/cm²/s ($p > 0.05$).

The ANOVA revealed a significant effect of retinal adaptation level (LA or DA) in static force ($p < 0.0001$), but not in gender, measured eye or any interactions [see Fig. 4(c)]. As expected, the static force that dilated the pupil was larger in DA than LA condition.

All the aforementioned *p*-values were Bonferroni corrected by multiplying the number of comparisons.

IV Discussion

Clarke [26] compared the pupil responses to light and to the electrical stimulation of the parasympathetic fibers in the III nerve in rats. The waveforms of pupillary constriction in response to single or brief trains of electrical impulses applied to the III nerve were virtually identical to those elicited with transient light flashes. It was suggested that the viscoelastic properties of the iris smooth muscle are presumably the major contributor to the PLR profile. Therefore, considering mechanical properties iris muscle is the key in modeling the pupillary light reflex. Our results appear to support this hypothesis. The PLR model developed based on the viscoelastic property of iris muscle and the interaction of autonomic nervous activities can fit those experimental data obtained at various stimulus intensities and ambient illumination conditions.

Based on our study, each element of this PLR model (8) was a necessity for a good fitting of the experimental data and cannot be reduced or simplified. For instance, if the viscosity term was removed from the model, or the elasticity terms were expressed as the first-order

relationship with the muscle length, the model output was not consistent with the experimental data when fitting algorithm converged.

We assumed that the waveforms of the parasympathetic and sympathetic modulations had the same square-wave profiles as the transient flash stimulation. This assumption was based on the earlier experimental observation by Smith *et al.* [27]. By recording the single midbrain neurons correlating with pupillary reflex in cats, Smith *et al.* [27] found that the fluctuation of the firing rate of neurons appeared a square wave while the stimulation was a sudden ON-and-OFF input. In fact, we achieved the best fitting results when the duration of parasympathetic activity was set to 100 ms as the optical stimulus. If longer durations were used, the fitting errors increased, as shown in Fig. 5.

A significant advantage of this proposed PLR model over others is its capability to extract separately the sympathetic and parasympathetic activities from experimental PLR data. We found that the timing of parasympathetic modulation coincided with the PLR latency. The sympathetic modulation was always activated after the pupil reached maximal constriction. These results were consistent with previous experimental observation that the transient PLR is mainly modulated by parasympathetic activities. Lowenstein and Loewenfeld [28] studied the transient PLR in cats, rabbits, and monkeys, and observed that the pupil constriction response to light pulse did not differ much before and after the sympathectomy. Subsequently, Heller *et al.* [29] studied the human PLR and observed that relaxing the dilator did not significantly affect the pupil response to transient flash, but only led to a smaller baseline pupil size. These observations suggest that sympathetic modulation in dilators does not play a significant role during pupillary constriction in response to transient light stimulation.

Earlier, we observed a weak gender difference in relative constriction amplitude (ratio of constriction amplitude to resting pupil size) of pupillary response to flash light [22]. This study further suggested that the observed gender difference in constriction amplitude was due to a higher parasympathetic and a lower sympathetic activity in females than in males, which was consistent with previous findings in cardiovascular system studies [30], [31]. In fact, a correlation analysis showed that 85% of variation in relative constriction amplitude could be explained by the parasympathetic input f'_p , the combined viscosity D , the initial ANS input P_0 , and the resting length of the dilator L_{0d} , while the f'_p alone contributed 55%. The aforementioned result also implied that the constriction amplitude may not be solely used to assess parasympathetic activities because of the involvement of other iris muscle properties.

In summary, we proposed a novel PLR model by considering mechanical properties of the iris muscle and ANS modulation. The model can reliably fit experimental PLR data to extract individual contributions from parasympathetic and sympathetic activities. Abnormal parasympathetic and/or sympathetic activities have been linked to many disorders, such as panic disorder [32], migraine [33], Parkinson's disease [34], Alzheimer's disease [35], and autism spectrum disorders [36]. The capability to extract specific parasympathetic- and sympathetic-related parameters from PLR measurements can provide more accurate diagnostic information on ANS functions. As an initial test, this model was applied to reveal

a higher parasympathetic but lower sympathetic activity in females than in males, which is in agreement with findings in earlier cardiovascular studies. Further studies are necessary to test this model in a large population and to compare ANS modulations derived from PLR measures to other physical measurement, such as heart rate variability. This new model may help advance our understanding of the PLR process and could be applied to analyze autonomic nervous interaction during pupillary responses.

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Fig. 1.

Model fitting results of experimentally recorded pupillary responses to 100-ms-flash light of various intensities. The data were obtained at (a) LA with three stimulus intensities and (b) DA condition with five stimulus intensities. The experimental data are shown in scatter plots, and the solid lines indicate the fitting results. The stimulation started at 1 s.

Fig. 2.

Model fitting results of binocular pupillary responses to 100-ms-flash light. The PLR data were obtained under DA conditions with stimulus intensities of (a) $10^{10.4}$, (b) $10^{11.4}$, and (c) $10^{12.5}$ photons/cm²/s. The stimulation started at 1 s.

Fig. 3.

Sample convergence trajectories of the model parameters K_d , K_c , and D obtained with three different sets of starting values. The PLR data were measured at DA $10^{12.5}$ photons/cm²/s (see Fig. 1). The figures show the trajectories (fitted values after each iteration) of (a) K_c versus K_d and (b) D versus K_d . The starting values are shown as circles and the fitted values upon convergence are marked as squares.

Fig. 4.

Fitted results of (a) parasympathetic activity, (b) sympathetic activity, and (c) static force from measured pupillary responses to flash light in 13 female and 12 male participants at LA and DA $10^{12.5}$ photons/cm²/s. The bars charts show the group mean and the standard deviation.

Fig. 5. Relation of the fitting error (RMSPE) with the duration of force originated from parasympathetic system. Five durations from 100 to 500 ms were assigned to parasympathetic modulations in the fitting process and the corresponding RMSPEs were shown as circles. The experimental data were obtained at DA $10^{12.5}$ photons/cm²/s (see Fig. 1) using a 100-ms stimulus.

TABLE I

Fitted Parameters From the Model Outputs in Fig. 1

Parameters	Light-adapted ($h\nu/cm^2/sec$)					Dark-adapted ($h\nu/cm^2/sec$)				
$10^{10.4}$	$10^{11.4}$	$10^{12.5}$	$10^{8.2}$	$10^{9.3}$	$10^{10.4}$	$10^{11.4}$	$10^{12.5}$			
$L_{00}(mm)$	3.59	3.59	3.57	3.57	3.59	3.59	3.59	3.59	3.59	3.59
$l_0(mm)$	0.91	0.92	0.93	0.93	0.92	0.92	0.92	0.92	0.92	0.92
$K_0(mN/g \cdot mm^2)$	1.24	1.29	1.24	1.24	1.30	1.32	1.28	1.28	1.24	1.24
$K_1(mN/g \cdot mm^2)$	0.059	0.065	0.047	0.057	0.057	0.058	0.062	0.061		
$(mN \cdot s/g \cdot mm)$	3.78	3.78	3.78	3.78	3.78	3.78	3.72	3.58		
$f'_p(mN/g)$	12.73	28.38	32.25	11.57	21.54	33.07	38.53	40.78		
$f_s(mN/g)$	9.10	10.65	9.43	6.38	9.21	7.16	7.18	6.35		
$P_0(mN/g)$	-0.760	-0.736	-1.007	-0.001	-0.002	-0.041	-0.015	-0.063		
$\tau_p(s)$	0.26	0.24	0.22	0.37	0.28	0.27	0.23	0.25		
$\tau_s(s)$	0.64	0.73	0.77	0.74	0.80	0.93	0.99	1.07		

TABLE II

Fitted Parameters From the Model Outputs in Fig. 2

Parameters	$10^{10.4} \text{ } h\nu/\text{cm}^2/\text{sec}$		$10^{11.4} \text{ } h\nu/\text{cm}^2/\text{sec}$		$10^{12.5} \text{ } h\nu/\text{cm}^2/\text{sec}$	
	Right	Left	Right	Left	Right	Left
$L_{00}(\text{mm})$	4.13	4.12	4.06	4.07	4.09	4.04
$l_0(\text{mm})$	1.02	1.02	1.02	1.03	1.01	1.01
$K_d(\text{mN/g}\cdot\text{mm}^2)$	0.85	0.92	0.77	0.81	0.82	0.84
$K_c(\text{mN/g}\cdot\text{mm}^2)$	0.049	0.050	0.041	0.047	0.048	0.050
$D(\text{mN}\cdot\text{s/g}\cdot\text{mm})$	3.22	3.33	3.23	3.32	3.12	3.33
$f''_p(\text{mN/g})$	46.36	49.38	47.89	51.26	48.78	51.01
$f_s(\text{mN/g})$	13.54	14.09	11.77	11.93	10.38	10.77
$P_0(\text{mN/g})$	0.042	0.094	0.084	0.162	0.009	0.000
$\tau_p(\text{s})$	0.23	0.23	0.23	0.22	0.23	0.21
$\tau_s(\text{s})$	0.99	1.04	1.09	1.02	1.12	1.10