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Gestational Week 20 as the Biomechanical Inflection Point of Retroperitoneal Fascial Lamination: A Mechanobiological Model Integrating Geometric Scaling and Evolutionary Front-Loading

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Abstract

The embryological basis for the lamination of the retroperitoneal fascia has long remained an anatomical paradox. Classical peritoneal fusion theories cannot account for either the highly organized multilaminar architecture of the mature fascia or the striking temporal lag between early visceral fixation (gestational weeks 9–18) and the abrupt, synchronized emergence of definitive fascial laminae around week 20. Integrating recent advances in fetal biomechanics, we propose that this developmental lag reflects a system-level mechanical transition driven by the geometric constraints of scaling and the evolutionary demands of obligate bipedalism.

Keywords: fascia; retroperitoneal space; mechanobiology; heterochrony; Poisson effect; tensegrity; evolutionary front-loading; renal agenesis

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Based on standard fetal growth curves, the internal fetal volume increases nearly sixfold between weeks 12 and 20, whereas the enclosing surface area only triples. This predictable geometric escalation—conceptually expressed as an increasing volume-to-surface-area ratio ($V/A \propto r$)—may contribute to a transition from a pressure-driven continuum to a tension-bearing architecture. As newly stiffened skeletal anchors (vertebrae, ribs, and the rapidly expanding iliac blades) begin to resist deformation, the expanding trunk generates a systemic multiaxial tension field. Stretching the highly hydrated retroperitoneal mesenchyme inevitably induces orthogonal Poisson compression, driving poroelastic fluid exudation and collapsing the collagen framework into discrete laminae. Fibroblast traction and subsequent lysyl-oxidase–mediated crosslinking stabilize these mechanically aligned layers, producing a primitive tensegrity-like load-bearing network.

To determine whether the outer lamina of the posterior renal fascia depends on renal expansion or reflects this broader tension field, we analyzed three adult cases of congenital renal agenesis identified among 5,509 abdominal CT scans. In all cases, the parietal (outer) lamina of the posterior renal fascia was preserved despite lifelong absence of the kidney, whereas the organ-dependent inner lamina was uniformly reduced. These findings are consistent with a model in which the outer fascial lamination can arise independently of local organ expansion and may be influenced by systemic mechanical forces emerging around mid-gestation.

Integrating fetal histology, biomechanics, and a natural subtraction experiment, these observations support a dual-mechanism model in which organ-specific hoop stress contributes to the inner lamina, while systemic tension fields emerging around mid-gestation may contribute to outer lamina formation. We hypothesize that this transition represents the morphological imprint of an

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evolutionarily front-loaded adaptation selected to protect the uniquely vulnerable human iliac flare. Further comparative and quantitative studies are required to test this mechanobiological hypothesis.

This mid-gestational transition likely reflects the interaction between a universal geometric scaling constraint and a uniquely human, genetically patterned iliac flare that necessitates an earlier mechanical shift.

1. Introduction

The developmental mechanisms underlying the multilaminar architecture of the retroperitoneal fascia have been debated for more than a century. Classical frameworks—including Toldt's (1879) peritoneal fusion hypothesis and the fixation apparatus models of Zuckerkandl (1883) and Gerota (1895)—provided early morphological descriptions but did not resolve the embryological origin of the bilaminar posterior renal fascia. With the advent of modern cross-sectional imaging, Raptopoulos et al. (1986) demonstrated that the posterior renal fascia consists of two distinct laminae, closely matching Gerota's original illustrations. Yet the fundamental question remains: how and why do these highly organized fascial layers emerge?

Despite extensive historical and contemporary research, two persistent paradoxes challenge fusion-based explanations. First, simple mesothelial adhesion cannot account for the consistently observed, sharply demarcated multilaminar planes described in high-resolution microanatomical studies (Kinugasa et al., 2007; Stecco et al., 2017). Second, multiple fetal studies reveal a striking temporal lag: early visceral fixation and the appearance of the inner renal fascia occur between gestational weeks 9–12 (Kanagasuntheram, 1957; Cho et al., 2009; Matsubara et al., 2009), and the initial adhesion of the ascending and descending mesocolon begins around weeks 17–18 (Baumann, 1945). Yet the definitive multilamination of the posterior renal fascia, retrocolic fascia, and retropancreatic fascia does not emerge until gestational week 20 (Cho et al., 2009; Matsubara et al., 2009). This reproducible delay suggests that lamination is not governed by local organ fixation alone but awaits a subsequent system-level mechanical transition.

Recent advances in fetal biomechanics provide a crucial conceptual foundation for understanding this transition. Nowlan (2015) demonstrated that the fetal musculoskeletal system undergoes a fundamental shift around week 20—from a compliant, pressure-driven continuum to a mechanically active, tension-bearing frame. However, the precise trigger for this abrupt transition has remained unclear.

Here, we hypothesize that the missing mechanistic link is a genetically timed mechanical collision. Based on standard fetal growth curves (Hadlock et al., 1991), the internal fetal volume increases nearly sixfold between weeks 12 and 20, whereas the enclosing surface area only triples. This predictable geometric escalation—conceptually expressed as a rapidly increasing V/A ratio ($V/A \propto r$)—imposes a rapidly rising internal load on the fetal trunk. As genetically programmed skeletal elements—such as vertebrae, ribs, and the uniquely expanding iliac blades—begin to stiffen, the trunk can no longer dissipate this load isotropically. Instead, it transitions into a tension-bearing architecture, generating a systemic multiaxial tension field.

This tension field acts upon the hydrated retroperitoneal mesenchyme, which is anchored medially by the duodenum and pancreas (weeks 9–12) and laterally by the ascending and descending mesocolon (weeks 17–18). Stretching this anchored mesenchyme inevitably induces orthogonal Poisson compression, driving poroelastic fluid exudation and collapsing the collagen framework into discrete laminae. Fibroblast traction and subsequent lysyl-oxidase–mediated crosslinking stabilize these mechanically aligned layers, producing a primitive tensegrity-like load-bearing network. Thus, the synchronized lamination observed at week 20 may represent the morphological imprint of this system-level mechanical transition.

A key unresolved question is whether the outer lamina of the posterior renal fascia depends on the expanding kidney or instead reflects this broader tension field. Congenital renal agenesis provides a natural "subtraction experiment" to distinguish organ-dependent from systemic

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mechanisms. If the outer lamina forms independently of renal expansion, its presence in renal agenesis would support a systemic, tension-driven origin.

Finally, comparative developmental anatomy suggests that this mid-gestational mechanical transition may be evolutionarily front-loaded in humans. Unlike most mammals, which maintain a compliant trunk until late gestation ("back-loading"), humans possess a uniquely laterally flared ilium essential for bipedalism but structurally vulnerable to isotropic pressure. We hypothesize that human ontogeny has shifted the timing of frame-locking forward to protect this fragile geometry.

In this study, integrating fetal histology, biomechanics, and radiological subtraction analysis, we propose an exploratory mechanobiological model in which the inner renal fascia arises from organ-specific hoop stress, whereas the outer fascial laminae emerge from a systemic tension network established at gestational week 20.

2. Materials and Methods

2.1. Study Design and Conceptual Framework

This study employed a hybrid design integrating (1) a retrospective radiological observational analysis of adult patients with unilateral renal agenesis, used as a natural subtraction experiment, and (2) a conceptual synthesis of published fetal histology and developmental biomechanics. The subtraction experiment was essential for distinguishing organ-dependent from system-level mechanisms of fascial lamination. If the outer lamina of the posterior renal fascia forms independently of renal expansion, its preservation in renal agenesis would support a tension-driven, systemic origin rather than a local organ-driven mechanism.

2.2. Case Selection and Retrospective Radiological Review

2.2.1. CT Acquisition

A systematic retrospective screening of 5,509 consecutive abdominal CT scans performed at a single institution (Gakkentoshi Hospital, Kyoto, Japan) between April 2018 and March 2024 was conducted to identify cases of unilateral renal agenesis. Renal vacancy was defined as:

- True congenital renal agenesis: complete absence of renal parenchyma and ureter.
- Severe renal involution/dysplasia: non-functional renal remnants < 3 cm.

Patients with prior nephrectomy, retroperitoneal surgery, major trauma, or local malignancy were excluded. Three adult patients met the inclusion criteria.

2.2.2. Measurement Protocol

CT scans were obtained on multiple scanners (manufacturers/models not consistently recorded). Slice thickness ranged from 1.0 mm to 5.0 mm (median 3.0 mm). In-plane pixel spacing ranged from 0.714 mm × 0.714 mm to 1.0 mm × 1.0 mm. Reconstructions used a standard soft-tissue kernel. Unenhanced CT studies were exclusively utilized; measurements were performed on axial images displayed with a specific window setting optimized for fascial visualization (window width: 250 HU, window level: 150 HU).

Fascial thickness was measured at predefined anatomical landmarks on axial images using 3D Slicer v5.10. Measurements were performed by a single observer (H.T.). Intra-rater reproducibility was not assessed due to retrospective constraints; inter-rater reproducibility was not assessed.

2.3. Literature Review and Embryological Integration

A structured review of fetal anatomical studies was conducted, focusing on the timing of renal ascent, emergence of the inner renal fascia, initial mesocolic adhesion, appearance of multilaminar fascial strata, developmental stiffening of skeletal anchors, and maturation of the fetal skin barrier. These data were integrated to reconstruct the mechanical timeline of retroperitoneal development,

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evaluating whether the timing and morphology of fascial lamination are better explained by local organ mechanics or by a systemic tension network.

2.4. Ethical Considerations

This study was approved by the Institutional Review Board of Gakkentoshi Hospital (Approval No. GT-R6-07-12-1). Due to its retrospective nature, written informed consent was waived, and an opt-out mechanism was provided through the hospital's official website. Radiological data are restricted to protect patient privacy but anonymized slices and measurement datasets can be provided upon reasonable request.

3. Results

3.1. Radiological Cohort of Renal Agenesis

Because the radiological cohort comprises only three cases, the following comparisons are descriptive and exploratory; statistical inference is not claimed.

Among the 5,509 abdominal CT scans screened, three adults met the strict inclusion criteria for unilateral renal vacancy without prior surgical intervention. Cases 1 and 2 demonstrated true congenital renal agenesis, each accompanied by the characteristic "lying-down" or pancake adrenal morphology (Kenney et al., 1985; Potter, 1972), confirming absence of the kidney from the earliest stages of development. Case 3 exhibited a severely involuted dysplastic renal remnant measuring approximately 2 cm.

3.2. Preservation of the Outer Posterior Fascial Plane

In all three cases, a continuous parietal (outer) lamina of the posterior renal fascia was clearly preserved at the predicted anatomical location of the retrorenal fascia (Figure 1). This lamina extended smoothly between the peritoneal sac and the posterior abdominal wall, anchored reliably to the psoas major or quadratus lumborum, and maintained typical anterior continuity with the lateroconal fascia (Congdon & Edson, 1941). Crucially, this fascial plane was identified despite the lifelong absence of an expanding renal parenchyma. This finding is consistent with the hypothesis that the outer lamina can be generated by a system-level tension field.

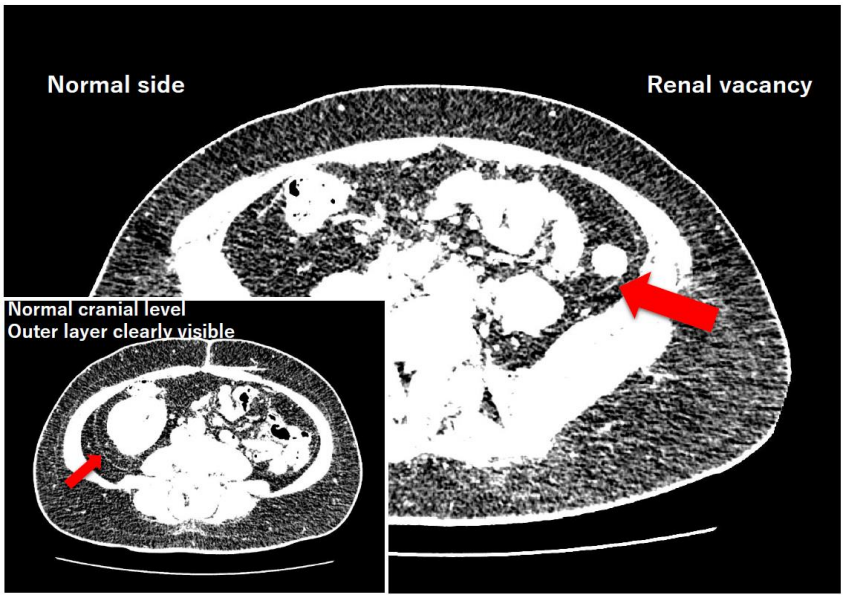

Figure 1. Radiological validation of the "natural subtraction experiment" in congenital renal agenesis. Axial unenhanced computed tomography (CT) image of Case 1 (53-year-old female) with true left renal agenesis. Despite the congenital and lifelong absence of the left kidney and ureter, a distinct, continuous hyperdense line (arrows) is clearly preserved in the predicted anatomical position of the posterior renal fascia. This structure

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represents the parietal (outer) lamina of Zuckerkandl's fascia. Note that while the healthy contralateral side (inset, bottom left) displays a robust, bilaminar composite fascia reflecting both organ-dependent and systemic influences, the affected side shows a thinner but well-defined solitary lamina (~1.52 mm) due to the absence of the organ-derived inner layer. This evidence demonstrates that the outer fascial lamina forms and persists independently of local renal parenchymal expansion. CT, computed tomography.

3.3. Quantitative Assessment of Fascial Thickness

Quantitative measurements are summarized in Table 1. Thickness values are presented per case; group statistical inference was not performed due to the small sample size.

- Affected side (renal vacancy): mean thickness 1.52 mm
- Contralateral healthy side: mean thickness 1.85 mm

Table 1. Clinical characteristics and quantitative radiological findings of the renal agenesis cohort (n = 3). Cases were extracted from a primary screen of 5,509 consecutive abdominal CT scans after strict exclusion of patients with a history of renal surgery.

Case	Age/Sex	Radiological Diagnosis	Adrenal Morphology	Fascial Thickness (Affected Side)	Fascial Thickness (Normal Side)	Difference (Δ)
1	53F	True left renal agenesis	"Lying-down" (pancake)	1.49 mm	1.88 mm	−0.39 mm
2	47F	True left renal agenesis	"Lying-down" (pancake)	1.46 mm	1.82 mm	−0.36 mm
3	89M	Severe left renal dysplasia/involution (renal remnant)	Normal	1.62 mm	Excluded*	N/A
Mean	—	—	—	**1.52 mm**	**1.85 mm**	**−0.38 mm**

*Excluded from measurement due to renal fascial thickening secondary to pyelonephritis on the unaffected side. F, female; M, male; CT, computed tomography.

The uniform reduction in thickness on the renal-vacant side reflects the absence of the organ-dependent inner lamina, while the preserved thickness corresponds to the outer lamina, which appears to form autonomously.

3.4. Chronological Integration of Fetal Fascial Development

Integration of published fetal histology revealed a two-phase developmental pattern (Table 2, Figure 2):

- Phase 1 (Weeks 9–18): Establishment of Mechanical Anchors. (Medial fixation of the duodenum/pancreas and emergence of the inner renal fascia, followed by lateral mesocolic adhesion).
- Phase 2 (~Week 20): System-Level Mechanical Transition. (V/A ratio increases ~3-fold, skeletal anchors stiffen, and multiaxial tension induces Poisson compression, collapsing collagen into discrete laminae) (Table 3).

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Table 2. Chronological integration of retroperitoneal fascial development and associated biomechanical events.

Gestational age	Key developmental events	Biomechanical context	References
Weeks 8.5–12 ***(Embryonic Day 59 onwards)***	**• Heterotopic shift of iliac growth axis (cartilaginous growth plate rotates 90° to expand horizontally).** **• Early central visceral fixation (pancreas and duodenum).** **• Appearance of inner renal fascial layer.**	**Initiation of the human-specific anatomical constraint.** Rotation of the growth axis patterns a transverse (horizontal) boundary; however, the early pelvic cartilage remains too compliant to generate global tension fields.	Kanagasuntheram 1957; Cho et al. 2009; Matsubara et al. 2009; Senevirathne et al. 2025
Weeks 14–16	**• Asymmetric pelvic stiffening commences. Posterior ilium undergoes perichondral ossification.** **• Anterior growth zone (AGZ) retains its chondrogenicity, expanding transversely.** **• Functional maturation of fetal lymphatic system.**	**Formation of the rigid transverse "wall."** While the AGZ drives lateral flaring, posterior ossification progressively limits isotropic pressure dissipation. Lymphatic drainage matures to prepare for poroelastic fluid movement.	van der Putte 1975; Swartz & Fleury 2007; Senevirathne et al. 2025
Weeks 17–18	**• Initiation of ascending/descending mesocolon adhesion to the posterior abdominal wall.** **• Continued expansion of the AGZ against the expanding abdominal volume.**	**Completion of the macroscopic anchor nodes.** Addition of lateral mechanical anchors fixes the retroperitoneal mesenchyme within the gradually tightening horizontal pelvic frame.	Baumann 1945; Senevirathne et al. 2025

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Weeks 18–20	**• Progressive vertebral and rib ossification.** **• Epidermal keratinization and cutaneous barrier maturation.** **• Onset of exponential truncal volumetric growth (sixfold volume surge vs. threefold surface area increase).**	**The mechanical incubation period reaches its limit.** Axial skeletal rigidity increases and the cutaneous envelope becomes inextensible, blocking all vertical and isotropic escape routes for the rapidly rising internal load.	Bagnall et al. 1977; Hardman et al. 1999; Hadlock et al. 1991
~Week 20 ***(Temporal Nexus)***	**• System-wide mechanical collision.** **• Pelvic expansion and ossification reach critical threshold.** **• Fascial lamination becomes histologically distinct.**	**The Biomechanical Inflection Point.** Trapped scaling energy violently collides with the evolutionarily derived horizontal constraints, generating powerful multiaxial tension fields that drive Poisson-mediated fascial lamination.	Cho et al. 2009; Matsubara et al. 2009; Verbruggen & Nowlan 2017
Weeks 21–24	**• AGZ finally reaches growth threshold (retention of cartilaginous properties concludes around week 24–25).** **• Definitive formation of the hominin-derived anterior inferior iliac spine (AIIS).**	**Stabilization of the bipedal architectural frame.** The permanent anchorage network for bipedal musculature is completed, permanently locking the tension-bearing fascial and skeletal infrastructure.	Senevirathne et al. 2025

Table 3. System-wide developmental changes occurring near gestational week 20.

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System	Developmental change	Mechanical significance	References
Musculoskeletal system	Transition from pressure-driven continuum to tension-bearing architecture	Establishes global tension-responsive network	Nowlan 2015; Verbruggen & Nowlan 2017
Axial skeleton	Vertebral ossification; iliac flare expansion	Increases axial rigidity; converts fetal volumetric expansion into directed strain	Bagnall et al. 1977; Baumgart et al. 2018; Senevirathne et al. 2025
Cutaneous envelope	Epidermal keratinization; barrier maturation	Converts skin into an inextensible boundary resisting internal load	Hardman et al. 1999
Lymphatic system	Functional drainage pathways established	Enables poroelastic fluid exudation during Poisson compression	van der Putte 1975; Swartz & Fleury 2007

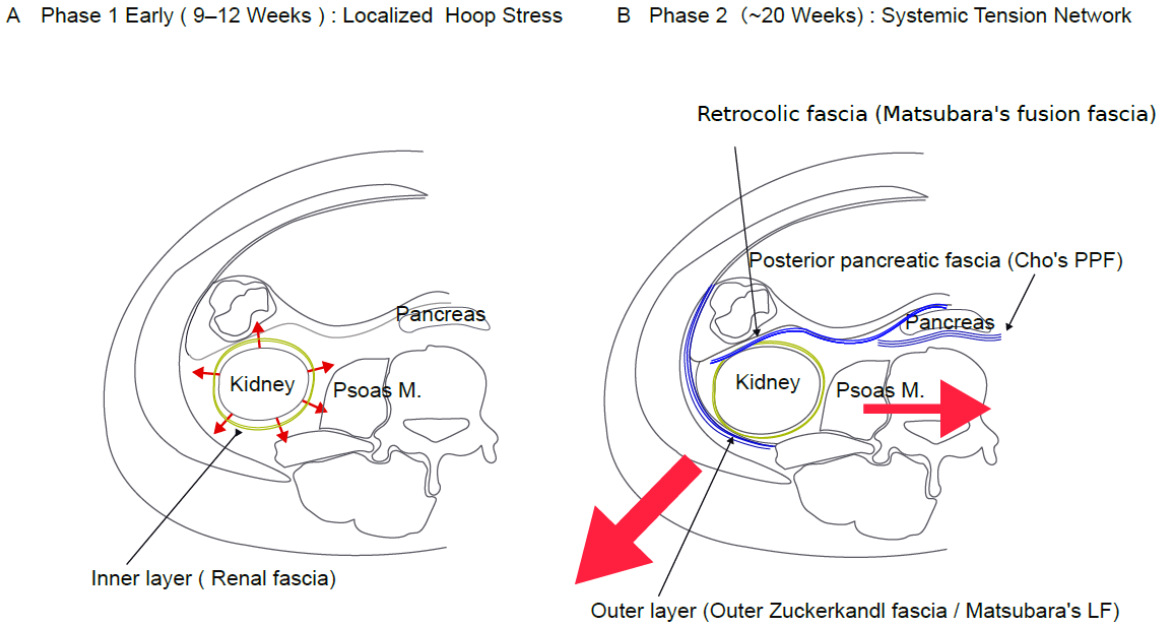

Figure 2. Spatiotemporal and biomechanical asymmetry in retroperitoneal fascial development. This diagram highlights the chronological discrepancy in fascial emergence. Phase 1 (weeks 9–18) involves the sequential establishment of central and lateral mechanical anchors. During the early part of this phase (weeks 9–12; Panel A), the inner renal fascia forms, driven by localized hoop stress from the expanding kidney. In contrast, Phase 2 (around week 20; Panel B) represents the mid-gestational systemic mechanical transition, during which the multilaminated outer layers are synchronously established across the macroscopic tension network completed between these central and lateral visceral anchors.

4. Discussion

4.1. The Developmental Lag Reveals a System-Level Mechanical Trigger

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The classical fusion-based model has long struggled to explain the reproducible temporal paradox in retroperitoneal development. Early visceral fixation occurs between weeks 9–12, yet definitive fascial lamination does not appear until week 20. This delay suggests that lamination is not a simple consequence of mesothelial adhesion. Our findings align with Nowlan's (2015) demonstration of a fundamental shift in the fetal musculoskeletal system around week 20. Geometric scaling ($V/A \propto r$) provides a compelling explanation, dramatically increasing internal load per unit surface area. The synchronized timing of skeletal stiffening and fascial lamination suggests that week 20 represents a mechanical inflection point where the fundamental physical forces of geometric scaling collide with the stiffening skeletal frame.

4.2. Local Hoop Stress vs. Systemic Poisson Compression

Our results are consistent with a dual-mechanism model of fascial development.

- Local organ-dependent hoop stress (weeks 9–12): The developing kidney generates intrinsic radial expansion pressure, producing circumferential hoop stress—a ring-like tensile force that arises when a cylindrical or spherical structure expands—that compacts adjacent mesenchyme. This mechanism explains the early emergence of the inner renal fascia, which behaves as an organ-specific investing fascia (Figure 3).
- Systemic tension-driven Poisson compression (week 20): As the fetal trunk transitions into a tension-bearing system, multiaxial tension stretches the hydrated retroperitoneal mesenchyme between established medial and lateral anchors. This inevitably induces orthogonal Poisson compression—a fundamental property of hydrated soft tissues when subjected to longitudinal strain. This mechanical compression drives poroelastic fluid exudation, directing interstitial fluid into the functionally maturing lymphatic system (van der Putte, 1975; Swartz & Fleury, 2007). This fluid shift causes the remaining collagen framework to collapse into discrete laminae (Mow et al., 1980). Fibroblasts respond to this strained environment by aligning along the principal tension vectors and exerting traction forces (Harris et al., 1981; Weiss, 1929), which further compacts and stabilizes the assembling extracellular matrix. Subsequent enzymatic crosslinking (Kagan & Li, 2003) permanently fixes these tension-aligned layers, producing a tensegrity-like load-bearing network (Figure 4).

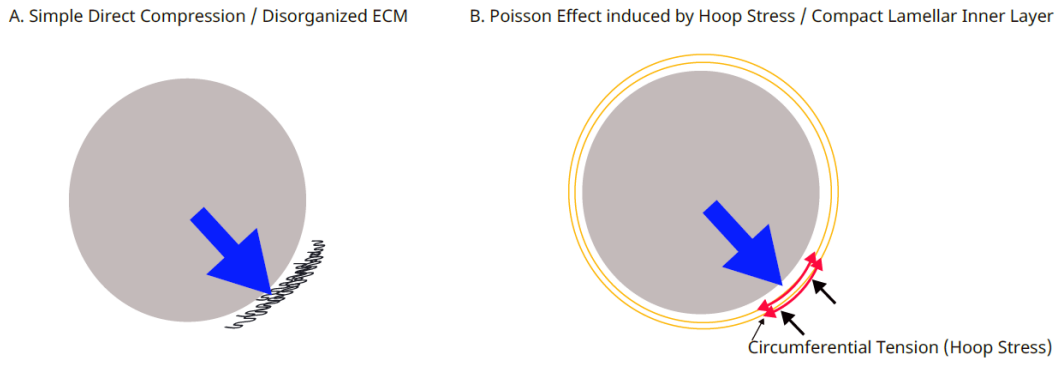

Figure 3. Theoretical comparison of tissue deformation: Direct Compression vs. Poisson Effect. (A) Simple Direct Compression: A classical assumption where localized compressive forces from expanding organs merely squash the mesenchyme without producing organized lamellar sheets, resulting in disorganized tissue compaction. (B) Hoop Stress-Induced Poisson Effect: Circumferential tension (hoop stress) from the expanding mass induces orthogonal (transverse) compression via the Poisson effect, leading to the orderly condensation of the mesenchyme into the compact lamellar structures characteristic of the investing renal fascia.

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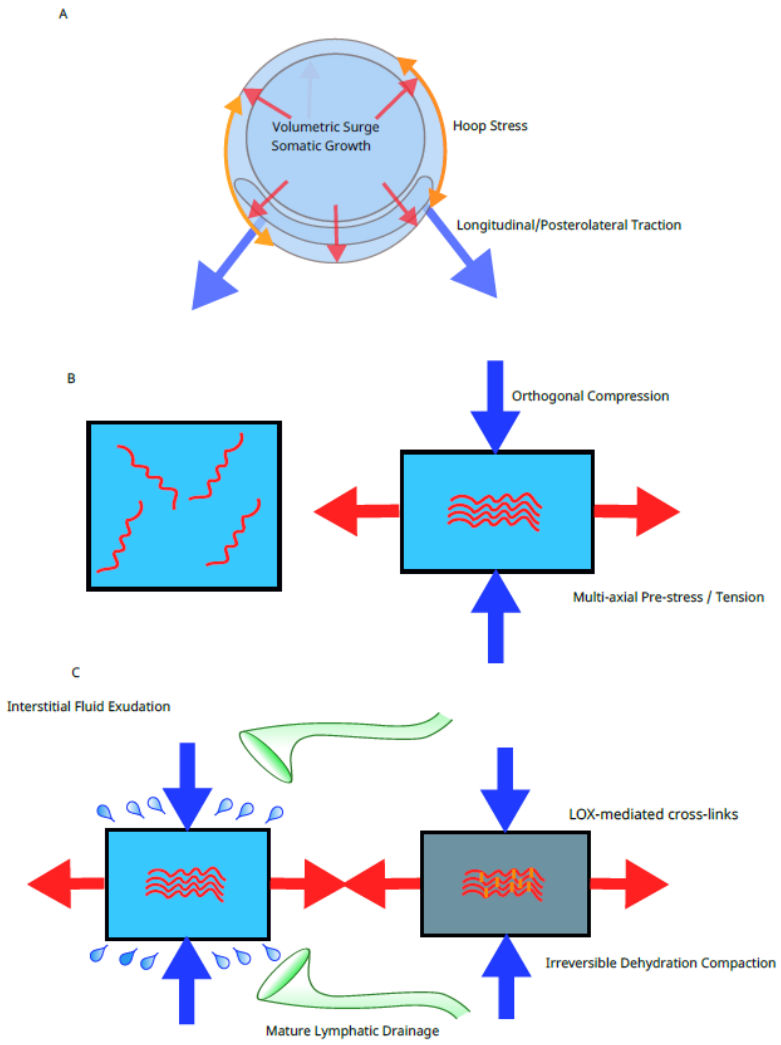

Figure 4. Square-cube-driven emergence of a fetal tension network and definitive lamination. (A) Volumetric Surge: Rapid somatic growth ($V/A \propto r$) dramatically increases internal mechanical load, generating hoop stress and longitudinal/posterolateral traction. (B) Orthogonal Compression: Because isotropic volumetric expansion is strictly restricted by early frame-locking (evolutionary front-loading), the trapped scaling energy is converted into a powerful multiaxial tension field, forcing the hydrated retroperitoneal mesenchyme to undergo obligatory orthogonal compression via the Poisson effect. (C) Consolidation and Fixation: This internal mechanical compression drives poroelastic fluid exudation into the maturing lymphatic system, while LOX-mediated cross-linking permanently stabilizes the collapsed collagen framework into the definitive, highly ordered fascial architecture. LOX, lysyl oxidase.

4.3. Evolutionary Front-Loading: Protecting the Iliac Flare

Importantly, geometric scaling ($V/A \propto r$) provides the universal physical background condition that increases internal load during mid-gestation, whereas the genetically patterned iliac flare represents the primary evolutionary driver that necessitates an earlier mechanical transition specifically in humans. In this framework, geometric scaling explains when the mechanical inflection point becomes unavoidable, while the iliac flare explains why humans reach this threshold earlier than other mammals.

While micro-scale fibroblast behaviors explain how lamination occurs under tension, geometric scaling addresses why this systemic multiaxial tension field emerges precisely around mid-gestation. Based on standard growth curves, the internal fetal volume increases nearly sixfold between weeks 12 and 20, whereas the enclosing surface area only triples. If the square-cube law alone were the sole physical trigger, massive tension and subsequent lamination would predictably occur in all large

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mammalian fetuses as they grow. However, most mammals adopt a "back-loading" developmental trajectory, maintaining a highly compliant truncal frame (cartilage and skin) throughout most of gestation, which safely dissipates expanding volume (Weisbecker et al., 2008). Similarly, non-human hominoids such as chimpanzees possess narrow iliac blades that do not flare laterally, thereby maintaining a more compliant and isotropically expandable pelvic geometry (Huseynov et al., 2017). This structural flexibility allows fetal volumetric expansion to be absorbed largely isotropically, preventing the kind of transverse constraint that would generate a pronounced multiaxial tension field. As a result, the mid-gestational mechanical collision observed in humans does not occur in these species.

Humans differ in a critical respect: the laterally flared ilium, essential for bipedalism (Senevirathne et al., 2025), is structurally vulnerable to isotropic pressure during mid-gestation. We hypothesize that human ontogeny may exhibit a relatively earlier timing of frame-locking, potentially reflecting an evolutionarily front-loaded pattern (vertebral stiffening and cutaneous keratinization) to protect this geometry. Consequently, human fetuses experience a unique mechanical collision around week 20 between the rapidly expanding internal volume and the stiffening external frame. The expanding energy, having lost its isotropic escape route, is converted into the powerful systemic multiaxial tension that drives definitive fascial lamination. However, comparative developmental data are limited, and this evolutionary interpretation remains provisional.

Recent work (Senevirathne et al., 2025) reveals that the root cause of this mechanical collision lies in a genetically patterned abrupt shift in the axis of iliac growth. While the vertically growing pelvis of other primates easily dissipates internal expansion pressure cranially, the human iliac cartilage growth plate undergoes a critical heterotopic shift as early as embryonic day 59 (~8.5 weeks), rotating 90 degrees to expand transversely (horizontally). Although this spatial reorientation is initiated early, the actual mechanical collision necessitates an incubation period. Coupled with a unique posterior ossification, this genetically programmed lateral flaring progressively constructs a rigid, anisotropic anatomical "wall." Consequently, as geometric scaling accelerates exponentially between weeks 12 and 20, the isotropic volumetric expansion loses its ancestral vertical escape route. The expanding fetal trunk becomes unable to dissipate the rapidly rising internal pressure within this gradually stiffening horizontal constraint. The systemic multiaxial tension field that emerges around week 20 is thus not the cause of the pelvic flare, but the inevitable mechanical consequence of volumetric scaling violently colliding with this evolutionarily derived transverse boundary.

4.4. Clinical Corroboration: Surgical Cleavage Planes as Fossils of Mid-Gestational Mechanics

Modern oncologic surgery routinely exploits avascular cleavage planes that correspond precisely to the laminae predicted by our model. Complete mesocolic excision and retroperitoneal mobilization reveal sharply defined fascial planes that behave as mechanically derived boundaries, not remnants of mesothelial fusion (Wedel et al., 2022). Pathologic fluid collections can hydraulically reopen these planes, further supporting their origin in poroelastic compression rather than adhesion (Molmenti et al., 1996).

4.5. Revisiting Tobin's 1944 Case: A Field Defect, Not a Falsification

The long-standing belief that the kidney is strictly required for fascial formation originates from Tobin's 1944 report of "absence of fascia" in renal agenesis. However, critical re-examination of his foundational 1944 specimen reveals that it lacked both the kidney and the adrenal gland—a combination occurring in approximately 11% of unilateral renal agenesis cases (Ashley & Mostofi, 1960; Dluhy & Gittes, 1986). In teratological principles, the simultaneous agenesis of developmentally distinct adjacent organs—the intermediate mesoderm-derived kidney and the coelomic epithelium/neural crest-derived adrenal gland—represents a classic developmental field defect (Opitz, 1985). Without an intact local field, the mesenchymal substrate required to receive and

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transmit mid-gestational tension is fundamentally absent; thus, the absence of fascia is a predictable consequence of the lost field, not evidence against tension-driven lamination.

In subsequent extensive studies, Benjamin and Tobin (1951) repeatedly used this 1944 illustration of a combined defect as a representative model. In the pre-CT era, lacking tomographic imaging to visualize local mesenchyme in vivo, this reliance on the gross presence or absence of the kidney was understandable. In contrast, modern multidetector CT (MDCT) allows non-invasive evaluation of the entire retroperitoneal field. All individuals in our cohort retained adrenal glands and therefore preserved the regional mesenchymal scaffold (Figure 5). The consistent presence of the continuous outer posterior fascial plane in these cases strongly supports a system-level mechanical origin independent of renal expansion.

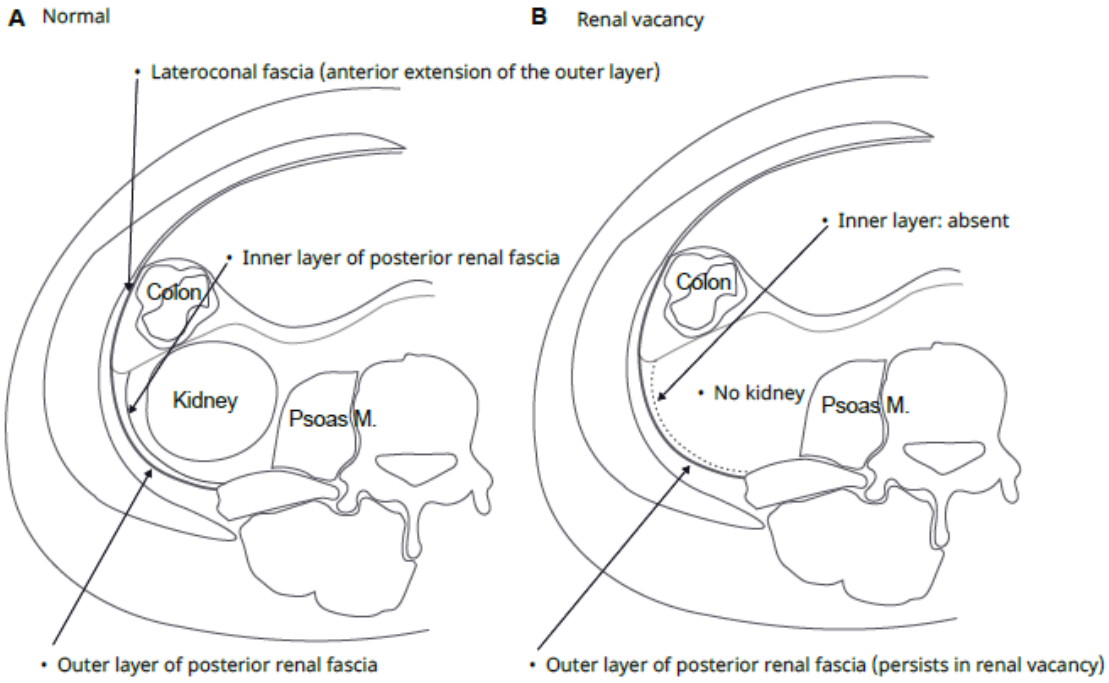

Figure 5. Conceptual schematic of retroperitoneal planes in normal anatomy versus renal agenesis. (A) Normal Anatomy: The kidney is enveloped by both the organ-dependent inner layer (derived from local hoop stress) and the continuous, system-derived outer layer (derived from systemic mid-gestational tension). (B) Renal Agenesis: In the absence of the kidney, the local inner layer fails to form. However, the outer layer persists autonomously and maintains its smooth continuity with the lateroconal fascia (Congdon & Edson, 1941). This isolates and illustrates that as long as the regional mesenchymal field remains intact to receive the systemic tension network, the formation of the outer lamina is independent of local organ expansion.

4.6. A Unified Mechanobiological Model of Retroperitoneal Fascial Development

Our findings support a unified model in which retroperitoneal fasciae arise through the sequential interplay of genetically patterned mesenchymal priming, organ-specific hoop stress (which forms the organ-dependent inner lamina), and system-level tension-driven Poisson compression. Crucially, this framework unifies the developmental origins of diverse multilaminar structures that emerge synchronously around week 20. Although classical embryology has divided these structures into distinct categories—such as the in situ mesenchymal condensation of the outer posterior renal fascia versus the supposedly adhesion-derived "fusion fasciae" (e.g., retropancreatic and retrocolic fasciae; Matsubara et al., 2009)—we propose that they share a common mechanobiological origin. Rather than resulting from disparate local events, all these fascial layers are better understood as the generalized morphological expressions of a single, system-wide mechanical transition.

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4.7. Limitations

This study has several limitations: the small radiological sample size (n=3), its retrospective single-center design, the heterogeneity of CT acquisition parameters, and the inferential nature of the mechanobiological model. These limitations temper our causal claims. The radiological observations are descriptive and serve primarily as a "subtraction experiment" to support theoretical biomechanical principles. These constraints motivate further comparative embryological and quantitative biomechanical studies to fully validate the proposed framework.

5. Conclusion

Congenital renal agenesis provides a natural subtraction experiment to explore the contribution of systemic mechanical forces to retroperitoneal fascial development. Across the three cases examined, the outer posterior lamina of the renal fascia was preserved despite the lifelong absence of renal parenchyma, whereas the inner lamina was uniformly reduced. This pattern is consistent with a dual-mechanism model in which the inner lamina is organ-dependent, but the outer lamina arises from system-level mechanical forces.

We propose that the synchronized lamination of retroperitoneal fasciae at gestational week 20 reflects a fundamental mechanical transition. As geometric scaling ($V/A \propto r$) drives an increase in internal load and skeletal anchors stiffen, the trunk shifts to a tension-bearing architecture. This multiaxial tension field induces Poisson-driven mesenchymal compression, poroelastic fluid exudation, and lamellar collapse.

Taken together, these findings suggest that retroperitoneal fascial lamination may represent a biomechanical footprint of human evolution, emerging from the interplay of geometric scaling, skeletal stiffening, and mid-gestational tension fields. These insights provide a theoretical foundation linking fetal mechanobiology to the adult surgical cleavage planes routinely exploited in clinical anatomy.

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Data Availability: Radiological data supporting the findings of this study are restricted to protect patient privacy but are available from the corresponding authFor upon reasonable request.

Ethics Statement: This retrospective study received approval from the Institutional Review Board of Gakkentoshi Hospital (Approval No. GT-R6-07-12-1). Written informed consent was waived, and an opt-out mechanism was provided through the hospital's official website.

Conflicts of Interest Statement: The author declares no conflicts of interest.

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