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Type V collagen fibrils in mouse metanephroi

Han-Hsiu Hsu [a], Yusuke Murasawa [b], Pan Qi [a], Yusuke Nishimura [a], Pi-Chao Wang [a,*]

[a] Graduate School of Life and Environmental Science, University of Tsukuba, Japan
[b] Department of Advanced Medicine, National Center for Geriatric and Gerontology, Japan

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ABSTRACT

Type V collagen (Col V) molecule, a minor component of kidney connective tissues, was found in adult cornea, and has been considered as a regulatory fibril-forming collagen that emerges into type I collagen to trigger the initiation of Col I fiber assembly. Col V was also found in injured, wound healing tissues or placenta, and was considered as a dysfunctional extracellular matrix (ECM). Reconstituted Col V fibril was characterized as an ECM to detach cells *in vitro*, and our previous study showed that the reconstituted Col V fibril facilitated the migration of glomerular endothelial cells and induced ECM remodeling, whereas Col V molecules stabilized cells. These facts suggest that not only the structure but also the function of Col V fibril are different from Col V molecule. Recently, Col V molecule has been reported existing in various developing tissues such as bone and lung, but Col V fibril has not been reported yet. In this study, we firstly explored the existence of Col V fibril in metanephroi, and found it distributed in the immature kidney tissues whereas disappeared when the tissues reached mature. It is likely that Col V fibril may form a prototype of pericellular microenvironment and the transient existence of Col V fibril may play a role as the pioneering ECM during metanephric tissue morphogenesis.

1. Introduction

Recently, type V collagen (Col V) molecule has been reported existing in various tissues and organs at the developing stage such as developing tendon and bone [1]. The existence of Col V may be related to the tissue and/or organ development such as cornea and tendons [1,2]. However, the structural fibril of Col V fibril in the developing tissue has not been reported yet. Col V molecule isolated from cornea has been considered as a regulatory fibril-forming collagen that is buried within type I collagen (Col I) [2] to trigger the initiation of Col I fiber assembly and form the heterotypic fibers [2–4]. However, fibril type of Col V in the normal mammals had not been identified and became a long controversy until it was isolated and reconstituted from human placenta [5]. Our previous study revealed that the reconstituted Col V fibril enhances the migration of renal glomerular cells whereas Col V molecule stabilizes cells in cell culture [6]. Therefore, not only the structure but the function of Col V fibril is considered different from Col V molecule. Col V exist with injured tissues such as collagen fibrionic glomerulopathy and progeria syndrome and in wound healing tissues [7,8], which rendered Col V as a dysfunctional ECM, but the structural types of the Col V molecule or fibril, is still in doubt.

Our previous research verified that Col V fibril not only facilitated the migration of glomerular endothelial cells, but also triggered the ECM outside-in signaling into cells, and induced ECM remodeling such as Col IV secretion after the degradation of Col V fibril [6]. These facts imply that Col V fibril may be a beneficial ECM rather than a dysfunctional ECM for the unstable tissues such as developing tissues. However, no report on the existence of Col V fibril in the developing kidney tissues has been investigated yet. In this study, we investigated both the existence and the distribution of Col V fibril at each stage of the developing metanephric kidney.

Metanephros, an embryonic kidney, is originated from two promordia, ureteric bud (UB) and metanephric mesenchyme (MM). UB appears at the early developing stage of E11.5 (embryonic day 11.5) with protruding tips for further branching and elongation to form thick collecting ducts and thin tubules, while MM interact with UB and differentiate into comma-shaped, S-shaped bodies, and glomeruli [9]. It has been reported some other types of collagen such as Col I, III, and IV appeared in the developing tissue such as MM nephrogenesis [10], angiogenesis [11], and chondrogenesis [12], which were related to ECM remodeling. In this study, we also found that Col V can provide a transitional microenvironment for developing process of metanephros. It is suggested that the transient existence of Col V fibril is likely to play a role as a pioneering ECM to lead maturing ECM like type I collagen fiber and type IV

* Corresponding author. Address: Graduate School of Life and Environmental Science, University of Tsukuba, 1-1-1 Tennodai, Tsukuba City, Ibaraki 305-8572, Japan.
E-mail address: wang.pichao.ft@u.tsukuba.ac.jp (P.-C. Wang).

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collagen basement membrane during the metanephric tissue morphogenesis.

2. Materials and methods

2.1. Isolation of metanephric kidneys

ICR female mice (Japan SLC) were mated overnight and vaginal plug was detected in the following morning, and this was considered 0.5 day of gestation (E0.5). Metanephric kidneys were isolated from embryos at E11.5, E13.5, E15.5, E17.5 and the first day of procreation (P0.5) with 30-gauge needles (Dentronics) under dissecting microscope (Olympus). Isolated metanephroi were maintained in DMEM medium (Nissui).

2.2. Reverse transcription polymerase chain reaction (RT-PCR)

mRNA from metanephroi was isolated by using an RNA extraction kit (Isogen, Nippon Genes), followed by RNA quantification with microvolume spectrophotometer (Thermo). An appropriate amount of mRNA was reverse transcribed to cDNA and amplified with SuperScript III One-Step kit (Invitrogen) using primers of Col V α1 chain (sense: 5′-CCGGATCCTGAGCCCACCGGTCTC and antisense: 5′-CCGAATTCCATCGGAAAGGCACGTG) and the primers of GAPDH (sense: 5′-TGTTCCTACCCCCAATGTGT and antisense: 5′-TGTGAGGGAGATGCTCAGTG) was used as a control.

2.3. ECM protein extraction from metanephroi and Western blot analysis

Murine metanephroi were extracted with 6 M guanidine hydrochloride (Gdn), 50 mM Tris–HCl, 1 mM PMSF, 1% (v/v) protease inhibitor cocktail (Sigma), pH 7.5 at 4 °C for 72 h, and the supernatant was collected by centrifugation at 12,000 rpm for 20 min. ECMs extracted without enzyme from tissue were analyzed by Western blotting as previously described [6,13]. Briefly, samples were resolved in 7.5% gels by SDS–PAGE under non reducing conditions. Separated proteins were transferred onto nitrocellulose membrane. The membrane was blocked with 5% nonfat skim milk (Dako) in TBST at room temperature for 1 h, followed by incubation with antibody against Col V (LSL) (1:1000) in TBST containing 2% milk. HRP-conjugated anti-rabbit IgG (Dako) was used for detection and development of the blots were facilitated by enhanced chemiluminescence (ECL; GE Healthcare, Bio-Sciences).

2.4. Preparation of metanephric kidney cross section

Isolated metanephroi were equilibrated with 30% sucrose for 12 h. Samples were then removed from sucrose to a stainless box (1 × 1 × 1 cm) filled with O.C.T. compound (Sakura Finetek), frozen in liquid nitrogen bath for 5 min and then stored at −80 °C for 1 h. Thickness of 10–20 μm sections were sliced with cryostat (Leica CM3050III) at −20 °C, mounted on slide glass (Matsunami), and air-dried for 10 min. Tissue sections were then washed with 0.01% detergent, distilled water, and fixed with 10% formalin/PBS (Wako) for 1 h.

2.5. Immunohistochemistry

Cross sections were fixed with 10% formalin (Wako), permeabilized in 0.1% saponin (Sigma), and subjected to blocking treatment with 0.1% BSA/PBS. Blocked samples were applied to primary antibodies (1:1000), incubated for 1 h at RT, washed in PBS 3 times for 10 min at RT, and incubated with secondary antibodies (1:1000) for another 60 min at RT. Samples were incubated with DBA for UB epithelia staining. For finalization, samples were washed with PBS 3 times for 10 min at RT, submerged in 20 μl fluoromount (Diagnostic BioSystems), covered by cover glasses (Iwaki) and sealed with mountquick (Daido Sangyo Co., Ltd). Negative control stained without primary antibodies was shown in Supplementary data, Fig. S1.

For primary antibodies, goat and rabbit polyclonal antibodies (pAb) against Col Vα1 chain and pAb against Col I (Santa Cruz Biotechnology) were used. Rabbit pAb against laminin α1 and α5 (Santa Cruz Biotechnology) were used to stain mature and immature basement membrane, respectively [14]. FITC-conjugated Dolichos Biflorus Agglutinin (DBA, Vector Lab) was used to stain UB epithelia [15]. For secondary antibody, HRP-conjugated anti-rabbit IgG (Dako), Alexa Fluor 568 donkey anti-goat IgG and donkey anti-rabbit (Invitrogen), Alexa Flour 488 donkey anti-goat IgG and chicken anti-rabbit (Invitrogen), Alexa Flour 633 goat anti-rabbit IgG (Invitrogen) and 5 nm gold conjugated-goat anti-rabbit IgG (GE Healthcare) were used.

2.6. Confocal imaging

Tissue sections were visualized using a confocal microscope (Carl Zeiss LSM700 V2URGB Stitch), which excluded nonspecific fluorescence from the metanephric kidneys. Dual-color images were captured in a sequential manner. Negative controls were scanned before sample scanning. Nonspecific fluorescence was not observed in negative controls (absence of primary antibodies) using 10% laser power, 720 gain, and −0.50 offset. Subsequently, each sample was scanned using these standard conditions. FITC and Alexa 568 fluorescent signals were detected at 488 and 543 nm laser excitation, respectively. Specimens were also observed by differential interference contrast (DIC) and double immunohistochemistry imaging. All images were obtained using a 40× water-immersion objective. Scanning was performed with a pinhole size of 1.0 airy unit and 8 times line averaging. The images were stored in a 512 × 512-pixel, 12-bit tiff file format.

2.7. Immunoelectron microscopy

Samples were prepared according to the detailed steps described previously [16]. Briefly, metanephric kidneys were fixed in solution A (1% glutaraldehyde and 2% formaldehyde in 0.1 M KHMg buffer adjusted to pH 7.4) for 2 h, followed by solution B (1% glutaraldehyde and 2% formaldehyde in 0.1 M NaHCa buffer adjusted to pH 7.4) overnight. The fixed samples were dehydrated in ascending ethanol series up to 99.5% and embedded in Lowicryl K4M resin (Polysciences, Warrington, PV). Ultrathin sections (95 nm) were mounted on nickel grids coated with formvar, and immune-labeled with anti-Col V antibody (LSL) (diluted 1/200). For control, anti-Col I antibody (LSL) (diluted 1/200) was used. Sections were washed with PBS and incubated with 5 nm gold conjugated-goat anti-rabbit IgG antibody (1:25 dilution: GE Healthcare). All sections were stained for 5 min with uranyl acetate before observation.

3. Results

3.1. Continuous Col V expression in metanephroi

Two subtypes of Col V, $[\alpha1(V)]_2\alpha2(V)$ and $\alpha1(V)\alpha2(V)\alpha3(V)$, have been discussed in Col V fibril structural formation [5]. As Col V α1 chain exists in both subtypes, its cDNA was investigated in metanephroi at E11.5, E13.5, E15.5, E17.5, P0.5, by RT-PCR amplification with GAPDH as internal control (Fig. 1A). Col Vα1

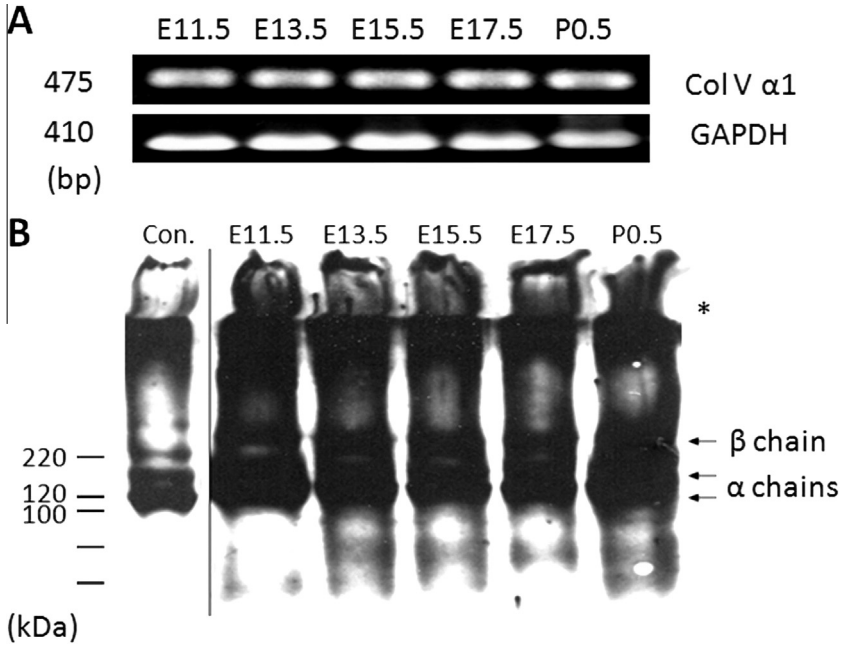

Fig. 1. Expression of Col V during metanephric development. Reverse transcription PCR (RT-PCR) and Western blotting showed Col V expression in E11.5–P0.5 metanephroi. (A) RT-PCR results showed cDNA of Col V α1 chain in all stages of metanephric development. GAPDH was used as control. (B) Multiple bands of Col V including α and β chains (arrows) were confirmed by Western blotting under non-reducing and non-digesting conditions. Migrating bands over 400 kDa at stacking gel showed high molecular super-structural aggregates of Col V (asterisk). Porcine corneal Col V was used as internal control (con.).

gene was continuously detected from E11.5 to P0.5 with little change.

Western blot analysis showed the same continuous expression as its gene expression. Extracted ECMs from immature tissue can be migrated and separated in gel of SDS–PAGE under non-proteinase digesting condition by protease. pAb against Col V revealed multiple bands present in extracts of metanephros prepared with 6 M guanidine. Multiple bands, α and β chains (arrows), were obviously detected (Fig. 1B). Moreover, the band migrating over 400 kDa (Fig. 1B, asterisk) and through the stacking gel, revealed the existence of Col V aggregates (Fig. 1B). These results indicated that Col V continuously expressed in the developing kidney from the early stage of E11.5 till the late stage of P0.5. In order to identify its spatial and temporal changes in metanephros, the distribution and localization of Col V in E11.5–P0.5 metanephroi were further investigated. Bands at lower molecular weight represent monomer of Col V molecule. Those around 220 kD represent dimmers and trimmers of Col V molecule and asterisk at high molecular weight over 400 kDa represent multimers and aggregates of Col V molecule.

3.2. Spatial and temporal changes of Col V in metanephroi

3.2.1. Col V localization during UB and renal tubule morphogenesis

Localization of Col V in metanephroi was investigated by immunohistochemistry (Fig. 2). FITC-DBA antibody (green color) was used as a UB marker, which was detected at newly forming UB tip E11.5 (Fig. 2A, arrow), thick trunks at E13.5 (Fig. 2B), tree-like structures at E15.5 (Fig. 2C), E17.5 (Fig. 2D) and P0.5 (Fig. 2E). Col V (red color) at early stage (E11.5–13.5) of metanephric development was observed in the whole metanephros, notably at renal capsule located at the edge (Fig. 2F), UB tip (Fig. 2F and P, arrow), cortex region (Fig. 2G), and UB trunks (Fig. 2G and Q, arrows). At the middle stage of E15.5, Col V was found localizing at nephrogenic zones in cortex region (Fig. 2H), and in UB tubules (Fig. 2H and R, arrows). These data showed Col V might be related to UB morphogenesis. Interestingly, at late stage of E17.5–P0.5, Col V expression in pre-mature collecting ducts turned to weak (mature UB, Fig. 2S and T, arrows), while that in the lumen of newly forming renal tubules remained strong (Fig. 2S and T, asterisks), indicating that Col V appears in immature tissues and disappear in mature ones.

3.2.2. Col V localization in premature glomerulus and its primordia

Col V expressions in glomeruli and its primordial including comma-shaped body and S-shaped body were confirmed by immunofluorescence and DIC (Fig. 3). Immunohistochemistry showed Col V expression in comma-shaped body (Fig. 3A-1), the expression continued and mainly distributed in S-shaped (Fig. 3B-1) at E15.5, and then concentrated in the immature basement membrane of glomerulus (Fig. 3C-1) at E17.5, and finally turned to weak when tissue reached maturation at P0.5 (Fig. 3D-1). Immature basement membrane (Fig. 3A-2, B-2, C-2, green) and mature one (Fig. 3D-2) were detected in glomerular primordial and glomeruli, respectively. Col V co-exists with immature basement membrane (Fig. 3A-3, B-3 and C-3), but was not observed in mature basement membrane (Fig. 3D-3). These results indicate that Col V distributed in immature tissues, and gradually disappeared in mature ones.

3.3. Fibril structure of Col V in developing metanephric kidney

3.3.1. Structure of Col V in cross sections

Col V fibril structure in metanephric kidney at the early stage of development was confirmed by immunogold electron microscopy (Fig. 4A–C). Col V fibril aggregations (arrows) were observed inside and outside of both UB epithelial cells (Fig. 4A) at E12.5. Very thin fibrils (arrows) and fibrils (arrow heads) were detected at the intercellular spaces of MM aggregations at E12.5 (Fig. 4B) and podocyte slit at E15.5 (Fig. 4C). As a control, Col I was observed as a thick fiber (Fig. 4D, asterisk) among cells of MM aggregations similar to the location of Col V fibrils. These results indicate that Col V presented as thin fibrils or in aggregated structure, whereas Col I presented as thick fibers in metanephric kidney. The temporal fiber formation of these two types of collagens in metanephric kidney was further investigated.

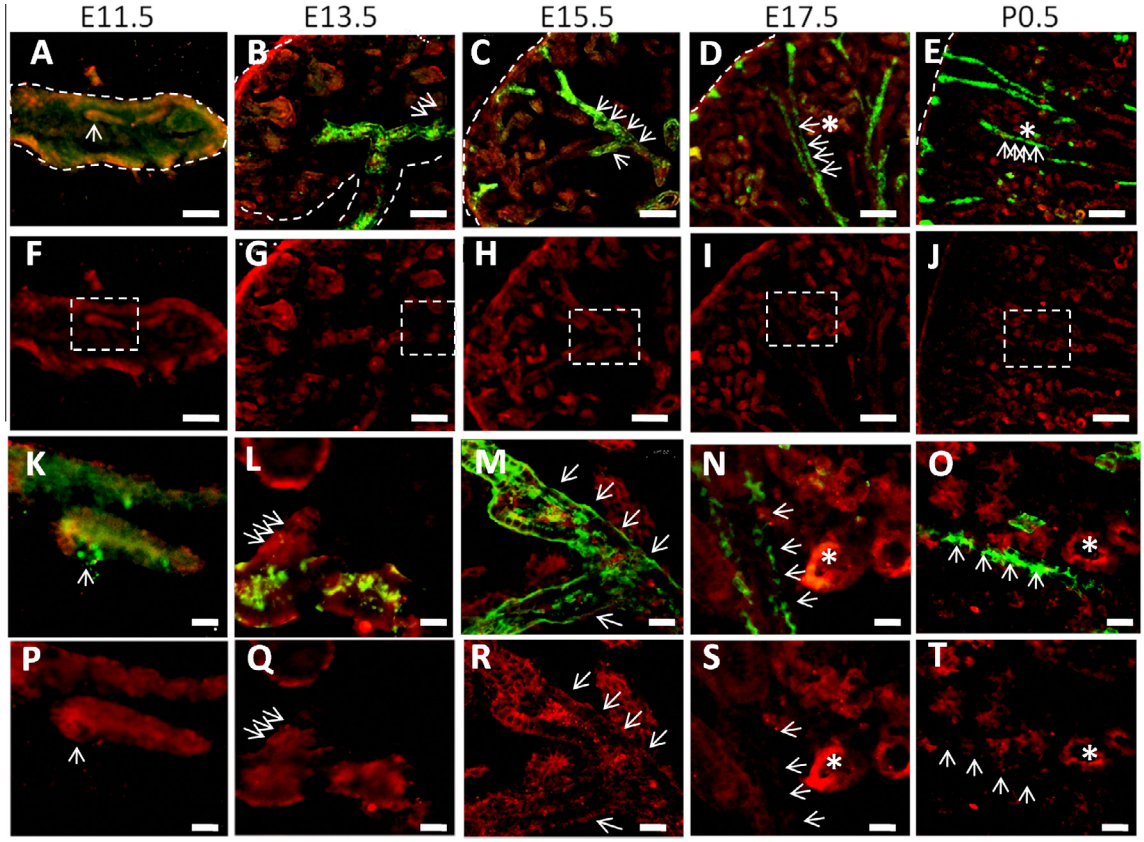

Fig. 2. Localizations of Col V during metanephric development. Immunohistochemistry showed localizations of Col V (red) in metanephroi during E11.5–P0.5. Developing UBs are stained by FITC-conjugated Dolichos Biflorus Agglutinin (DBA, green). (A–E) Merged images of Col V (red) and DBA staining (green); (F–J) Col V staining images (red); (K–O) enlarged merged images and (P–T) enlarged Col V staining from dotted squares in (F–J), respectively. Scale bars indicated 100 μm for (A–J), 20 μm for (K–T). White dot lines in (A–E) indicated edges of metanephroi. Arrows indicated branched or elongated UBs and/or collecting ducts. Asterisks indicated immature renal tubules. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

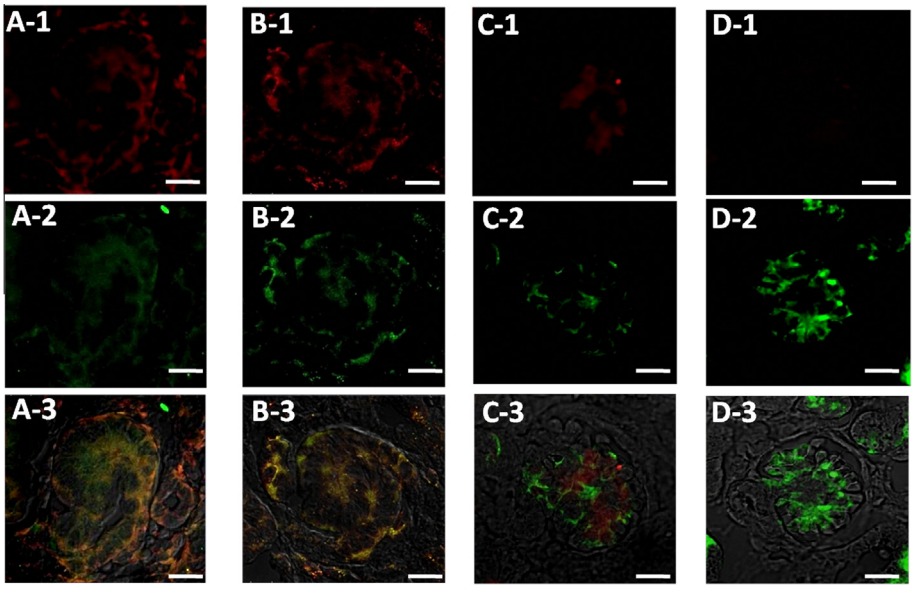

Fig. 3. Localizations of Col V in glomerular primordia. Immunohistochemistry of Col V (red), immature basement membrane marker laminin α5 (A–C, green), and mature basement membrane marker laminin α1 (D, green) merged with differential interference contrast (DIC, bottom) showed Col V mainly localized in glomerular primordial: comma-shaped body (A), S-shaped body (B), premature glomerulus (C), but very few in mature glomerulus (D). Scale bars indicated 20 μm. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

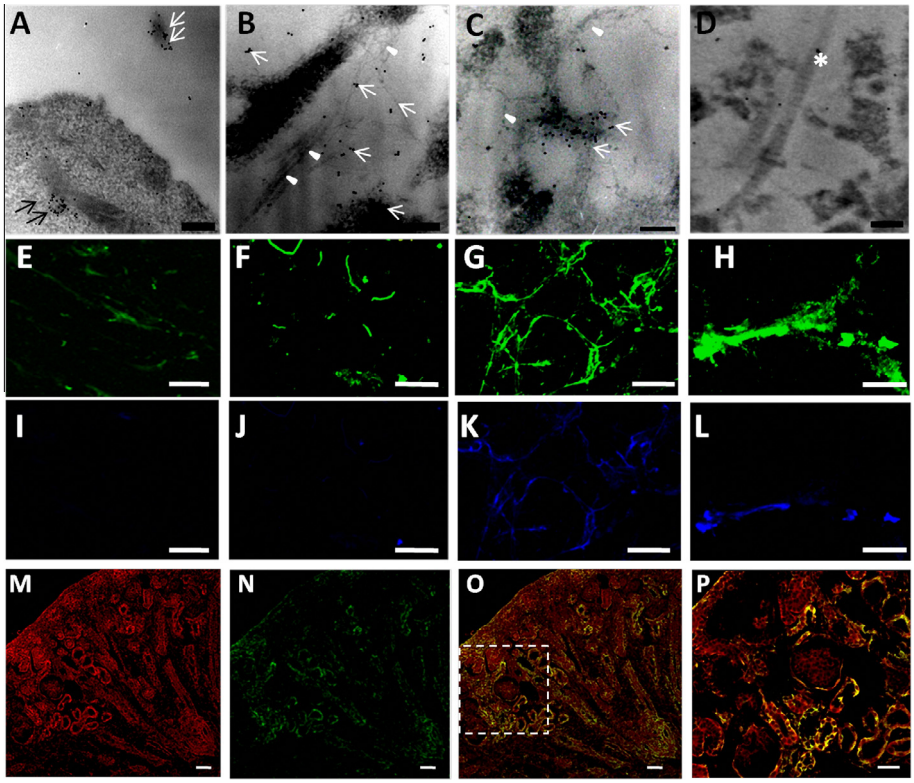

Fig. 4. Col V fibril in metanephroi. Collagen structures were confirmed by immunoelectron microscopy (A–D) and confocal microscope (E–L). Immunocytochemistry of Col V (A–C) and Col I (D) in metanephroi cross sections at E12.5 (A, B and D) and E15.5 (C) showed Col V aggregations, thin fibrils (arrows) and fibrils (arrow-heads) at following regions: (A) UB; (B) MM; (C) podocyte slit. Col I fibril was observed within MM aggregations (D), with thicker diameter than that of Col V (asterisk). Isolated ECMs form E13.5 (E and I). E15.5 (F and J), E17.5 (G and K), and P0.5 (H and L) metanephroi were coated on slide glasses under air dry and immune stained. Col V fibrils (E–H, green) was detected by goat anti Col V pAb with anti goat Alexa 488. Col I fibers (I–L, blue) were detected by rabbit anti Col I pAb with anti rabbit Alexa 633. Immunohistochemistry of whole metanephros at E15.5 showed major localization of Col V (M, red), Col I (N, green) and minor co-localization of Col V/I (O and P, yellow as merged color). Only Col V localized within glomeruli (P, red). Scale bars indicated 100 nm (A–D), 10 μm (E–L), 50 μm (M–O), and 20 μm (P), respectively. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

3.3.2. Collagen fibers isolated from metanephros detected by Col V and Col I antibodies

ECM proteins extracted from E13.5 to P0.5 metanephros were coated on slide glasses under air dry and then immune stained by antibodies against Col V (green) and Col I (blue). Occurrence of Col V fibrils initiated at E13.5 and the fibril thickness gradually increased to form bundles at P0.5 (Fig. 4E–H). At the early stage of development, Col V fibrils (Fig. 4E and F) were found independently of Col I fibers (Fig. 4I and J) whereas thick Col I fibers and bundles (Fig. 4K and L) were detected co-localizing with Col V (Fig. 4G and H) at the late stage of E17.5–P0.5.

3.3.3. Major independent localization and minor co-localization of Col V and Col I

Immunohistochemistry E15.5 metanephroi section shows Col V and Col I mainly exist independently in overall sight of whole metanephroi and immature glomeruli (Fig. 4M–P, red and green) although minor co-existence was observed (Fig. 4O and P, yellow). Compared with Col I, Col V was detected as stronger ECM at early development stage of metanephroi, indicating that most of Col V localized independently of Col I and few Col V co-localized with Col I during kidney development.

4. Discussion

Col V molecule control the initiation of Col I fibril assembly [2–4]. However, low abundant Col V fibril has seldom been reported until successful reconstituted. *In vitro* fibroblast cell culture on the reconstituted Col V fibril resulted in the accumulation of Col V fibril in the intercellular spaces due to cell aggregation, which rendered Col V fibril to be a cementing material for cell clump formation [17,18]. Our previous study revealed that reconstituted the Col V fibril has a different effect from Col V molecule when these two materials were utilized as scaffolds for glomerular endothelial cells culture [6]. Cells showed high mobility with less cellular skeleton of stress actin fiber on Col V fibril as compared to those on Col V molecule. On the other hand, Col V had been detected in the injured tissue [19]. Its increment of Col V in the interstitium matrices of glomerular basement membrane and the induction on over-expression of Col IV resulted in over-maturation of glomerular and caused collagen fibrotic glomerulopathy and progeria syndrome [7,20]. Therefore, Col V is considered as a dysfunctional component. However the structural type of the Col V, either molecule or fibril, is still doubtful.

In this study, we firstly detected the existence of Col V fibril in the process of metanephric morphogenesis, and showed the temporal and spatial changes of Col V expression and distribution in metanephroi. Both RT-PCT and Western blotting showed that Col V expression continued at all stages of development (Fig. 1). Multiple bands appeared in Western blotting may be due to the non-reducing and non-digesting conditions for ECM extraction, which caused multi-binding of Col V molecules to form dimmer, trimmer, and high molecular weight of aggregates. Col V mostly localized at the newly forming tissues in UB and glomeruli (Figs. 2 and 3). For UB tubulogenesis, Col V initiated its expression at UB from E11.5

and expanded to cortex region where UB are branching and elongating until E15.5 (Fig. 2P–R). Interestingly, Col V disappeared from the matured collecting duct (Fig. 2N, S, O and T, arrows) while maintained its expression in the immature tubules (Fig. 2S and T, asterisks) at E17.5 and P0.5. For developing glomerular primordia, Col V expression initiated from comma-shaped body, where MM aggregated around UB tips (Fig. 3A). The expression continued at the next stage of nephrogenesis during which, S-shaped bodies (Fig. 3B) and premature glomeruli (Fig. 3C) are differentiated. The expression then turns to weak when glomeruli became mature (Fig. 3D). Immunoelectron microscopy showed that Col V not only existed inside and outside of UB (Fig. 4A and B) but localized at intercellular spaces around MM aggregation in a thin fibril structure (Fig. 4C), whereas Col I appeared at the same region in a thick fiber structure (Fig. 4D).

Immune stains of extracted collagens and section of metanephroi showed that Col V existed as an independent fibril of Col I at early stage of development (Fig. 4E, F, I, J, M, and N). However, Col I fibers and bundles co-localizing with Col V at late stage of E17.5–P0.5 (Fig. 4G, H, K and L) may suggest that Col V initiates as an independent fibril and then Col I follows and co-localizes with Col V to form Col I–V bundles when tissues reach maturation. Collagen bundle has been reported to be essential to enhance epithelial clefts [21], it may imply that Col I–V bundles are essential to UB branching.

ECM microenvironmental remodeling and collagen types shifting have been discussed in angiogenesis [11], mesenchymal stem cell differentiation [12], MM development [10], and fibrotic diseases [22,23], which provide dynamic niches for cell movement and development. In this study, Col V expression appeared in immature UB and glomerular primordial, and gradually disappeared in the mature collecting duct and glomeruli. These facts proved the temporal and spatial expression shift of Col V, which suggest that Col V fibril is a transitional ECM between newly forming and mature tissue.

This study unveiled the existence of Col V fibril in metanephros. Although Col V fibril had been regarded as dysfunctional component in the injured or pathologic tissues so far, our results suggest that Col V fibril may provide a transitional microenvironment from immature to mature tissues and may be considered as a pioneering ECM to form a prototype of pericellular microenvironment during tissue morphogenesis. The possibility to utilize Col V fibril in tissue regeneration will be further investigated.

Acknowledgments

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