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The Science of Cellular Communication

From Paracrine Signalling to Environmental Conditioning

Paper 01 in the CFT Advantage Series

WHITE PAPER

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Abstract

Cells do not work in isolation. Across every tissue in the body, cells maintain a steady conversation, sending biochemical messages that coordinate growth, repair, immune balance, and metabolism. Much of this conversation travels through the secretome: the complete set of soluble proteins, lipids, metabolites, matrix-associated factors, and membrane-bound vesicles that cells release into their surroundings. Extracellular vesicles (EVs), nanometer-scale carriers of biological cargo, are an important part of this signalling system, working alongside soluble growth factors, cytokines, and lipids rather than in place of them.

This paper introduces the science that the rest of the CFT Advantage Series builds on. The first half describes how cells communicate naturally: paracrine, autocrine, and endocrine signalling; the biogenesis pathways that produce exosomes, microvesicles, and apoptotic bodies; and the cargo-loading mechanisms that determine what each vesicle carries. The second half describes how environmental conditioning, the controlled exposure of cells to physiological stressors such as low oxygen and serum-free conditions, leverages ancient stress-response pathways (including HIF and ATF4) to upregulate the secretion of bioactive factors. The paper closes by connecting these two threads to the way Cell-Free Therapy (CFT) is manufactured: a personalised, autologous secretome derived from the patient's own conditioned blood-derived cells, prepared to support normal biological function through the same signalling pathways the body already uses.

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1. Introduction: Cells in Conversation

A multicellular organism is, in one sense, a coordination problem. Trillions of cells must keep their behaviour aligned with the needs of the tissues they occupy, the organs those tissues compose, and the body those organs sustain. Direct cell-to-cell contact, through tight junctions, gap junctions, and surface receptors, accounts for some of that coordination. The rest depends on a quieter and more pervasive system: the constant secretion of biochemical messages into the extracellular space, where they reach neighbouring cells, drift further afield, or arrive packaged inside membrane-bound vesicles.

The complete catalogue of what a cell secretes is called the secretome. It includes soluble proteins (growth factors, cytokines, enzymes), lipids (signalling phospholipids, sphingolipids), nucleic acids (microRNAs, mRNAs, fragments of long non-coding RNA), metabolites, matrix-associated factors, and extracellular vesicles. Extracellular vesicles (EVs), are one important class within that mix. Their lipid bilayer can protect cargo from degradation in biological fluids, and a single particle can carry multiple signalling molecules at once. Soluble factors and matrix-bound signals carry much of the rest of the load.

This paper sets out, in plain terms, what the biology underneath Cell-Free Therapy (CFT) actually is. It begins with how cells communicate by default. It then describes how that communication can be amplified through environmental conditioning, in which cells are deliberately exposed to mild physiological stressors that switch on adaptive secretion programmes. It closes by tracing how those two pieces meet in CFT manufacturing.

2. Modes of Cellular Signalling

2a. Paracrine, Autocrine, Endocrine, Juxtacrine

Cell signalling is usually classified by how far the message travels.

Paracrine signalling describes the secretion of factors that act on nearby cells, typically within 100 to 200 micrometres. The Greek prefix “para-”, meaning beside, captures the local nature of the exchange. Paracrine signals diffuse through the extracellular matrix or interstitial fluid and bind receptors on adjacent cells, triggering intracellular cascades that change those cells’ behaviour. Most tissue-level coordination, including immune response orchestration, wound healing, vascular maintenance, and tissue patterning during development, runs on paracrine traffic.

Autocrine signalling describes the same chemistry directed at the secreting cell itself. A cell may release a growth factor that binds back to its own receptors, reinforcing or sustaining its current state. Autocrine and paracrine effects often happen at the same time: a single secreted factor can act on the cell that produced it and on cells nearby.

Endocrine signalling describes long-range chemistry. Hormones such as insulin, thyroid hormone, and adrenaline travel through the bloodstream to act on distant tissues. The endocrine system enables whole-body coordination but pays a price in speed and specificity, as messages must be diluted, travel far, and pass hepatic and renal clearance.

Juxtacrine signalling, the fourth modality, requires direct cell-to-cell contact through membrane-bound ligands and their receptors. The Notch-Delta system and many T-cell receptor interactions work this way. Juxtacrine signalling provides exquisite specificity but only operates between physically touching cells.

In intact tissues, these modalities run in parallel. Paracrine traffic carries most of the load for local tissue coordination; autocrine signalling reinforces activated states; juxtacrine signalling enables contact-dependent decisions; endocrine signalling integrates across organ systems.

2b. Why Local Signalling Suits Tissue Coordination

Paracrine communication has practical advantages over its longer-range counterpart. Local diffusion is fast, so tissues can respond on the timescale of seconds to minutes rather than waiting for systemic distribution. The same secreted factor can build a concentration gradient across a small region, giving spatial precision without committing the whole organism to a response. Multiple secreting cells can fine-tune the message by adjusting the volume and composition of their secretion in real time. And paracrine signalling avoids the metabolic cost of clearing factors through the liver and kidneys, because the messengers are taken up and processed close to where they were released.

EVs are an important part of paracrine traffic, alongside soluble growth factors, cytokines, lipids, metabolites, and matrix-associated signals. Together, these channels carry the local coordination work that keeps tissues maintained.

3. Extracellular Vesicles: Carriers of Cellular Information

3a. Three Classes: Exosomes, Microvesicles, Apoptotic Bodies

Most, if not all, cell types release extracellular vesicles as part of normal physiology. EVs are usually classified by size and biogenesis pathway (Raposo & Stoorvogel, 2013; Yáñez-Mó et al., 2015).

Exosomes (30 to 150 nm) form inside multivesicular bodies (MVBs), an internal compartment of the endosomal system. When MVBs fuse with the plasma membrane, the small intralumenal vesicles inside are released into the extracellular space. Exosomes are the smallest and most uniformly sized class.

Microvesicles, also called ectosomes (50 to 1,000 nm), bud directly from the plasma membrane. They retain the membrane orientation of the cell that made them, so their outer surface presents the same molecules that face the extracellular environment on intact cells.

Apoptotic bodies (500 to 5,000 nm) are larger vesicles released during programmed cell death. They contain cytoplasmic, and sometimes nuclear, material from the dying cell.

These categories overlap in size, especially microvesicles and exosomes in the 100 to 200 nm range, and any biological sample contains a mixture of all three. The International Society for Extracellular Vesicles guidelines, MISEV2018 and the updated MISEV2023, recommend that papers report what they actually measured (size, density, marker profile, morphology) rather than relying on size alone to assign vesicles to a class (Théry et al., 2018; Welsh et al., 2024).

3b. Biogenesis: ESCRT-Dependent and ESCRT-Independent Pathways

The classical exosome biogenesis pathway uses the ESCRT machinery (endosomal sorting complexes required for transport). ESCRT-0, -I, -II, and -III complexes are recruited sequentially to the limiting membrane of an MVB. They recognise ubiquitinated cargo proteins, deform the membrane inward, and pinch off intralumenal vesicles into the MVB lumen. The result is selective in many systems: cargo is loaded based on ubiquitin tags, and the ESCRT-associated proteins TSG101 and ALIX are commonly enriched in exosome preparations and are frequently used as EV-associated markers, with the caveat that no single marker is universal across every vesicle or every preparation (Théry et al., 2018).

A parallel set of ESCRT-independent pathways also produces intralumenal vesicles. These pathways depend on lipid composition (particularly ceramide-rich domains), tetraspanin interactions, and direct protein-protein contacts. The relative balance between ESCRT-dependent and ESCRT-independent biogenesis varies between cell types and conditions (van Niel et al., 2018).

Microvesicle budding works through a different mechanism. Plasma membrane budding is driven by lipid reorganisation, especially the externalisation of phosphatidylserine on the outer leaflet, which exposes recognition signals for vesicle uptake. Cholesterol- and sphingomyelin-rich microdomains at the budding site help concentrate cargo before scission.

Cellular stress accelerates EV secretion across all classes. Hypoxia, nutrient deprivation, oxidative stress, mechanical loading, and thermal stress all increase EV release. This is not incidental; it is a regulated response that boosts intercellular communication when conditions demand it.

3c. Cargo: Proteins, Lipids, Nucleic Acids

Extracellular vesicles can carry proteins, lipids, and nucleic acids, though cargo composition varies by cell type, physiological state, and isolation method. EV populations are heterogeneous, and not

every cargo class is consistently or functionally meaningful in every preparation. The descriptions below summarise what has been reported across multiple cell types and conditions in the literature.

Protein cargo can include structural proteins (the tetraspanins CD9, CD63, CD81), signalling proteins (growth factors such as VEGF, FGF, PDGF; cytokines and their receptors), enzymes (proteases, kinases), adhesion molecules, and metabolic enzymes. Cargo loading appears selective in many systems: protein concentrations inside vesicles can differ markedly from those in the cytoplasm of the secreting cell.

Lipid cargo can differ from the bulk plasma membrane. Many EV populations are enriched in phosphatidylserine, sphingolipids (ceramides, sphingomyelin), and cholesterol. Externalised phosphatidylserine can act as an “eat me” signal that promotes vesicle uptake by target cells.

Nucleic acid cargo has been reported across many EV populations. EVs can carry microRNAs (typically 18 to 25 nucleotides), longer non-coding RNAs, and messenger RNAs. Some EV populations also carry fragments of nuclear or mitochondrial DNA. When delivered to a recipient cell, such RNA species can modify gene expression: microRNAs bind complementary sequences in the 3′ untranslated regions of target mRNAs to repress translation, and intact mRNAs can be translated by recipient ribosomes. The functional weight of any individual cargo class in a given preparation depends on the cells of origin, their state, and how the vesicles were prepared. However, recent evidence suggests that EV-associated microRNAs should be interpreted cautiously as functional mediators of intercellular communication, because only a small fraction of EVs may carry microRNAs and, even when present, their delivery to recipient cells may be inefficient or biologically undetectable (Albanese et al., 2021).

Where multiple cargo classes are present in the same particle, EVs have the potential to transmit information at several levels at once, much like a single sentence carries vocabulary, grammar, and tone together.

4. How Target Cells Receive the Signal

4a. Surface Recognition and Multiple Uptake Routes

EV uptake is influenced by surface proteins, lipids, glycans, and receptor-ligand interactions (Mathieu et al., 2019; van Niel et al., 2018). Integrins, tetraspanins, externalised phosphatidylserine, and surface glycosylation all contribute, and the relative weight of each pathway depends on the EV population, the target cell, and the local milieu. Autologous vesicles carry patient-derived membrane markers, including self-HLA on relevant vesicle populations, which may reduce alloimmune recognition. Uptake itself, however, is governed by multiple overlapping pathways rather than by HLA matching alone.

Several uptake routes operate in parallel.

Receptor-mediated endocytosis. EV-surface ligands such as integrins, tetraspanins, and growth factors bind cognate receptors on the target cell. Receptor clustering then triggers clathrin-mediated or clathrin-independent endocytosis.

Lipid-based uptake. Externalised phosphatidylserine on the EV surface is recognised by phosphatidylserine receptors (such as TIM family receptors) on the target cell, prompting endocytic engulfment. This route is especially important for microvesicles and apoptotic bodies.

Direct membrane fusion. Vesicles rich in cholesterol and sphingomyelin can fuse with the target cell's plasma membrane, releasing their contents directly into the cytoplasm without passing through the endosomal system. This is the fastest route to delivery.

Macropinocytosis. Bulk fluid uptake can carry EVs into the cell along with the surrounding medium. It is non-selective but contributes meaningfully when EV concentrations are high.

4b. Intracellular Trafficking and Cascade Activation

Once inside, an EV can take several paths. It may sit briefly in an early endosome before maturing to a late endosome, which provides a regulated environment for cargo release. Also EVs may be trafficked to lysosomes, where the membrane is broken down and the cargo degraded; thus EV uptake should not be equated with productive signalling, since substantial evidence indicates that many internalised EVs are recycled or routed to lysosomes for degradation rather than releasing functional cargo into the recipient cell. This is a regulatory mechanism that limits signalling. It may pass through recycling endosomes, prolonging interaction with the recipient cell's machinery. Or, if it fused at the surface, it may have already deposited its cargo in the cytoplasm.

Once cargo is released, several signalling cascades can be activated. Vesicle-associated growth factors engage receptor tyrosine kinases, triggering PI3K-Akt, MAPK/ERK, and JAK-STAT pathways that change gene expression, survival, and metabolism. Pattern recognition receptors (TLRs and others) may detect molecular signatures on the vesicle surface, initiating innate immune signals. EV-associated microRNAs and mRNAs can directly modify the recipient cell's gene expression programme.

The point worth holding is that one vesicle can simultaneously deliver protein-level, lipid-level, and gene-expression-level signals. EVs are not single-purpose carriers; they are multilingual.

5. The Stress-Response Paradigm

5a. Why Cells Secrete More Under Pressure

Cells are sensitive to the chemistry of their environment. When a tissue runs short on oxygen, when nutrients become scarce, when mechanical strain rises, or when local pH shifts, cells detect those

changes through molecular sensors and respond through coordinated transcriptional and translational programmes.

Under physiological pressure, cells alter their secretion toward signals involved in adaptation, repair, immune recruitment, and survival. Hypoxic cells, for example, secrete more angiogenic factors (VEGF, FGF, PlGF). Nutrient-deprived cells release more growth factors and survival-supporting cytokines (HGF, IL-6, IL-10). Mechanically stressed cells produce more matrix-supporting and angiogenic factors (TGF-β, PDGF, VEGF). The broad pattern is that stress shifts the secretome toward factors involved in coordinating adaptation.

Not every stress-induced signal is beneficial in isolation. Stress responses can also produce inflammatory mediators or damage-associated signals, and over-stressing cells can shift the balance toward apoptotic debris rather than viable secretion. This is precisely why conditioning intensity, timing, and viability control matter: a defined protocol concentrates the adaptive component while limiting the unwanted one. The use of mild, defined stress applied to autologous cells in a manufacturing setting is the design choice that allows this balance to be targeted, with batch characterisation reported in Paper 02.

5b. An Evolutionarily Conserved Logic

Stress-response signalling is ancient. Bacteria use the SOS response under DNA damage; yeast and other unicellular eukaryotes use the heat shock response; invertebrates retain HSF and DAF-16 as master stress transcription factors; mammals layer in more elaborate systems including HIF, p53, NF-κB, and ATF4.

The conservation across phylogeny is informative. It indicates that stress-induced secretion of cytoprotective and tissue-supportive factors is one of the fundamental tools that life uses to persist under adverse conditions. Manufacturing approaches that lean on these pathways are working with biology rather than against it.

6. Environmental Conditioning: Tuning the Secretome

Environmental conditioning is the controlled exposure of cells to defined physiological stressors during manufacturing, with the aim of activating natural stress-response pathways and concentrating their secreted output in the resulting preparation. CFT's conditioning protocol uses two of these stressors directly: reduced oxygen tension and serum-free medium (covered in 6a and 6b below). Mechanical, pH, and osmotic cues are described in 6c and 6d as background biology only; the operational conditioning levers in CFT are hypoxia and serum deprivation. Paper 02 in this series describes the full manufacturing protocol, characterisation methodology, and release specifications.

6a. Hypoxia and HIF Signalling

Lowering oxygen below physiological levels (typically below 5 percent) is the most thoroughly characterised conditioning stimulus. Hypoxia stabilises hypoxia-inducible factor (HIF), a heterodimeric transcription factor (HIF-α and HIF-β). Under normal oxygen, prolyl hydroxylase enzymes use oxygen to mark HIF-α for proteasomal degradation. Under low oxygen, those enzymes lose activity, HIF-α accumulates in the nucleus, and binds hypoxia response elements (HREs) on the promoters of hypoxia-responsive genes (Semenza, 2012; Kaelin & Ratcliffe, 2008).

The HIF-driven output is broad. Angiogenic factors (VEGF, FGF, PlGF) are upregulated to support new vessel growth and oxygen delivery. Glucose transporters and glycolytic enzymes are upregulated to maintain ATP production under low oxygen. Anti-apoptotic factors such as survivin and Bcl-2 family proteins are upregulated to protect cells through the stressed window. Immune-recruitment factors (CXCL1, CXCL12, CSF-2) are upregulated to support tissue repair.

NF-κB and p53 signalling also contribute under hypoxia, broadening the secreted output beyond pure HIF targets.

6b. Serum Deprivation and Nutrient Stress

Removing serum from culture medium denies cells access to growth factors, lipoproteins, transferrin, and other inputs they normally rely on. Nutrient sensors detect the deprivation and trigger an adaptive response.

mTORC1, the master integrator of growth signals, is inhibited as growth factor signalling drops; this releases the brakes on autophagy and stress-response transcription (Saxton & Sabatini, 2017). The GCN2-ATF4 axis is activated by uncharged tRNAs accumulating when amino acids are scarce; ATF4 then drives the transcription of amino acid biosynthesis enzymes and a wide stress-response programme. AMPK is activated when ATP falls, phosphorylating mTORC1 and shifting metabolism toward catabolism (Hardie et al., 2012).

The secreted output is again broad. Growth factors (FGF1, FGF2, VEGF, HGF) and cytokines (IL-6, IL-8, IL-10) increase, alongside autophagy components and heat shock proteins. The pattern is paradoxical only on first glance: under nutrient stress, cells increase secretion of the factors that would normally support their own survival and stimulate vascular growth.

6c. Mechanical Stress and Mechanotransduction (Background Biology)

For completeness, mechanical inputs are transduced into biochemical signals through several systems: integrin clusters at focal adhesions, the FAK-Src-PI3K cascade, the RhoA-ROCK pathway, mechanosensitive ion channels (Piezo1, Piezo2, TRPM7), and the YAP/TAZ transcriptional co-activators (Hynes, 2002; Dupont et al., 2011).

When cells are subjected to compression, fluid shear, or stretching, these systems activate in parallel and shift secretion toward matrix proteins and angiogenic factors. This section is included as background biology rather than as a description of the CFT process; the CFT conditioning protocol relies on hypoxia and serum-free conditions, not mechanical conditioning.

6d. pH and Osmotic Cues (Background Biology)

For completeness, cells also sense pH and osmotic shifts, and these inputs feed into the same broad stress-response toolkit. This is included only as illustrative background biology; CFT does not materially rely on pH or osmotic conditioning.

6e. Inter-Donor Variability and Autologous Consistency

Large-scale studies of peripheral blood mononuclear cells (PBMCs) have demonstrated substantial inter-donor variation in transcriptional responses to the same stimulus, including differences in cytokine, chemokine, and growth-factor expression (Fairfax et al., 2014). These differences arise from underlying genetic, epigenetic, and physiological variation between individuals.

For an autologous product, such variability is expected and reflects the biology of the source material rather than inconsistency in process execution. Consequently, manufacturing consistency is assessed through control of the conditioning process and compliance with predefined release criteria, rather than through identical molecular composition across donors. The objective is to produce a reproducible, well-characterised secretome while acknowledging that some degree of donor-specific variation is an inherent feature of autologous cell-derived preparations.

7. Synergy and Timing

7a. Combined Stressors Compound the Response

Individual stressors upregulate specific gene sets. Combinations often produce more than the sum of the parts.

Hypoxia plus serum deprivation activates HIF targets (from low oxygen) alongside ATF4-driven nutrient-stress targets (from low nutrients), with mTORC1 inhibited at the same time. The result is maximal upregulation of angiogenic factors, immunomodulatory cytokines, and stress-response proteins. Hypoxia plus mechanical stress combines HIF and NF-κB signalling with FAK-Src, RhoA, and YAP/TAZ; the output is robust upregulation of angiogenic factors, matrix proteins, and immune factors, mimicking the chemistry of wounded, hypoxic tissue.

Two molecular reasons explain the synergy. First, gene promoters often carry binding sites for multiple stress-responsive transcription factors; cooperative occupation produces supraadditive

transcription. Second, individual stressors remove repressive signals: serum deprivation inhibits mTORC1, which normally represses autophagy and parts of the stress programme, releasing brakes that other stressors can then exploit.

An important caveat is that synergistic responses are highly cell-type and context dependent, and the effect must ultimately be confirmed empirically in the specific cell population used for manufacturing. Studies in PBMCs and other primary immune-cell preparations have shown that combined stressors can increase not only regenerative and adaptive-response mediators, but also pro-inflammatory cytokines as mentioned above.

7b. Timing the Harvest

Stress-induced secretion unfolds in a recognisable order. Transcription factor activation comes first, with HIF, ATF4, NF-κB, or YAP/TAZ accumulating in the nucleus depending on the stressor. Primary target mRNAs rise next, followed by secondary target mRNAs encoding the larger set of growth factors, cytokines, and matrix-supporting proteins. Translated protein then begins to appear in the medium, and EV release rises in parallel. If stress continues unchecked, cells eventually exhaust their reserves: viability declines, apoptotic markers appear, and intracellular contents leak into the medium as debris.

Each phase has a different signature in the harvested preparation. Too early, and there is little secreted bioactive content because translation and export have not caught up with transcription. Too late, and apoptotic debris contaminates the secretome, diluting what is functional with what is not. The exact shape of this curve depends on the cells, the stressor combination, and the readout used to measure it.

CFT's conditioning protocol is designed to harvest within a window that targets peak secreted output while preserving cell viability and limiting apoptotic debris. Release criteria for total protein, growth factor concentrations, and EV concentration, together with the batch characterisation that supports them, are considered in Paper 02.

8. From Science to Product: How CFT Applies These Principles

Cell-Free Therapy (CFT) is an autologous, cell-free biological preparation derived from the patient's own peripheral blood. The manufacturing process applies the science set out above in a defined and protocol-controlled way.

8a. Autologous Starting Material

Each preparation begins with a peripheral blood draw from the patient. Blood-derived cells, predominantly peripheral blood mononuclear cells, are isolated and used as the conditioning substrate. Because the starting material is the patient’s own cells, the secretome ultimately

produced carries the patient's own HLA profile and broader patient-derived biological signature. This helps avoid the donor-recipient alloimmune mismatch and potential clearance challenges that allogeneic preparations must manage.

8b. Defined Conditioning

Cells are exposed to a defined set of physiological stressors, selected to activate adaptive secretion programmes without compromising viability. The harvest window is designed to capture peak bioactive output while cell viability remains high. Processing avoids high-shear ultracentrifugation, a method commonly used in research-scale EV isolation, helping preserve the broader secretome rather than narrowing the preparation to a single isolated particle fraction. Other isolation approaches, including tangential flow filtration, size-exclusion chromatography, precipitation, and affinity methods, are also in use across the field of EV-only research.

8c. The Resulting Preparation

The resulting CFT preparation contains the bioactive output of the patient's conditioned cells: extracellular vesicles (exosomes and microvesicles), growth factors and regulatory cytokines across the families well documented in the PBMC and conditioned-secretome literature (covering members of the VEGF, FGF, HGF, PDGF, and TGF families, alongside members of the interleukin family), adhesion molecules, and EV-associated microRNAs. Published characterisations of cell-derived conditioned secretomes typically report extracellular vesicle concentrations on the order of 10^9 to 10^{10} particles per mL alongside a panel of soluble bioactive factors at physiologically relevant concentrations (Yáñez-Mó et al., 2015; Mathieu et al., 2019). Each CFT batch is quantified against a defined panel of proteins, cytokines, and EV parameters and released only when within established release criteria. Paper 02 in this series "From blood draw to biology" will describe the assay panel, release criteria, and characterisation methodology in detail.

The preparation is delivered as a personalised biological product calibrated to the patient's own physiology. CFT supports the body's normal biological function through mechanisms consistent with the paracrine signalling and adaptive secretion systems described here. Demonstration of clinical efficacy requires appropriately designed clinical studies and cannot be inferred solely from molecular composition or in vitro biological activity.

9. Conclusion

Cellular communication is not a side feature of biology; it is one of the foundations on which multicellular life rests. Paracrine signalling carries much of the day-to-day coordination work, through soluble factors, lipids, metabolites, matrix-associated signals, and extracellular vesicles operating in parallel. Cells modulate the volume and composition of their secretion in response to

physiological pressure, increasing the output of factors that support adaptation when conditions demand it.

Environmental conditioning works with these systems rather than around them. By applying defined, mild stressors during manufacturing, cells can be steered to upregulate the secretion of bioactive factors in a controlled, reproducible way. The resulting secretome is calibrated to the same biological logic the body already uses to maintain homeostasis.

CFT applies these principles to autologous cells, harvesting the full conditioned secretome and delivering it as a personalised preparation. The remaining papers in this series develop individual aspects of this picture. Paper 02 covers the full manufacturing workflow, quality control, and the United States regulatory framework. Papers 03, 05, and 06 examine how CFT differs from adjacent modalities: Paper 03 compares CFT with platelet-rich plasma, Paper 05 takes apart the particle-count claim used to market commercial allogeneic exosome preparations, and Paper 06 sets out the case for preserving the whole secretome rather than isolating purified exosome fractions. Paper 07 develops the personalisation argument that follows from using the patient's own cells. Paper 04 addresses neurological applications via the blood-brain barrier. Paper 08 covers cell banking and repeated dosing from a single blood draw. Papers 09 and 10 turn to specific use cases: athletic recovery and performance (Paper 09) and longevity and healthy ageing (Paper 10).

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Disclaimer: *Individual results vary. Cell-Free Therapy is not intended to diagnose, treat, cure, or prevent any disease. The information in this paper is provided for educational purposes and does not constitute medical advice. CFT supports the body's normal biological function through autologous, cell-free biological preparations.*

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