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Posted Date: 7 April 2026

doi: 10.20944/preprints202603.1933.v3

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Article

Fetal Week 20 as the Mechanical Turning Point of Retroperitoneal Fascial Lamination: A Poisson-Effect Framework

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Abstract

This study proposes a mechanobiological model explaining how multilaminated retroperitoneal fasciae arise through the interplay of localized and systemic tension fields. Classical peritoneal fusion theories account for neither the organized laminar architecture nor the 10-week developmental delay between early visceral fixation and definitive fascial formation. The present framework proposes that localized tension at 10–12 gestational weeks generates the inner renal fascial layer, whereas a systemic tension field emerging around 20 weeks—driven by axial skeletal ossification, pelvic expansion, and exponential volumetric growth—establishes a fetal-scale tensegrity network. This systemic tension induces orthogonal Poisson-effect compression, poroelastic fluid exudation, and LOX-mediated cross-linking, collectively generating the laminated outer layer. To provide empirical illustration of this framework, a pure cohort of adult renal vacancy ($n=3$) was identified from 5,509 consecutive CT scans. Despite the lifelong absence of the kidney, a continuous outer fascial layer persisted in all cases, indicating that its formation is tension-driven rather than organ-dependent. This natural subtraction experiment resolves the longstanding discrepancy between classical dissection and modern imaging, and supports a systemic mechanobiological origin for retroperitoneal fascial lamination.

Keywords: fascia; retroperitoneal space; mechanobiology; Poisson effect; tensegrity; embryology; poroelasticity; lysyl oxidase; renal agenesis

1. Introduction

The developmental mechanisms underlying the complex fascial planes of the retroperitoneal space have been debated since the late nineteenth century. Historically, retroperitoneal fasciae were classified according to classical peritoneal fusion theories (Toldt, 1879) and distinct fixation apparatus models (Zuckerkanl, 1883; Gerota, 1895). Although Gerota's original illustration (1895) depicted the renal fascia as a two-layered structure, later authors—notably Marks and subsequently Raptopoulos—noted that this bilaminar configuration bore a striking resemblance to modern radiological observations, though its developmental significance remained obscure for decades. Hayes (1950) later introduced the concept of 'migration fasciae,' proposing that mechanical stress from visceral growth induces localized mesenchymal condensation.

These compartmentalized descriptive models, however, leave several critical developmental enigmas unresolved.

- **Structural inadequacy:** Simple mesothelial apposition cannot account for the highly organized, multilaminated architecture consistently observed in modern high-resolution microanatomy.
- **Temporal discrepancy:** A substantial 10-week latency exists between early organ fixation (10 weeks) and definitive fascial lamination (20 weeks) in the posterior pancreatic and renal regions (Cho et al., 2009).
- **Clinical contradiction:** Historical macroscopic dissections suggest complete fascial agenesis in the absence of a kidney (Tobin, 1944), whereas modern cross-sectional imaging reveals that the

normal posterior renal fascia is a bilaminar structure (Raptopoulos et al., 1986). How the connective tissue meshwork behaves in the developmental absence of the primary organ thus remains a contentious and unresolved question.

Central to the present framework is the Poisson effect—the principle whereby a material stretched along one axis undergoes proportional compression in the orthogonal directions. The present study aims to resolve this 150-year contradiction by integrating fetal histology, biomechanics, and a natural subtraction experiment. It is hypothesized that the systemic tension field converging at 20 gestational weeks—driven by fetal volumetric expansion and musculoskeletal rigidification—constitutes the primary mechanobiological stimulus sculpting the outer layer of the posterior renal fascia through Poisson-effect orthogonal compression and poroelastic compaction, ultimately integrating the retroperitoneum into a body-wide tensegrity framework.

2. Methods (Conceptual and Analytical Approach)

This section describes a conceptual and analytical framework rather than a conventional experimental methodology; no primary data were generated.

To elucidate the physical drivers of fascial morphogenesis, this study employed a theoretical synthesis integrating diverse developmental data with clinical radiological illustration. First, classical and contemporary high-resolution fetal histological literature was re-evaluated along a rigorously aligned chronological axis to identify synchronization phenomena in fascial emergence. Second, fundamental principles of solid and fluid mechanics—specifically the Poisson effect, hoop stress governed by the square–cube law, and biphasic poroelasticity (Mow et al., 1980)—were applied to the undifferentiated three-dimensional mesenchymal meshwork. Third, to interrogate whether fascial lamination is strictly organ-dependent or driven by broader systemic tension, a retrospective radiological review of 5,509 consecutive abdominal CT scans was performed. After explicitly excluding cases with a history of renal surgery, a pure cohort of three cases of adult 'natural renal vacancy' (congenital agenesis or severe dysplasia/involution) was identified. By analyzing the fascial architecture in the developmental absence of the localized tension generator—the kidney—this cohort was employed as a biomechanical 'subtraction experiment' to assess the physical autonomy of the remaining fascial planes.

Regarding manuscript preparation, generative AI tools (Gemini, Google; Copilot, Microsoft; Claude, Anthropic) were used to improve English readability and to assist with the structural organization of the theoretical arguments. Following use of these tools, the author thoroughly reviewed, edited, and verified all content, and takes full intellectual responsibility for the final manuscript.

3. Results

3.1. Radiological Subtraction Experiment (Adult Renal Vacancy)

To empirically validate the systemic, Poisson-effect-driven framework proposed here, a natural subtraction experiment comprising cases of adult renal vacancy was analyzed. From 5,509 CT scans, three rare cases of pure renal vacancy were identified: Cases 1 and 3 presented with true unilateral agenesis and 'pancake' adrenal glands, while Case 2 showed severe dysplasia/involution leaving only a 2-cm dysplastic remnant. In all three cases, despite the lifelong absence of an expanding renal 'core,' a continuous fascial plane was unambiguously preserved (Figure 5).

A. Simple Direct Compression / Disorganized ECM

B. Poisson Effect induced by Hoop Stress / Compact Lamellar Inner Layer

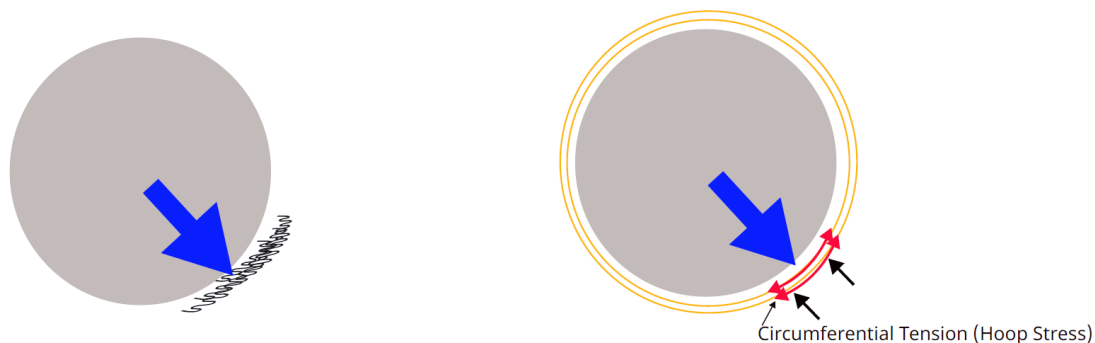


Figure 1. Theoretical comparison of mechanical tissue deformation: direct compression versus the Poisson effect. (A) Simple Direct Compression / Disorganized ECM: The classical model implies that multidirectional or localized compressive forces (e.g., from visceral expansion) act directly upon the undifferentiated mesenchymal meshwork. Physically, this merely squashes the extracellular matrix (ECM) in a disorganized manner, failing to produce aligned fascial sheets. (B) Poisson Effect induced by Hoop Stress / Compact Lamellar Layer: In the present model, when the internal mass expands against an inextensible envelope, it generates circumferential tension (hoop stress). This sustained multi-axial tension forces an obligatory orthogonal compression (the Poisson effect) within the intervening tissue. This highly directional compaction actively aligns and condenses the ECM into the organized, compact lamellar structures characteristic of true retroperitoneal fasciae.

A 10–12 Weeks (Localized Tension Field)

B 20 Weeks (Systemic Tension Network)

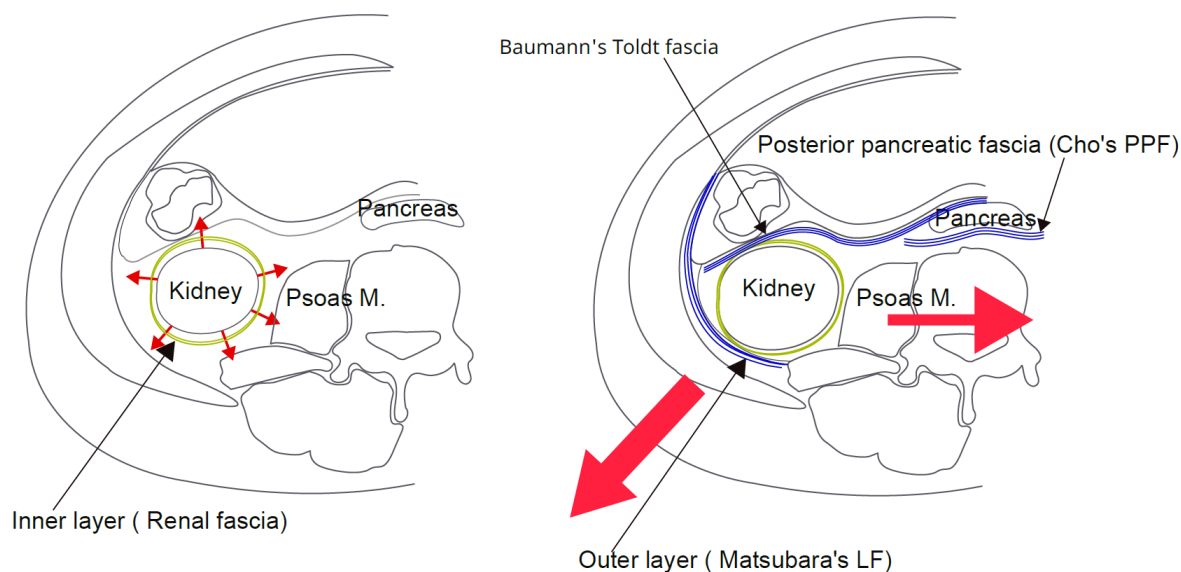


Figure 2. Spatiotemporal and biomechanical asymmetry in the development of the posterior renal fascia. This diagram illustrates the chronological discrepancy in fascial emergence. The inner layer of the renal fascia forms early (10–12 weeks), driven by localized hoop stress from the expanding kidney, whereas the multilaminated outer layer is established significantly later (around 20 weeks) as a direct consequence of the systemic biomechanical convergence and the completion of the macroscopic tension network.

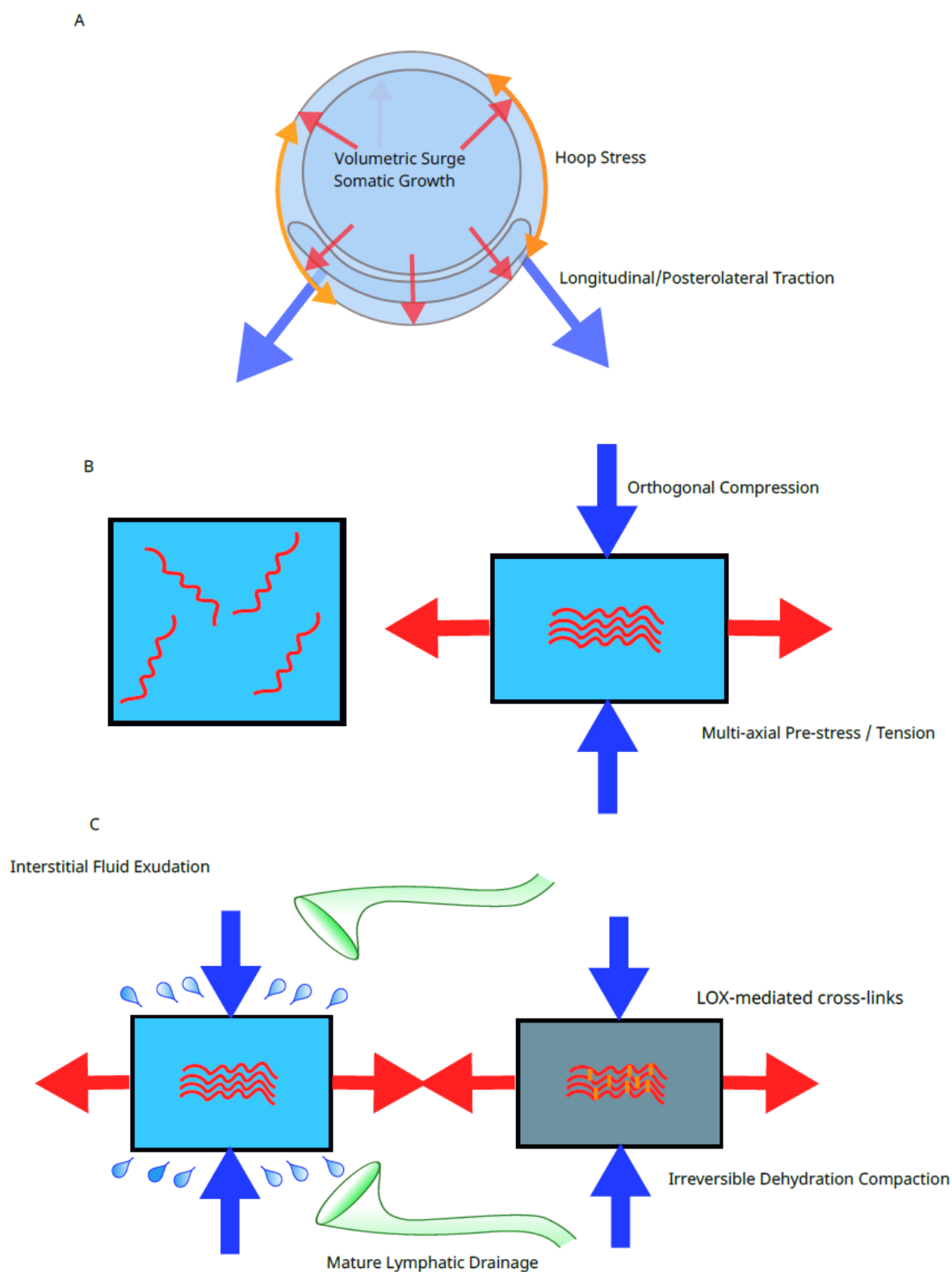


Figure 3. *Mechanobiological model illustrating how the 20-week systemic tension field produces orthogonal Poisson-effect compression, driving lamellar separation.* (A) Macroscopic Tension Convergence: At approximately 20 weeks, the fetal trunk experiences a unique convergence of mechanical forces. Rapid visceral volumetric expansion (red arrows) encounters the resistance of the inextensible keratinized epidermis (hoop stress; orange arrows), while the iliac flare provides a potent posterolateral anchor (blue arrows), creating a high-pressure mechanical field within the extraperitoneal space. (B) Geometric Transformation via the Poisson Effect: Under multi-axial tension, the loose, hydrated mesenchyme undergoes a geometric shift. Stretching along the longitudinal and circumferential axes triggers orthogonal compression (blue arrows), flattening the tissue and forcing

disorganized collagen fibers into a dense, laminated planar arrangement. (C) Irreversible Fixation via Poroelastic Compaction and Cross-Linking: The mechanical compression drives interstitial fluid out of the extracellular matrix (exudation; blue droplets). The functional maturation of the lymphatic system (green) at this stage ensures the permanent removal of this fluid, leading to poroelastic compaction. LOX-mediated covalent cross-linking then chemically stabilizes the approximated collagen fibers, completing the transition from a transient fluid state to definitive solid-state laminae and thereby establishing adult-type fascia.

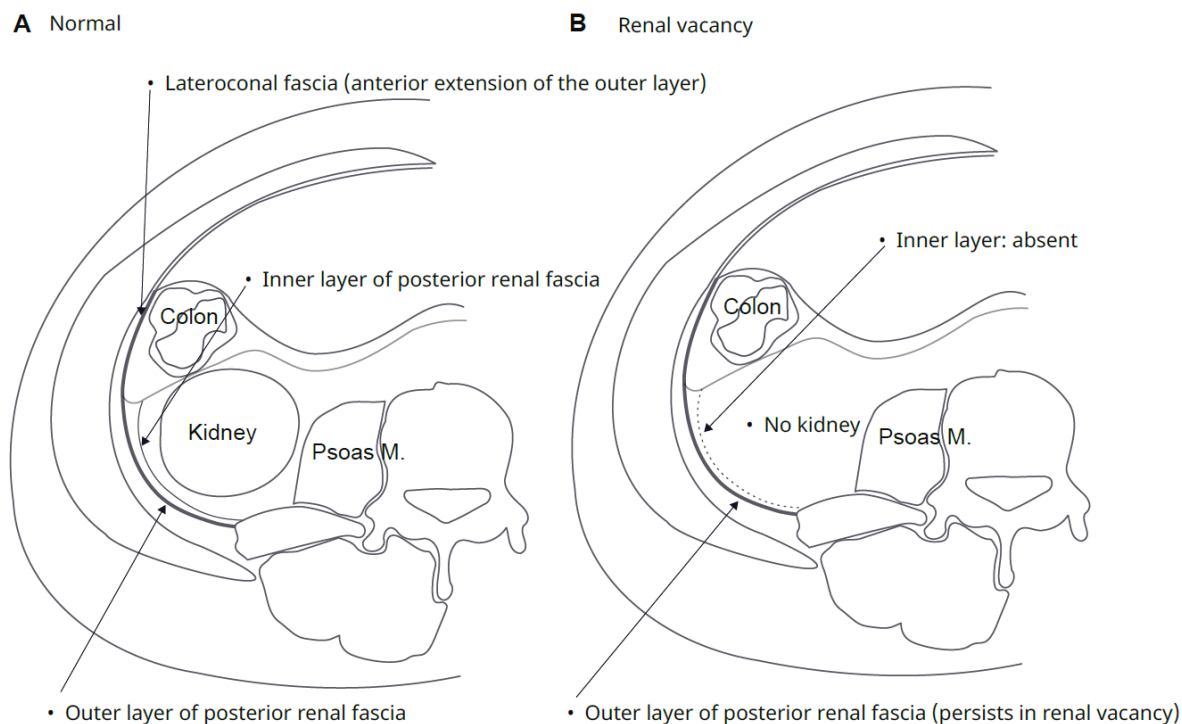


Figure 4. Conceptual and terminological mapping of the retroperitoneal fasciae. This schematic provides a terminology map to resolve historical discrepancies. It highlights the evolution from Congdon's (1941) macroscopic definition (historical 'migration fascia') to Matsubara's (2009) microscopic re-evaluation. To avoid terminological ambiguity, the posterior structure is defined herein as the 'outer layer of the posterior renal fascia,' with the 'lateroconal fascia' treated as its continuous anterior extension.



Figure 5. Radiological validation through the 'subtraction experiment' of renal vacancy. Axial unenhanced computed tomography (CT) demonstrating persistence of the outer fascial layer in the absence of a localized renal mass (Case 1, left renal agenesis). The main image shows a single axial slice comparing both sides. On the right side of the image (patient's left side, renal vacancy), a continuous outer fascial plane is unambiguously preserved despite the lifelong absence of an organ-derived inner layer, resulting in a proportionally reduced total fascial thickness of approximately 1.5 mm. This plane actively anchors the descending colon and peritoneal sac to the posterior retroperitoneal wall, providing compelling evidence for its autonomous formation via the systemic tension network. On the left side of the image (patient's right side, normal anatomy), the posterior renal fascia is not visible at this specific slice level. Inset (bottom left): An axial CT slice at a more cranial level on the normal right side, clearly showing the thick, bilaminar composite posterior renal fascia for comparison. CT images are displayed with soft-tissue window settings (W: 250, L: 150). This radiological 'subtraction experiment' isolates the tension-derived outer layer by removing the organ-dependent inner layer from the developmental equation.

Quantitative measurements performed with 3D Slicer demonstrated that this persisting plane (mean thickness ~1.52 mm, compared with the normal bilaminar composite of 1.85 mm) continuously tethered the peritoneal sac to the posterior abdominal wall. This uniform reduction in thickness is interpreted as reflecting the developmental subtraction of the organ-dependent inner layer, leaving the tension-driven outer layer intact. This structural autonomy provides compelling evidence that macroscopic fascial lamination is governed primarily by a systemic tension field rather than by local visceral growth alone.

3.2. Chronological Synthesis of Fetal Fascial Development

Histological studies demonstrate that the inner layer of the renal fascia initially emerges circumferentially around the developing kidney at 10–12 weeks of gestation (Matsubara et al., 2009; Figure 2). This early, localized formation is biomechanically attributable to hoop stress—the circumferential tension generated within the surrounding envelope by radial expansion of the growing kidney (Fung, 1990)—which in turn triggers a localized Poisson effect.

In contrast, a comparative synthesis reveals a striking synchronicity in the broader, systemic maturation of retroperitoneal fasciae. Baumann (1945) documented the establishment of Toldt's fascia at approximately 18–20 weeks. Cho et al. (2009) identified a critical transition beginning at 20 weeks, at which the posterior pancreatic fascia transforms into a distinct laminated structure. Matsubara et al. (2009) similarly confirmed that the multilaminated architecture of the outer layer of the posterior renal fascia becomes histologically definitive around the same 20-week period. This chronological convergence identifies gestational week 20 as a critical biomechanical threshold for macroscopic fascial lamination.

4. Theoretical Integration and Discussion

4.1. Mechanobiological Interpretation of the 20-Week Transition

The formation of distinct fascial layers may be understood through three sequential mechanobiological processes:

- Fibroblast alignment along dominant macroscopic tension vectors (Ingber, 2003).
- Orthogonal extracellular matrix (ECM) compression dictated by the Poisson effect and poroelasticity (Lakes, 1991).
- Enzymatic stabilization via LOX-mediated cross-linking.

As the undifferentiated mesenchymal field is stretched laterally and axially by somatic growth, the tissue necessarily thins in the perpendicular direction. In hydrated embryonic tissue, this orthogonal compaction forces the exudation of interstitial fluid, resulting in laminar separation according to biphasic poroelastic principles (Mow et al., 1980). Lysyl oxidase (LOX) then catalyzes covalent cross-links between these densely packed collagen fibers (Kagan & Li, 2003), ensuring the irreversible stabilization of the emergent structures as distinct laminae (Figure 3).

The synchronous clarification of multiple retroperitoneal fasciae around 20 weeks strongly implies a shared mechanobiological driver, with the Poisson effect serving as a unifying principle. The stabilizing kidney creates early local tension, forming the inner layer of the renal fascia at 10–12 weeks. Later, as systemic structures integrate around 20 weeks, multi-axial tension induces orthogonal Poisson-effect compression, generating the laminated outer layer. The 10-week latency suggests that early visceral fixation serves to integrate the organs into a developing tension network; definitive lamination occurs only once the systemic tension field reaches a critical threshold.

4.2. Systemic Tension Field and Poisson-Effect Lamination

Constrained between rigidifying osseous anchors and an inextensible external envelope, the intervening retroperitoneal mesenchyme undergoes an abrupt escalation of multi-axial tension. In accordance with the physical principles of Poisson's ratio, this stretching forces orthogonal compression within the tissue, directly driving the sudden lamination of the outer layer of the posterior renal fascia.

This biomechanical convergence represents a systemic phase transition extending well beyond the retroperitoneum. Notably, the perineurial tight junctions of the human sciatic nerve undergo marked structural compaction beginning around 21–22 weeks (Pummi et al., 2004), a timing that cannot be explained by local nerve growth alone. This synchronous maturation strongly suggests a system-wide mechanical transition, consistent with the proposed 20-week tension field. Because the perineurium is a tension-sensitive, multilayered connective tissue sheath, its maturation timing provides an independent indicator of systemic mechanical transitions during fetal development.

Furthermore, this static tension network is powerfully amplified by a positive biomechanical feedback loop involving fetal motor activity. Before 15 weeks, the highly compliant fetal skeleton fails to transmit muscular forces effectively (Nowlan, 2015). However, as the vertebral column ossifies (Bagnall et al., 1977) and the iliac flare expands (Baumgart et al., 2018) toward week 20, these rigidified structures provide the essential mechanical anchors for efficient muscle contraction. As a

result, gross fetal body movements—particularly coordinated kicking—increase substantially in both frequency and transmissible force from 20 weeks onward (de Vries et al., 1982; Patrick et al., 1982). This transition generates rhythmic, dynamic traction on the retroperitoneal mesenchyme. It is proposed that these dynamic tension spikes synergize with static volumetric expansion to actively align fibroblasts and accelerate the poroelastic fluid exudation required for definitive fascial lamination.

4.3. Terminological Clarification and Clinical Implications

To avoid terminological ambiguity, the posterior structure is defined herein as the 'outer layer of the posterior renal fascia,' with the 'lateroconal fascia' treated as its continuous anterior extension (Figure 4). With respect to the lateroconal fascia, which Congdon et al. (1941) historically defined as the lateral fusion of the anterior and posterior renal fasciae, Matsubara (2009) used the term more broadly to encompass both the outer layer of the posterior renal fascia and its anterior extension.

Clinically, this mechanobiological framework offers a principled explanation for the avascular dissection planes exploited in oncological surgery. Kinugasa et al. (2008) demonstrated that optimal retroperitoneal dissection should proceed along specific multi-layered fascial boundaries defined by embryological architecture. The present model reveals that these empirically established surgical planes correspond precisely to the tension-aligned layers generated by poroelastic compaction. Recognizing these protective planes as mechanically derived structures—rather than mere fusion scars—provides a robust anatomical foundation for standardizing embryologically sound surgical approaches. This framework also explains why pathological high-pressure fluid accumulation—such as occurs in acute pancreatitis—acts as a hydraulic separator, re-infiltrating the ECM and reopening the orthogonal cleavage planes originally established by the Poisson effect (Ishikawa et al., 2006).

4.4. Resolving the Historical Debate on the Bilaminar Renal Fascia

Although Gerota's original illustration (1895) depicted the renal fascia as a two-layered structure, the developmental and functional significance of this bilaminar arrangement went unrecognized for nearly a century. A renewed appreciation emerged only in the mid-1980s, when Marks et al. (1986) reported that their radiological observations of posterior renal fascial planes bore a striking resemblance to Gerota's original depiction—an unexpected concordance that prompted reconsideration of the anatomical complexity of this region. Shortly thereafter, Raptopoulos et al. (1986) provided a more systematic radiological characterization, demonstrating that the posterior renal fascia is not a single sheet but a bilaminar structure, thereby establishing the modern imaging-based framework that classical dissection had failed to capture.

Classical dissection studies—most notably that of Tobin (1944)—concluded that the renal fascia may be entirely absent in renal agenesis, reinforcing the longstanding assumption that fascial formation depends strictly on renal presence. Subsequent fetal studies further clarified that the two layers arise from distinct developmental mechanisms (Matsubara et al., 2009), suggesting that the outer layer is not simply a passive extension of the organ-dependent inner layer.

The radiological cohort of renal vacancy ($n=3$) assembled in the present study provides a decisive subtraction experiment that directly tests this historical debate. When the kidney—the presumed generator of localized radial tension—is absent from the developmental equation, the inner layer predictably fails to form. Nevertheless, a continuous outer fascial layer persisted into adulthood in all three cases. This finding reconciles the longstanding discrepancy between classical dissection and modern imaging: the inner layer is organ-dependent, whereas the outer layer is a tension-derived, systemically generated structure that forms independently of renal presence. By isolating the outer layer in its 'naturally subtracted' state, these data reveal its functional autonomy and identify it as a product of the systemic tension field converging around 20 gestational weeks.

4.5. The Square–Cube Law and the Emergence of a Fetal Tensegrity System

Below approximately 15 weeks of gestation, the fetal trunk behaves as a pressure-dominated structure. Internal viscera and fluid are contained by the compliant body wall, and internal pressure is distributed almost isotropically, enabling global form to be maintained without specialized load-bearing fascial planes.

As fetal size increases, however, the square–cube law imposes a fundamental mechanical constraint. For a growing body of radius r , the surface-area-to-volume ratio is $3/r$; its reciprocal—representing the internal mechanical load borne by each unit area of the cutaneous envelope—is therefore proportional to $r/3$. Based on standard biometric curves, this internal load increases nearly threefold between 12 and 20 weeks. As $r/3$ rises, the simple 'hydrostatic sac' strategy becomes mechanically untenable: the body wall alone can no longer contain the expanding viscera.

Simultaneously, the internal anchors of the system are stiffening. Ossification of the vertebral column (Bagnall et al., 1977), lateral expansion of the ilium (Baumgart et al., 2018), and outward migration of the abdominal wall create rigid boundaries between which the retroperitoneal mesenchyme is stretched. The stress field consequently becomes anisotropic, with forces preferentially transmitted between the rigidifying axial skeleton and the expanding abdominal wall.

In this configuration, the retroperitoneal mesenchyme functions as a temporary fluid-supported strut. Rich in glycosaminoglycans and interstitial fluid, it initially resists deformation through fluid incompressibility. As multi-axial tension escalates around 20 weeks, Poisson-effect orthogonal compression forces fluid out of the matrix and into the maturing lymphatic system (Bekker et al., 2005; Mow et al., 1980). Once this poroelastic exudation occurs, the tissue can no longer rely on fluid support; the remaining collagen framework collapses and condenses into fiber-supported, tension-bearing laminae.

Around 20 weeks, the fetal trunk therefore undergoes a mechanical regime shift: from a pressure-dominated, isotropic, fluid-supported continuum to a tension-dominated, anisotropic, fiber-supported network. This transition represents the emergence of a fetal-scale tensegrity-like structural system (Ingber, 2003). The ossifying vertebral column, the laterally expanding iliac blades, and the inextensible cutaneous envelope function as discrete compression-bearing elements, while the retroperitoneal mesenchyme and fascial continuum form a continuous tension network. Within this architecture, Poisson-effect orthogonal compression constitutes the local expression of these global compressive struts. Together, these interacting tension and compression elements generate a mechanically integrated framework analogous to a tensegrity structure, enabling efficient force transmission and stabilizing the laminated retroperitoneal fasciae that emerge at mid-gestation. The outer layer of the posterior renal fascia represents one of the clearest anatomical manifestations of this regime shift—its formation reflects the rising $r/3$ -driven internal load and the emergence of a system-wide tension network, rather than any dependence on renal presence.

4.5.1. Developmental Prerequisites from Pelvic Morphogenesis

Building on this structural transformation, recent developmental work by Senevirathne et al. (2025) provides an essential upstream framework for interpreting the present mechanobiological model. Their study demonstrated that the human ilium acquires its uniquely broad, laterally flared geometry through two key developmental innovations: a heterotopic reorientation of the iliac growth plate and a heterochronic, externally biased ossification sequence. These morphogenetic shifts—absent in other primates—establish the geometric preconditions for the lateral traction vectors that become mechanically relevant in mid-gestation.

When these findings are integrated with the temporal synthesis of Verbruggen and Nowlan (2017), who showed that the most rapid phase of iliac expansion occurs precisely during mid-gestation, a coherent developmental hierarchy becomes apparent. The genetic and cellular mechanisms described by Senevirathne et al. define the shape and material distribution of the pelvis, whereas the present model identifies the moment—around 20 gestational weeks—at which this genetically specified geometry becomes mechanically integrated into the systemic tension network. In this view, the 20-week convergence represents the point at which structure enables function: the

rigidified spine and expanded iliac flare begin to transmit fetal motor forces efficiently, amplifying both static volumetric tension and dynamic traction. The findings of Senevirathne et al. therefore do not compete with the present mechanobiological model; rather, they furnish its essential morphogenetic substrate, clarifying why the Poisson-effect-driven lamination of retroperitoneal fasciae emerges only after the pelvis has acquired its human-specific geometry.

4.6. *Lymphatic Maturation, Poroelastic Compaction, and Cross-Linking*

Although the Poisson effect dictates orthogonal compression, irreversible fixation requires both fluid drainage and enzymatic stabilization. The fetal lymphatic system acquires functional systemic drainage capacity between 14 and 16 weeks (Bekker et al., 2005). As Poisson-effect compression occurs around 20 weeks, mature lymphatics facilitate poroelastic fluid exudation, bringing dispersed collagen fibers into intimate physical proximity. This proximity constitutes the essential spatial prerequisite for LOX-mediated covalent cross-linking (Kagan & Li, 2003), which chemically stabilizes the lamination and accounts for the irreversibility of fascial compaction.

4.7. *Comparative Anatomy Implications*

Although the Poisson effect is universal in mammalian soft tissues, the highly specialized multilaminated architecture of the human retroperitoneum appears intrinsically linked to hominin bipedalism. Quadrupedal mammals lack the uniquely flared ilium required for upright posture, suggesting that this specific lateral traction vector is a geometric prerequisite for human-like fascial lamination. A detailed comparative biomechanical analysis, however, lies beyond the scope of the present study.

4.8. *Limitations and Future Directions*

While this mechanobiological model is consistent with histological observations and is supported by the cross-sectional radiological subtraction experiment described herein, it remains a theoretical synthesis. Future empirical validation—such as direct quantification of in vivo retroperitoneal tissue stiffness and tension vectors using high-resolution fetal MRI or ultrasound elastography—will be essential to substantiate the proposed tension network.

5. Conclusions

The systemic tension field converging at 20 gestational weeks constitutes the primary mechanobiological stimulus sculpting the outer layer of the posterior renal fascia through Poisson-effect orthogonal compression. The natural subtraction experiment in adult renal vacancy conducted in the present study provides compelling evidence for this structural autonomy, ultimately resolving a 150-year anatomical contradiction regarding retroperitoneal fascial formation.

By identifying the 20-week systemic tension field as the mechanobiological trigger for fascial lamination, the present study offers a unifying framework that integrates fetal biomechanics, developmental anatomy, and clinical radiology.

Table 1. Chronological Synthesis of Retroperitoneal Fascial Development and Associated Mechanobiological Events.

Clinical Gestational Age	Anatomical / Biomechanical Events	Mechanobiological Significance	Key References
10–12 weeks	Early pancreatic fixation to the posterior wall; circumferential emergence of the inner layer of the renal fascia; morphogenetic blueprint of iliac flare geometry established via cartilage shift	Localized tension and spatial vector preparation: Viscera asynchronously integrate into the nascent tension network. Pelvic cartilage establishes the future posterolateral traction vector, though it remains too compliant at this stage to generate systemic tension.	Cho et al. (2009); Matsubara et al. (2009); Senevirathne et al. (2025)
14–16 weeks	Functional maturation of the fetal lymphatic system (terminal venous connections)	Physiological preparation: Establishes systemic drainage capacity required for poroelastic fluid exudation during the subsequent Poisson-effect compression phase.	Berger (1999); Bekker et al. (2005)
18–20 weeks	Progressive ossification of the vertebral column; epidermal keratinization and completion of the inextensible cutaneous envelope; volumetric growth begins to surge exponentially (square-cube law)	Dynamic preconditioning and the square-cube law: The spine stiffens into a central pillar. Volumetric expansion outpaces surface area, generating massive outward pressure. This collides with epidermal maturation to produce profound systemic hoop stress.	Bagnall et al. (1977); Singh & Archana (2008); Hadlock et al. (1991)
Around 20 weeks (The Chronological Intersection)	Somatic rigidification: cumulative three-dimensional pelvic expansion and ossification reach a biomechanical	The temporal mechanical trigger and Poisson effect: As anchors stiffen into rigid levers, growth forces are transmitted without dissipation. The resulting	Baumann (1945); Cho et al. (2009); Matsubara et al. (2009); Verbruggen & Nowlan (2017); (Current Model)

	threshold; fascial lamination: synchronous, definitive clarification of Toldt's fascia, the posterior pancreatic fascia, and the multilaminated outer layer of the posterior renal fascia	tension spike induces orthogonal compression (Poisson effect), forcing fluid exudation and subsequent LOX-mediated cross-linking to complete fascial lamination.	
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Table 2. Systemic Manifestations of the Biomechanical Convergence Around 20 Weeks.

Anatomical System	Event at ~20 Weeks	Biomechanical Significance	Key References
Skeletal System	Ossification of the vertebral column	Rigid central pillar for tension transmission	Bagnall et al. (1977)
	Expansion and ossification of the iliac flare	Establishes the lateral lever arm for whole-body tension	Baumgart et al. (2018); Senevirathne et al. (2025)
Cutaneous Envelope	Epidermal keratinization	Inextensible shell generating systemic hoop stress	Hardman et al. (1999)
Somatic Growth	Exponential volumetric expansion	Square-cube law drives global tension	Hadlock et al. (1991)
Musculoskeletal Motor System	Intensification of fetal kicking and gross body movements	Dynamic tension spikes actively align fibroblasts and accelerate poroelastic compaction	Nowlan (2015); de Vries et al. (1982); Patrick et al. (1982)
Respiratory Physiology	Sharp increase in fetal breathing movements (FBMs)	Rhythmic loading reinforces the tension network	Nowlan (2015)
	Rib cage rigidity increases	Thorax becomes a stable mechanical frame	Bagnall et al. (1977); Verbruggen & Nowlan (2017)
	Surfactant production begins	Stabilizes alveoli, sustaining consistent FBMs	Avery & Fletcher (1974); Clements (1957)

Lymphatic System	Systemic drainage capacity matures	Promotes irreversible poroelastic compaction	Bekker et al. (2005)
Fascial Structures	Lamination of Toldt's fascia	Reflects the systemic tension threshold	Baumann (1945)
	Lamination of the posterior pancreatic fascia	Occurs after a 10-week latency period	Cho et al. (2009)
	Lamination of the outer layer of the posterior renal fascia	Multi-axial tension combined with Poisson-effect compression	Matsubara et al. (2009)
Peripheral Nervous System	Compaction of sciatic nerve sheaths	Pelvic traction induces Poisson-effect compression	Pummi et al. (2004)

Table 3. Clinical Characteristics and Quantitative Radiological Analysis of the Renal Vacancy Cohort (n=3). Extracted from a primary screening of 5,509 consecutive abdominal CT scans, strictly excluding patients with a history of renal surgery.

Case	Age/Sex	Radiological Diagnosis	Adrenal Morphology	Fascial Thickness (Vacancy Side)	Fascial Thickness (Normal Side)	Difference (Δ)
1	53/F	True Left Renal Agenesis	"Pancake" (lying-down)	1.49 mm	1.88 mm	-0.39 mm
2	47/F	Severe Left Renal Dysplasia / Involution (renal nubbin)	Normal	1.46 mm	1.82 mm	-0.36 mm
3	89/M	True Left Renal Agenesis	"Pancake" (lying-down)	1.62 mm	Excluded*	N/A
Mean				1.52 mm	1.85 mm	-0.38 mm

*Case 3 normal side was excluded from the mean due to the presence of localized contralateral inflammation affecting the fascial planes.

Author Contributions: H.T. conceived the study, performed the literature synthesis, developed the mechanobiological model, analyzed the radiological data, and wrote the manuscript.

Ethics Statement: This retrospective study was approved by the Institutional Review Board (IRB) of Gakkentoshi Hospital (Approval No. GT-R6-07-12-1). The requirement for written informed consent was waived due to the retrospective nature of the study, and an opt-out mechanism was provided via the hospital's official website in accordance with national ethical guidelines.

Data Availability: The radiological data supporting the findings of this study are restricted due to patient privacy protocols but may be requested from the corresponding author. All other information is derived from previously published literature.

Acknowledgments: The author thanks colleagues in the Department of Urology at Gakkentoshi Hospital for their support and for providing a clinical environment that continually inspires anatomical inquiry. Sincere gratitude is extended to the radiological staff at Gakkentoshi Hospital for their exceptional technical assistance in the systematic retrieval of clinical imaging data. The author also acknowledges the foundational contributions of classical anatomists whose meticulous observations continue to guide modern reinterpretations.

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

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