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*Review*

# Rigor Mortis Revisited: Does Fascia Contribute to Post-Mortem Stiffness?

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## Abstract

Rigor mortis has conventionally been explained as the consequence of adenosine triphosphate depletion and persistent actin-myosin cross-bridge formation within skeletal muscle fibres. More than two centuries after Nysten's description of post-mortem rigidity, the explanatory framework has remained overwhelmingly muscle-centred. This hypothesis-generating narrative review examined whether fascia may contribute to post-mortem rigidity alongside skeletal muscle. Evidence from living tissues and experimental models was synthesised to identify three candidate processes: altered calcium-sensitive myofibroblast activity, changes in extracellular matrix hydration and viscoelastic behaviour, and loss of distributed force transmission across the fascial-muscular continuum. Their specific contributions after death have not been established. Regional variation in fascial density, hydration, cellular composition and loading history may help explain uneven onset, variable timing and non-linear resolution. The proposed fascia-inclusive model did not replace the established actomyosin account. It broadened the anatomical field of enquiry and identified testable questions for direct forensic and anatomical investigation.

**Key words:** post-mortem physiology; calcium signalling; tensegrity; forensic science; anatomy; thanatology; mechanotransduction; extracellular matrix; myofibroblasts; post-mortem interval.
Abbreviations: ATP, adenosine triphosphate; ECM, extracellular matrix.

## 1. INTRODUCTION

### 1.1 Challenging the Muscle-Only Paradigm

Rigor mortis has long been explained in relatively simple biochemical terms. Following death, adenosine triphosphate (ATP) depletion prevents the dissociation of actin–myosin cross-bridges, resulting in progressive joint and muscular stiffening increasing resistance to passive movement (Saukko and Knight, 2004; Shedge and Zubair, 2026). Contemporary forensic reviews and standard reference texts, including Spitz and Fisher's Medicolegal investigation of Death, continue to frame rigor mortis almost exclusively as a muscular phenomenon, with little or no consideration given to fascia or connective tissue contributions (Spitz and Spitz, 2006). This omission does not demonstrate rejection of a broader interpretation but indicates that the question has not yet been examined directly. Consequently, this hypothesis-generating narrative review offers a systems-informed conceptual synthesis rather than an experimental demonstration. Developments within fascia science over the last two decades have substantially altered anatomical and physiological understanding through advances in dissection methodology, histology, imaging and mechano-

biological research (Schleip et al., 2005; Stecco et al., 2011).

Research within this field indicates that fascia participates in mechanotransduction, tissue metabolism and force transmission. Some fascial tissues contain contractile cells and possess intrinsic biochemical and cellular dynamics that contribute to structural integrity (Schleip et al., 2005). Considered within a post-mortem context, these findings raise a question that extends across anatomy, physiology and forensic science: might connective tissues contribute to the stiffness currently attributed almost exclusively to skeletal muscle?

Drawing upon contemporary fascia research, this review considers whether rigor mortis may be more appropriately investigated as a whole-body phenomenon involving both skeletal muscle and the fascial tissues that surround, permeate and support it (Stecco et al., 2011).

## 2. SYSTEMS PERSPECTIVE: FASCIA AND WHOLE-BODY STRUCTURAL COHERENCE

This paper introduces a straightforward proposition: fascia may contribute to the stiffness observed following death. Anatomical examination has long encountered connective tissue tautness. However, it has not been systematically incorporated into rigor mortis models or tested as an independent contributor to post-mortem stiffness. The proposed interpretation broadens the conventional account without replacing it. Rigor mortis may involve both actomyosin cross-bridge formation and changes within the wider fascial continuum as ionic regulation and active biological maintenance cease (Kirkness et al., 2025).

This perspective places established muscle biochemistry within a continuous, multi-tissue anatomy. During life, cells and extracellular matrix respond to changing mechanical conditions through mechanotransduction (Sukharev and Corey, 2004). Following death, that active regulation ceases. Fascial tissues then change under the influence of ionic failure, fluid redistribution and their existing material properties. This does not establish a fascial mechanism of rigour. It identifies a tissue domain that has not yet been measured directly. Fascia is not mechanically inert. It senses load, adapts to use and contributes to force transmission (Zügel et al., 2018). Calcium signalling and tissue geometry also influence how structure is maintained across scales (Kirkness et al., 2025). Once energetic regulation ceases, fascial tissues may retain or redistribute tension according to their regional architecture and viscoelastic properties (Kodama et al., 2023). Whether these changes measurably alter rigor mortis remains unknown.

## 3. COHERENCE, MECHANOTRANSDUCTION AND THE LOSS OF ORGANISATION

Within anatomy and physiology, coherence describes the capacity of tissues to remain in mechanical and biochemical relationship (van der Wal, 2009). Fascia contributes to this relationship by transmitting force across multiple spatial scales (Huijing, 2009). Its behaviour arises through continuing interaction among cells, extracellular matrix and the forces passing through them (Noble, 2006). This organisation is actively maintained during life. It cannot be assumed to persist unchanged after death. The systems comparison used here is instructive rather than literal. Its relevance is limited to one principle: organised biological behaviour depends upon continuing exchange and regulation. Following death, ionic gradients fail and active cellular regulation diminishes. Tissue behaviour then becomes increasingly governed by passive material properties. This paper does not redefine rigor mortis as a chronological marker or propose a replacement post-mortem timeline. It asks whether the loss of regulation across the fascial-muscular continuum contributes to regional asymmetry, variable onset or non-linear resolution.

## 4. REVIEW APPROACH AND SCOPE

The observations discussed throughout this manuscript are qualitative, illustrative, and hypothesis-generating in nature. They are informed by anatomical dissection, fascia-focused investigation, and forensic observation. No claim is made regarding standardised scoring systems, inter-observer validation, or controlled post-mortem experimentation. The purpose of this narrative review is conceptual, not experimental, offering a theoretical framework intended to stimulate further anatomical and forensic investigation. The distinction between the established muscle-centred account, the proposed fascia-inclusive interpretation and the evidence required to evaluate it is summarised in Table 2 before the candidate mechanisms are examined in detail. Narrative reviews carry recognised limitations. The evidence considered here comes from different tissues, experimental models and research settings. The literature was selected for its relevance to the proposed mechanisms rather than through a registered systematic-review protocol. Publication bias and selective interpretation therefore cannot be excluded. Most importantly, evidence from living or cultured tissues cannot be transferred directly to the early post-mortem state. The present synthesis should be read as a basis for testing, not as evidence that fascia has already been shown to contribute to rigor mortis. Proposed sequence for determining whether fascia changes stiffness after death is presented in Figure 1.

## 5. CANDIDATE MECHANISMS OF FASCIAL STIFFENING

The following section summarises established biological properties of fascial tissues demonstrated in living systems and experimental models, without presupposing their exact behaviour in the post-mortem state (Ahmed et al., 2019).

**Table 1.** Candidate fascial processes relevant to post-mortem stiffening.

| Candidate process | Potential relevance | Citations |
|---|---|---|
| Myofibroblast contractility | Calcium-sensitive tension generation demonstrated in living and experimental tissues; post-mortem persistence requires testing | (Klingler et al., 2014; Schleip and Klingler, 2019; Zügel et al., 2018) |
| ECM hydration and material change | Changes in collagen–proteoglycan organisation, hydration and viscoelasticity may alter tissue stiffness | (Lampi and Reinhart-King, 2018; Loomis et al., 2022; Pirri et al., 2021; Zügel et al., 2018) |
| Ionic dysregulation | Loss of ionic homeostasis may alter cellular and extracellular tissue behaviour | (Schleip and Klingler, 2019; Zügel et al., 2018) |
| Loss of distributed force transmission | Failure of active regulation may redistribute or release pre-existing tension across the fascial–muscular continuum | (Ingber, 2008; Sharkey, 2021; Zügel et al., 2018) |

### 5.1 Active Contractility and Myofibroblasts

Fascial tissues form connective tissue specialities along a continuum (see Fig 2). Some contain myofibroblasts capable of calcium-dependent contractile activity that can influence tissue stiffness over timescales ranging from minutes to hours in living and experimental tissues (Fede et al., 2021; Hinz, 2007; Klingler et al., 2014; Schleip and Klingler, 2019; Tomasek et al., 2002; Zügel et al., 2018). Following death, ATP availability declines and physiological calcium regulation progressively fails. These events are central to skeletal-muscle rigor because calcium-pump failure favours actomyosin binding while ATP depletion prevents cross-bridge release. The corresponding sequence has not been established in fascial myofibroblasts. Fibroblasts can be cultured from some human tissues obtained after death, including dura and scalp (Bliss et al., 2012). This demonstrates that somatic death does not produce simultaneous cellular death throughout the body. It does not demonstrate continued contraction in situ. Cellular survival, capacity for culture and the ability to generate mechanically relevant tension are different questions. No defined post-mortem contractile window for fascial myofibroblasts was identified. Direct studies must therefore compare ATP availability, intracellular calcium handling, membrane integrity and force generation in fascia and skeletal muscle over the same post-mortem intervals. Temperature, tissue depth and refrigeration would need to be controlled. Until such evidence is available, active myofibroblast contraction should remain a candidate mechanism rather than an assumed component of rigor mortis.

### 5.2 Extracellular Matrix Remodelling and Material Change

Following death, ATP depletion is accompanied by calcium-pump failure and progressive disruption of ionic homeostasis in skeletal muscle. Fascia occupies the same changing biochemical environment. Its ECM behaviour depends upon composition, hydration and structural organisation (Yahia et al., 1993). Post-mortem fluid redistribution may therefore alter fascial stiffness and viscoelastic behaviour.

The collagen cross-linking evidence cited here concerns bone and disease-related matrix biology, not early post-mortem fascia (Paschalis et al., 2001). It identifies measurable matrix properties. It does not show that new collagen or proteoglycan cross-links form during rigor mortis. Comparisons with fibrosis, ageing, diabetes and chronic loading (although these occur on a different time-scale) may help to identify candidate variables, but those conditions cannot be assumed to reproduce the post-mortem state (Lampi and Reinhart-King, 2018; Loomis et al., 2022). Direct measurement remains necessary.

### 5.3 Tensegrity and Force Transmission

Fascial tissues represent a continuous embryologically derived connective tissue network capable of transmitting

**Table 2.** From the established muscle-centred account to a fascia-inclusive research framework.

| Domain | Established muscle-centred account | Fascia-inclusive hypothesis | Measurement required |
|---|---|---|---|
| ATP and calcium | ATP depletion prevents actin–myosin dissociation. | Loss of ionic regulation may also alter fascial cellular and extracellular behaviour. | Regional ATP, calcium and tissue-stiffness measurements. |
| Tissue responsible for stiffness | Rigidity is attributed predominantly to skeletal muscle. | Fascia may modify resistance to passive movement alongside muscular rigor. | Simultaneous muscular and fascial elastography or mechanical testing. |
| Cellular tension | Persistent cross-bridge formation stiffens muscle fibres. | Fascial myofibroblast viability or contractility may differ from skeletal-muscle behaviour after death. | Matched post-mortem assays of viability, ATP, calcium handling and force generation. |
| Extracellular matrix | The ECM is not central to the conventional explanation. | Hydration, proteoglycan behaviour and viscoelastic change may influence stiffness. | Serial measurement of water content, hyaluronan behaviour and matrix stiffness. |
| Force transmission | Considered mainly within individual muscles. | The fascial–muscular continuum may redistribute or release pre-existing tension. | Regional deformation and force-transmission mapping. |
| Regional variability | Attributed to temperature, exertion, metabolic state and muscle mass. | Fascial architecture, hydration and cellular composition may provide additional variation. | Comparison across anatomical regions, individuals and post-mortem intervals. |
| Resolution | Rigidity resolves through proteolysis of muscle proteins. | Matrix degradation and fluid redistribution may influence the pattern of resolution. | Parallel biochemical and material testing over time. |

and distributing mechanical forces throughout the body (Pirri et al., 2021). Through this continuity, fascia contributes to whole-body structural coherence by balancing tension and compression across multiple regions simultaneously (Sharkey, 2021). During life, force transmission reflects the combined behaviour of muscle, intramuscular connective tissues, aponeuroses, septa and surrounding fascia (Huijing, 2009). Following death, these tissues remain anatomically continuous while their active regulation changes. A fascia-inclusive contribution would therefore need to be separated experimentally from stiffness generated within muscle fibres. One approach would combine regional elastography with controlled passive movement before and after a limited fascial release. The release region should be compared with a sham incision and an untreated contralateral site. A change in passive resistance or deformation after fascial release would indicate mechanical participation by the released tissue, but would not by itself establish a cellular mechanism. Regional displacement mapping, direct force measurement and matched histological sampling would be required to distinguish passive matrix behaviour from myofibroblast activity and skeletal-muscle rigor (Gennisson et al., 2013; Huijing, 2009).

## 6. DISCUSSION AND IMPLICATIONS

### 6.1 Implications for Forensics and Anatomy

The forensic value of rigor mortis lies primarily in pattern recognition and interpretation alongside other post-mortem findings, including lividity, body temperature, scene context, and individual history. The fascia-inclusive framework proposed here is not intended to function as a competing post-mortem “clock” (Henssge and Madea, 2007). Recognition of fascia as a potential contributor to post-mortem stiffening may broaden anatomical and forensic understanding of the body after death while opening new avenues for investigation. Importantly, such a framework remains compatible with established forensic practice and classical descriptions of rigor mortis (Saukko and Knight, 2004; Shedge and Zubair, 2026; Spitz and Spitz, 2006). A fascia-inclusive interpretation may help explain longstanding observations such as uneven

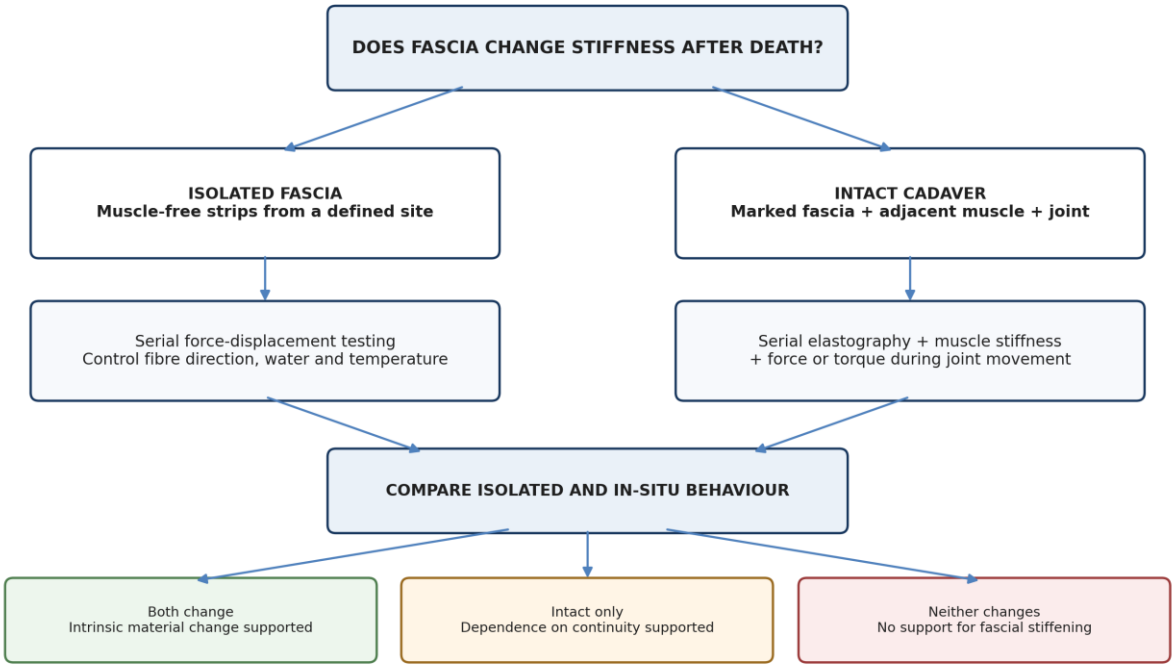


**Figure 1.** Proposed sequence for determining whether fascia changes stiffness after death. Isolated testing addresses intrinsic material change. Measurement in the intact cadaver addresses behaviour retained through anatomical continuity. Mechanistic studies follow only if a mechanical change is demonstrated.

**Table 3** provides a staged investigation of a possible fascia contribution to post-mortem stiffness.

| **Stage** | **Preparation** | **Primary measurement** | **Interpretation** |
|---|---|---|---|
| 1. Isolated fascia | Muscle-free strips from a defined site; matched dimensions and fibre direction. | Force-displacement curve at serial post-mortem intervals. | Tests whether the material stiffness of fascia changes after death. |
| 2. Repeated loading control | Paired strips tested once or repeatedly. | Change attributable to testing history. | Separates post-mortem change from preconditioning or tissue damage. |
| 3. Intact fascia | A large accessible structure such as thoracolumbar fascia. | Serial shear-wave elastography at marked sites. | Tests apparent stiffness while anatomical continuity is preserved. |
| 4. Adjacent muscle | Muscle beneath or beside the fascial site. | Muscle elastography and, where possible, ATP and temperature. | Separates fascial findings from established muscular rigor. |
| 5. Joint resistance | Standardised joint angle and movement rate. | Torque or force required through the same range. | Relates tissue measurements to the clinical sign called rigor. |

**Table 3 Cont.**

| | | | |
|---|---|---|---|
| 6. Release test | Limited fascial division with sham and contralateral controls. | Change in deformation and joint resistance. | Tests whether intact fascial continuity contributes mechanically. |
| 7. Mechanism | Only after a stiffness change is demonstrated. | Histology, cell phenotype, viability, water content and matrix chemistry. | Distinguishes cellular activity, passive material change and an effect dependent upon intact continuity. |

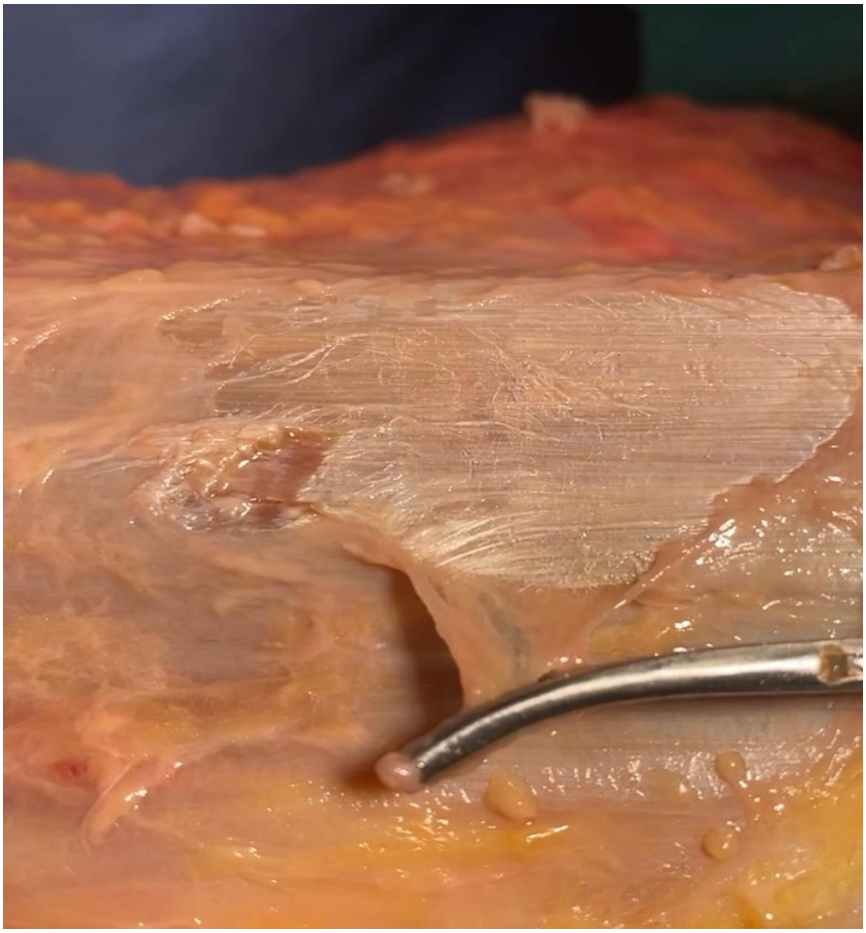

**Figure 2**. Dissection of the lateral thigh showing the organised macroscopic architectural continuum of deep fascia and its collagenous fibre arrangement. The image illustrates continuity and tension-bearing architecture. Image: John Sharkey, 2024.

regional rigidity, variability in onset, and non-linear progression of stiffening. Fascial tissues possess material properties distinct from skeletal muscle and are influenced by hydration state, extracellular matrix organisation, and connective tissue architecture (Fede et al., 2021; Kodama et al., 2023; Yahia et al., 1993).

Interaction between these properties and ATP-dependent muscular stiffening offers a testable explanation for regional and inter-individual variability observed during post-mortem examination (Shedge and Zubair, 2026). While this conceptual framework does not eliminate uncertainty in post-mortem interval estimation, it may

clarify why uncertainty persists within muscle-only models. Fascial hydration, architecture and passive material properties provide candidate influences on the overall resistance encountered during examination. Active fascial stiffening after death has not yet been demonstrated. Any interaction between connective tissue change and muscular rigidity should be treated as a testable hypothesis and not an established component of rigor mortis (Webber and Connor, 2025).

### 6.2 The Variability Challenge in Traditional Models

Forensic literature has long recognised that rigor mortis rarely follows the orderly and predictable sequence described in older manuals. In practical mortuary experience, the process frequently presents considerable variability, suggesting influences extending beyond skeletal muscle chemistry alone. Contemporary reviews emphasise that established post-mortem interval methods remain subject to substantial biological and environmental variation and should not be interpreted in isolation (Strete et al., 2025; Teixeira et al., 2025). An autopsy study by Joshi et al. (2021) likewise demonstrated variability in the onset and progression of rigor mortis. Temperature, premortem exertion, metabolic condition, body composition and perimortem activity may all alter the timing and pattern of stiffness (Shedge and Zubair, 2026). Rigor mortis consequently cannot function as a precise independent measure of the post-mortem interval. These observations do not show that fascia explains variation currently attributed to muscle. Temperature, premortem exertion, metabolic state, body composition and muscle mass remain established influences on muscular rigor. They must be measured before any additional fascial contribution can be inferred. The proposed model is therefore additive and interactive rather than alternative. Regional fascial density, hydration, cellular composition and architecture may modify resistance to passive movement after the muscular contribution and recognised environmental factors have been accounted for.

### 6.3 Fascia as an Explanatory Framework

Introducing the fascial continuum into research on rigor mortis provides an additional anatomical framework for investigating variation. It does not convert unexplained variation into evidence for fascia. If muscular and fascial tissues contribute differently across time and anatomical region, variability would be expected. That proposition can be tested only by measuring both tissue domains under the same conditions (Schleip et al., 2005; Shedge and Zubair, 2026). If apparent fascial stiffness rises and later falls, the same measurements used during onset can determine whether this follows water redistribution, loss of force transmitted from degrading muscle, or a reversible change within the fascia. Collagen or proteoglycan cross-linking should not be invoked unless it is measured directly and shown to occur on the observed timescale.

### 6.4 What Anatomists and Forensic Investigators Gain

For a limited period following death, fascial tissues retain architectural and material properties formed during life, although those properties begin changing immediately. Anatomists may encounter persistent tautness in retinacula, aponeuroses and intermuscular septa, but such observations cannot presently be attributed to myofibroblast activity, new extracellular matrix cross-linking or tensegrity failure without measurement. Documenting regional stiffness, hydration and deformation alongside the stage of muscular rigor could determine whether reproducible fascial patterns exist. Reconstruction of an individual's loading or movement history from post-mortem fascial tension should remain a long-term research question. The resistance felt when a joint is moved can be recorded as force or torque. Fascia and adjacent muscle can be measured separately at the same site and time. This would show whether the intact body expresses more than muscle rigor alone.

## 7. CONCLUSIONS

This hypothesis-generating narrative review proposes three testable conclusions:

1. Fascia may contribute to post-mortem rigidity through changes in cellular tension, extracellular matrix hydration and viscoelasticity, and force transmission across the fascial–muscular continuum. These candidate processes may occur alongside ATP-dependent muscle fibre stiffening, but their magnitude and timing require direct measurement.

**2.** Regional variation in fascial density, hydration, cellular composition and loading history may help to explain uneven onset, variable timing and non-linear resolution of post-mortem stiffness. This proposed association remains to be tested against muscle-only explanations and other recognised sources of variability.

**3.** A fascia-inclusive model generates practical research priorities. These include longitudinal measurement of fascial and muscular stiffness, hydration, ionic change, proteoglycan behaviour and collagen organisation during the early post-mortem interval (Çevik Saldıran et al., 2025; Gennisson et al., 2013). Fascial biomarkers and reconstruction of loading history should be regarded as possibilities for validation, not current forensic or anatomical tools.

This fascia-inclusive framework extends the muscle-centred model by recognising that observable post-mortem stiffness arises within an integrated, multi-tissue architecture. It does not present original experimental

data. Its contribution is to synthesise established fascia research into testable hypotheses concerning post-mortem rigidity and to define the direct measurements required to evaluate them.

## SIGNIFICANCE STATEMENT

Rigor mortis is currently explained as a consequence of ATP depletion within skeletal muscle fibres. This established account does not fully explain uneven distribution, variable timing and inconsistent progression of post-mortem stiffness across the body. The authors propose that fascia may contribute to this variability because the connective tissue continuum remains structurally continuous with muscle as ionic regulation and active biological maintenance cease.
Considering fascia does not replace the classical explanation of rigor mortis. It broadens the anatomical field of enquiry and offers testable questions for anatomists and forensic scientists. The authors hope that this framework will encourage direct investigation of a previously under-explored topic in forensic science.

## DATA AVAILABILITY STATEMENT

**No new data are associated with this article.**

## ETHICAL AND CONSENT STATEMENT

The image was obtained during a fascia-focused dissection course at Trecchi Human, Cremona, Italy, under the auspices of its Scientific Committee and in accordance with Italian Law No. 10 of 10 February 2020, 'Rules regarding the disposition of one's body and post-mortem tissues for study, training, and scientific research purposes'. The authors adhere to the ethical and legal requirements governing the scholarly use of donated human material.
No identifying features are visible, and the image is used solely for anatomical and scientific illustration.


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## CONFLICTS OF INTEREST

**The authors declare no conflicts of interest.**

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