Wellbeing International Foundation

Personalised by Nature

Why Your Own Biology Is the Best Biology

Paper 07 in the CFT Advantage Series

WHITE PAPER

Wellbeing International Foundation

Abstract

The human immune system is fundamentally designed to distinguish self from non-self. This capability is both the body's most sophisticated protective mechanism and the primary barrier to allogeneic therapeutic products. When a preparation contains donor material, the recipient's immune system recognises it as foreign, triggering clearance, inflammatory responses, and potential sensitisation. Autologous therapy avoids donor-recipient HLA mismatch and is therefore expected to substantially reduce alloimmune recognition. Yet the advantage extends beyond safety. Because every patient's cellular biology is distinct (shaped by immune history, inflammatory phenotype, age, genetic background, and metabolic state), an autologous preparation is inherently personalised at the molecular level. The secretome produced by a patient's own conditioned cells reflects that individual's unique physiology, not a generic donor profile. This paper examines both dimensions of the autologous advantage: the immunological safety of self-derived material and the molecular personalisation that autologous manufacturing delivers. We review the biology of immune recognition, published evidence of immune responses to allogeneic preparations, the documented inter-patient variability in cellular secretomes, and the precision medicine trajectory that positions autologous approaches as the future of regenerative biology. Cell-Free Therapy (CFT) operationalises both principles through a GMP-compliant workflow in which the patient is the sole source and sole recipient of biological material.

Table of Contents

1. Introduction: The Two Dimensions of the Autologous Advantage

Two separate but connected arguments underpin the case for autologous therapy over allogeneic alternatives. The first is immunological: the patient's own material is recognised as self and is not attacked by the immune system. The second is biological: the patient's own material carries a molecular signature that reflects their unique physiology, making it inherently personalised in a way no donor-derived product can replicate.

These two arguments are frequently made separately. Papers on immune compatibility focus on HLA matching and rejection. Papers on personalised medicine focus on secretome variability and precision therapeutics. In practice, they are two faces of the same coin. The same biological fact (that every individual's cells are molecularly unique) simultaneously creates the immune rejection problem for allogeneic products and the personalisation advantage for autologous ones.

This paper examines both dimensions together. Understanding why autologous material avoids donor-recipient alloimmune mismatch requires the same biology that explains why it is molecularly personalised. The patient's HLA molecules that avoid alloimmune rejection are the same molecules that contribute to biological compatibility. The patient's unique cytokine production profile that makes their secretome distinct from every other patient's is the same profile that ensures cargo relevance. Safety and personalisation are not separate features of autologous therapy. They are consequences of a single biological principle: the therapy comes from the patient themselves.

Cell-Free Therapy (CFT) operationalises both principles. A minimum volume of peripheral blood is collected from the patient, processed in a GMP facility in Germany, and the resulting cell-free secretome (containing extracellular vesicles, growth factors, cytokines, and signalling molecules) is returned exclusively to that patient. No donor material, no pooling, no foreign HLA. The patient is both the source and the recipient.

2. The Biology of Immune Recognition

2a. The MHC/HLA System: Your Molecular ID Card

The immune system's ability to distinguish self from non-self rests primarily on the Major Histocompatibility Complex (MHC), known in humans as the Human Leukocyte Antigen (HLA) system. HLA molecules are proteins displayed on the surface of nearly every nucleated cell in the body. They function as a molecular identification badge, telling immune surveillance cells that the cell is part of the individual's own body. HLA class I molecules (A, B, C) help the immune system check what is happening inside a cell, such as whether it is infected or abnormal. HLA class II

molecules (DP, DQ, DR) help show the immune system material that comes from outside the cell, such as parts of bacteria or other foreign substances.

HLA molecules are extraordinarily polymorphic: they vary dramatically between individuals. This polymorphism is so extensive that HLA matching is a core requirement for organ transplantation, and even with matched organs, recipients typically require lifelong immunosuppression to prevent rejection. Without matching, rejection occurs within days to weeks or even hours.

Allogeneic cell-derived products, including extracellular vesicles from donor cells, display donor HLA molecules on their surface. One example are Medicinal Signaling Cells (MSCs), traditionally known as Mesenchymal Stem/Stromal Cells, that can be used allogeneically because they express low HLA-class I, little to no HLA-class II or costimulatory molecules, and exert immunomodulatory effects, resulting in weak alloreactive activation. However, compared with autologous MSCs, allogeneic MSCs may still carry a risk of immune recognition, reduced persistence, donor variability, or repeated-dose sensitization. The recipient's immune system immediately recognises these molecules as foreign. This is the beginning of alloimmunisation, the process of becoming sensitised to foreign HLA antigens.

2b. How the Immune System Responds to Foreign Material

The immune response to foreign HLA follows two main pathways. Innate immunity provides the first line of defence: pattern recognition receptors on macrophages and dendritic cells detect allogeneic material as danger signals, triggering release of pro-inflammatory cytokines (IL-6, TNF-α, IL-1β) and initiating acute inflammation.

Adaptive immunity mounts a more targeted and persistent response. Helper T cells (CD4+) promote the production of anti-donor antibodies by B cells. These antibodies bind to foreign material, marking it for destruction by complement activation and antibody-dependent cellular cytotoxicity. Cytotoxic T cells (CD8+) directly attack cells displaying foreign HLA. Critically, memory T cells and B cells persist long-term, meaning subsequent exposures trigger faster and stronger immune responses.

The result is not only destruction of the therapeutic material but also immune sensitisation. Patients exposed to allogeneic material develop antibodies and memory cells against those donor HLA types. If they later receive another allogeneic product from the same or cross-matching donor, the secondary immune response is typically much more vigorous. This has direct clinical consequences: sensitised patients may be excluded from future transplant programs entirely.

2c. Why Autologous Material Escapes Immune Attack

Autologous material displays the patient's own HLA molecules. These molecules have been 'educated' in the patient's thymus during immune system development. The patient's T cells have already undergone positive and negative selection, a process that ensures they recognise self-HLA as safe and do not attack self-tissue.

When autologous material is administered, the immune system encounters only self HLA. Self-recognition is expected to avoid the alloimmune T cell activation, antibody formation, and inflammatory cascades associated with foreign HLA exposure. The material is recognised as part of the body itself. This is not a theoretical advantage; it is the same biological principle that allows blood transfusions between identical twins without immunosuppression. It is worth noting that autologous origin does not guarantee complete absence of any immune response in every clinical context; processing steps, adjuvants, or aggregation could theoretically trigger innate immune pathways. However, the fundamental alloimmune barrier is removed.

3. Published Evidence of Immune Responses to Allogeneic Preparations

3a. Immune Responses to Allogeneic Extracellular Vesicles

Extracellular vesicles (EVs), including exosomes and microvesicles, retain donor HLA and other donor-derived antigens on their surface when derived from allogeneic sources. A growing body of research demonstrates immune responses to such material. Larssen et al. (2019) showed that allogeneic dendritic cell-derived EVs enhanced antigen-specific CD8+ T cell responses, follicular helper T cell activation, and antibody production with long-term immunological memory, demonstrating that allogeneic origin amplifies EV-mediated immune activation. In a transplant alloimmunity model, Chen et al. (2025) demonstrated that graft-derived allogeneic EVs activate complement via IgM binding, with complement-opsonised vesicles promoting B cell activation, germinal centre formation, and donor-specific antibody generation. This was a transplant model rather than a secretome context, but it establishes a mechanistic pathway by which allogeneic EVs can drive humoral alloimmunity. Prunevieille et al. (2021) showed that allogeneic exosomes activate T cells in vivo and sensitise recipients to alloantigens, particularly in inflammatory environments.

These findings indicate that cell-derived products can trigger adaptive immune responses when derived from allogeneic donors. It is important to present the evidence base honestly. Some recent literature describes MSC-derived EVs as having low immunogenicity (Li et al., 2025), and a 2024 meta-analysis of human EV-based therapy reported low overall serious adverse event rates with no statistically significant difference between autologous and allogeneic administration in subgroup analysis (Van Delen et al., 2024). HLA-G may partly explain this apparent low immunogenicity because it is a non-classical HLA class I molecule with immunomodulatory functions, capable of dampening NK-cell and T-cell responses and promoting tolerogenic immune pathways; however, its expression in MSCs and MSC-derived EVs can vary by donor, tissue source, culture conditions, and inflammatory priming. A 2024 perspective identified an urgent need to address EV immunogenicity more rigorously for clinical development, noting that absence of acute immunotoxicity does not equate to absence of immunogenicity (Xia et al., 2024). The field is actively

working through these questions. What is established is that allogeneic EVs carry foreign HLA and can trigger immune recognition; what remains under investigation is the magnitude and clinical significance of that recognition across different product types and clinical contexts.

The position of this paper is that the immunological principles are well established (foreign biological material triggers recognition) and that autologous origin removes the most fundamental trigger of alloimmune activation. We do not claim that all allogeneic cell-free products are clinically dangerous; rather, we claim that autologous products avoid a biologically well-established immunobarrier.

3b. Clinical Evidence: Transplantation and Transfusion

Clinical data from allogeneic cell transplantation programmes reinforce the principle. Graft-versus-host disease remains one of the most significant complications of allogeneic stem cell transplantation, occurring even with HLA-matched donors. Host-versus-graft rejection occurs when the recipient's immune system destroys the transplanted material. Transfusion-related acute lung injury (TRALI) has been associated with HLA alloantibodies in transfused blood products, demonstrating that even routine biological transfusions can trigger serious immune complications (Toy et al., 2012; Kopko et al., 2002).

These examples involve cellular products and represent the severe end of the alloimmune spectrum. Cell-free products may elicit less pronounced responses, but the underlying principle (that foreign HLA triggers immune recognition) applies across biological preparations. The degree to which this translates into clinically meaningful effects for cell-free secretomes is an active area of investigation.

4. The Safety Advantage of Self-Derived Material

4a. No Cross-Reactivity, No Sensitisation

Cross-reactivity occurs when antibodies or T cells formed against one foreign antigen recognise and attack antigens from a different donor. With autologous therapy, cross-reactivity is impossible, because the material comes from the patient alone. There are no foreign HLA molecules to cross-react with.

Sensitisation (the development of antibodies to foreign HLA antigens) is one of the most serious long-term risks of allogeneic therapy. Once sensitised, patients face higher rejection risk for any future allogeneic material and may be excluded from transplant programmes. Autologous therapy avoids this risk. Without exposure to foreign HLA, alloimmune sensitisation does not arise.

4b. No Immunosuppression Required

Allogeneic transplant recipients typically require long-term immunosuppressive drugs (calcineurin inhibitors, corticosteroids, and others), each carrying significant side effects including increased infection risk, secondary malignancy, renal toxicity, and metabolic complications. Autologous therapy requires no systemic immunosuppression. Because the material is recognised as self, there is no immune activation to suppress. This eliminates an entire class of medication-related risks.

5. Beyond Safety: How Autologous Manufacturing Delivers True Personalisation

The term 'personalised medicine' has become ubiquitous in healthcare marketing, often applied to products manufactured from generic donor cell lines with minimal variation between batches. True personalised medicine means the therapy itself is shaped by the recipient's biology. In autologous CFT, that principle is realised at the molecular level.

5a. Immune History and Inflammatory Phenotype

A patient's lifetime of microbial exposures, vaccinations, and prior infections shapes their T cell and B cell populations, their monocyte priming, and their baseline cytokine production capacity. When cells are harvested from that patient and conditioned in vitro, they carry this immunological history with them.

A patient with a history of acute inflammatory disease may have monocytes primed toward pro-inflammatory secretion patterns. A patient with chronic low-grade inflammation may display different baseline IL-6 and TNF-α production. These differences are not noise; they are biological information about that patient's physiology, encoded in their unique secretome.

5b. Age and Metabolic State

Cellular ageing is associated with changes in metabolic capacity, mitochondrial function, and stress response machinery. Older cells show reduced glycolytic capacity, altered NAD+ metabolism, and shifts in autophagy competence. These changes propagate into the secretome: cells from an older patient produce different patterns of growth factors and matrix-modulating enzymes than cells from a younger one.

A patient with metabolic syndrome will have circulating cells that express a different proteomic signature than a metabolically healthy individual. When those cells are conditioned, their secretory output reflects that metabolic context. The preparation is not generic; it is a molecular readout of that patient's current biological state.

5c. Genetic Background and Epigenetic Imprint

Individual genetic variation in cytokine genes, growth factor receptors, and inflammatory pathway components creates person-to-person differences in baseline secretory capacity. Beyond DNA sequence, epigenetic modifications accumulated over a patient's lifetime (DNA methylation patterns, histone acetylation marks, chromatin accessibility) determine which genes are poised for expression. These epigenetic marks persist in cultured cells and shape what that patient's cells will produce under conditioning.

6. The Personalisation Limits of Allogeneic Products

When mesenchymal stromal cells (MSCs) are derived from a single donor, expanded through multiple passages, and frozen in batches, every recipient receives cells bearing that donor's immune profile, age, health status, and epigenetic history. A 75-year-old patient with rheumatoid arthritis cannot receive a secretome that reflects their own inflammatory history; they receive the donor's secretome instead.

Marketing language often frames this as 'consistency': the same product every time. But consistency of what? Consistency of a product that has no biological relationship to the patient being treated. In true personalised medicine, consistency is not the goal; alignment with the patient's own physiology is.

This is not a subtle distinction. Published research demonstrates substantial inter-patient variability in cell-derived secretomes, meaning that the gap between any single donor's secretome and any individual recipient's optimal biological support may be considerable. The allogeneic model deliberately erases this personalisation. The autologous model preserves it.

7. Published Evidence on Inter-Patient Variability

The scientific literature documents substantial inter-individual variation in cell-derived secretomes. Zhukareva et al. (2010) demonstrated that human bone marrow stromal cells from different donors produce markedly different cytokine and chemokine secretion profiles under standardised culture conditions, with considerable variability in both baseline output and response to inflammatory stimuli. Turlo et al. (2023) used proteomic analysis to show that donor age significantly influences MSC secretome composition, with age-related decreases in specific proteins across both bone marrow and adipose-derived MSCs.

Extracellular vesicle cargo shows a similar pattern. Sanz-Rubio et al. (2018) measured circulating exosomal miRNA profiles across healthy subjects over consecutive weeks and found measurable inter-individual variation in exosomal miRNA signatures, even after controlling for temporal stability within individuals, suggesting that EV miRNA cargo carries a degree of individual biological specificity. Chen et al. (2013) showed that MSCs from donors at different developmental stages produce exosomes with different yields and cardioprotective efficacy, demonstrating that donor origin influences both the quantity and functional properties of the resulting EV preparation, though this study compared developmental maturity rather than individual-level variation among adult donors.

It should be noted that published inter-patient variability data derives primarily from MSC secretome studies. The degree of variation in white blood cell-derived secretomes produced under CFT conditioning protocols requires further systematic characterisation, though the underlying biological principle (that each patient's immune history shapes their cellular secretory output) applies to WBC biology as well. This makes deep proteomic analysis directly relevant to regenerative medicine-related products, as it provides the analytical resolution needed to characterise donor- or patient-specific secretome composition, assess batch-to-batch consistency, and link molecular profiles to intended biological function.

8. The Blood Type Analogy: Personalisation You Can Understand

Consider blood transfusion. Before blood typing was understood, transfusions were frequently fatal. Doctors assumed blood was blood, that what worked for one patient would work for another. The discovery of blood types revealed that even something as apparently simple as blood has critical molecular differences between individuals. Transfusing the wrong type triggers immune destruction of the transfused cells, with potentially lethal consequences.

A similar principle applies to regenerative biologics, though at a different scale of clinical consequence. A secretome produced by one individual's cells carries that individual's molecular fingerprint. Administering a donor-derived secretome to a recipient represents a form of biological mismatch: the material is less tailored to the recipient's immune and inflammatory state and may trigger some degree of immune recognition. This is not analogous to a dangerous transfusion reaction, but it does mean the preparation is standardised rather than intrinsically personalised to the recipient.

Autologous therapy is the closest biological equivalent to a fully self-matched preparation. The patient receives material produced by their own cells, carrying their own HLA, reflecting their own physiology. There is no donor-recipient mismatch because there is no second individual involved. This is the same immunological principle that makes autologous blood banking the preferred option for patients who can plan ahead for surgery.

9. CFT's Autologous Workflow: Both Principles in Practice

Cell-Free Therapy operationalises both the immunological and personalisation principles through a rigorous, GMP-compliant workflow. A minimum volume of whole blood is collected from the patient under standard phlebotomy protocols. This blood contains the patient's own platelets, circulating extracellular vesicles, plasma proteins, and growth factors. Platelets and many extracellular vesicles carry patient-specific membrane markers, including self-HLA, while the soluble components reflect the patient's own biochemical state.

White blood cells are isolated and subjected to environmental conditioning, a process in which cells are exposed to controlled microenvironmental stimuli (hypoxia, serum deprivation) that enhance their secretory output. The resulting cell-free secretome, containing no living cells, is collected, characterised, and cryopreserved for administration.

Several design features are critical. The patient's material is never combined with material from other donors, never supplemented with foreign growth factors or biologics, and never exposed to animal-derived products. Full chain-of-custody documentation is maintained from draw to delivery, with each sample tracked through a unique patient identifier that links collection, processing, testing, and release records.

Manufacturing occurs in a GMP-certified laboratory in Germany under strict quality-control standards. Each batch undergoes sterility testing (bacterial and fungal culture), endotoxin quantification (limulus amebocyte lysate assay), cell count verification to confirm acellularity, and potency assessment. Release criteria must be met before any preparation is approved for administration. These are not theoretical safeguards; they are documented, auditable quality gates applied to every individual patient's preparation. For physicians evaluating CFT, these release records provide objective evidence of product safety and consistency that complements the biological rationale for autologous sourcing.

Because the conditioning interacts with the patient's own cellular phenotype, the resulting secretome is doubly personalised: it reflects both the patient's baseline biology and their cells' specific response to the conditioning environment. A conditioning protocol designed to enhance anti-inflammatory secretion will engage differently with cells from a patient with active inflammation versus a patient with a quiescent immune profile. The conditioning is standardised; the output is personalised.

10. Implications for Physicians and Patients

For physicians, the dual advantage of autologous therapy simplifies clinical decision-making. Donor-recipient alloimmune mismatch and sensitisation are greatly reduced concerns, so post-treatment

monitoring can focus on clinical response rather than systemic immune complications. No immunosuppressive co-medication is required, reducing pharmacological burden and drug interactions. If multiple treatments are needed, autologous therapy can be repeated without risk escalation from increasing alloimmunisation.

Patient communication is similarly straightforward. The explanation is empowering and transparent: 'This treatment uses your own biological material. Your immune system is expected to recognise it as your own rather than as foreign material. The preparation reflects your unique biology rather than a generic donor product.'

The growing consumer preference for treatments derived from the individual's own biology reinforces this positioning. This preference is not driven by anti-medical sentiment; it reflects a mature, informed demand for transparency, autonomy, and biological compatibility. When asked 'What is in this treatment?', the answer is unambiguous: 'It is your own biological material.'

In this context, personal biobanking becomes a logical extension of autologous regenerative medicine: preserving one's own biological material while it is available and clinically usable creates a future-facing reserve of personalised therapeutic potential.

11. Conclusion

The human immune system's ability to distinguish self from non-self is simultaneously a protective mechanism and a barrier to allogeneic therapy. The same molecular uniqueness that triggers immune rejection of foreign material ensures that autologous preparations are more immunologically compatible than allogeneic alternatives and biologically personalised.

Three levels of evidence support the autologous case. First, established immunology: foreign HLA triggers alloimmune recognition, and autologous material avoids this trigger. This is not contested. Second, published evidence from EV research demonstrates that allogeneic vesicles can activate dendritic cells, trigger complement, and induce donor-specific antibodies, though the clinical significance of these findings for cell-free secretomes specifically is still being characterised. Third, a reasonable inference: if immune recognition of foreign material is a real biological event, then avoiding it through autologous sourcing is a meaningful advantage, even where the magnitude of clinical benefit remains to be fully quantified.

Beyond safety, autologous therapy delivers true personalisation at the molecular level. The secretome produced by a patient's own conditioned cells reflects that patient's immune history, inflammatory phenotype, age, metabolic state, and epigenetic background. Published evidence demonstrates substantial inter-patient variability in cell-derived secretomes, confirming that one donor's product cannot serve all recipients equally. In autologous manufacturing, this personalisation is preserved. In allogeneic manufacturing, it is deliberately erased.

Cell-Free Therapy operationalises both principles through a GMP-compliant workflow where the patient is the sole source and recipient of biological material. The material is processed in a

regulated environment, tested for safety and potency, and returned to the patient, never mixed with foreign material, never standardised to a donor profile.

From immunological principle to molecular personalisation to manufacturing practice, autologous approaches represent a fundamentally safer, more biologically relevant approach to cell-free therapy. The patient's own biology offers what no donor-derived product can: immune compatibility and molecular alignment with the recipient's unique physiology.

References

1. Larssen, P., Veerman, R. E., Güçlüler Akpinar, G., et al. (2019). Allogenicity boosts extracellular vesicle-induced antigen-specific immunity and mediates tumor protection and long-term memory in vivo. Journal of Immunology, 203(4), 825–834. [Link]

2. Chen, J., Powell, D. J., Jr., et al. (2025). Transplants foster B cell alloimmunity by relaying extracellular vesicles to follicular dendritic cells. Cell Reports, 44(6), 115832. [Link]

3. Prunevieille, A., et al. (2021). T cell antigenicity and immunogenicity of allogeneic exosomes. American Journal of Transplantation, 21, 2583–2589. [Link]

4. Toy, P., Gajic, O., Bacchetti, P., et al. (2012). Transfusion-related acute lung injury: incidence and risk factors. Blood, 119(7), 1757–1767. [Link]

5. Kopko, P. M., Marshall, C. S., MacKenzie, M. R., et al. (2002). Transfusion-related acute lung injury: report of a clinical look-back investigation. JAMA, 287(15), 1968–1971. [Link]

6. Zhukareva, V., Obrocka, M., Houle, J. D., et al. (2010). Secretion profile of human bone marrow stromal cells: donor variability and response to inflammatory stimuli. Cytokine, 50(3), 317–321. [Link]

7. Turlo, A. J., Herath, T. D. A., Peffers, M. J., et al. (2023). Mesenchymal stromal cell secretome is affected by tissue source and donor age. Stem Cells, 41(11), 1047–1059. [Link]

8. Sanz-Rubio, D., Martin-Burriel, I., Gil, A., et al. (2018). Stability of circulating exosomal miRNAs in healthy subjects. Scientific Reports, 8, 10306. [Link]

9. Chen, T. S., Yeo, R. W., Arslan, F., et al. (2013). Efficiency of exosome production correlates inversely with the developmental maturity of MSC donor. Journal of Stem Cell Research and Therapy, 3(2), 145. [Link]

10. Opelz, G., & Döhler, B. (2007). Effect of human leukocyte antigen compatibility on kidney graft survival: comparative analysis of two decades. Transplantation, 84(2), 137–143. [Link]

11. Yao, Y., Huang, J., Geng, Y., et al. (2015). Paracrine action of mesenchymal stem cells revealed by single cell gene profiling in infarcted murine hearts. PLoS ONE, 10(6), e0129164. [Link]

12. Théry, C., Witwer, K. W., Aikawa, E., et al. (2018). Minimal information for studies of extracellular vesicles 2018 (MISEV2018). Journal of Extracellular Vesicles, 7(1), 1535750. [Link]

13. Raposo, G., & Stoorvogel, W. (2013). Extracellular vesicles: Exosomes, microvesicles, and friends. Journal of Cell Biology, 200(4), 373–383. [Link]

14. Pittenger, M. F., Mackay, A. M., Beck, S. C., et al. (1999). Multilineage potential of adult human mesenchymal stem cells. Science, 284(5411), 143–147. [Link]

15. Xia, Y., Zhang, J., Liu, G., & Wolfram, J. (2024). Immunogenicity of extracellular vesicles. Advanced Materials, 36(33), e2403199. [Link]

16. Van Delen, M., Derdelinckx, J., Wouters, K., Nelissen, I., & Cools, N. (2024). A systematic review and meta-analysis of clinical trials assessing safety and efficacy of human extracellular vesicle-based therapy. Journal of Extracellular Vesicles, 13, e12458. [Link]

17. Li, S., Zhang, J., Sun, L., et al. (2025). Mesenchymal stem cell-derived extracellular vesicles: current advances in preparation and therapeutic applications for neurological disorders. Frontiers in Cell and Developmental Biology, 13, 1626996. [Link]

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.

Wellbeing International Foundation
Prepared with the guidance of, and reviewed under, the Scientific & Medical Advisory Committee.

June 2026