It has been demonstrated that VC protects the rat lens from oxidative damage induced by UVR type B (UVR-B) (Reddy and Bhat, 1999; Reddy et al., 1998) or hydrogen peroxide (Shang et al., 2003) in vitro. A few published studies have confirmed the efficacy of VC in preventing cataracts in vivo (Malik et al., 1995). Although a UVR-B exposure experimental system has been established using a representative animal model of cataracts, the effect of VC in preventing cataracts in this model has been demonstrated in only one report, describing a study of guinea pigs in which cataracts progressed when the animals were fed a low VC diet (Malik et al., 1995). But the cataract seen in that experiment was atypical because the lens opacity was located in the vicinity of the posterior capsule, unlike the anterior sub-capsular opacity often seen following exposure of mice (Jose, 1986; Meyer et al., 2005), rats (Wu et al., 1997), and guinea pigs (Mody et al., 2008) to UVR-B.

It has been shown that SMP30 decrease in amount in kidney and liver with aging, a decrease that is androgen-independent (Fujita et al., 1992). To clarify the physiological role of SMP30 in age-associated organ disorders, we used gene targeting to establish the SMP30 knockout (KO) mouse model from C57/BL6 mice (Ishigami et al., 2002). We recently reported that SMP30 KO mice have no gluconolactonase (GNL) activity (Kondo et al., 2006). Since GNL is a key enzyme in the VC biosynthetic pathway of mammals, mice deprived of GNL (SMP30 KO mice) lack the ability to synthesize VC (Ishigami and Maruyama, 2007).

We investigated whether the decreased VC in this mouse model increases the ratio of lens opacity induction by in vivo exposure to UVR-B.

2. Materials and methods

2.1. Animals

SMP30 KO mice were established and maintained as described previously. SMP30 KO mice were fed commercial chow (CRF-1; Oriental Kobo, Tokyo, Japan) and had free access to water containing 1.5 g/L VC in 10 μM EDTA until they were weaned at the age of 30 days. After weaning, the mice were fed VC-deficient chow (CL-2; CLEA Japan, Tokyo, Japan) and divided into 2 groups: VC sufficient (VC (+)) and VC deficient (VC (−)). The VC (+) group had free access to water containing 1.5 g/L VC in 10 μM EDTA, the VC (−) group free access to water containing 0.0375 g/L VC in 10 μM EDTA. Wild-type (WT) mice were fed commercial chow containing 12 mg/100 g VC (CRF-1; Oriental Kobo. Tokyo, Japan) and had free access to plain water without VC. The animals were maintained on a 12:12-h light–dark cycle in a controlled environment throughout the experiments. All animals were treated according to the Association for Research in Vision and Ophthalmology (ARVO) Statement for the Use of Animals in Ophthalmic and Vision Research.

2.2. Determination of total VC levels in lenses

After the mouse sacrificed, the eyes were enucleated and the lenses were extracted microsurgically. Lenses were homogenized in 5% metaphosphate/1 mM EDTA with a handy homogenizer (Mojimojikun; Nippon Genetics, Tokyo, Japan). The supernatant was obtained by centrifugation at 21,000 g for 15 min at 4 °C and immediately frozen at −80 °C until use. Total VC levels were determined using a high performance liquid chromatography (HPLC) electrochemical detection method as described previously (Sato et al., 2010). Samples were analyzed by HPLC using an Atlantis dC18 5 μm column (4.6–150 mm, Nihon Waters, Tokyo, Japan). The mobile phase was 50 mM phosphate buffer (pH 2.8), 0.2 g/L EDTA, and 2% methanol at a flow rate of 1.3 mL/min, and electrical signals were recorded using an electrochemical detector with a glassy carbon electrode at +0.6 V.

2.3. UVR-B exposure

UVR-B in the 302 nm wavelength region was generated with a transilluminator (TFML-20; UVP, Upland California, USA). UVR-B intensity was 200 mW/cm^2, measured with a radiometer (UV-340; CUSTOM, Tokyo, Japan) as irradiance in the corneal plane. The transilluminator was covered with aluminum foil except for a 5 mm hole. Each mouse was manually held (without anesthesia), such that the right eye was irradiated through the hole. The left eye was not irradiated and was used as control. Five minutes before UVR-B exposure, 1% tropicamide and 0.1% atropine sulfate hydrate were instilled in both eyes to induce mydriasis. Prior to exposure, all animals were checked with a slit lamp to exclude pre-existing cataracts. One eye of each mouse was exposed in vivo to UVR-B for 100 s twice a week for 3 weeks (total: 1200 mJ/cm^2). Each animal was kept for a latency period of 48 h from the last UVR-B exposure, based on data showing cataract progression after in vivo exposure to UVR-B, as described previously (Meyer et al., 2005).

2.4. Cataract morphology

Cataract development was observed with a slit-lamp microscope (Kowa SL-15, Nagoya, Japan). Immediately after lens extraction from the eyes, cataract morphology was documented by dark-field illumination with a microscope photography system.

Digital images of the anterior capsules were captured, and areas of cataract were measured using freely available National Institutes of Health Image J software. The ratio of the cataract area to the anterior capsule area was calculated as the cataract area ratio (Fig. 2a–d).

2.5. Histology

Eyes were fixed in Superfix (Kurabo, Osaka, Japan) as described previously (Yamamoto et al., 2008) and embedded in paraffin. Paraffin sections (3 μm thick) were prepared and stained with hematoxylin and eosin (HE).

2.6. Determination of lens protein contents

Radio immunoprecipitation assay buffer was added to isolated lenses, followed by crushing with a homogenizer and centrifugation at 4 °C and 1000 rpm for 30 min. The supernatant was harvested as a sample. Each 160 μL sample was combined with 40 μL of protein assay fluid (Bio-Rad, California, USA). The mixture was agitated and left standing at room temperature for 20 min. Then, absorbance at 595 nm was measured with a microplate reader.

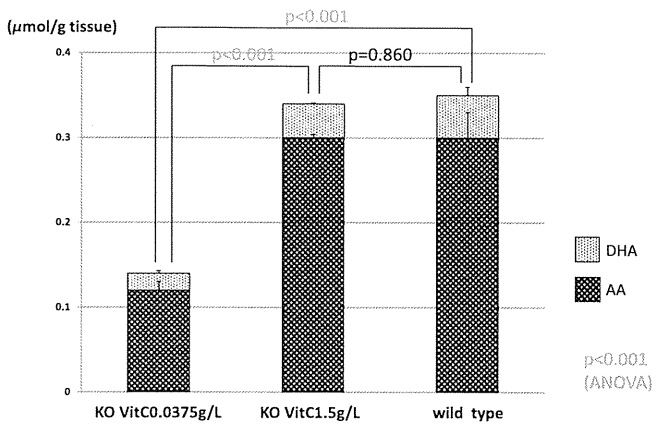

Fig. 1. Total vitamin C (VC) levels in lenses from SMP30 KO VC (−), SMP30 KO VC (+) and WT mice at age 16 weeks. Values are expressed as the means ± SD of 4 animals. DHA = dehydroascorbic acid; AA = ascorbic acid.

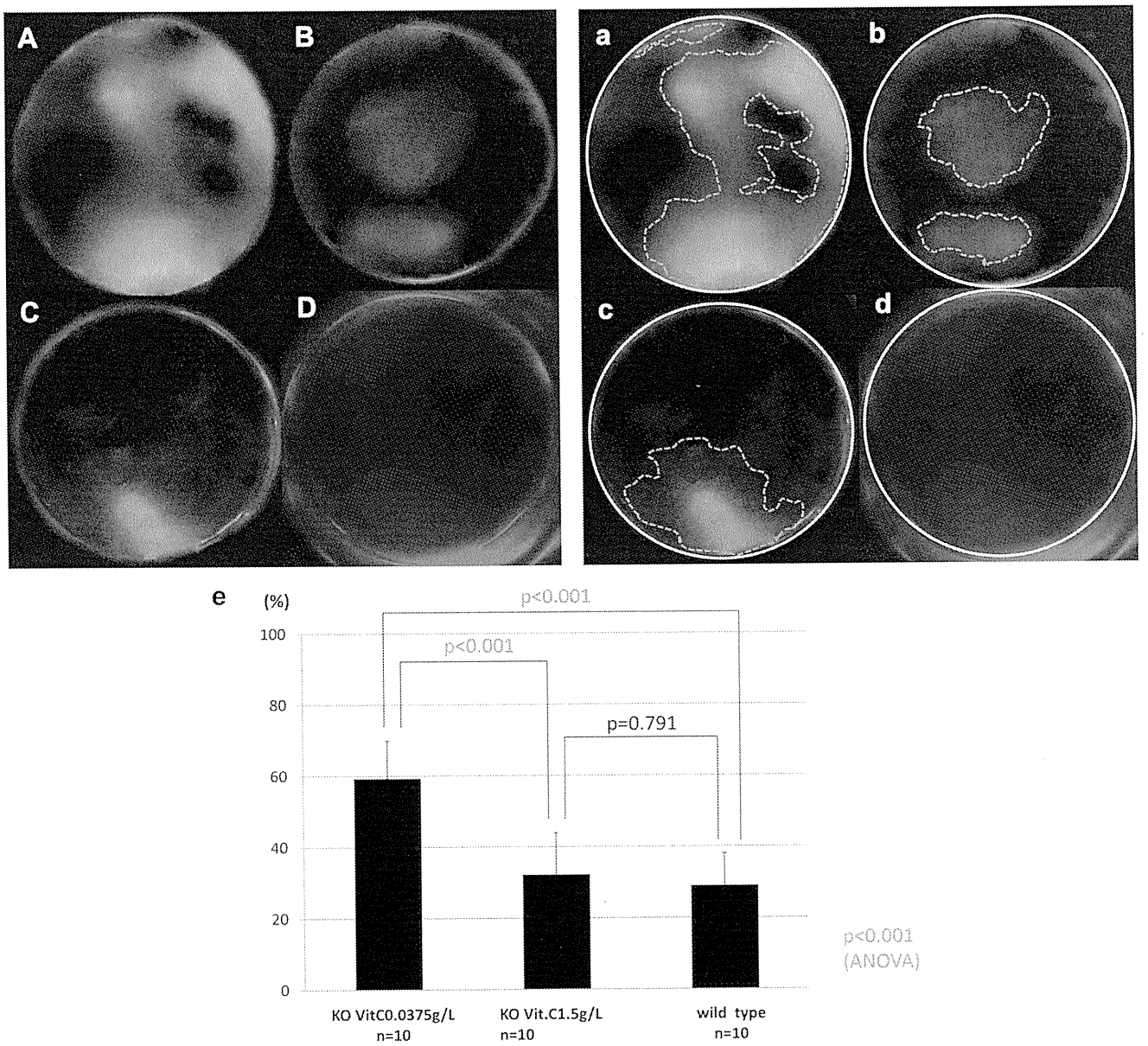

Fig. 2. Stereomicroscopic images of lens opacities were obtained 48 h after the last exposure to UVR-B. SMP30 KO VC (−) (A), SMP30 KO VC (+) (B), and WT (C). Unexposed lens of SMP30 KO VC (−) was clear (D). The cataract area was the range surrounded with a dotted line, and the anterior capsule area was the range surrounded with a solid line. (a, b, c, and d) As the actual area ratio, a is 58.2%, b is 27.6%, c is 26.4%, and d is 0.0%. The cataract area ratios were 59.3% ± 10.6% in the SMP30 KO VC (−) group, 32.2% ± 11.7% in the SMP30 KO VC (+) group and 29.0% ± 9.0% in the WT group. In the SMP30 KO VC (−) group the cataract area ratio was significantly higher than in the SMP30 KO VC (+) group and the WT group ($P < 0.001$) (e).

2.7. Statistical parameters

Data are expressed as means ± SD. Statistical analyses were performed using one-way ANOVA followed by the Tukey post hoc test for multi-group comparisons. SPSS for Windows (version 11.0, SPSS Inc., Chicago, IL, USA) was used for all statistical analyses. $P < 0.05$ was considered statistically significant.

3. Results

3.1. Total VC levels in lenses

We measured VC content in the lenses of SMP30 KO mice to confirm that eliminating VC from the diet led to VC deficiency and that supplementation via ad libitum access to VC-containing water resulted in essentially normal levels (Fig. 1). The total VC level for WT mice was approximately 0.36 ± 0.03 μmol/g lens tissue. The total VC level in lenses from the SMP30 KO VC (−) group was significantly reduced (0.14 ± 0.01 μmol/g lens tissue) at age 16 weeks ($P < 0.001$). The SMP30 KO VC (+) group (0.34 ± 0.004 μmol/g lens tissue), however, had VC levels similar to those of the WT group ($P = 0.860$).

3.2. Cataract morphology

48 h after the last exposure to UVR-B, lens opacity was observed mainly at anterior sub-capsular with a slit-lamp microscope. Slit-lamp examination revealed no lens opacity at 1 and 2 weeks. All exposed lenses of SMP30 KO VC (−), SMP30 KO VC (+), and WT mice developed lens opacity (Fig. 2A–C). The left (control) eye, not exposed to UVR-B, was free of opacity in all mice (Fig. 2D). Opacity of the UVR-B-exposed eye was more extensive in the SMP30 KO VC (−) group than in the SMP30 KO VC (+) and WT groups, with the

cataract area ratio being 59.3% ± 10.6% in the SMP30 KO VC (−) group, approximately double the ratios in the SMP30 KO VC (+) group (32.2% ± 11.7%) and the WT group (29.0% ± 9.0%) ($P < 0.001$; Fig. 2e).

3.3. Histology

HE staining findings were similar in the 3 groups. Anterior sub-capsular cell proliferation, a sign of anterior sub-capsular cataract, was accompanied by disturbed arrangement of surrounding lens fibers (Fig. 3).

4. Discussion

We investigated cataract development 48 h after unilateral in vivo exposure to UVR-B for 100 s twice a week for 3 weeks (total: 1200 mJ/cm^2) in SMP30 KO, a vitamin C deficient mouse model, and age-matched WT mice. In the SMP30 KO (−) group, exposure to UVR-B resulted in cataract nearly twice as extensive as that seen in the SMP30 KO (+) and WT groups. These findings suggest that decrease of VC increase the ratio of lens opacity induced by in vivo exposure to UVR-B. The free radicals formed in the lens exposed to UVR-B are scavenged primarily by glutathione and VC (Niki, 1991; Pirie, 1965). SMP30 KO (−) mice, characterized by low lens VC levels, were more susceptible to UVR-B damage, probably resulting in more extensive lens opacity. The severity of lens opacity in SMP30 KO (+) mice was similar to that in WT mice. It seems that the VC administered to these mice scavenged free radicals (formed in the lens following UVR-B exposure) to a degree similar to the scavenging of free radicals by endogenous VC in WT mice.

SMP30 KO mice develop scurvy-like symptoms and die at about 2 months after they are fed a VC-free diet and water. Keeping these mice alive requires administration of the minimum amount of VC needed for avoidance of manifestations of deficiency. For this reason, as in previously reported experiments, mice fed the minimum necessary amount of VC (0.0375 g/L, in aqueous solution) served as the VC deficient group (Iwama et al., 2011; Kashio et al., 2009). In view of a previous report demonstrating that administration of an aqueous solution with a higher concentration of VC (1.5 g/L) resulted in brain (Koike et al., 2010; Kondo et al., 2008; Sato et al., 2010), lung (Koike et al., 2010) and muscle (Sato et al., 2010)

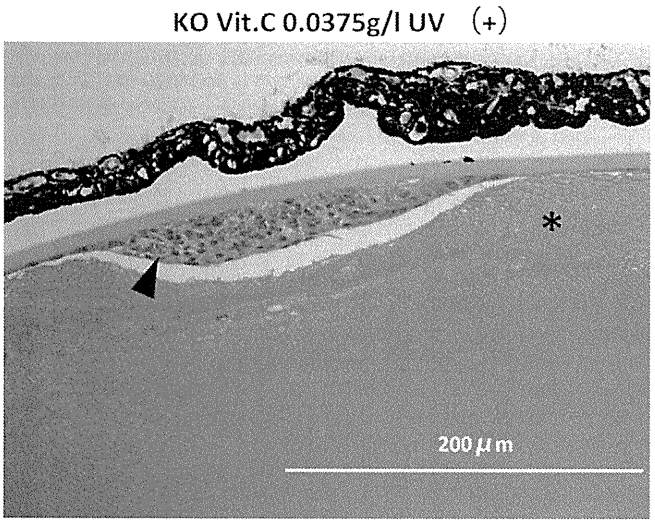

Fig. 3. Histological changes in the lenses of SMP30 KO VC (−) mice exposed to UVR-B. Cell proliferation in the anterior sub-capsular area (arrow head) is accompanied by disturbed arrangement of lens fibers in the surrounding region (*).

VC levels similar to those in WT mice under normal conditions, mice given this VC-containing aqueous solution served as the VC sufficient group. In mice given the 0.0375 g/L aqueous solution of VC, the lens VC level decreased to approximately 1/3 of that in WT mice. In mice given the 1.5 g/L aqueous solution of VC, the lens VC level was similar to that in WT mice. Thus, the 2 VC doses used in this study can be viewed as appropriate for achieving VC deficiency and sufficiency, respectively, in the SMP30 KO mouse lens.

There is one report describing observation of the lenses of VC-deficient guinea pigs exposed to UVB-R. In that experiment, lens opacity increased (Malik et al., 1995), as in the present study. However, the lens opacity seen in that experiment appeared to reflect not only the influence of UVR-B exposure but also nutritional disorders, for the following reasons: (1) the lens opacity was located in the vicinity of the posterior capsule, unlike the anterior sub-capsular opacity often seen following exposure of mice (Jose, 1986; Meyer et al., 2005), rats (Wu et al., 1997), and guinea pigs (Mody et al., 2008) to UVR-B; (2) the VC-deficient guinea pigs showed weight loss and were evidently in poor general condition; and (3) opacity was also seen in guinea pigs not exposed to UVR-B (Malik et al., 1995). In the animal model used in our study, it is unlikely that pathological conditions possibly causing nutritional disorders were present and evaluation of the influence of VC deficiency under conditions close to normal was apparently possible, taking into account the following facts: (1) the animals showed no evident external signs of health disorders at the time [14–17 weeks of age] of the experiments (Kashio et al., 2009); (2) the lens opacity was located in the anterior sub-capsular region, a finding similar to those in previously reported animal studies of UVR-B exposure; and (3) the eye not exposed to UVR-B was free of opacity.

Despite the development of lens opacity, there was no change in lens protein levels in the present study (data not shown). UVR-B with a wavelength close to 300 nm is known to attenuate on the anterior plane of the lens, and lenses exposed to UVR-B show changes largely confined to the sub-capsular area (Löfgren and Söderberg, 2001). In the present study, we analyzed the entire lens in the form of a homogenate. With this method, we cannot rule out the possibility that even if there had been changes in the anterior sub-capsular region, such changes might have been masked by proteins levels in the core and posterior cortex, which are not reached by UVR-B. Also, any changes might have been minimal and thus undetectable. VC is considered to play important roles in the maintenance of homeostasis and transparency of the lens through reducing and scavenging free radicals formed by various factors (Chiu and Taylor, 2007; Lou, 2003; Pirie, 1965). In the present study, administration of VC reduced the lens opacity induced by UVR-B and thereby exerted a protective effect. A possible mechanism underlying the activity of VC is that this vitamin reduces and scavenges the free radicals formed in the lens following exposure to UVR-B. However, since the present study did not incorporate measurement of VC levels (dehydroascorbic acid, ascorbic acid) after exposure to UVR-B, whether or not VC was directly oxidized as a free radical scavenger remains unknown.

Excessive administration of VC can stimulate the progression of cataracts (Cheng et al., 2001, 2006; Linetsky et al., 1999, 2008; Nagaraj et al., 1999). This is considered to be attributable to denaturation of cross-link-associated lens proteins caused by products of the oxidative degradation of VC, which in turn leads to the induction of cataracts (Nagaraj et al., 1999). In the present study, SMP30 KO mice were exposed to UVR-B in either a state of VC deficiency (1/3 of the VC level for WT mice) or a state similar to that in WT mice. To evaluate the influences of an excessive VC dose on cataract development, further study involving massive VC administration is needed.

We have shown that in SMP30 KO VC (−) group lens opacities induced by UVR-B were more extensive than in SMP30 KO VC (+)

group or in WT group, and in SMP30 KO VC (−) the lens VC level decreased to 1/3 of that in SMP30 VC (+) group or in WT group. These findings suggest that VC deficiency increases lens susceptibility to in vivo UVR-B induced oxidative stress in the mouse.

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特　集
眼内レンズにまつわるトラブル

5. 小児の場合

— Troubles after intraocular lens implantation in children —

田中三知子*　黒坂大次郎*

はじめに

小児への眼内レンズ（IOL）挿入は，慎重に適応を考える必要がある。成人の白内障と比較して手術の難易度が高いこともさることながら，小児白内障の症例数の少なさ，IOL挿入眼の合併症に付随する術後管理の特殊性を理解する必要がある。また，特に乳児におけるIOL挿入眼の視力予後が人工的無水晶体眼（コンタクトレンズや眼鏡矯正）に比べ，格段に優れているという明確なデータがないこと[1]もIOLの適応が慎重になる理由である。本稿では，小児IOLの現状と可能性，これまでに報告されている術後合併症と対策について解説する。

1. 小児IOLの可能性と現状

小児でも，年長児のIOLでは成人と同等なメリットがあり，多数の良好な術後成績が報告されている。では，より年少の，特に乳児においてはどうであろうか。現状での乳児IOLのデメリットは，術後合併症の多さであり，術後視力においてもコンタクトレンズよりも優れた結果を得るに至っていない[1]。しかし，aphakiaと決定的に違う点は，IOL挿入眼の術後屈折異常はaphakiaに比べて格段に軽く，裸眼でも大まかな視機能を恒常的に有していることである。IOLでも眼鏡やコンタクトレンズでの屈折矯正が必要だが，aphakiaの眼鏡・コンタクトレンズと比べて重量が軽く，重みで偏心することも少ない。したがって，術後合併症の問題を克服し，屈折変化に適切に対応できれば，IOL挿入眼はaphakiaよりも優れた視機能をもたらす可能性が十分にあるといえる。

現在，小児へのIOLの適応としてコンセンサスが得られているのは，2歳以上，白内障以外の眼合併症がない例であり，IOLは原則として嚢内固定である。素材はfoldableの疎水性アクリルが広く用いられるようになり，一部では多焦点IOLの有効性も検討されはじめている。眼合併症がある場合でも，程度によってはIOL挿入が行われるようになった。最近では，ぶどう膜炎患者の併発白内障においてもIOL挿入が行われ，IOLの有効性と安全性が検討されている。しかし，小眼球・小角膜を含め，前眼部形成不全では水晶体嚢の大きさが十分でないこともあり，IOLは積極的に挿入されていない。

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Key words：小児，眼内レンズ（IOL），合併症

表1

	IOL	Aphakia
平均月齢(カ月)	1.1～3.1	1.2～3.2
症例数(眼)	同一症例の，それぞれ57眼ずつ	
最も多い術後合併症	後発白内障 71.9%	緑内障 9%
2番目に多い合併症	瞳孔偏位 19%	硝子体出血 網膜出血 網膜剝離 皮質の残余 それぞれ5%
3番目に多い合併症	緑内障 15.7%	
4番目に多い合併症	硝子体出血 7%	
合併症発生率	77%	25%
12カ月での視力(logMAR)	0.97	0.80

II. IOLの有無による術後合併症の違い

The Infant Aphakia Treatment Study Group[1]は，生後6カ月までの両眼の先天白内障において，片眼をIOLで，もう片眼をaphakia＋コンタクトレンズで矯正した114名，それぞれ57眼の予後を調査した(**表1**：文献1より改変して転載)。この調査により，生後6カ月未満の小児IOLでは，特に後発白内障が多くみられることが判明した。瞳孔偏位もIOL群で有意に多くみられるが，緑内障以下には有意差がない。すべての合併症を合わせた発症率はIOL群で77%，aphakia群で25%と，IOL群で有意に多い結果であるとともに，生後12カ月での術後視力には両群間で有意差がみられなかった。

国内では，このような同一症例でのIOL/aphakiaの比較はないが，稲富ら[2]が行った小児IOLのアンケート調査により，小児IOLにおける術後合併症の内訳が明らかにされている(**表2**：文献2より改変して転載)。白内障の成因にかかわらず，3割以上の症例に後発白内障がみられる。先天/発達白内障に含まれた3歳未満の症例は45/359例であった。外傷性白内障では虹彩後癒着，角膜内皮細胞減少，緑内障の割合が高く，併発白内障はアトピー性白内障を含んでいるため網膜剝離の割合が高い。

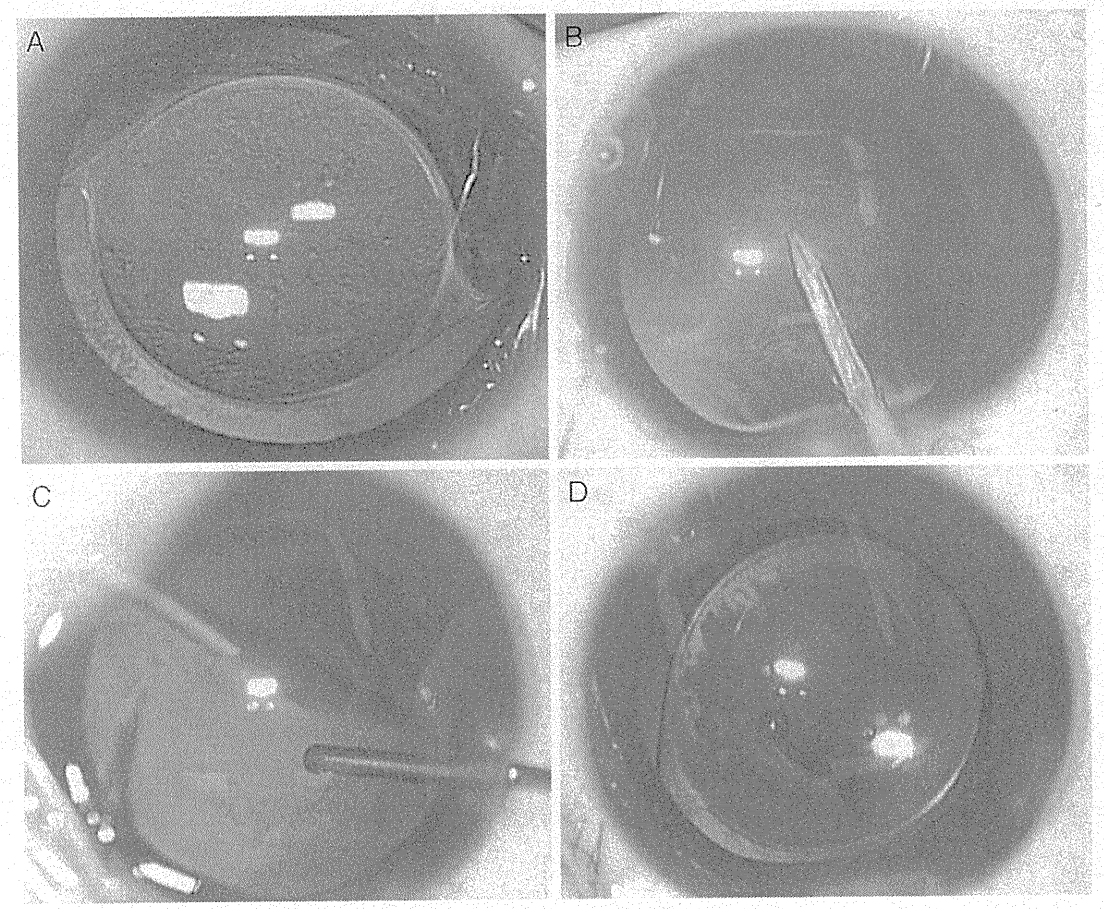
❶ 後発白内障(visual axis opacification：VAO)[3]

VAO〔狭義の後発白内障(**図A**)だけでなく，水晶体嚢の収縮，deposits，膜増殖などの視軸の混濁を含める〕は，最も多くみられる合併症で，患者が年少であるほど高度にみられ，視力予後を左右する。通常はVAOの予防目的に，PCCC(posterior continuous curvilinear capsulorrhexis)と前部硝子体切除を行う(**図B～D**)が，小児のIOLにおいては，PCCCと前部硝子体切除をしている場合でも高率にVAOがみられ，なかでも，6カ月未満の症例では71.9%[1]から80%[3)～7)]と，群を抜いて多くみられる。2歳以上で，同じくPCCCと前部硝子体切除をしている場合には平均5.1%と発生率が低下することから[3]，患児の年齢(月齢)が低いことは，後発白内障の発生を高めるリスクファクターである。

以前に行われていたVAO予防のための，

表 2

	先天 / 発達白内障	外傷性白内障	併発白内障
平均年齢(歳)	8.1	9.4	12.6
症例数(眼)	359	69	70
最も多い術後合併症	後発白内障 35.7%	後発白内障 36.2%	後発白内障 35.7%
2 番目に多い合併症	一過性炎症 10%	瞳孔変形 / 後癒着 17%	網膜剝離 15%
3 番目に多い合併症	瞳孔変形 / 後癒着 6%	角膜内皮細胞減少 17%	一過性炎症 7%
4 番目に多い合併症	IOL 偏位，網膜剝離 それぞれ 2%	緑内障 11%	一過性高眼圧 3%
合併症発生率	56.5%	82.6%	67.1%
術後視力 0.5 以上	66.3%	81.2%	80%



図

PCCC を行わなかった例の後発白内障(A)。PCCC(B)と前部硝子体切除(C)。現在は外来で YAG レーザーができない症例には PCCC と前部硝子体切除術をルーチンで行っている。眼内レンズ挿入後(D)。

IOLの光学部をPCCCに陥頓させるoptic capture[8]は，前嚢収縮により前嚢切開が必要になり[9]，硝子体切除を併施した場合には硝子体脱出を助長し[10]，併施しない場合には前部硝子体膜上に線維性の混濁をきたすこと[11]から，現在はあまり行われていない[10][12]。

VAOの治療としては，6～8歳をひとつの目安に，外来でYAGレーザーを行う施設が多い。それ以下の年齢では，硝子体カッターでの混濁切除がメインとなる。

IOL表面の瞳孔膜やdepositsは，炎症が強いほど，また患児が低年齢であるほど高率にみられる[7]。発生すると除去が困難であり，術後の十分な消炎が求められる。ステロイドおよび非ステロイド系消炎剤の点眼に加えて，アトロピン点眼を用いて消炎に努める。全身ステロイド投与は症例に応じて用いる。

❷ 虹彩後癒着，瞳孔偏位[3]

虹彩後癒着や瞳孔偏位も，炎症が強く，患児が低年齢であるほど高率にみられる[9]。虹彩が全周で癒着すれば瞳孔ブロックによる緑内障を発症する。特にぶどう膜炎症例では虹彩後癒着を起こしやすいので，周辺虹彩切除，瞳孔管理，全身ステロイド投与などの対策が必要である[13]～[15]。

❸ 緑内障[3]

IOLの有無にかかわらず，小児の白内障術後には緑内障を合併することが知られている。その原因としては，術後の炎症や水晶体起因物質の目詰まり，隅角形成異常や瞳孔ブロックなどが考えられ，開放/閉塞のどちらの緑内障も発症し得る[16]。小児の白内障術後の緑内障の発症頻度は，IOL群で0.3～3.8％に対して，aphakia群では11.3～17％と，IOL群で有意に低い[17]。しかし，生後4.5カ月までに白内障を手術した例における緑内障の発症頻度は，aphakia群では19％，IOL群では24.4％とIOLの有無による有意差はなくなる[17]。これはthe Infant Aphakia Treatment Study Groupの調査でも同様の結果で[3]，6カ月未満では，IOLの有無にかかわらず，等しく術後緑内障の発症リスクがあるといえる。

緑内障の発症例では，角膜の透明性・直径，視神経の所見に注意し，点眼で無効な場合には，緑内障手術を検討しなければならない。

❹ 網膜剝離

小児IOL術後の網膜剝離の発症頻度は，aphakiaよりも低い（アトピー性白内障を除く）[3]。IOL挿入例では，IOLが前眼部と後眼部を隔てているので硝子体が前房に脱出しにくく，またpars plicata lensectomyを行わないので，毛様体に裂孔を生じる危険性が低いことがその理由と考えられる。IOL挿入後の網膜剝離の原因ははっきりしないが，PCCCからの硝子体の脱出や手術による硝子体の液化の進行が原因と考えられている[18]。

❺ その他

乳児では，水晶体嚢の容積が小さく，かつ収縮が強いので，IOLが嚢外や前房に脱出する可能性がある。水晶体嚢の容積が小さい前眼部形成不全でIOLの適応が慎重なのは，これがひとつの理由である。CCCが流れた場合は，特に慎重に観察する必要がある。術後の屈折および弱視管理も重要な要素であるが，これは術前の検査が万全に行われることが前提である。それでもIOL挿入後の屈折が予測と異なることも起こり得る[1]。どの程度の度数までなら許されるか，明確なデータはないが，少なくとも眼軸長が大きく変化する3歳未満で強度近視であれば，その後さらに近視化し問題になる可能性があると思われる。

まとめ

小児のIOL挿入後は，VAOが起こりやすく，その他の合併症も含めて，炎症のコントロール

が要のひとつであり，特に乳幼児のVAOには早急な対応を要する。これまでの症例のひとつひとつの積み重ねから，トラブルとIOLの適応/不適応が徐々に明らかになってきた。症例の少なさと特殊性ゆえに，今後も専門施設での手術がメインになる。さらなる術後症例の検討が進めば，長期予後と安全性，また，どの時期にどんな方法でIOL挿入を選択すれば(あるいは選択しなければ)aphakiaよりも視力予後がよいのか，より明確になることが期待される。

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