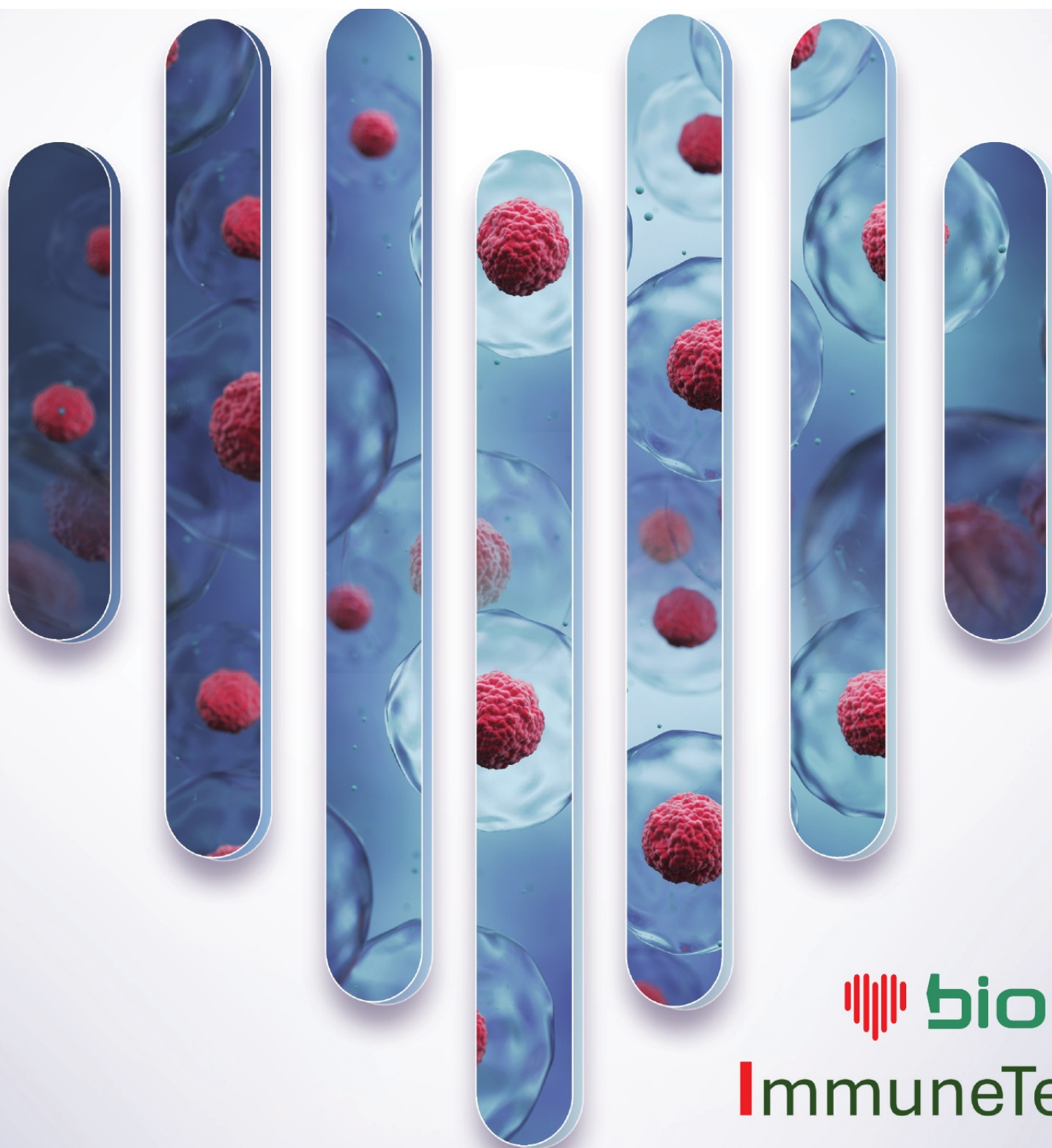


Understanding Your Immune System, Strengthening Clinical Decisions

A scientific approach to personalised health management through advanced immunoprofiling analyses.



 **bioart**
ImmuneTesTR

Clinical Application Areas

| ImmuneTesTR **inflammation**

| ImmuneTesTR **longevity**

| ImmuneTesTR **tissue repair**

| ImmuneTesTR **oncology**

Focus on the patient's biology,
not only on the disease.



A Clinical Necessity Beyond Routine Testing

Medicine is entering an era in which patient-specific biological responses shape clinical decisions beyond standard-of-care protocols. Routine tests can outline the overall picture, but in complex cases they may not explain why treatment has failed or what the next step should be.

The ImmuneTesTR immunoprofiling service is designed to identify the missing link that routine methods cannot reach. Its value lies in supporting the clinician's aim to intervene with the highest possible precision. Cellular dynamics and microenvironmental factors that cannot be resolved by standard laboratory capability become visible through this specialised service.

The main challenge in current practice is not a lack of data, but access to the right, specific data. This work is the product of advanced technology and specialist expertise beyond conventional diagnostic capacity. Its purpose is to support clinical predictability.

The ImmuneTesTR report is designed to:

- clarify biological mechanisms when standard tests are negative or inconclusive;
- detect treatment resistance at the cellular level before it becomes clinically apparent; and
- create a personalised roadmap that reflects each patient's biological history.

This is not a routine laboratory output, but boutique scientific support combining advanced cell characterisation and analytical methodologies available in only a limited number of centres worldwide. It equips clinicians not only to treat disease, but to manage its biological basis.

With our best wishes for its clinical usefulness.



Defining the Strategic Distribution of the Immune System

We characterise immune-system strategy through T-lymphocyte memory, response capacity, cytokine and cytotoxic-molecule production, exhaustion status and regulatory T-cell (Treg) levels. A small peripheral-blood sample is sufficient.

Multiparameter immunophenotyping includes: CD3, CD4, CD8, CD56, CD45RA, CCR7, TIGIT, PD-1, TIM-3, LAG-3, CTLA-4, CD28, CD27, CD69, HLA-DR, CD38, 4-1BB, CD25, CD127 and FoxP3.

Following immune-cell stimulation, T-cell responses are assessed using a broad cytokine and cytotoxic panel including IL-5, IL-13, IL-2, IL-6, IL-9, IL-10, IFN-gamma, TNF-alpha, IL-17A, IL-17F, IL-4, IL-22, Fas, FasL, granzyme A, granzyme B, perforin and granulysin.

Immune-profile data are further detailed through natural killer (NK) cell, inflammatory monocyte and immature-neutrophil subsets. Immunophenotyping of these populations focuses on CD56, CD14, CD16, CD19, CD66b and HLA-DR.

Together, these parameters define not only immune-cell subsets but functional capacity, creating the comprehensive picture termed immunoprofiling. Such a detailed marker panel is not a routine full blood count: it is used in advanced clinical research to investigate personalised immunotherapy and complex immune reactions. Our work combines advanced immunophenotyping, cell-culture stimulation, multiplex cytokine arrays and multiparametric flow cytometry.

Our immunoprofiling research supports the transition from trial-and-error medicine to precision medicine.





HIGH SE

MID

PA

Naive, Effector-Memory and Central-Memory T Cells

The relative proportions of naive, effector-memory and central-memory T cells provide a profile of the immune system's past experience, current activity and capacity to respond to future risk.

1. Naive T cells - insurance for the future

Role: Untrained cells waiting to recognise antigens not previously encountered.

Why it matters: A higher proportion suggests capacity to generate new antigen-specific responses against a novel virus or mutated malignant cell.

Clinical interpretation: A low proportion may indicate immunosenescence or depletion under chronic stress.

2. Effector-memory T cells - front-line responders

Role: Cells actively responding to a current threat.

Why it matters: A higher proportion may reflect a current or recent infection, chronic inflammation or readiness to respond.

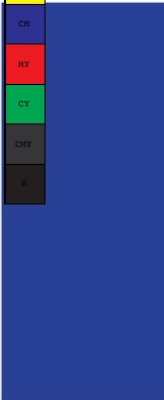
Clinical interpretation: Markedly elevated levels without clinical infection may point towards autoimmunity or persistent occult inflammation.

3. Central-memory T cells - archive and rapid recall

Role: Long-lived cells that retain information from previous infections or vaccination and expand rapidly on re-exposure.

Why it matters: They reflect immune experience and the durability of vaccine- or infection-derived protection.

Clinical interpretation: Deficiency may indicate impaired immune memory and vulnerability to recurrent infection.



Comparative Analysis of These Proportions

Cell type - higher proportion - lower proportion

Naive: strong adaptive capacity and a younger immune profile - immune fatigue, ageing and higher infection risk.

Effector: active infection, acute response or inflammation - weak defence or suppressed immunity.

Central memory: robust immune history and durable protection - memory loss and recurrent-infection risk.

Where and why is this used?

1. Cancer immunotherapy: assesses the patient's capacity to mount a cell-mediated response.
2. HIV/AIDS monitoring: tracks depletion of naive-cell immune reserves.
3. Chronic fatigue and inflammation: evaluates whether the body remains in a persistent high-effector response state.
4. Vaccine response: helps distinguish transient protection from durable immune memory.

Functional Markers on T Lymphocytes

The proportions of function-related molecules on T lymphocytes reveal cellular activation strategy, exhaustion and evidence of an active threat. These markers are functional status signals rather than arbitrary proteins.

1. Activation markers - Is the cell currently engaged?

HLA-DR, CD38, CD69, 4-1BB

- CD69 indicates very early activation.
- Co-expression of CD38 and HLA-DR, particularly in viral infections such as HIV or COVID-19, indicates marked activation and may reflect substantial inflammation.
- 4-1BB (CD137) is a strong survival and activation signal; in cancer immunotherapy it may support evidence that a cell is primed for attack.

2. Immune checkpoints and exhaustion - Is the cell losing function?

PD-1, TIM-3, LAG-3, CTLA-4, TIGIT

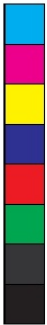
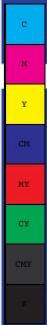
These inhibitory receptors normally prevent excessive responses, but sustained elevation in chronic disease can indicate T-cell exhaustion.

- PD-1 and CTLA-4 deliver inhibitory signals. Tumour cells may exploit these pathways to suppress T cells; high expression may therefore reflect immune inhibition.
- Co-expression of TIM-3 or LAG-3 with PD-1 is associated with deeper exhaustion.
- TIGIT restrains cytotoxic capacity in both NK and T cells.

3. Co-stimulatory markers - Is the cell still functionally young?

CD27, CD28

These molecules provide the second signal required for T-cell survival and proliferation. Preserved expression supports replicative and memory capacity. Loss of CD28 suggests repeated division and cellular senescence; CD28-negative cells are more frequent in older individuals with chronic inflammation.



How Can These Data Support Clinical Practice?

1. Cancer immunotherapy: High PD-1 or CTLA-4 expression may help identify inhibitory checkpoint activity and inform consideration of checkpoint-based treatment.
2. Chronic-infection monitoring: In HIV, hepatitis and other persistent infections, the proportion of exhausted cells helps characterise treatment response and disease course.
3. Autoimmune-disease analysis: Identifying the cells driving tissue-directed immune attack may support more targeted treatment.
4. Transplantation: Post-transplant T-cell activation markers such as CD69 and CD38 may provide early information about rejection risk.

Summary

Activation - HLA-DR, CD38, 4-1BB: indicates whether an active immune response is under way.

Exhaustion - PD-1, TIM-3, LAG-3: indicates whether immune function is becoming suppressed or ineffective.

Cellular youth/strength - CD27, CD28: reflects proliferative and survival potential.



Regulatory T Cells (Treg): The Immune System's Braking Mechanism

The proportion of regulatory T cells indicates the strength of immune restraint. If CD4 and CD8 T cells are the soldiers, Treg cells are the peacekeeping unit that prevents them from attacking civilians and calls a ceasefire when the battle has ended. Treg cells are typically identified as CD4+, CD25+, CD127-low/- and FoxP3+.

1. Autoimmune-disease risk and monitoring

Treg cells help prevent immune attack against self-tissue. Low numbers or impaired function are commonly reported in type 1 diabetes, multiple sclerosis, rheumatoid arthritis and lupus. Measurement can support assessment of autoimmune susceptibility or disease severity.

2. Cancer and tumour immunity

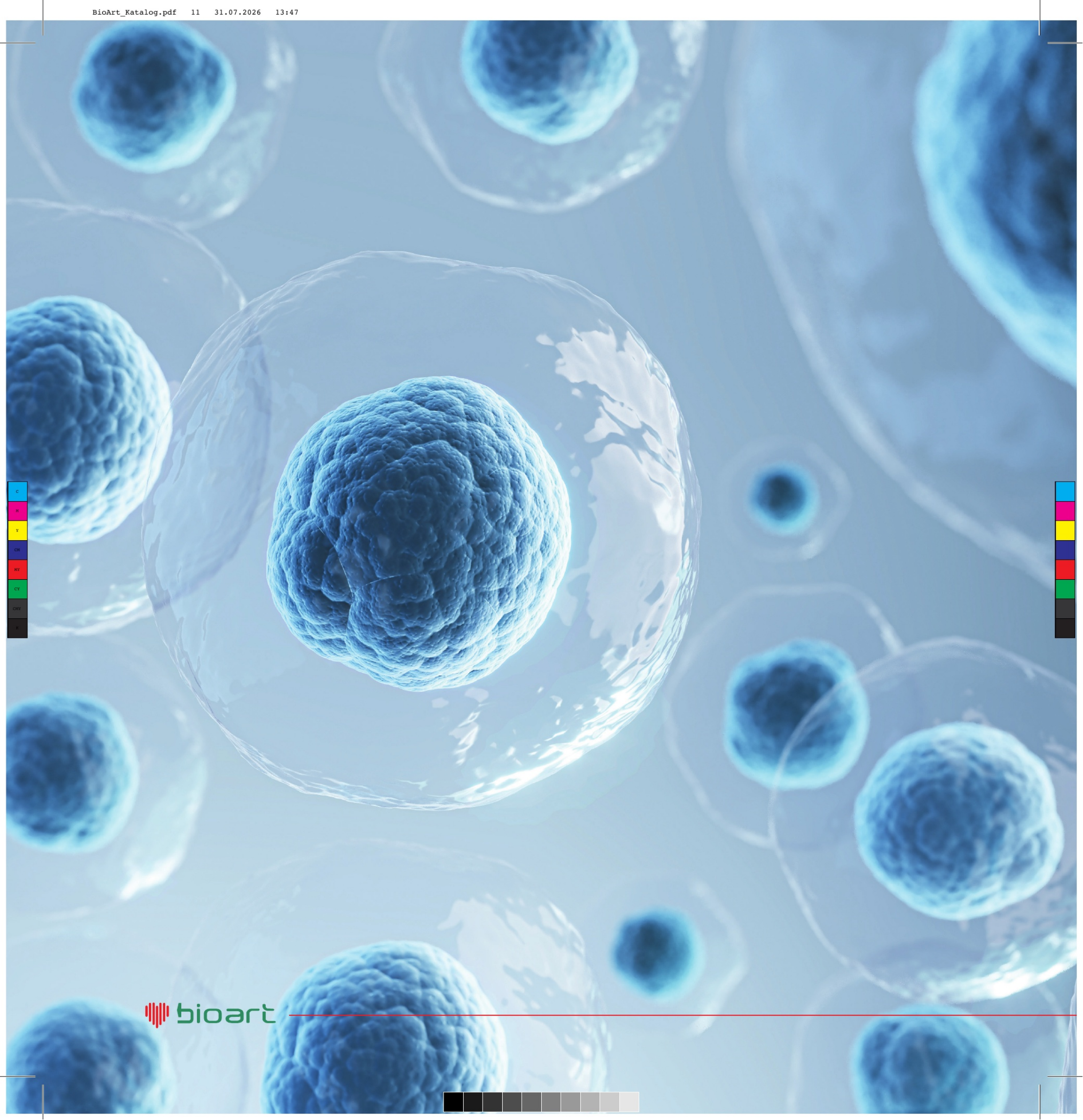
In cancer, a high Treg proportion can be unfavourable. Tumours may recruit Treg cells to create a protective immunosuppressive shield that limits cytotoxic T-cell activity. High intratumoural Treg levels may therefore indicate suppression of antitumour immunity and can inform immunotherapy research.

3. Allergy and hypersensitivity

Treg cells limit inappropriate responses to harmless stimuli such as pollen. Low proportions are observed in asthma, food allergy and eczema, where immune restraint may be insufficient.

4. Pregnancy and transplant tolerance

Treg cells contribute to tolerance of foreign tissue. Healthy pregnancy requires increased uterine tolerance to the semi-allogeneic embryo; low Treg levels may be associated with recurrent miscarriage. In transplantation, higher Treg activity may support graft tolerance.



Treg Interpretation and Functional Immune Profiling

Treg measurement commonly assesses FoxP3, CD25 and CD127 expression. In unresolved chronic inflammation or poor response to cancer treatment, clinicians may examine the immune system's accelerator-brake balance.

What Treg proportions may suggest
Autoimmunity - usually lower: inadequate restraint of self-reactive immunity.
Cancer - often higher: excessive suppression and reduced antitumour activity.
Allergy - usually lower: unnecessary responses to harmless substances.
Healthy pregnancy - higher locally: successful tolerance of fetal tissue.

Why perform this functional panel?
After in vitro stimulation of immunophenotyped cells, analysis of a broad cytokine and cytotoxic-molecule panel provides a functional fingerprint. Cell proportions tell us how many soldiers are present and their ranks; functional markers indicate how strongly they respond and which weapons they use.

1. Determining response type: Th1, Th2 and Th17 balance
- Th1 - IFN-gamma, TNF-alpha, IL-2: intracellular pathogens and cancer; high levels indicate strong cellular defence, while excessive levels may suggest chronic inflammation or autoimmunity.
 - Th2 - IL-4, IL-5, IL-13, IL-9: parasite defence and allergic responses; elevation is associated with asthma, eczema and hay fever.
 - Th17/Th22 - IL-17A, IL-17F, IL-22, IL-6: fungal defence and mucosal-barrier maintenance; dysregulation can contribute to tissue injury in psoriasis, Crohn's disease and rheumatoid arthritis.



2. Measuring Cytotoxic Capacity

Granzyme A, granzyme B, perforin and granulysin
These are the effector molecules used by CD8+ T cells and NK cells. They indicate the potential to kill virus-infected or malignant cells. Cell counts may be normal, yet low production of these proteins can indicate inadequate cytotoxic armament.

3. Cell Death and Regulation: Apoptosis and Control sFas, sFasL and IL-10

- sFas/sFasL: part of programmed cell death. Disruption may allow immune cells to survive when they should be deleted, contributing to lymphoproliferative disease, autoimmunity or tumour escape.
- IL-10: an anti-inflammatory stop signal. Low levels can permit persistent inflammation; excessive levels may be associated with immunosuppression.

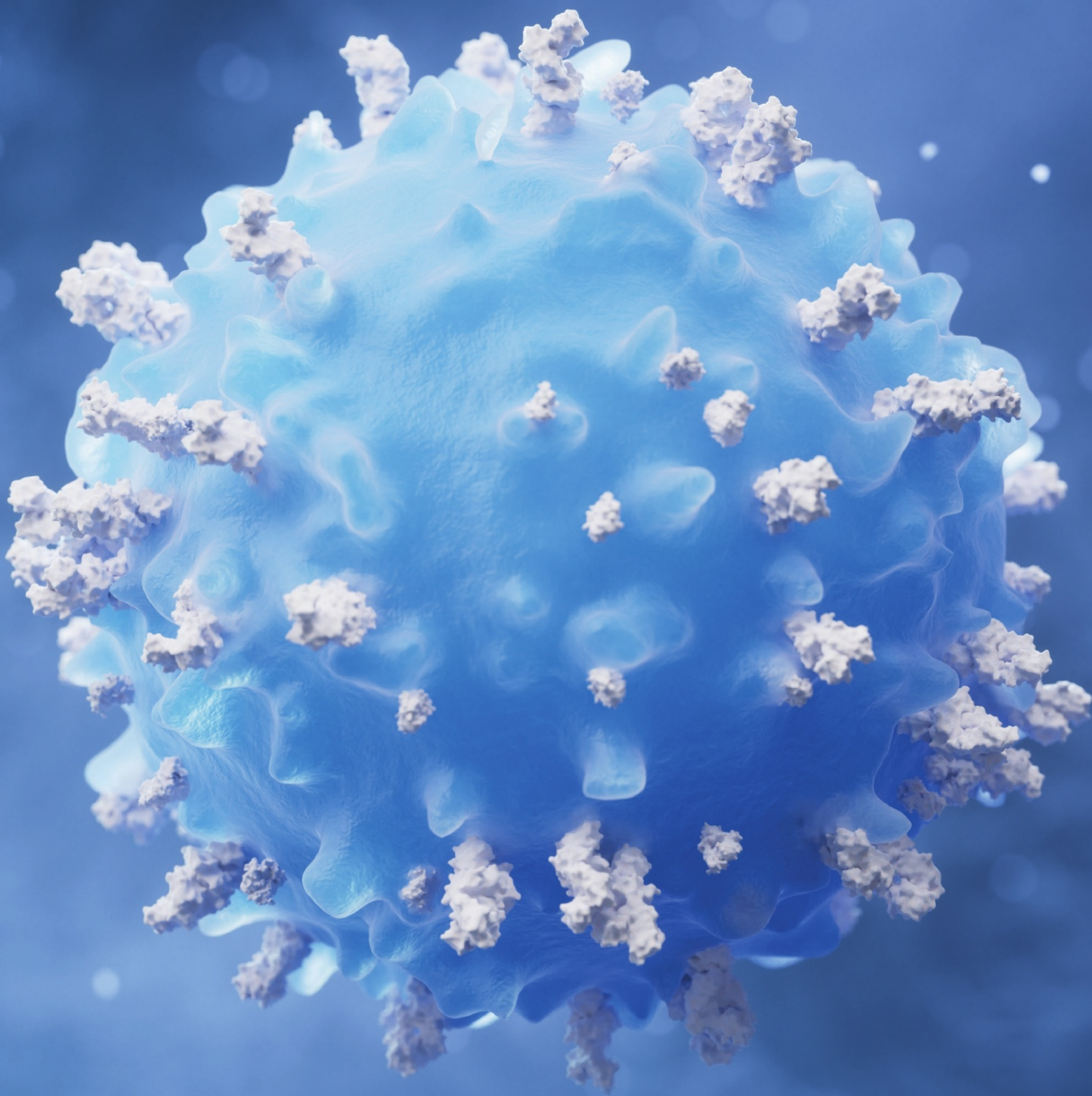
4. What Does This Information Provide in Clinical Practice?

- 1. Cytokine-storm monitoring: In severe viral infection, sepsis and severe COVID-19, IL-6, TNF-alpha and IL-17 can provide early evidence of hyperinflammation and risk of deterioration.
- 2. Personalised biologic selection: A dominant IL-17 or TNF-alpha pathway may help inform selection of pathway-directed therapies such as anti-IL-17 or anti-TNF agents where clinically indicated.
- 3. Allergy and asthma phenotyping: Determining whether asthma is driven by an IL-5-associated eosinophilic pathway can materially alter treatment strategy.
- 4. Immunodeficiency analysis: Failure to produce IFN-gamma or IL-2 after stimulation may indicate a profound cellular immune defect.

Parameter map
Inflammation/Th1 - TNF-alpha, IFN-gamma: antiviral and antitumour activity; overall inflammatory intensity.
Allergy/Th2 - IL-4, IL-5, IL-13: allergic tendency and parasite defence.
Autoimmunity/mucosa - IL-17A/F, IL-22, IL-6: tissue-damage risk and barrier protection.
Cytotoxicity - perforin, granzymes: the cell's direct killing capacity.
Suppression - IL-10, sFas, sFasL: the system's ability to terminate a response.

In short, this panel measures not only the presence of immune cells but their performance and direction. It is generally used in advanced research and complex cases that do not respond to standard treatment.





Natural Killer (NK) Cell Subsets

NK cell subsets are the immune system's rapid-response force. Their proportions provide information about antiviral defence, tumour-cell killing capacity and reproductive immunology. NK cells are divided principally by CD56 and CD16 expression.

1. CD56bright - cytokine-producing regulators

Approximately 10% of NK cells. Rather than acting primarily through direct killing, they secrete cytokines that coordinate other immune cells.

- Function: regulation of inflammatory balance.
- Higher levels: may occur in lymphoid tissue, chronic inflammatory states or early immune responses.
- Lower levels: may suggest reduced immune coordination and have been described in conditions such as chronic fatigue syndrome.

2. CD56dim - cytotoxic effectors

Approximately 90% of circulating NK cells.

- Function: direct cytotoxic elimination of virus-infected and potentially malignant cells.
- Higher levels: may indicate an active defensive state or robust cytotoxic reserve.
- Lower levels: may be associated with recurrent viral infections such as HSV, HPV or EBV, immune deficiency and impaired tumour surveillance.



Specific Uses of NK-Cell Subset Analysis

A. Cancer surveillance and monitoring

The proportion and cytotoxic competence of NK cells, particularly the CD56dim subset, reflect elements of tumour immune surveillance. Serial measurement during cancer treatment may provide information about response, including immunotherapy-associated change.

B. Reproductive health: infertility and miscarriage

Uterine NK-cell number and activity require careful balance for implantation. In recurrent miscarriage or repeated IVF failure, excessive activation is sometimes investigated as a possible contributor to embryo-directed immune responses.

C. Control of viral infection

In chronic hepatitis, HIV or herpesvirus infection, NK-subset distribution may help indicate whether antiviral containment is effective. A low cytotoxic subset can be associated with reduced viral control.

D. Ageing and immunity: immunosenescence

NK-cell numbers may rise with age while cytotoxic quality declines. Subset analysis can help characterise biological immune resilience.

Summary

CD56bright: fewer cells; cytokine-producing regulation - immune coordination and reproductive immune balance.
CD56dim: predominant cytotoxic cells - tumour surveillance and viral clearance.

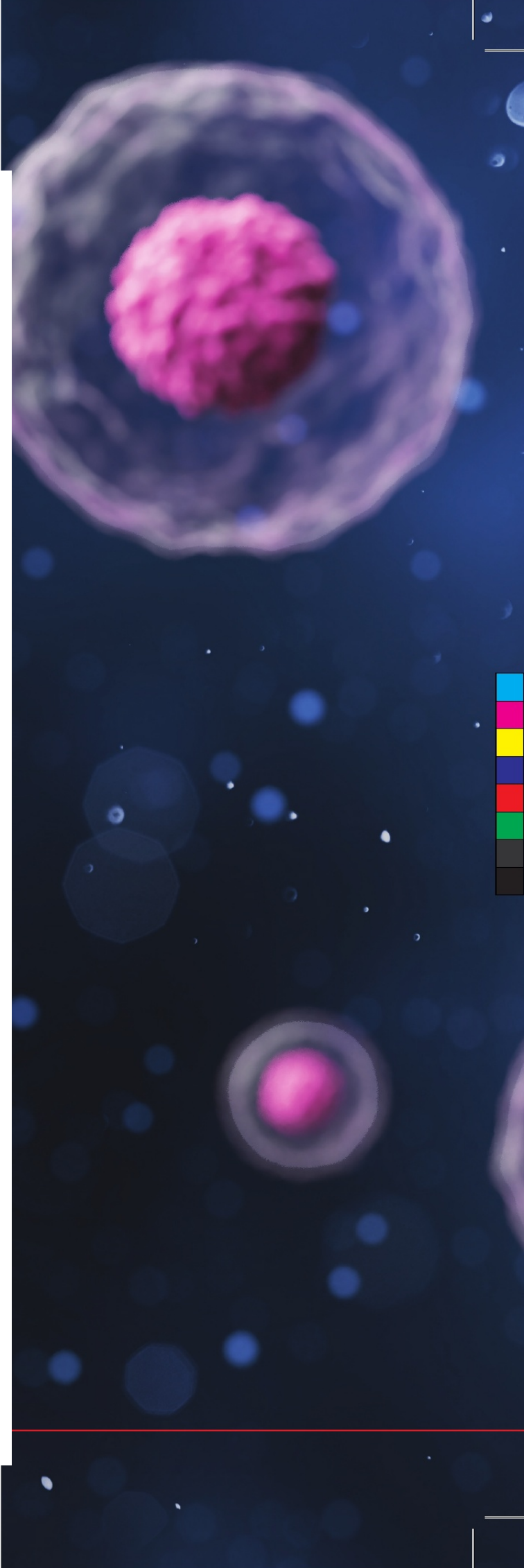
NK-subset analysis therefore examines not only how many cells are present, but whether they are regulatory or cytotoxic and whether they are performing their expected roles.

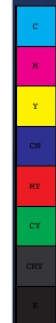
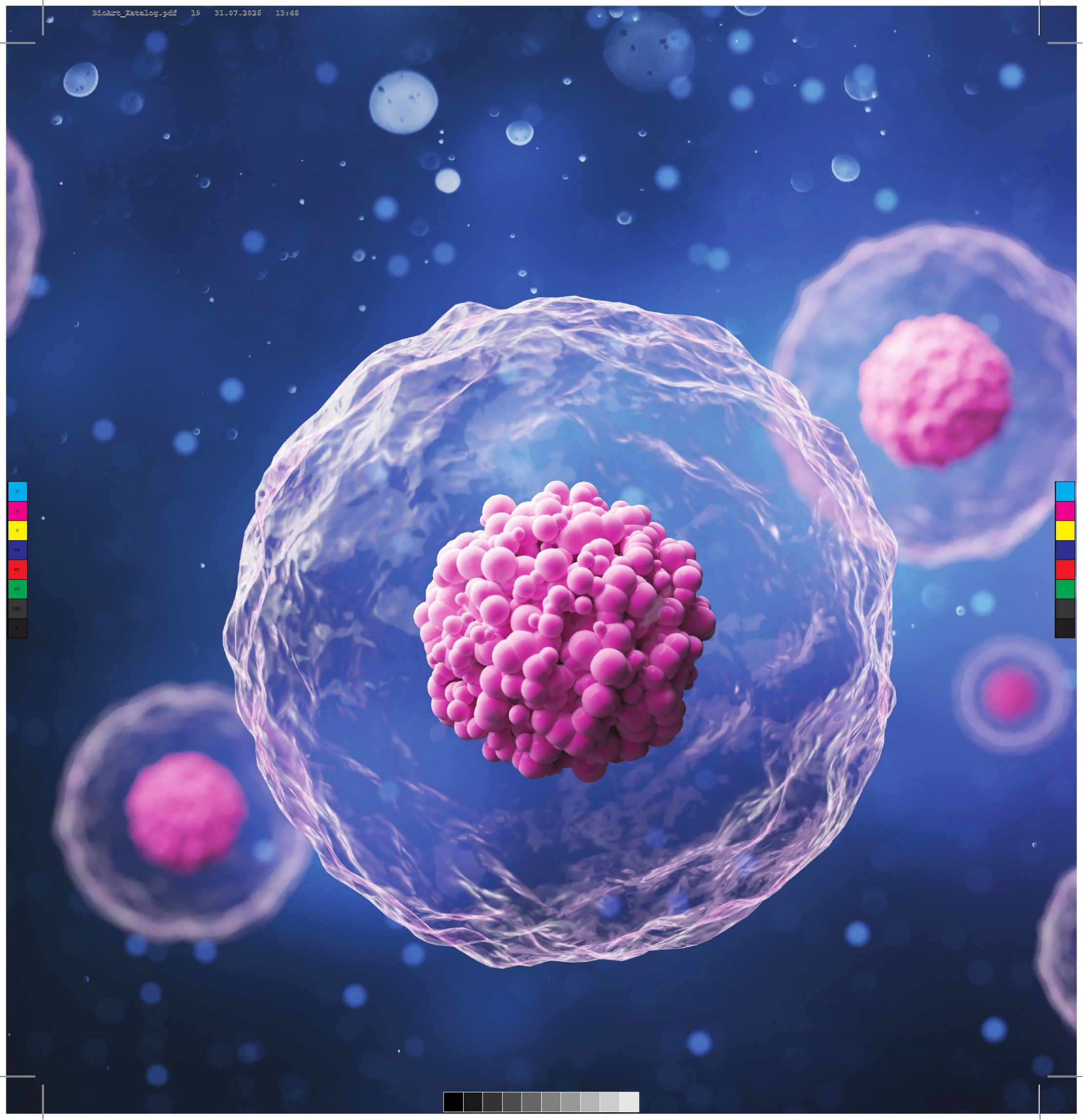


Monocyte Subsets: A Sensitive Mirror of Inflammation

Monocyte-subset proportions help characterise the type and intensity of inflammation and the capacity for tissue repair. Monocytes comprise three dynamic circulating subsets.

1. Classical monocytes - CD14++ CD16-
Approximately 85-90% of the monocyte pool and the immune system's first-response team.
 - Migrate early to infection, phagocytose bacteria and initiate repair.
 - Rise in acute infection, physical stress and after heavy exercise.
 - Low levels may occur with marrow suppression or severe sepsis.
2. Intermediate monocytes - CD14++ CD16+
Approximately 5% of monocytes and often the most clinically informative subset.
 - Combine phagocytosis with high cytokine output, acting as alarm cells.
 - Elevation may indicate substantial chronic inflammation or vascular dysfunction and has relevance in cardiovascular risk, asthma exacerbation and kidney-disease monitoring.
3. Non-classical monocytes - CD14+ CD16++
Approximately 5-10% of monocytes; vascular patrolling cells.
 - Survey vessel walls and contribute to damage control.
 - May increase in chronic viral infection, including HIV, and in some autoimmune diseases such as lupus.





Clinical Value of Monocyte-Subset Proportions

- A. Cardiovascular-risk analysis
Elevated intermediate monocytes are associated with atherosclerosis and cardiovascular risk. Classical-monocyte abundance may also provide information about tissue repair after myocardial infarction.
- B. Autoimmune and inflammatory-disease monitoring
In rheumatoid arthritis, Crohn's disease and related conditions, increased non-classical monocytes may accompany disease activation or flare.
- C. Infection differentiation
Classical monocytes often predominate in bacterial infection, whereas chronic viral burden may alter the non-classical compartment.
- D. Cancer and the tumour microenvironment
In some cancers, monocytes migrate into tumours and differentiate into tumour-associated macrophages (TAMs). Subset distribution may help characterise whether the myeloid response supports antitumour immunity or inadvertently favours tumour progression.

Summary
Classical - 85-90%: elevation in acute infection, tissue injury and stress.
Intermediate - about 5%: elevation in cardiovascular disease, chronic kidney disease and metabolic syndrome.
Non-classical - 5-10%: elevation in viral infection, vascular-wall inflammation and autoimmunity.

Mature and Immature Neutrophil Subsets

Neutrophils are the body's front-line defence against bacterial infection. Separating mature from immature forms provides information about bone-marrow stress and infection severity.

1. Assessing infection severity

Most circulating neutrophils are mature, segmented and CD16++. An increase in immature, low-CD16 forms, particularly band forms, indicates accelerated marrow release before full maturation. This can help distinguish a limited viral illness from serious bacterial infection such as sepsis.

2. Evaluating bone-marrow function

An immature-cell rise during infection may be reactive and appropriate. Very immature cells or blasts in the absence of infection may indicate marrow pathology, including leukaemia, and require formal diagnostic assessment.

3. The immature-granulocyte parameter

Modern analysers report immature granulocytes automatically. An increase can precede fever in systemic inflammation and may support early sepsis assessment. Serial values can also assist postoperative monitoring for recovery or infectious complications.

Mature versus immature neutrophils

Mature: segmented 3-5-lobed nucleus; full microbial killing capacity; normally approximately 95-100%.

Immature: band-shaped U- or C-shaped nucleus; less developed killing capacity; normally approximately 0-5%.

Reasons for increase: severe infection, physiological stress, trauma or haematological disease.

These proportions help clinicians judge whether the body is controlling the response or exhausting marrow reserves. Monitoring can be particularly important in intensive care and neonatal medicine.



Revealing the Hidden Mechanisms of Wound Healing

Immune profiling of wound tissue is designed to reduce trial-and-error in chronic-wound management, optimise treatment costs and support limb-preservation decisions with biological data. Only a small tissue sample is required.

By integrating macrophage, monocyte and neutrophil subsets with cytokines and growth factors released by stimulated cells, we create a personalised map of regenerative biology.

Wound-tissue leukocyte immunophenotyping: CD45, CD14, CD16, CD66b, CD163, CD206, CD80, CD38 and CD209.

Stimulated-cell growth-factor analysis: TGF-beta1, angiopoietin-2, EGF, EPO, G-CSF, HGF, M-CSF, PDGF-AA, PDGF-BB, SCF, FGF-basic and VEGF.

Macrophage Subtypes in Wound Healing

M1- and M2-oriented macrophage subsets help identify where healing has stalled and whether tissue-directed activity is destructive or reparative. Macrophages coordinate both the demolition and construction phases of healing.

1. Inflammation-repair balance: M1 versus M2

M1 macrophages - classically activated, pro-inflammatory

Predominate early, kill bacteria, clear necrotic tissue and release inflammatory mediators such as TNF-alpha and IL-1beta. Persistent elevation can trap the wound in the inflammatory phase and drive ongoing tissue injury, a hallmark of chronic wounds including diabetic foot ulcers.

M2 macrophages - alternatively activated, reparative

Emerge later, resolve inflammation, promote collagen production and initiate angiogenesis. A rising M2 proportion indicates progression into the proliferative and repair phase.

2. Estimating chronic-wound risk

Failure of the phenotypic switch from M1 to M2 supports classification as a chronic wound and may indicate a need for immune-modulating interventions beyond dressings alone.

3. Predicting fibrosis and scarring

Although M2 cells support repair, excessive or uncontrolled activation may drive collagen accumulation. Detailed analysis of M2a, M2b and M2c subsets may help estimate risk of keloid or hypertrophic scarring.

4. Monitoring treatment response

Changes in macrophage polarisation can provide biological evidence of response to advanced wound therapies including cell-based treatments, exosomes, growth factors and negative-pressure wound therapy.



Macrophage Summary and Neutrophil Phenotypes in Wound Tissue

Macrophage-subtype summary

M1 - pro-inflammatory/destructive - inflammation, days 0-3: excess activity may prevent closure and increase tissue death.
M2 - anti-inflammatory/reparative - proliferation, days 3-21: excess activity may increase fibrosis and scarring.
Macrophage subtype analysis therefore helps estimate the wound's biological age and reparative direction.

Assessing CD14 and HLA-DR on wound neutrophils provides information not only about their presence but their functional phenotype. These markers are normally low, but change within the wound microenvironment.

1. Acquisition of antigen-presenting properties

Neutrophils normally focus on phagocytosis. Increased HLA-DR (MHC class II) suggests the capacity to present antigen to T cells, indicating a more complex, regulated immune phase rather than simple inflammation alone.

2. Inflammatory intensity and sepsis risk

CD14 is chiefly a monocyte marker but can appear on neutrophils. Increased expression may indicate heightened sensitivity to bacterial products such as lipopolysaccharide. Wound neutrophils expressing high CD14 and HLA-DR may provide early evidence of persistent local infection or systemic inflammatory-response risk.

3. Wound-healing neutrophils: the N2 phenotype

Like the M1/M2 macrophage distinction, neutrophils may be described as N1-like destructive or N2-like reparative. HLA-DR and CD14 expression may indicate a hybrid phenotype capable of regulatory or repair signalling. Balanced expression can accompany transition into remodelling; excessive expression may indicate immunopathology and uncontrolled tissue injury.



Summary of Neutrophil Subtypes in Wound Tissue

Marker - status - interpretation in wound tissue

HLA-DR - high: neutrophils may acquire antigen-presenting properties and engage T-cell responses.

CD14 - high: heightened alert to bacterial products and possible active-infection risk.

Both increased: transition towards a hybrid regulatory or reparative phenotype may be occurring.

Why assess these markers?

In diabetic foot wounds and non-healing chronic ulcers, advanced flow-cytometric research can determine whether neutrophils express these markers, helping reveal whether the wound-site immune response is functionally impaired or following an inappropriate defence strategy.

Monocyte Subtypes in Wound Tissue

Wound-tissue monocyte subsets indicate whether the wound remains in acute clearance or has progressed to repair and remodelling. Because monocytes differentiate into macrophages after entering tissue, their profile provides a current field report of healing potential.

1. Classical monocytes - CD14++ CD16-: the clearance team

Arrive early, kill pathogens, remove debris and release inflammatory cytokines. They reflect defensive capacity in the inflammatory phase. Persistent elevation can indicate failure to transition into repair and risk of chronicity.
2. Intermediate monocytes - CD14++ CD16+: alarm and coordination

Bridge inflammation and repair through phagocytosis and intense signalling-molecule release. An increase may indicate attempted transition from defence to repair. In cardiovascular or chronic kidney disease, analysis may help characterise wound-healing capacity.
3. Non-classical monocytes - CD14+ CD16++: surveillance and repair

Associated with vascular surveillance and tissue renewal. Their presence can support evidence that the wound has entered angiogenesis and remodelling.

Clinical uses

- Non-healing wounds: identifies failure to switch from classical CD16- to reparative CD16+ phenotypes.
- Inflammatory characterisation: elevated non-classical cells may point towards chronic viral burden or autoimmune activity.
- Treatment planning: low reparative subsets may support consideration of growth-promoting factors such as VEGF or PDGF, or immunomodulatory strategies where appropriate.

In summary, monocyte subsets indicate whether the wound is biologically in a defence phase or a construction phase.





Growth-Factor Profiling of Wound Tissue

Following in vitro stimulation of wound-derived cells, a broad cytokine and growth-factor panel provides critical information on healing speed, repair quality and vascularisation. TGF-beta1, angiopoietin-2, EGF, EPO, G-CSF, HGF, M-CSF, PDGF-AA, PDGF-BB, SCF, FGF-basic and VEGF are classified as growth factors.

1. Capacity for new-vessel formation: angiogenesis

VEGF, angiopoietin-2, FGF-basic

New vessels are essential for oxygen and nutrient delivery. Low values can indicate an ischaemic wound bed and help explain stalled healing, particularly in diabetic foot ulcers.

2. Tissue construction and cell proliferation

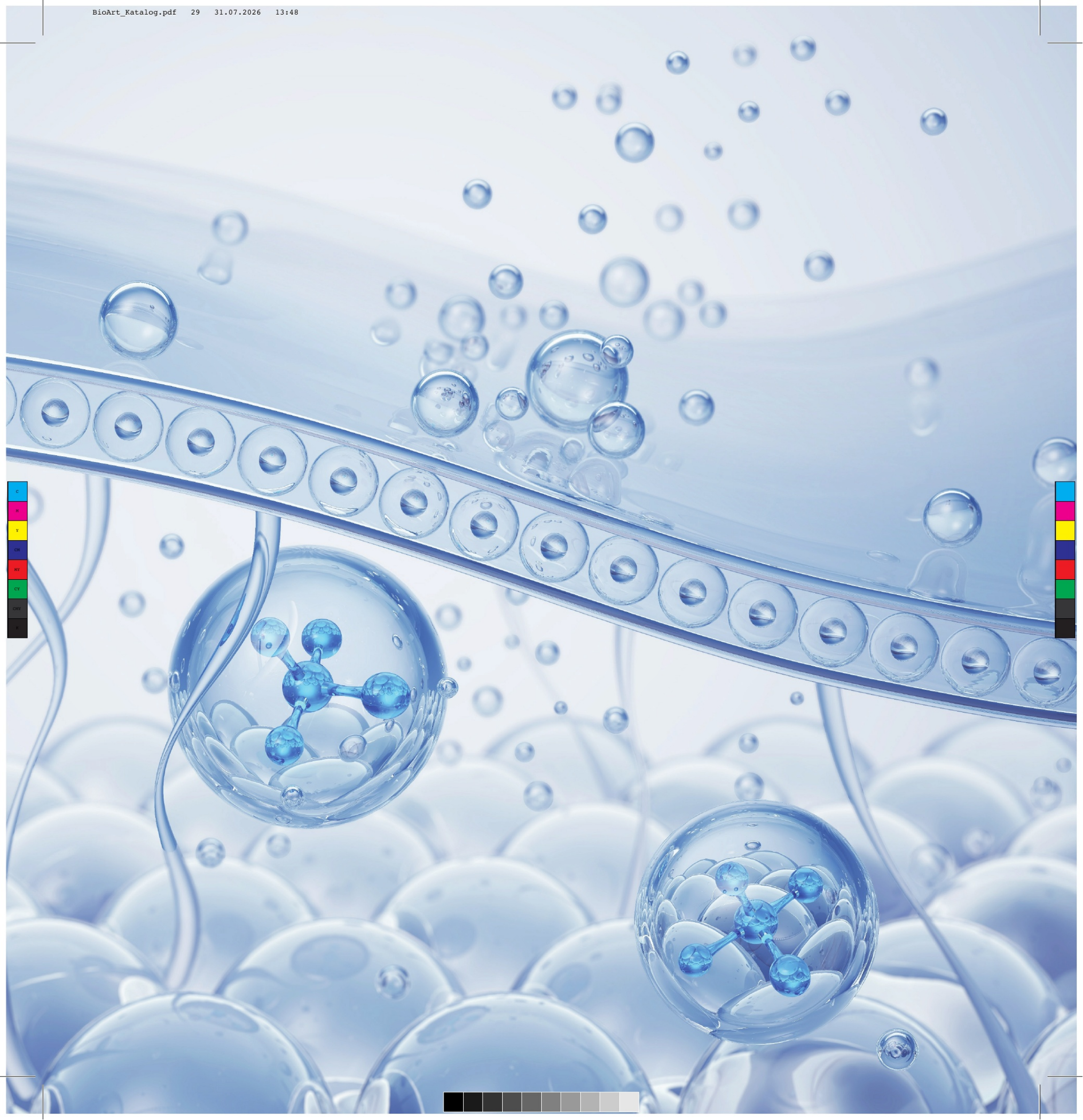
EGF, PDGF-AA/BB, FGF-basic, HGF, SCF

These factors stimulate keratinocytes and fibroblasts to proliferate and close the wound. Their relative levels help characterise closure capacity.

3. Scarring and risk of chronicity

TGF-beta1

TGF-beta1 is a central architect of repair but excess can drive keloid or hypertrophic scarring. Low levels may indicate failure to progress into the closure phase.



Concise Functional Map of the Parameters

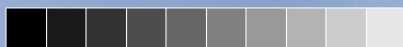
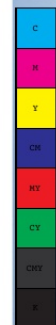
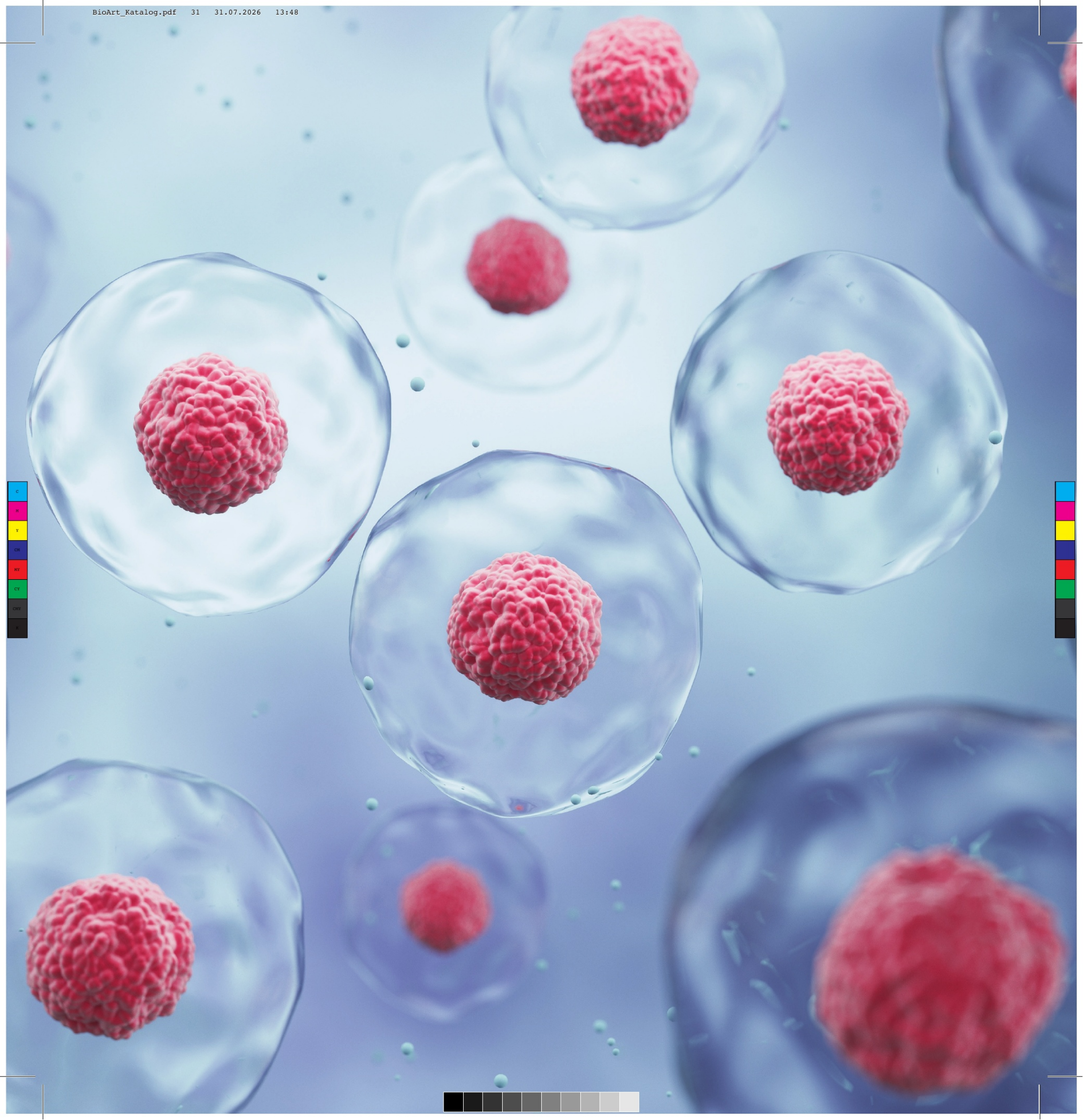
Vessel-forming factors - VEGF, Ang-2, FGF-basic: support perfusion and oxygenation.
Cell-proliferation factors - EGF, HGF, PDGF: fill tissue deficits with new tissue.
Cell-recruitment factors - G-CSF, M-CSF, SCF: attract immune and repair cells to the site.
Remodelling factor - TGF-beta1: regulates collagen production and tissue stiffness.
Haematopoietic/oxygen-support factor - EPO: supports local oxygen capacity.

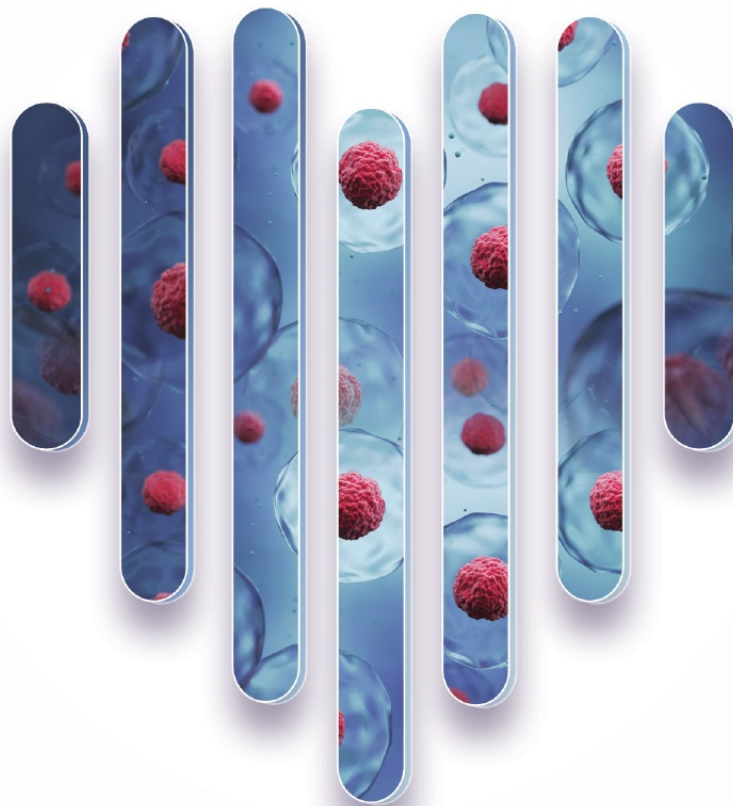
Clinical applications

- 1. Chronic-wound analysis: identifies the biochemical phase in which healing has stalled in diabetic, venous or pressure ulcers.
- 2. Personalised treatment: low VEGF may support vascularisation-focused strategies; low PDGF may inform consideration of growth-factor products or platelet-rich plasma where clinically appropriate.
- 3. Prediction of surgical success: helps estimate tissue viability before major surgery or skin grafting.
- 4. Aesthetic and plastic surgery: may support assessment of persistent scarring and the likely quality of tissue repair.

Overall, the analysis indicates how efficiently the wound's biological construction site is working, whether the required cellular materials are present and whether additional growth-factor support may be needed.







Don't Lose Time!

 info@immunetestr.com.tr

 immunetestr.com.tr