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Article

# Gestational Week 20 as the Mechanobiological Inflection Point of Retroperitoneal Fascial Lamination: A Poisson Effect Model

Running title: Mechanobiology of the Retroperitoneal Fascia

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## Abstract

This study proposes a mechanobiological model explaining how the multilayered retroperitoneal fascia forms through the interplay of local and systemic tension fields. The classical peritoneal fusion hypothesis (Toldt, 1879) cannot account for the regular lamellar architecture observed in this region, nor for the 10-week temporal lag between early visceral fixation (gestational week 10) and definitive fascial lamination (gestational week 20). We hypothesize that early local tension at gestational weeks 10–12 forms the inner layer of the renal fascia, while a “systemic tension field”—driven by axial skeletal ossification, pelvic expansion, and exponential volumetric growth, converging near gestational week 20—establishes a fetal-scale **tensegrity-like** network. This systemic tension triggers orthogonal Poisson effect compression, poroelastic fluid exudation, and lysyl oxidase (LOX)-mediated cross-linking, the integration of which generates the multilayered outer fascial layers. To provide empirical grounding for this theoretical framework, we identified a cohort of adults with pure renal absence (empty renal fossa;  $n = 3$ ) from 5,509 consecutive CT scans. Despite the absence of a sustained, expanding renal mass (due to true agenesis or severe involution), continuous outer fascial layers were unambiguously preserved in all cases, demonstrating that their formation is tension-driven rather than organ-dependent. This natural “subtraction experiment” resolves a long-standing discrepancy between classical gross anatomy and modern cross-sectional imaging, supporting a 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 layers of the retroperitoneal space have been debated since the late nineteenth century. Historically, the retroperitoneal fascia has been classified on the basis of the classical peritoneal fusion hypothesis (Toldt, 1879) and independent fixation-apparatus models (Zuckerkanndl, 1883; Gerota, 1895). Hayes (1950) subsequently introduced the concept of “migration fasciae,” proposing that mechanical stress generated by visceral migration induces focal mesenchymal condensation.

Although Gerota’s original illustration (1895) depicted the renal fascia as a bilaminar structure, the **anatomical significance** of this arrangement went unrecognized for nearly a century. Only with the advent of modern cross-sectional imaging did Raptopoulos et al. (1986) systematically demonstrated the bilayered nature of the posterior renal fascia. Subsequently, in a companion anatomical study, Marks et al. (1986) noted that this radiological configuration bore a striking resemblance to Gerota’s classical depiction, praising the accuracy of the century-old drawings.

Building on these radiological insights, the concept of “interfascial planes” has emerged as the prevailing clinical paradigm, redefining retroperitoneal fasciae as potential spaces and dynamic

pathways for disease spread (Ishikawa et al., 2016). However, despite this modernized clinical consensus, the developmental etiology taught in contemporary authoritative textbooks (e.g., *Campbell-Walsh-Wein Urology*, 12th ed.) continues to rely fundamentally on classical descriptive embryology—specifically, Tobin’s (1944) three-stratum fusion model. Consequently, these compartmentalized descriptive models leave several critical developmental questions unresolved:

- **Structural inadequacy:** Simple mesothelial apposition (fusion) alone cannot account for the highly organized multilayered (lamellar) architecture consistently observed in high-resolution micro-anatomical studies.
- **Temporal paradox:** Classical models assume that fascial lamination occurs concurrently with visceral fixation. However, a pronounced temporal lag exists. In the posterior pancreatic region, early visceral fixation (gestational week 10) precedes definitive fascial lamination by 10 weeks (Cho et al., 2009). Similarly, in the renal region, anatomical fixation (completion of ascent) occurs by weeks 7–9, followed by the formation of the organ-dependent inner fascial layer at weeks 10–12. However, the definitive multi-layered outer lamination is delayed until the week-20 threshold (Matsubara et al., 2009). **This 10-week developmental lag represents the central paradox that classical descriptive embryology cannot explain.** This asynchronous development suggests that definitive outer lamination is governed not by initial organ fixation, but by a later, systemic trigger.
- **Clinical contradiction:** Classical gross anatomy suggested that the renal fascia is entirely absent when the kidney is missing (Tobin, 1944), whereas modern cross-sectional imaging clearly demonstrates a bilayered posterior renal fascia in the normal state (Raptopoulos et al., 1986). The behavior of connective tissue meshwork in the absence of the principal organ remains an unresolved question.

Although Hayes (1950) introduced a mechanical perspective, his “migration fasciae” model attributed fascial formation to the stress generated by the active migration of organs. In contrast, recent high-resolution fetal histological observations have revealed that definitive fascial lamination occurs long after organs have reached their final anatomical positions and fixed to the posterior wall. Consequently, modern anatomists speculate that a different form of mechanical stress—arising from **localized post-fixation visceral dynamics, such as gastrointestinal peristalsis and adjacent organ expansion**—forces the reinforcement of initially vague mesenchymal remnants into definitive fascial layers (Cho et al., 2009; Wedel et al., 2022). However, they implicitly assume that direct compression exerted by **these adjacent visceral movements** drives this fascial thickening. This **strictly localized perspective** fails to capture the true morphogenetic principles and leaves the specific, system-wide biophysical mechanisms governing this synchronized structural transformation unidentified.

Central to the theoretical framework of this study is the Poisson effect—the physical phenomenon whereby stretching a material along one axis produces proportional compression along the orthogonal axes. Integrating fetal histology, biomechanics, and a natural subtraction experiment, this study aims to resolve a 150-year-old anatomical paradox. We hypothesize that gestational week 20 marks a fundamental biomechanical inflection point where the fetal trunk transitions from a pressure-driven isotropic state to a systemic tension-driven **anisotropic** network. Driven by exponential volumetric growth and musculoskeletal rigidification, this emerging systemic tension field generates Poisson effect orthogonal compression. This orthogonal compression, combined with poroelastic consolidation, is the true mechanobiological stimulus that sculpts the outer layer of the posterior renal fascia, entirely replacing the classical illusion of direct local compression.

## 2. Methods: Conceptual and Analytical Approach

This section describes the conceptual and analytical framework adopted; primary experimental data were not generated.

To elucidate the physical determinants of fascial morphogenesis, we employed a theoretical approach integrating diverse embryological data with clinical imaging examples. First, classical and

contemporary high-resolution fetal histological literature was re-evaluated along a rigorously aligned timeline to identify synchronization events in fascial emergence. Second, fundamental principles of solid and fluid mechanics—specifically the square-cube law (which necessitates the systemic mechanical phase transition), along with hoop stress, the Poisson effect, and biphasic poroelasticity (Mow et al., 1980)—were applied to undifferentiated three-dimensional mesenchymal meshwork. Third, to demonstrate whether fascial lamination is strictly organ-dependent or driven by a broader systemic tension field, we conducted a retrospective radiological review of 5,509 consecutive abdominal CT scans. After explicit exclusion of patients with a history of renal surgery, a pure cohort of three adults with “natural renal absence” (congenital agenesis or severe dysplasia/involution) was identified. By analyzing fascial architecture in the absence of the local tension source (the kidney), this cohort served as a biomechanical subtraction experiment to interrogate the physical autonomy of the residual fascial plane.

Use of AI-assisted tools: AI-based tools were used to support the clarification and organization of conceptual ideas and to improve English readability. No generative AI system contributed original scientific content, data interpretation, or theoretical conclusions. All scientific arguments, mechanobiological models, and interpretations were independently developed, verified, and fully validated by the author, who assumes complete responsibility for the accuracy and integrity of the manuscript.

### 3. Results

#### 3.1. Radiological Subtraction Experiment (Adult Renal Absence)

To empirically test the systemic Poisson effect-driven model, we analyzed a natural subtraction experiment comprising adults with renal absence. From 5,509 CT scans, three rare cases of pure renal absence were identified: Cases 1 and 3 represented true unilateral renal agenesis with a “pancake” adrenal configuration, and Case 2 showed severe renal dysplasia/involution retaining only a 2-cm dysplastic remnant. While true unilateral agenesis (Cases 1 and 3) provides an absolute developmental subtraction of the localized tension generator, severe involution (Case 2) represents a failure to sustain an expanding mass, even if transiently present during early gestation. Crucially, across all three functional subtraction scenarios, continuous fascial planes were unambiguously preserved (Figure 5) despite the developmental failure to provide a sustained, localized expanding “core.”

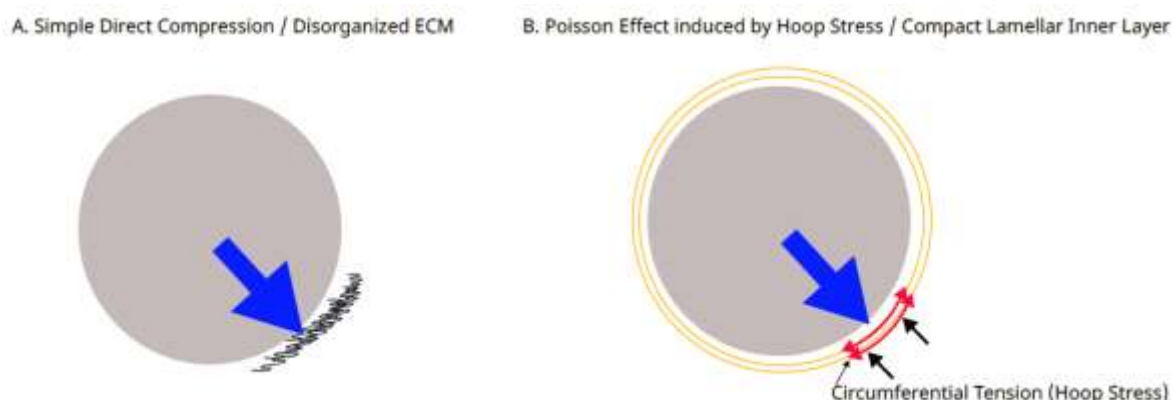
Quantitative measurement using 3D Slicer demonstrated that the residual fascial plane (mean thickness approximately 1.52 mm, compared with 1.85 mm for the normal bilaminar composite) continuously tethered the peritoneal sac to the posterior abdominal wall. This uniform reduction in thickness is interpreted as reflecting subtraction of the organ-dependent “inner layer” during development, with the tension-driven “outer layer” remaining intact. This structural autonomy provides strong evidence that macroscopic fascial lamination is not solely a product of local visceral growth but is ultimately governed by the systemic tension field.

#### 3.2. Temporal Integration of Fetal Fascial Development

Histological studies indicate that the inner layer of the renal fascia first appears circumferentially around the developing kidney at gestational weeks 10–12 (Matsubara et al., 2009; Figure 2). This early local formation is biomechanically attributable to hoop stress—circumferential tension generated within the surrounding envelope by the radial growth of the expanding kidney (Fung, 1990)—which in turn induces local Poisson effect compression. (Figure 1 near here)

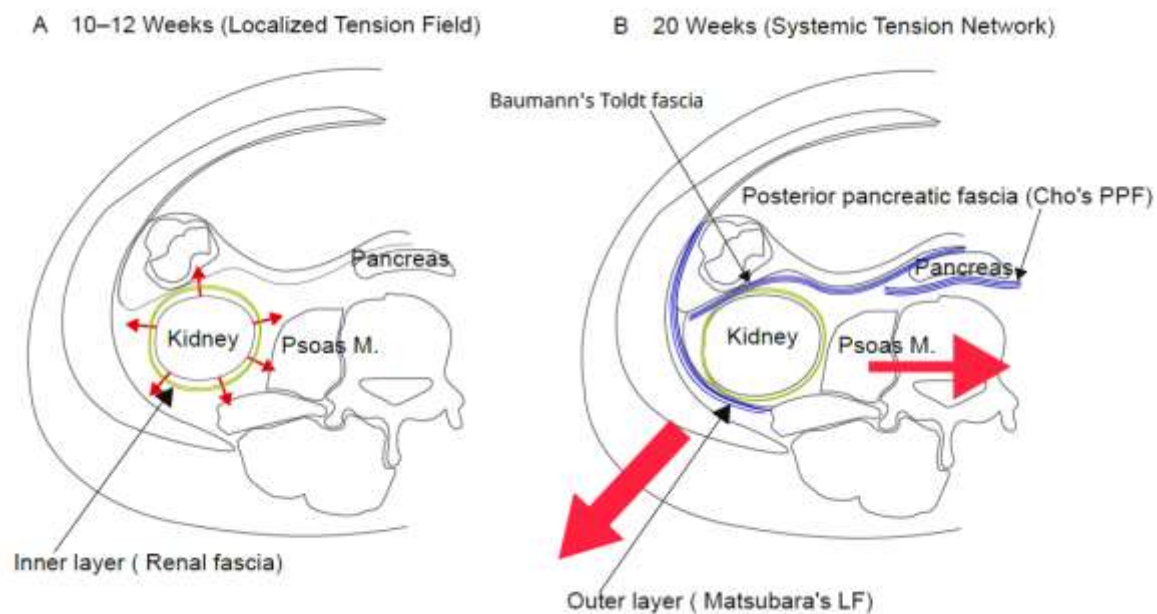
By contrast, comparative integration revealed a striking synchrony in the broader systemic maturation of the retroperitoneal fascia. Baumann (1945) documented establishment of the Toldt fascia at approximately gestational weeks 18–20. Cho et al. (2009) identified a critical inflection point beginning at gestational week 20, at which the posterior pancreatic fascia transitions to a distinct lamellar architecture. Matsubara et al. (2009) similarly confirmed that the multilayered structure of

the outer posterior renal fascia becomes histologically definitive around the same week-20 threshold. This temporal convergence identifies gestational week 20 as the critical biomechanical threshold for macroscopic fascial lamination. (Figure 2 near here)



**Figure 1. Theoretical comparison of mechanical tissue deformation: Direct Compression vs. 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.



**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 macroscopic tension network completion.

## 4. Theoretical Integration and Discussion

### 4.1. Mechanobiological Interpretation of the Week-20 Inflection Point

Formation of distinct fascial layers can be understood through continuous mechanobiological tissue remodeling. Soft biological tissues dynamically restructure their internal architecture in response to sustained mechanical loading (Humphrey, 2003). As the multiaxial tension field generated by somatic growth stretches the undifferentiated mesenchyme, it inevitably produces a strong orthogonal compressive force—the Poisson effect (Lakes, 1991). Crucially, the highly ordered alignment of collagen fibers along the principal tension vectors (Provenzano & Vanderby, 2006) is itself powerfully driven by this orthogonal spatial constraint.

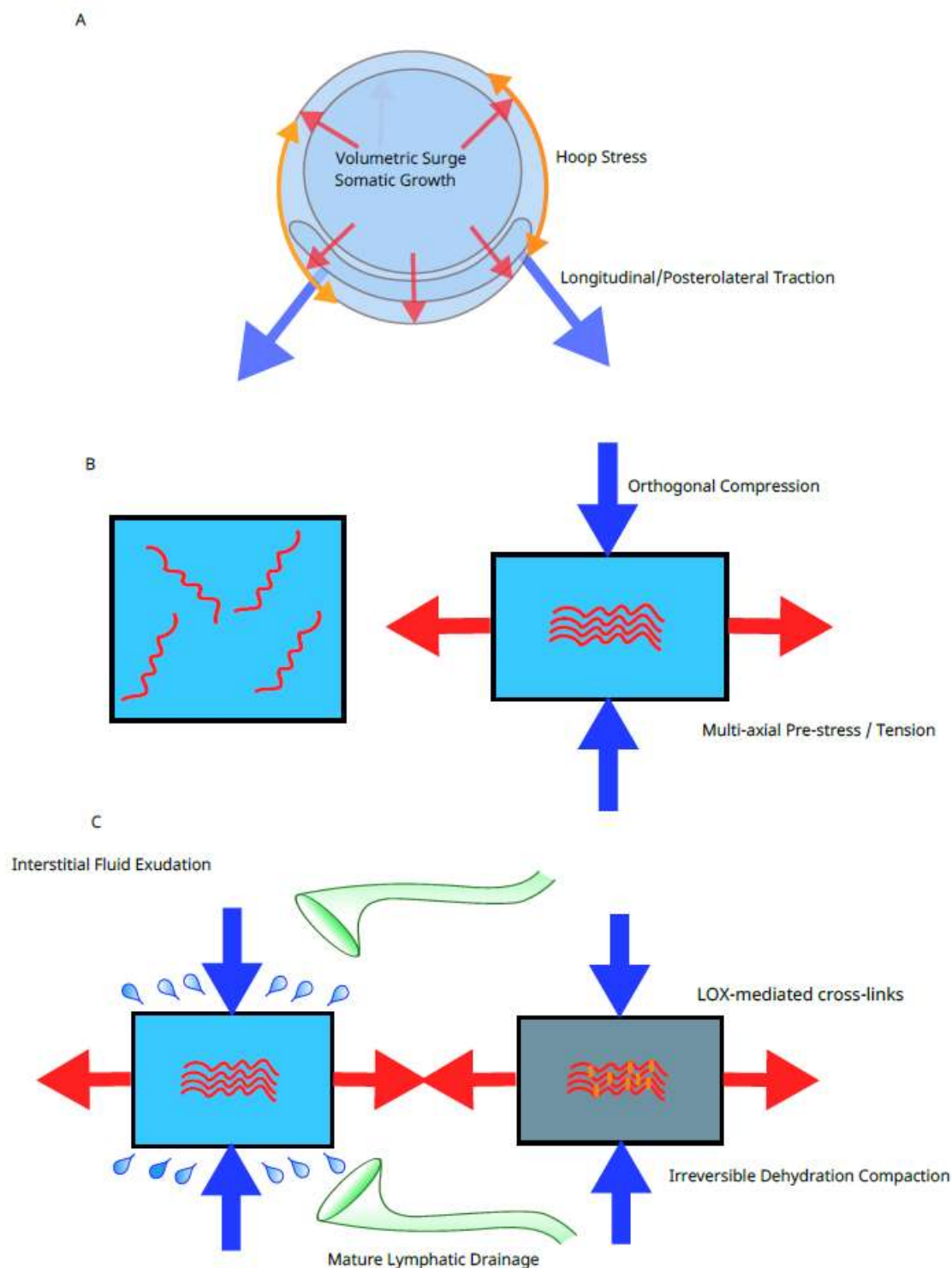
In the highly hydrated fetal tissue, this Poisson-effect compression not only forces the disorganized fibers into a planar alignment but also drives the exudation of interstitial fluid. In regions where primitive adipose tissue is concurrently developing, such as the lateroconal fascia, this highly hydrated mass acts as a localized incompressible body. Indeed, Matsubara et al. (2009) morphologically observed that this primitive adipose tissue overtly compresses adjacent loose connective tissue into a membranous sheet—an appearance that perfectly represents a **localized physical footprint** of this systemic orthogonal Poisson compression. However, the universal driver of fascial lamination across all retroperitoneal regions remains the poroelastic consolidation of the GAG-rich mesenchyme itself, regardless of local adipogenesis. According to the principles of biphasic poroelasticity (Mow et al., 1980), this process separates and compacts the fibrous network into the distinctive multilaminated cleavage planes characteristic of the retroperitoneum. Although mature adipose tissue is not yet present at gestational week 20, the retroperitoneal mesenchyme—derived from the dorsal mesentery and retaining its mesenchymal, GAG-rich characteristics (Matsubara et al., 2009)—contains preadipocytic, highly hydrated, GAG-rich stromal cells (Carlson, 2014; Chau et al., 2014; Han et al., 2011; Coffey & O’Leary, 2016). Their poroelastic behavior likely contributes to the fluid-supported phase of the emerging tensegrity system, modulating local stiffness and facilitating Poisson-effect-driven lamination. Finally, lysyl oxidase (LOX) catalyzes covalent cross-linking between these densely packed fibers (Kagan & Li, 2003), ensuring irreversible stabilization of the resulting distinct layers (Figure 3).

The synchronized clarification of multiple retroperitoneal fasciae near gestational week 20 strongly suggests a shared mechanobiological determinant. Within this framework, the Poisson effect operates as a unifying principle. The stabilizing kidney generates early local tension, forming the inner renal fascial layer at weeks 10–12. As the overall body framework integrates near week 20, multiaxial tension induces orthogonal Poisson compression, generating the multilayered outer posterior renal fascia. This 10-week lag indicates that early fixation incorporates the viscera into the developing tension network, while definitive lamination occurs only once the systemic tension field reaches a critical threshold. (Figure 3 near here)

### 4.2. Systemic Tension Field and Poisson Effect-Driven Lamination

Confined between stiffening skeletal anchors and an inextensible cutaneous envelope, the retroperitoneal mesenchyme experiences a sharp rise in multiaxial tension. In accordance with the physics of the Poisson ratio, this stretching forces orthogonal compression within the tissue, directly driving the abrupt lamination of the outer posterior renal fascia.

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



**Figure 3. Mechanobiological model illustrating how the 20-week systemic tension field produces orthogonal Poisson-effect compression, driving laminar 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). This creates 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. This ‘Poisson effect’ forces disorganized collagen fibers into a dense, laminated planar arrangement. (C) Irreversible Fixation via Poroelastic Compaction and Cross-linking: The mechanical compression drives

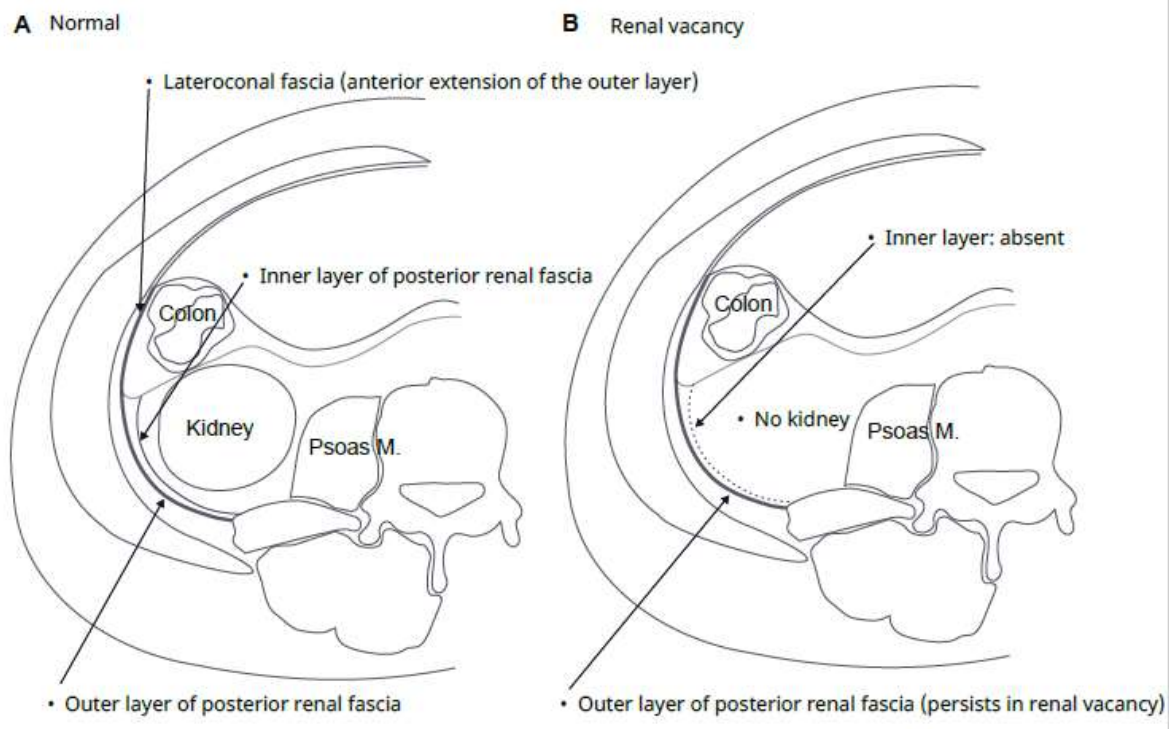
interstitial fluid out of the extracellular matrix (exudation; blue droplets). The functional maturation of the lymphatic system (green) at this stage ensures the permanent removal of this fluid, leading to poroelastic compaction. Finally, LOX-mediated covalent cross-linking chemically stabilizes the approximated collagen fibers. This transition from a transient fluid state to a definitive solid-state laminae completes the formation of the adult-type fascia.

This static tension network is powerfully amplified by a biomechanical positive-feedback loop driven by fetal movement. Before gestational week 15, the highly compliant fetal skeleton cannot efficiently transmit muscular force (Nowlan, 2015). However, as the vertebral column ossifies (Bagnall et al., 1977) and the iliac flare expands toward week 20 (Baumgart et al., 2018), these stiffened structures provide the mechanical anchors essential for efficient muscular contraction. Consequently, global fetal body movements—particularly coordinated kicks—increase dramatically in both frequency and transmitted force after week 20 (de Vries et al., 1982; Patrick et al., 1982). These changes generate rhythmic, dynamic traction on the retroperitoneal mesenchyme. Dynamic tension spikes are thought to synergize with static volumetric expansion to actively align fibroblasts and accelerate the poroelastic fluid exudation required for definitive fascial lamination.

#### 4.3. Terminology and Clinical Relevance

To avoid terminological ambiguity, the posterior structure is defined throughout this paper as the “outer layer of the posterior renal fascia,” and the lateroconal fascia is treated as its continuous anterior extension (Figure 4). Historically, Congdon et al. (1941) defined the lateroconal fascia as the lateral fusion site of the anterior and posterior renal fasciae, whereas Matsubara (2009) used the term to encompass both the outer layer of the posterior renal fascia and its anterior extension. (Figure 4 near here) (Figure 5 near here)

Clinically, this mechanobiological framework provides a principled explanation for the avascular dissection planes exploited in oncological surgery. Kinugasa et al. (2008) elegantly demonstrated that optimal retroperitoneal dissection should proceed along specific multilayered fascial boundaries dictated by embryological architecture. Furthermore, recent anatomical studies optimizing complete mesocolic excision (CME) advocate dissecting specifically along the interfascial plane between the parietal peritoneal fascia and the prerenal fascia (Wedel et al., 2022). The present model reveals that these empirically established surgical planes correspond precisely to the tension-aligned laminae generated by poroelastic consolidation. Recognizing these planes as mechanically derived structures rather than mere fusion vestiges provides a rigorous anatomical and scientific basis for standardizing embryologically sound surgical dissection. Indeed, pathological high-pressure fluid accumulation acts as a hydraulic separator, re-infiltrating the ECM and reopening the orthogonal cleavage planes originally established by the Poisson effect—a clinical phenomenon initially suggested through radiological observations (Feldberg, 1983), experimentally demonstrated (Raptopoulos et al., 1986), subsequently conceptualized as interfascial planes (Molmenti et al., 1996), and later systematized for clinical diagnostics (Ishikawa et al., 2006).



**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 the 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 ~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.

#### 4.4. Resolution of the Historical Controversy Regarding the Bilaminar Renal Fascia

Gerota’s original illustration (1895) depicted the renal fascia as a bilaminar structure, yet the **full significance** of this arrangement went unrecognized for nearly a century. A renewed appreciation emerged only in the mid-1980s, when Raptopoulos et al. (1986) performed systematic imaging evaluation and demonstrated that the posterior renal fascia comprises two layers rather than a single sheet. Subsequently, in a companion anatomical study, Marks et al. (1986) reported a striking correspondence between these imaging findings and Gerota’s original depiction, praising the anatomical accuracy of the century-old drawings. This unexpected concordance prompted a fundamental reconsideration of the anatomical complexity of this region and ultimately established a modern imaging-based framework that classical gross anatomy had failed to capture.

**Even after this anatomical clarification, the embryological explanation for the bilaminar fascia remained trapped in classical paradigms.** Classical anatomical studies—most prominently that of Tobin (1944)—**had concluded** that renal agenesis may be associated with complete absence of the renal fascia, reinforcing the long-held assumption that fascial formation depends strictly on the presence of the kidney. Subsequent fetal studies further clarified that the two layers arise from distinct developmental mechanisms (Matsubara et al., 2009), suggesting that the outer layer may not simply represent passive elongation of the inner, organ-dependent layer.

The radiological cohort of renal absence cases assembled in this study (n = 3) provides a decisive subtraction experiment for directly testing this historical controversy. When the kidney—the presumed source of local radial tension—is absent from the developmental equation, the inner layer predictably fails to form. Nevertheless, a continuous outer fascial layer was preserved into adulthood in all cases. This finding reconciles the long-standing discrepancy between classical anatomy and modern imaging: the inner layer is organ-dependent, whereas the outer layer is a tension-derived, system-generated structure that forms independently of the kidney’s 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 near gestational week 20.

#### 4.5. The Square-Cube Law and the Emergence of a Fetal Tensegrity-like System

Before gestational week 15, the fetal trunk behaves as a pressure-driven structure. Viscera and fluid are enclosed within a compliant body wall, and internal pressure distributes nearly isotropically, allowing overall shape to be maintained without specialized fascial planes to bear load.

As fetal size increases, however, the square-cube law imposes a fundamental mechanical constraint. Approximating the fetal trunk as a sphere of radius  $r$ , the surface-area-to-volume ratio is  $3/r$ . The inverse—the internal mechanical load borne per unit area of the outer envelope—therefore scales as  $r/3$ . Based on standard fetal growth curves (Hadlock et al., 1991), this internal load approximately triples between gestational weeks 12 and 20.

As  $r/3$  rises, the simple strategy of a compliant “hydrostatic bag” becomes mechanically untenable. To prevent mechanical failure of the boundary, the fetal epidermis undergoes progressive

keratinization and stratification by gestational weeks 18–20 (Hardman et al., 1999), transforming the previously compliant body wall into an inextensible cutaneous envelope. This rigidified outer boundary actively resists the surging internal volume, generating profound systemic hoop stress.

Simultaneously, the internal anchors of the system gain stiffness. Vertebral ossification (Bagnall et al., 1977), lateral expansion of the iliac wings (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 stiffening axial skeleton and the expanding abdominal wall.

In this configuration, the retroperitoneal mesenchyme functions as a transient fluid support strut. Rich in glycosaminoglycans and interstitial fluid, this tissue initially resists deformation through fluid incompressibility. As multiaxial tension rises near gestational week 20, however, Poisson effect orthogonal compression forces fluid from the tissue 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 consolidates into tension-bearing fibrous laminae.

Near gestational week 20, therefore, the fetal trunk undergoes a mechanical regime shift from an isotropic, pressure-driven continuum to an anisotropic, tension-dominated network. This emerging architecture is **tensegrity-like** in that discrete compression-bearing elements—namely, the ossifying vertebrae, expanding iliac wings, and the inextensible cutaneous envelope—interact with a continuous tension network formed by the retroperitoneal mesenchyme. **While not proposing a strict tensegrity model, this analogy effectively clarifies how anisotropic force transmission becomes organized at the whole-body scale**, providing the necessary mechanical environment for definitive fascial lamination. The outer layer of the posterior renal fascia represents one of the clearest anatomical manifestations of this transition: its formation reflects not the presence of the kidney, but rather the rise in internal load driven by  $r/3$  and the emergence of the system-wide tension network.

#### 4.5.1. Embryological Prerequisites Derived from Pelvic Morphogenesis

Building on this structural remodeling, a recent embryological study by Senevirathne et al. (2025) provides an indispensable upstream framework for interpreting the present mechanobiological model. Their work demonstrates that the human ilium acquires its characteristically broad, laterally flared geometry through two key developmental innovations: ectopic reorientation of the iliac growth plate and a laterally biased heterochronic ossification sequence. These morphogenetic changes, absent in other primates, establish the fundamental geometric prerequisites for the lateral traction vectors that become mechanically relevant in the second trimester.

Integrating these findings with the temporal data of Verbruggen and Nowlan (2017)—who showed that the most rapid phase of iliac expansion occurs precisely in the second trimester—reveals a coherent developmental hierarchy. The genetic and cellular mechanisms described by Senevirathne et al. specify pelvic shape and material distribution, while the present model identifies the moment at which this genetically specified geometry becomes mechanically integrated into the systemic tension network: approximately gestational week 20. From this perspective, the week-20 convergence represents the point at which “structure enables function”: the stiffened vertebrae and expanded iliac flare begin efficiently transmitting fetal movement forces, amplifying both static volumetric tension and dynamic traction. The findings of Senevirathne et al. are therefore not in competition with the present mechanobiological model; rather, they provide its indispensable morphogenetic foundation, clarifying how **this laterally flared pelvic geometry** provides the essential biomechanical anchors for Poisson effect-driven retroperitoneal fascial lamination.

#### 4.6. Lymphatic Maturation, Poroelastic Consolidation, and Cross-Linking

Although the Poisson effect specifies orthogonal compression, irreversible fixation requires fluid evacuation and enzymatic stabilization. The fetal lymphatic system acquires functional whole-body

drainage capacity between gestational weeks 14 and 16 (Bekker et al., 2005). When Poisson compression occurs near week 20, mature lymphatic vessels facilitate poroelastic fluid exudation, bringing dispersed collagen fibers into extreme physical proximity. This proximity constitutes the spatial prerequisite for LOX-mediated covalent cross-linking (Kagan & Li, 2003), chemically stabilizing the lamellar architecture and accounting for the irreversibility of fascial consolidation.

#### 4.7. Implications for Comparative Anatomy

Although the Poisson effect is a **universal property of vertebrate soft tissue** (Exposito et al., 2010), the highly specialized multilayered structure of the human retroperitoneum appears intrinsically linked to hominin bipedalism. Quadrupedal mammals lack the characteristic iliac flare required for upright posture, suggesting that this specific lateral traction vector constitutes a geometric prerequisite for human-type fascial lamination. A detailed comparative biomechanical analysis, however, lies beyond the scope of this study.

#### 4.8. Limitations and Future Directions

Although the present mechanobiological model is consistent with histological observations and supported by the cross-sectional radiological subtraction experiment described here, it remains a theoretical integration. Empirical validation—including direct *in vivo* quantification of retroperitoneal tissue stiffness and tension vectors using high-resolution fetal MRI or ultrasound elastography—will be essential to demonstrate the proposed tension network.

## 5. Conclusion

In conclusion, the systemic tension field converging near gestational week 20 constitutes the principal mechanobiological stimulus that sculpts the outer layer of the posterior renal fascia through Poisson effect orthogonal compression. The natural subtraction experiment conducted in adults with renal absence provides compelling evidence of this structural autonomy and definitively resolves a 150-year-old anatomical discrepancy regarding retroperitoneal fascial formation. By identifying the week-20 systemic tension field as the mechanobiological trigger of fascial lamination, this study offers a unifying framework integrating fetal biomechanics, developmental anatomy, and clinical imaging.

**Table 1.** Chronological integration of retroperitoneal fascial development and associated mechanobiological events.

Gestational Age	Anatomical / Biomechanical Event	Mechanobiological Significance	Key References
Weeks 10–12	<ul style="list-style-type: none"> <li>• Early pancreatic fixation to the posterior wall</li> <li>• Circumferential appearance of the inner renal fascial layer</li> <li>• Morphogenetic blueprint of iliac flare geometry established via cartilaginous migration</li> </ul>	<p>Local tension and spatial vector priming: viscera are asynchronously integrated into the nascent tension network.</p> <p>Pelvic cartilage establishes future posterolateral traction vectors but remains too compliant to generate systemic tension.</p>	<p>Cho et al. (2009); Matsubara et al. (2009); Senevirathne et al. (2025)</p>
Weeks 14–16	<ul style="list-style-type: none"> <li>• Functional maturation of the fetal lymphatic system (connection to terminal veins)</li> </ul>	<p>Physiological preparation: systemic drainage capacity required for poroelastic fluid exudation during subsequent Poisson effect compression is</p>	<p>Berger (1999); Bekker et al. (2005)</p>

Gestational Age	Anatomical / Biomechanical Event	Mechanobiological Significance	Key References
Weeks 18–20	<ul style="list-style-type: none"> <li>• Progressive vertebral ossification</li> <li>• Epidermal keratinization and completion of the inextensible cutaneous envelope</li> <li>• Onset of exponential volumetric growth (square-cube law)</li> </ul>	<p>established.</p> <p>Dynamic priming and the square-cube law: the vertebral column stiffens into a central pillar; volumetric growth outpaces surface area, generating immense outward pressure that collides with the maturing epidermis to produce powerful whole-body hoop stress.</p>	<p>Bagnall et al. (1977); Singh &amp; Archana (2008); Hadlock et al. (1991)</p>
~Week 20 (temporal nexus)	<ul style="list-style-type: none"> <li>• Trunk stiffening: cumulative 3D pelvic expansion and ossification reach a mechanical threshold</li> <li>• Fascial lamination: multilayered architecture of the Toldt fascia, posterior pancreatic fascia, and outer posterior renal fascia simultaneously becomes definitive</li> </ul>	<p>Temporal and mechanical trigger with Poisson effect: once anchors stiffen into rigid levers, growth forces are transmitted rather than dissipated. The resulting tension spike induces orthogonal compression (Poisson effect), forcing fluid exudation and subsequent LOX cross-linking that completes fascial lamination.</p>	<p>Baumann (1945); Cho et al. (2009); Matsubara et al. (2009); Verbruggen &amp; Nowlan (2017); (present model)</p>

**Table 2.** System-wide manifestations of the biomechanical convergence near gestational week 20.

Anatomical System	Event at ~Week 20	Biomechanical Significance	Key References
Skeletal system	Vertebral ossification	Rigid central pillar for tension transmission	Bagnall et al. (1977)
	Iliac flare expansion/ossification	Establishment of lateral levers for systemic tension	Baumgart et al. (2018); Senevirathne et al. (2025)
Cutaneous envelope	Epidermal keratinization	Inextensible shell generating whole-body hoop stress	Hardman et al. (1999)
Trunk growth	Exponential volumetric increase	Square-cube law drives systemic tension	Hadlock et al. (1991)
Musculoskeletal movement	Intensification of fetal kicking and gross body movement	Dynamic tension spikes actively align fibroblasts and accelerate poroelastic consolidation	Nowlan (2015); de Vries et al. (1982); Patrick et al. (1982)
Respiratory physiology	Surge in fetal breathing movements	Rhythmic loading reinforces tension network	Nowlan (2015)

Anatomical System	Event at ~Week 20	Biomechanical Significance	Key References
	(FBM)		
	Increasing thoracic rigidity	Thorax becomes a stable mechanical frame	Bagnall et al. (1977); Verbruggen & Nowlan (2017)
	Onset of surfactant production	Alveolar stabilization → stable FBM	Avery & Fletcher (1974); Clements (1957)
Lymphatic system	Maturation of whole-body drainage capacity	Facilitates irreversible poroelastic consolidation	Bekker et al. (2005)
Fascial structures	Lamination of the Toldt fascia	Reflects systemic tension threshold	Baumann (1945)
	Lamination of the posterior pancreatic fascia	Occurs after the 10-week lag	Cho et al. (2009)
	Lamination of the outer posterior renal fascia	Multiaxial tension + Poisson effect compression	Matsubara et al. (2009)
Peripheral nervous system	Compaction of the sciatic nerve sheath	Pelvic traction induces Poisson compression	Pummi et al. (2004)

**Table 3.** Clinical characteristics and quantitative radiological findings of the renal absence 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 ( $\Delta$ )
1	53F	True left renal agenesis	“Pancake” (recumbent)	1.49 mm	1.88 mm	-0.39 mm
2	47F	Severe left renal dysplasia/involution (renal remnant)	Normal	1.46 mm	1.82 mm	-0.36 mm
3	89M	True left renal agenesis	“Pancake” (recumbent)	1.62 mm	Excluded*	N/A
			Mean	1.52 mm	1.85 mm	-0.38 mm

\*The contralateral side of Case 3 was excluded from the mean calculation owing to local contralateral inflammation affecting the fascial plane.

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**Data Availability:** The radiological data supporting the findings of this study are restricted to protect patient privacy, but are available from the corresponding author upon reasonable request. All other information is based on previously published literature.

**Ethics Statement:** This retrospective study received approval from the Institutional Review Board (IRB) of Gakkentoshi Hospital (approval number: GT-R6-07-12-1). Written informed consent was waived given the retrospective nature of the study, and an opt-out mechanism was made available through the hospital's official website in accordance with national ethical guidelines.

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## References

- Avery ME, Fletcher BD (1974) *The Lung and Its Disorders in the Newborn Infant*. 3rd ed. WB Saunders, Philadelphia.
- Bagnall KM, Harris PF, Jones PR (1977) The appearance of ossification centers in the human fetal spine. *J Anat* 124, 791–802.
- Baumann JA (1945) Développement et anatomie de la loge rénale chez l'homme. *Acta Anat (Basel)* 1, 15–65.
- Baumgart M, Wiśniewski M, Grzonkowska M, Badura M, Biernacki M, Siedlecki Z, et al. (2018) Quantitative anatomy of the ilium's primary ossification center in the human fetus. *Surg Radiol Anat* 40, 1047–1054.
- Bekker MN, van den Akker NM, de Ruiten MC, Gittenberger-de Groot AC (2005) The human fetal lymphatic system: a morphological study. *Anat Embryol (Berl)* 210, 167–175.
- Berger G (1999) First-trimester nuchal translucency and the fetal lymphatic system. *Ultrasound Obstet Gynecol* 13, 12–17.
- Carlson BM (2014) *Human Embryology and Developmental Biology*. 5th ed. Elsevier, Philadelphia.
- Chau YY, Bandiera R, Serrels A, Saunders PTK, Hastie ND (2014) Visceral adipose tissue formation depends on mesothelial cells. *Development* 141, 2752–2762.
- Cho BH, Kimura W, Song CH, Fujimiya M, Murakami G (2009) Investigation into the embryological development of the fasciae used as the basis for pancreatoduodenal mobilization. *J Hepatobiliary Pancreat Surg* 16, 824–831.
- Clements JA (1957) Surface tension of lung extracts. *Proc Soc Exp Biol Med* 95, 170–172.
- Coffey JC, O'Leary DP (2016) The mesentery: structure, function, and role in disease. *Nat Rev Gastroenterol Hepatol* 13, 477–486.
- Congdon ED, Edson JN (1941) The cone of renal fascia in the adult white male. *Anat Rec* 80, 289–313.
- de Vries JJ, Visser GH, Prechtl HF (1982) The emergence of fetal behaviour. I. Qualitative aspects. *Early Hum Dev* 7, 301–322.
- Exposito JY, Lethias C, Ricard-Blum F (2010) The fibrillar collagen family. *Structure* 18, 160–170.
- Feldberg MAM (1983) *Computed Tomography of the Retroperitoneum: An Anatomical and Pathological Atlas with Emphasis on the Fascial Planes*. Martinus Nijhoff, Boston.
- Fung YC (1990) *Biomechanics: Motion, Flow, Stress, and Growth*. Springer-Verlag, New York.
- Gerota D (1895) Beiträge zur Kenntniss des Befestigungsapparates der Niere. *Arch Anat Entwicklungsgesch* 265–285.
- Hadlock FP, Harrist RB, Martinez-Poyer J (1991) In utero analysis of fetal growth: a sonographic weight standard. *Radiology* 181, 129–133.
- Han J, Lee JE, Jin J, Lim JS, Oh N, Kim K, et al. (2011) The origin and development of mesenteric adipose tissue. *Cell Metab* 13, 376–388.
- Hardman MJ, Sisi P, Banbury DN, Byrne C (1999) Barrier formation in the human fetus is patterned. *J Invest Dermatol* 113, 1106–1113.

- Hayes MA (1950) Abdominopelvic fasciae. *Am J Anat* 87, 119–161.
- Humphrey JD (2003) Continuum biomechanics of soft biological tissues. *Proc Math Phys Eng Sci* 459, 3–46.
- Ingber DE (2003) Tensegrity I: cell structure and hierarchical systems biology. *J Cell Sci* 116, 1157–1173.
- Ishikawa K, Idoguchi K, Tanaka H, Tohma Y, Ukai I, Watanabe H, et al. (2006) Classification of acute pancreatitis based on retroperitoneal extension: application of the concept of interfascial planes. *Eur J Radiol* 60, 445–452.
- Ishikawa K, Nakao S, Nakamuro M, Huang TP, Nakano H (2016) The retroperitoneal interfascial planes: current overview and future perspectives. *Acute Med Surg* 3, 219–229.
- Kagan HM, Li W (2003) Lysyl oxidase: properties, specificity, and biological roles inside and outside of the cell. *J Cell Biochem* 88, 660–672.
- Kinugasa Y, Niikura H, Murakami G, Suzuki D, Saito S, Tatsumi H, et al. (2008) Development of the human hypogastric nerve sheath with special reference to the topohistology between the nerve sheath and other prevertebral fascial structures. *Clin Anat* 21, 558–567.
- Lakes R (1991) Deformation mechanisms in negative Poisson's ratio materials: structural aspects. *J Mater Sci* 26, 2287–2292.
- Marks SC Jr, Raptopoulos V, Kleinman P, Snyder M (1986) The anatomical basis for retrorenal extensions of pancreatic effusions: the role of the renal fasciae. *Surg Radiol Anat* 8, 89–97.
- Matsubara A, Kinugasa Y, Murakami G, Suzuki D, Fujimiya M, Sugihara K (2009) Development of the lateroconal fascia in human fetuses. *Cells Tissues Organs* 190, 286–296.
- Molmenti EP, Balfe DM, Kanterman RY, Bennett HF (1996) Anatomy of the retroperitoneum: observations of the distribution of pathologic fluid collections. *Radiology* 200, 95–103.
- Mow VC, Kuei SC, Lai WM, Armstrong CG (1980) Biphasic creep and stress relaxation of articular cartilage in compression: theory and experiments. *J Biomech Eng* 102, 73–84.
- Nowlan NC (2015) Biomechanics of fetal movement. *Eur Cell Mater* 29, 1–21.
- Partin AW, Dmochowski RR, Kavoussi LR, Peters CA, eds. (2020) *Campbell-Walsh-Wein Urology*. 12th ed. Elsevier, Philadelphia.
- Patrick J, Campbell K, Carmichael L, Natale R, Richardson B (1982) Patterns of gross fetal body movements over 24-hour observation intervals during the last 10 weeks of pregnancy. *Am J Obstet Gynecol* 142, 363–371.
- Provenzano PP, Vanderby R (2006) Collagen fibril morphology and organization: implications for force transmission in ligament and tendon. *Matrix Biol* 25, 71–84.
- Pummi K, Heape AM, Grenell S, Peltonen J, Peltonen S (2004) Tight junction proteins ZO-1, occludin, and claudins in developing and adult human perineurium. *J Histochem Cytochem* 52, 1037–1046.
- Raptopoulos V, Kleinman PK, Marks S, Snyder M, Silverman PM (1986) Renal fascial pathway: posterior extension of pancreatic effusions within the anterior pararenal space. *Radiology* 158, 367–374.
- Senevirathne G, Fernandopulle SC, Richard D, Baumgart SL, Christensen AL, Fabbri M, et al. (2025) The evolution of hominin bipedalism in two steps. *Nature* 645, 952–963.
- Singh G, Archana G (2008) Unraveling the mystery of vernix caseosa. *Indian J Dermatol* 53, 54–60.
- Tobin CE (1944) The renal fascia and its relation to the transversalis fascia. *Anat Rec* 89, 295–311.
- Toldt C (1879) Bau und Wachstumsveränderungen der Gekröse des menschlichen Darmkanales. *Denkschr Akad Wiss Wien* 41, 1–56.
- Verbruggen SW, Nowlan NC (2017) Ontogeny of the human pelvis. *Anat Rec (Hoboken)* 300, 643–652.
- Wedel T, Heimke M, Fletcher J, Miskovic D, Benz S, Stelzner S, et al. (2022) The retrocolic fascial system revisited for right hemicolectomy with complete mesocolic excision based on anatomical terminology: do we need the eponyms Toldt, Gerota, Fredet and Treitz? *Colorectal Dis* 24, 1612–1622.
- Zuckerkindl E (1883) Ueber den Fixationsapparat der Nieren. *Med Jahrb* 1883, 59–67.

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