Is Myopia Accelerated In Type 1 Diabetes Mellitus Children? Analyses From The Ocular Parameters

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Abstract

Purpose: Compare ocular biometry with or without myopia in children with type 1 diabetes mellitus (T1DM) and healthy children in China, to analyze the difference between myopia T1DM children and myopia healthy children.

Methods: A case-control study was conducted at Children's Hospital of Fudan University, the children were divided into one of four subgroups depended on myopia or non-myopia, T1DM or non-DM. The participants were evaluated for anterior chamber depth (ACD), lens thickness (LT), axial length (AL), average keratometry (K), and lens power (P); also cycloplegic refraction was performed, and spherical equivalent (SE) was acquired.

Results: Two hundred and four eyes of 102 patients with T1DM and 174 eyes of 87 healthy subjects were included. In age-sex adjusted analysis, myopia T1DM subgroup showed thicker LT ($p < 0.001$), shallower ACD ($p = 0.009$), larger P ($p < 0.001$), shorter AL ($p = 0.004$), similar K and SE (all $p > 0.05$) than myopia control subgroup. Also in age-sex adjusted analysis, myopia T1DM subgroup showed deeper ACD ($p = 0.041$), longer AL ($p < 0.001$), similar LT, K and P (all $p > 0.05$) than non-myopia T1DM subgroup. For the relationship of ACD, LT, AL, and P with myopia, in T1DM patients, eyes with longer AL (OR, 10.665; $p < 0.001$) and larger P (OR, 1.348; $p = 0.033$) were more likely to have myopia; in healthy controls, eyes with longer AL were more likely to have myopia (OR, 11.956; $p < 0.001$).

Conclusions: In myopia T1DM children, ACD deepened than non-myopia T1DM children, yet not profound enough than healthy myopia children, while LT remained unchanged comparing to non-myopia T1DM children, therefore the lens lacked the ability to lose power as compensation to AL growth, these were the evidence that myopia would be accelerated in T1DM children.

Introduction

Axial length (AL), corneal power, and lens power (P) were kept in balance to maintain refractive status. AL elongation contributed more than 70% of myopia progression, followed by P and corneal power¹, since the cornea does not change much in diameter and power after year 2², the role of corneal power in myopia progression was limited, beside AL, attention should be paid more to P. In the study of chickens' eyes, from age 10 to 90 days, AL increased while the cornea and the lens lost power accordingly. The lens became larger and the curvatures flattened, in the meantime, the equivalent index decreased, with all these changes, the lens lost 30 diopters of power in these days of eye growth in chickens, thus the refractions were maintained in the low hyperopic range³. For human beings, the lens compacted inside the nucleus, with a slow rate of addition of new fibers in the newly developed cortex after birth, so the lens became thinning in the first 10 years of life; it also decreased gradient index power because of the nucleus compaction with a more abrupt climbing gradient profile, then the lens lose internal power². Therefore, the lens compensates for AL elongation in childhood to maintain refractive status. But myopia can be developed once the lens reaches its limit in power loss because of its internal structure². In

CLEERE Study found that myopia onset was characterized by an abrupt loss of lens compensatory changes that continue in emmetropes throughout childhood AL elongation⁴. The AL elongation accelerated before the onset of myopia, and spherical equivalent (SE) progression was the highest during the year of onset⁵. Though myopic eyes have thinner lenses, lower P, and longer anterior segments lengths to compensate longer AL^{2,6}, less reduction in P in myopes compared to non-myopes was found, the decreased ability of P loss may not only because of the limitation of internal structure but also associated with growing age and increasing AL⁷.

Above all, the compensation of lens for AL elongation was not endless, myopia happens when AL grows to a certain extent. What if the lens was suffered from some other circumstance, like hyperglycemia? In our previous study of ocular biometry in type 1 diabetes mellitus (T1DM) children, lens thickness (LT) was increased accompanied with anterior chamber depth (ACD) decrease, while SE was unaffected compared to healthy controls, it was suspected a compensation from lens refractive index⁸, but the other half contribution of P loss were changes from LT and curvature². Whether the compensation from lens refractive index is enough to confront both LT growth and AL elongation in myopia T1DM children? How does P progress in myopia T1DM children? Doubts aroused about the lens compensation ability and whether myopia is accelerated in T1DM children. So we conducted this study to further elucidate the differences of ocular biometry changes in both T1DM children myopes and non-DM children myopes.

Methods

This was a hospital-based case-control study approved by the ethics committee of both Children's Hospital of Fudan University in Shanghai (approval number: No. 01 (2018)) and Shanghai General Hospitals (approval number: 2016KY005). This study conformed to the guidelines proposed in the Helsinki Convention. It was a part of the Shanghai Children and Adolescent DM Eye study (SCADE). SCADE is a study aiming to investigate the ocular disorders of DM children since January 2018, and we arranged yearly for follow-up and new enrollment around every January, the deadline here was 2020.

Patients with T1DM enrolled between the 3 years were included in the study. Healthy subjects were chosen from consecutive children who came to the clinic for routine vision examination and were willing to participate in our research in January 2018. We excluded those with other metabolic disorders (i.e., Prader-Willi syndrome), and those under myopia control measures (i.e., orthokeratology or atropine eye drops). Eyes with a history of ocular trauma and diseases (i.e., corneal pathology, cataract, glaucoma, optic nerve atrophy, retinopathy, strabismus) were also excluded. Written informed consent and medical history questionnaire were obtained from each participant's parent.

The methodology has been published⁸. Examinations were performed by the same team. Refractive error and K1, K2 keratometry were measured by an autorefractor (ARK-1; Nidek, Tokyo, Japan), the keratometry data was converted to average K, $K = (K1 + K2)/2$; ACD, LT, and AL were acquired by IOL Master (700; Carl Zeiss Meditec, Dublin, CA). Then, the pupil was dilated by 1% cyclopentolate. After that, subjective refraction was performed. The refraction data were converted to spherical equivalent (SE; SE = sphere

power + 1/2 cylinder power). The refractive power of the lens (P) was calculated using the modified Bennette-Rabbetts formula^{9, 10}.

Eyes were classified into one of two groups based on the SE, myopia group was defined as SE with at least - 0.50 D (inclusive), the rest were defined as non-myopia group, each group was then defined into two subgroups as T1DM group or control group.

Statistical analysis of the data was performed with SPSS version 26.0. Mean values and standard deviation (SD) were used for descriptive analyses, Independent T-test was used to compare the differences of continuous variables between subgroups, Pearson chi-square test was used for the gender analyses between subgroups, Univariate General Linear Models was used to compare the differences of ocular biometry between subgroups adjusting for age-sex. Binary logistic regression models were performed to explore the association between myopia and P, AL, ACD, LT after adjusting for age and sex of both DM and non-DM groups. Both eyes were included in this study. All data were approximately normally distributed. Statistical significance was set as $p < 0.05$.

Results

Two hundred and four eyes of 102 patients with T1DM and 174 eyes of 87 healthy subjects were included in the study. The mean age of myopia T1DM subgroup and myopia control subgroup was 12.30 ± 2.80 years and 10.37 ± 2.12 years respectively, the differences between 2 groups were significant ($p < 0.001$), the mean age of the non-myopia T1DM subgroup and non-myopia control subgroup was 9.85 ± 2.89 years and 8.20 ± 1.87 years respectively, the differences between 2 groups were significant ($p < 0.001$). There were 37 male and 63 female eyes in the myopia T1DM group, 69 male and 55 female eyes were in the myopia control group, the differences between 2 groups were significant ($p = 0.005$); there were 49 male and 55 female eyes in non-myopia T1DM group, 26 male and 24 female eyes were in the non-myopia control group, the differences between 2 groups were not significant ($p = 0.570$).

Table 1 presents the means and SDs of the ocular biometry of myopia T1DM subgroup vs myopia control subgroup and non-myopia T1DM subgroup vs non-myopia control subgroup. The data named below were all adjusted for age-sex. The LT was significantly thicker in the myopia T1DM group than the myopia control group ($p < 0.001$), the ACD of the myopia T1DM group was much shallower than the myopia control group ($p = 0.009$). The AL was longer in myopia control group ($p = 0.004$), while K and SE showed no significant difference ($p = 0.319$, $p = 0.646$). P was significantly larger in the myopia T1DM group than the myopia control group ($p < 0.001$). However, non-myopia T1DM subgroup and non-myopia control subgroup showed no difference in all parameters (ACD, LT, AL, K, P, and SE, all $p > 0.05$).

Table 1

Ocular parameters comparisons between subgroups of T1DM patients and healthy controls, Mean $\pm$ SD

	Myopia T1DM N = 100	Myopia control N = 124	p- value	Non-myopia T1DM N = 104	Non-myopia control N = 50	p- value
ACD, mm						
Unadjusted	3.44 $\pm$ 0.30	3.55 $\pm$ 0.32	0.009	3.30 $\pm$ 0.29	3.25 $\pm$ 0.39	0.401
Age-sex adjusted	3.44 $\pm$ 0.03	3.56 $\pm$ 0.03	0.009	3.29 $\pm$ 0.03	3.28 $\pm$ 0.05	0.911
LT, mm						
Unadjusted	3.45 $\pm$ 0.16	3.35 $\pm$ 0.15	< 0.001	3.48 $\pm$ 0.18	3.50 $\pm$ 0.17	0.539
Age-sex adjusted	3.47 $\pm$ 0.02	3.34 $\pm$ 0.01	< 0.001	3.49 $\pm$ 0.02	3.48 $\pm$ 0.03	0.803
AL, mm						
Unadjusted	24.50 $\pm$ 1.04	24.71 $\pm$ 1.02	0.125	23.10 $\pm$ 0.78	22.98 $\pm$ 0.97	0.405
Age-sex adjusted	24.40 $\pm$ 0.10	24.79 $\pm$ 0.09	0.004	23.04 $\pm$ 0.08	23.10 $\pm$ 0.12	0.680
K, D						
Unadjusted	43.10 $\pm$ 1.59	42.98 $\pm$ 1.36	0.557	43.17 $\pm$ 1.41	43.03 $\pm$ 1.49	0.577
Age-sex adjusted	42.93 $\pm$ 0.14	43.14 $\pm$ 0.14	0.319	43.15 $\pm$ 0.14	43.10 $\pm$ 0.22	0.806
P, D						
Unadjusted	22.42 $\pm$ 1.85	21.54 $\pm$ 1.43	< 0.001	23.17 $\pm$ 2.04	22.77 $\pm$ 2.36	0.385
Age-sex adjusted	22.60 $\pm$ 0.17	21.37 $\pm$ 0.16	< 0.001	23.25 $\pm$ 0.22	22.53 $\pm$ 0.38	0.104
SE, D						
Unadjusted	-2.80 $\pm$ 2.05	-2.38 $\pm$ 1.73	0.107	0.69 $\pm$ 0.71	0.78 $\pm$ 1.12	0.666
Age-sex adjusted	-2.50 $\pm$ 0.18	-2.65 $\pm$ 0.16	0.646	0.74 $\pm$ 0.09	0.68 $\pm$ 0.14	0.739

Data of both eyes. ACD: anterior chamber depth; LT: lens thickness; AL: axial length; K: average keratometry; P: lens power; SE: spherical equivalent; D: diopters; SD: standard deviation. p value < 0.05

significant.

Table 2 presents the means and SDs of the ocular biometry of myopia T1DM subgroup vs non-myopia T1DM subgroup and myopia control subgroup vs non-myopia control subgroup. The data named below were all for age-sex adjusted data. Between two control subgroups, myopia subgroup showed deeper ACD ($p < 0.001$) and thinner LT ($p < 0.001$), longer AL ($p < 0.001$), smaller P ($p = 0.039$). Between two T1DM subgroups, myopia subgroup showed deeper ACD ($p = 0.041$), longer AL ($p < 0.001$), while similar LT ($p = 0.982$) and P ($p = 0.203$). K were stable in two pairs of subgroups (all $p > 0.05$).

Table 2
Ocular parameters comparisons between subgroups of myopia subjects and non-myopia subjects,
Mean $\pm$ SD

	Myopia T1DM N = 100	Non-myopia T1DM N = 104	p- value	Myopia control N = 124	Non-myopia control N = 50	p- value
ACD, mm						
Unadjusted	3.44 $\pm$ 0.30	3.30 $\pm$ 0.29	0.001	3.55 $\pm$ 0.32	3.25 $\pm$ 0.39	< 0.001
Age-sex adjusted	3.42 $\pm$ 0.03	3.33 $\pm$ 0.03	0.041	3.55 $\pm$ 0.03	3.27 $\pm$ 0.05	< 0.001
LT, mm						
Unadjusted	3.45 $\pm$ 0.16	3.48 $\pm$ 0.18	0.288	3.35 $\pm$ 0.15	3.50 $\pm$ 0.17	< 0.001
Age-sex adjusted	3.47 $\pm$ 0.02	3.47 $\pm$ 0.02	0.982	3.36 $\pm$ 0.01	3.47 $\pm$ 0.02	< 0.001
AL, mm						
Unadjusted	24.50 $\pm$ 1.04	23.10 $\pm$ 0.78	< 0.001	24.71 $\pm$ 1.02	22.98 $\pm$ 0.97	< 0.001
Age-sex adjusted	24.38 $\pm$ 0.09	23.22 $\pm$ 0.09	< 0.001	24.58 $\pm$ 0.81	23.30 $\pm$ 0.13	< 0.001
K, D						
Unadjusted	43.10 $\pm$ 1.59	43.17 $\pm$ 1.41	0.744	42.98 $\pm$ 1.36	43.03 $\pm$ 1.49	0.851
Age-sex adjusted	42.97 $\pm$ 0.15	43.31 $\pm$ 0.15	0.139	43.00 $\pm$ 0.13	42.99 $\pm$ 0.21	0.954
P, D						
Unadjusted	22.42 $\pm$ 1.85	23.17 $\pm$ 2.04	0.010	21.54 $\pm$ 1.43	22.77 $\pm$ 2.36	0.011
Age-sex adjusted	22.58 $\pm$ 0.20	22.98 $\pm$ 0.22	0.203	21.65 $\pm$ 0.15	22.35 $\pm$ 0.29	0.039
SE, D						
Unadjusted	-2.80 $\pm$ 2.05	0.69 $\pm$ 0.71	< 0.001	-2.38 $\pm$ 1.73	0.78 $\pm$ 1.12	< 0.001
Age-sex adjusted	-2.56 $\pm$ 0.15	0.43 $\pm$ 0.16	< 0.001	-2.26 $\pm$ 0.14	0.40 $\pm$ 0.27	< 0.001

Data of both eyes. ACD: anterior chamber depth; LT: lens thickness; AL: axial length; K: average keratometry; P: lens power; SE: spherical equivalent; D: diopters; SD: standard deviation. p value < 0.05 significant.

Table 3 shows the relationship results of ACD, LT, AL, and P with myopia or non-myopia in age- and sex-adjusted logistic models. In T1DM patients, eyes with longer AL were more likely to have myopia (OR, 10.665; p < 0.001), eyes with larger P were more likely to have myopia (OR, 1.348; p = 0.033), there were no significant associations between ACD or LT and myopia. In healthy controls, eyes with longer AL were more likely to have myopia (OR, 11.956; p < 0.001), there were no significant associations between ACD, P, LT, and myopia.

Table 3
Binary logistic regression with Dependent Variable myopia/non-myopia and Independent Variables ACD, LT, AL and P, Adjusted for Age and Sex in T1DM and Non-DM groups

	T1DM		Non-DM	
	OR (95 % CI)	p value	OR (95 % CI)	p-value
ACD	0.527 (0.09–3066)	0.476	0.277 (0.037–2.086)	0.213
LT	3.996 (0.244–65.505)	0.332	0.039 (0.001–2.961)	0.142
AL	10.665 (4.526–25.135)	< 0.001	11.956 (3.970-36.009)	< 0.001
P	1.348 (1.025–1.772)	0.033	1.400 (0.849–2.310)	0.187
Data of both eyes. ACD: anterior chamber depth; LT: lens thickness; AL: axial length; P: lens power; T1DM: type 1 diabetes mellitus; DM: diabetes mellitus; p-value < 0.05 significant				

In Fig. 1, the ACD curves showed an inverted U-shape in both subgroups, whereas the shape of non-DM controls was much deeper than T1DM patients. The peak of the non-DM group was around 8–10 years, and the peak of the T1DM group was around 11–13 years. The non-DM group showed shallower ACD in early and elder ages, while deepened in around 8 to 13 years when compared with the T1DM group. On contrary, the LT curves displayed like a U-shape in both subgroups, there was no cross of the two curves, the level of T1DM patients was consistently higher than the non-DM controls. The trough of the T1DM group was around 8–10 years, and the trough of the non-DM group was around 11–13 years. The changing trend of AL was approximately in a linear fashion, with the AL grew with age in both subgroups. AL was consistently higher in the non-DM controls, the non-DM group had faster axial elongation before 8–10 years then slowed down, T1DM group had faster axial elongation before 11–13 years then slowed down. No cross in the AL lines indicated the growth rate of the two subgroups were similar for all ages. The P curves were displayed in different ways of two subgroups. In the T1DM group, the P curves showed a transverse Z-shape, indicated no relationship with age; different from that, in the non-DM group, the P declined with age, with a marked reduction over the age from 5 to 10 years, and the decrease slowed down thereafter.

Discussion

In comparison between two myopia subgroups, which agreed with our previous study⁸, LT was found to be one of the main differences between myopia T1DM children and myopia controls. The myopia T1DM children showed a marked increase in LT accompanied by a significantly ACD decrease than myopia controls; P was further analyzed in this study, which showed a markedly increase in myopia T1DM children compared to the myopia controls. K was still identical within the two subgroups in the present study; AL was longer in myopia controls after adjustment of age and sex. Nevertheless, the lucubrate research now revealed that the differences between T1DM patients and healthy controls in ocular parameters(ACD, LT, AL, and P) only exist in myopia subjects rather than non-myopia subjects. A question arose that why only the myopic eye and how does the ocular biometry change in the myopic eye? We could read from the comparison between two healthy control subgroups, the myopia controls had longer AL than non-myopia controls, but the eye seemed to try to refocus on the retina again as the flattened LT and smaller P appeared to contend with it. When turning to the myopia T1DM children, notwithstanding the elongated AL versus non-myopia T1DM children, the unchanged LT and P made the compensation mechanism more passive. This was the evidence that myopia was suspected to be accelerated in T1DM children. In the Atropine for Treatment of Myopia study¹¹, less myopic progression accompanied by less increase in LT was found in atropine-treated eyes compared with placebo-treated eyes, when atropine was stopped, a marked increase in myopia and increase in LT was found compared with placebo-treated eyes. Although this was suspected to be the pharmacological effect of atropine, we still can see the relevance of LT increase and myopia increase. We have discussed the possible reasons for LT growth in T1DM children in the previous study, lens overhydration and less able to flatten due to ciliary muscles⁸. It was proved by MRI that the unaccommodated shapes of lenses in people with T1DM mimic the accommodated shape of lenses in people without T1DM¹². It probably the very reason that the lens lacks the ability to lose power in myopia T1DM children compared to myopia controls.

When we added LT in the logistic regression model, there was no significant relationship between LT and myopia both in T1DM and Non-DM groups, for LT was correlated with P¹³, therefore, the association between LT and myopia seemed to be reflected in the impact of P. Despite larger P was associated with a higher risk of having myopia in the T1DM group (OR 1.348, P = 0.033), it was not a risk of having myopia in the non-DM group (OR 1.400, P = 0.187). Owing to better elasticity in non-DM lens, a more relaxed lens avoid lens power to become a risk of myopia. Gao et al. examined the ocular components before and after cycloplegia, a significant decrease in LT and backward movement of the lens appeared after cycloplegia. That means during accommodation, there were both an increase in LT and a forward movement of the lens, while the myopia eyes showed the least change in LT and lens movement compared to hyperopic and emmetropic eyes. Also, before cycloplegia, the myopic eyes held the thinnest LT and the deepest ACD compared to the other groups¹⁴. In other words, the thinnest LT with the least thickness increase, meanwhile the deepest ACD with the least lens forward movement, which means that the myopia eyes maintained the thinnest LT and deepest ACD regardless of accommodative or not, which was the same with Li et al⁶. Deeper ACD and thinner LT also appeared in our study of myopia controls when compared to non-myopia controls, but deeper ACD and unchanged LT were found in our myopia T1DM children compared to non-myopia T1DM children. Namely, the lens in myopia T1DM children held

the ability to move backward in relaxed accommodation but cannot flatten. According to the Bennette-Rabbetts formula, ACD is a determinant of P here. However, P was smaller in myopia controls than myopia T1DM children (21.37 ± 0.16 vs 22.60 ± 0.17 , $p < 0.001$), while showed no significant difference in comparison between myopia T1DM children and non-myopia T1DM children after adjustment of age and sex (22.58 ± 0.20 vs 22.98 ± 0.22 , $p = 0.203$). In the logistic regression model, there was no significant relationship between ACD and myopia both in T1DM and non-DM groups. It was obvious that ACD change alone was not profound enough to down regulate P in myopia T1DM children.

The growth curves of ACD and LT were both displayed in a two-phase pattern, with the convex pointing to the opposite direction, U-shape for LT, and invert U-shape for ACD in not only myopia controls but also in myopia T1DM children. Our result agreed with the previous study of SCORM. It was speculated that the first decrease phase of LT was caused by stretching of the elongating eyeball, and the followed increase phase was by the thickness growth of the lens outpaced the stretching. Furthermore, the hyperopia children displayed a flat line and the emmetrope children showed a less concave line than myopia children¹⁵. Hence, we deduced the initial decrease of LT also attributed to the more flattened pattern in healthy myopia children. In our study, the myopia controls showed constant thinner LT comparing to the myopia T1DM group since the latter lack the ability to flatten. We inferred the ACD trend consist of several reasons, first, the two-phase pattern of the lens front surface; second, the first increase phase also caused by the backward movement of the lens in myopes, since in the SCROM study, the ACD showed an invert U-shape in children with myopia, while was constant in the children with hyperopia¹⁵; third, the increase phase caused by the growth of AL, but the unidirectional increase pattern of AL probably provided the least contribution. In our study, the additional thinning of the lens in myopia controls made the reciprocal ACD convex more profound than the myopia T1DM children.

The influence of LT and ACD would eventually lead to the influence of P. From the previous study, there was no doubt that P decrease with age in children^{7, 13, 16, 17}. Our result agreed with Xiong et al. that in myopia controls, P decreased rapidly before 10 years and slowed down after 10 years. They also analyzed the associations of P with SE or AL in healthy Chinese children and found P showed a positive correlation with SE when SE > -5.00 D meanwhile exhibited a stronger negative association with AL in non-myopes but a weaker negative association in myopes. They made it clear in their article that before the onset of myopia, P reduces in the compensation of AL elongation to maintain emmetropia, which is a co-effect of changes in the flattening and thinning of the lens and the decline in internal power due to the gradient is compacted, but P loss might be limited, while AL growth might have no endpoint, myopia develops when the rate of AL growth outpaces the compensatory loss of P, thus the compensatory ability of P became smaller not only with increasing age for its natural development but also in myopes as AL elongating⁷. In our study, AL grew with age, while P declined with age only in myopia controls but showed no trend of declination in the myopia T1DM group. The same as the above P compensation theory of myopes and non-myopes, in T1DM, the thickness of the lens and the gradient refractive index of the lens were suffered from hyperglycemia, which made the lens less capable to compensate for AL growth in myopia T1DM. As we can see from two myopia subgroups in our study, with the same level of SE ($p =$

0.064), longer AL ($p = 0.004$) and smaller P ($p < 0.001$) were found in myopia controls than myopia T1DM group, indicated that P was more tolerant to longer AL in healthy myopes.

AL elongation contributed most of the SE progression¹, it was not an exception here, the OR of AL was as high as 10.665 ($p < 0.001$) in the T1DM group and 11.956 ($p < 0.001$) in the non-DM group, compared to P, AL elongation played a more dominant role in having myopia. However, as for our knowledge, AL change was not caused by T1DM, so, we would not further discuss it in our study.

To conclude, in myopia T1DM children, though ACD deepened to a certain degree, LT failed to get thinner comparing to non-myopia T1DM children, still, the lens lacked the ability to lose power as compensation to AL growth, these were the evidence that myopia would be accelerated in T1DM children.

Declarations

Ethics approval and consent to participate

This study approved by the ethics committee of both Children's Hospital of Fudan University in Shanghai (approval number: No. 01 (2018)) and Shanghai General Hospitals (approval number: 2016KY005). This study conformed to the guidelines proposed in the Helsinki Convention. Written informed consent was obtained from each participant's parent in the study.

Consent for publication

The authors were made consent for publication of the paper.

Availability of data and material

The data used to support the findings of this study are available from the corresponding authors upon request.

Competing interests

The authors report no competing interests.

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Authors' contributions

Ying Xiao and Yu Qian contributed equally to this work.

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Figures

Figure 1

The curves of ACD (anterior chamber depth), LT (lens thickness), AL (axial length), P (lens power) changes with age from 5 to 16 years in two myopia subgroups (DM: myopia type 1 diabetes mellitus children, Non-DM: myopia controls), with every 3 years as a time point.