

Chemotaxis of Leukocytes

Chemical guidance, receptor signaling, cell migration and inflammatory patterns

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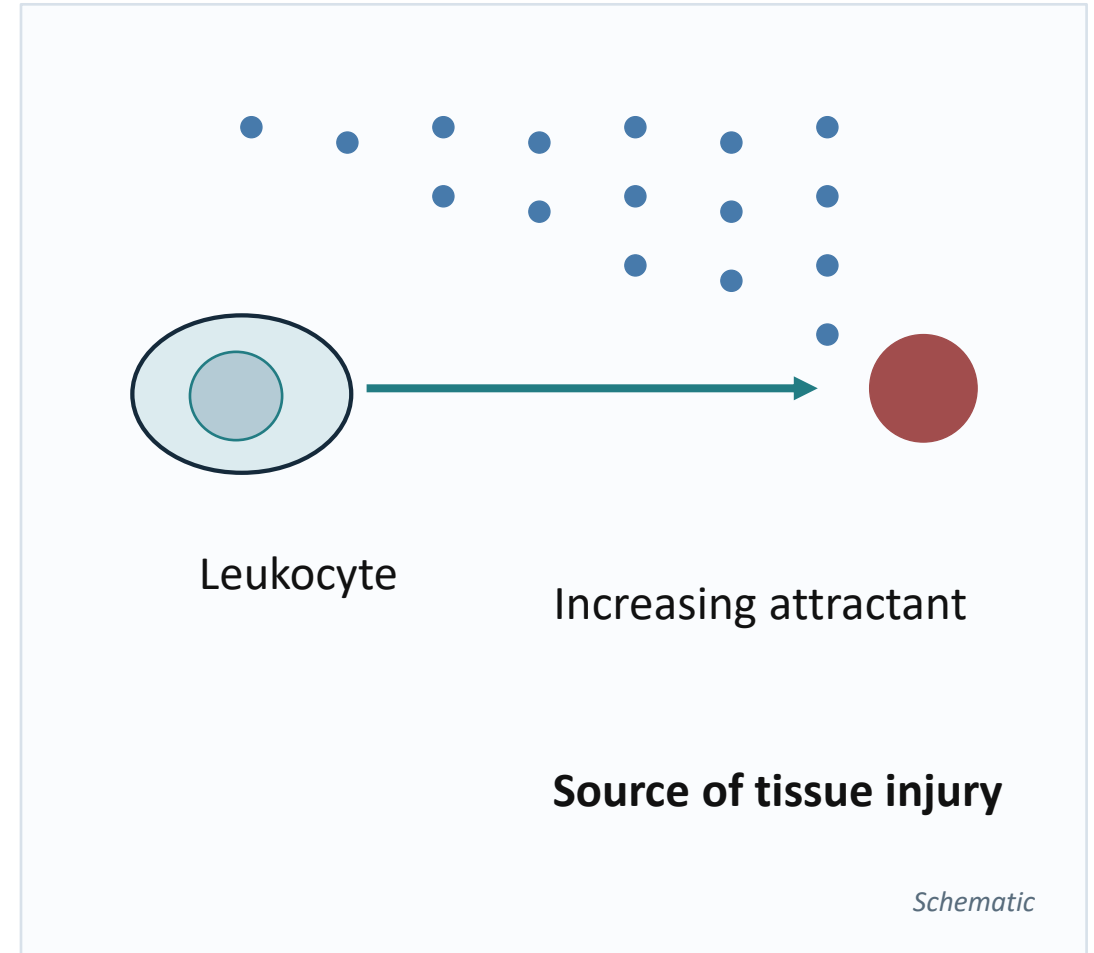
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Learning objectives

- Define chemotaxis and distinguish it from leukocyte adhesion and diapedesis.
- Identify microbial and host-derived chemoattractants and their receptors.
- Explain GPCR-to-cytoskeleton signaling and polarized movement.
- Interpret time- and stimulus-dependent patterns of leukocyte recruitment.
- Connect chemotaxis with host defense and its clinical limitations.

Chemotaxis is directional leukocyte locomotion

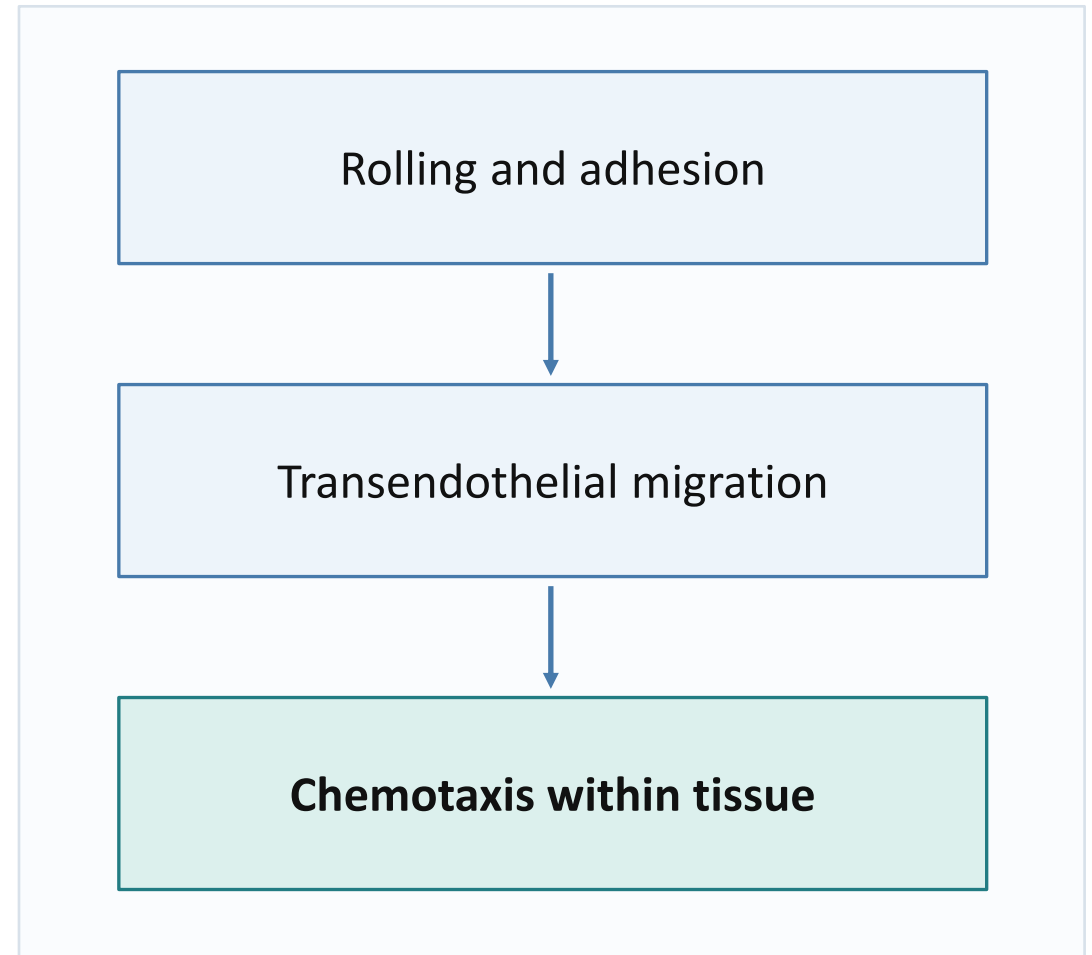
- Chemotaxis means movement along a chemical concentration gradient.
- Leukocytes in tissues move toward a source of infection or cell injury.
- Guidance differs from simply increasing the number of circulating cells.



The gradient supplies orientation; Cellular motility supplies displacement.

Chemotaxis occurs after leukocyte extravasation

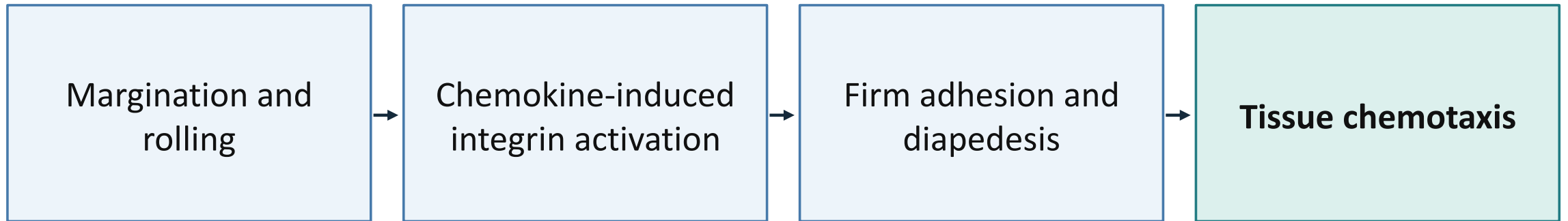
- Bloodborne leukocytes first interact with activated vascular endothelium.
- They then traverse the endothelial barrier and basement membrane.
- Directional tissue migration brings them to the offending stimulus.



Chemotaxis is a tissue-migration phase within a broader recruitment process.

Leukocyte recruitment is a multistep process

Each stage permits the next; Different molecular systems predominate.



Defect at any upstream stage can reduce delivery to an injury site.

Rolling is not the same as chemotaxis

- Selectins mediate initial transient endothelial contacts.
- Integrins stabilize attachment before vascular exit.
- Chemoattractants guide cellular movement in the interstitium.

Rolling

Transient adhesive contacts under blood flow.

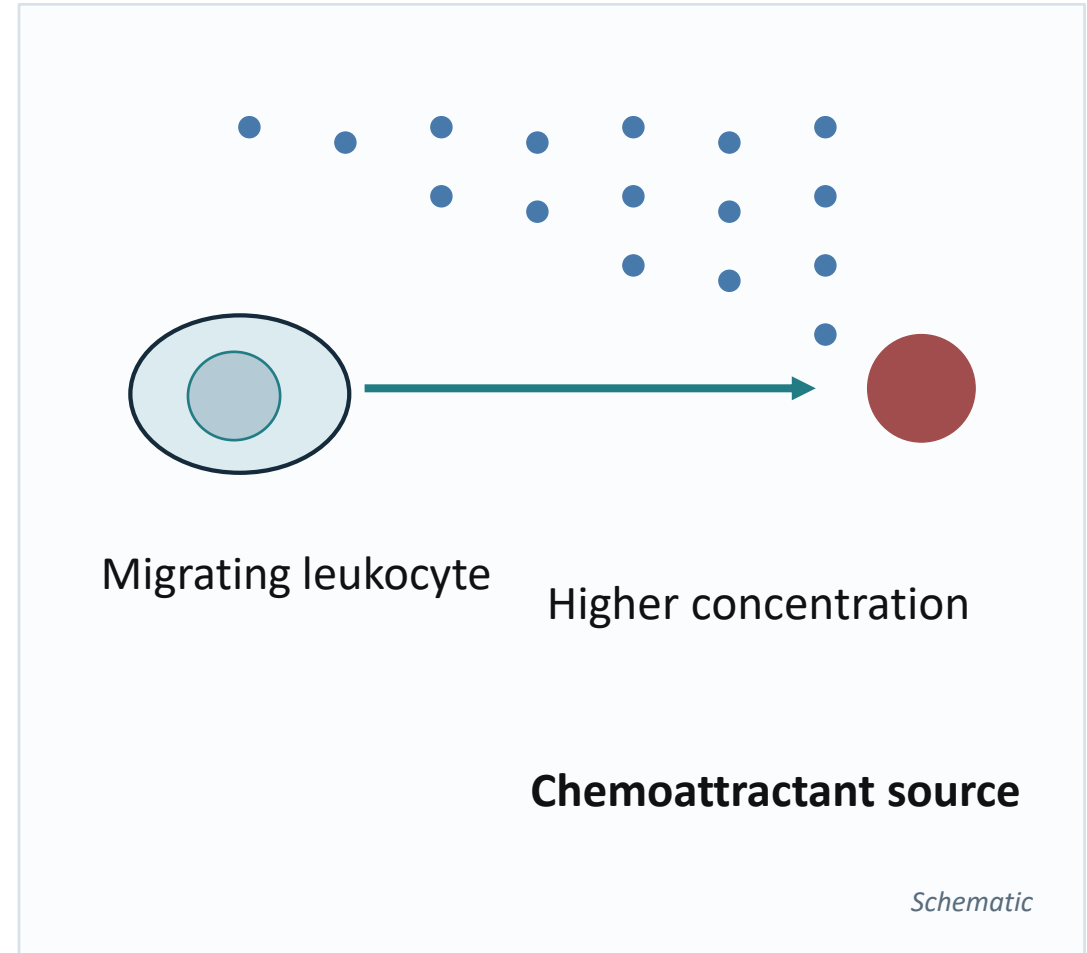
Chemotaxis

Directional locomotion along a tissue chemical gradient.

Terminology reflects distinct mechanisms and anatomic compartments.

A chemical gradient creates spatial information

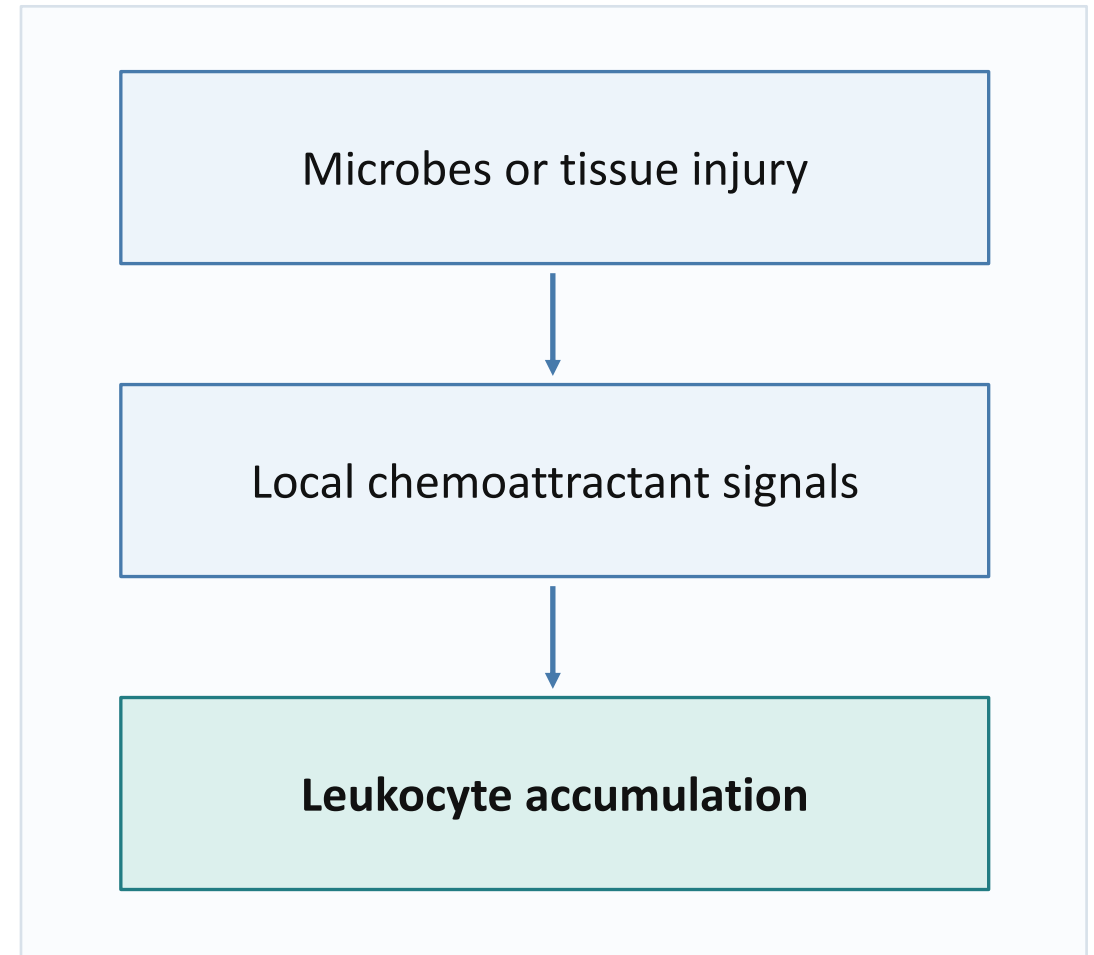
- The source of injury or microbes contributes chemoattractant molecules.
- Concentration differences orient leukocyte movement.
- The cell migrates toward the stronger chemotactic stimulus.



The orientation cue is chemical rather than mechanical.

Sources of chemoattractants cluster at injured tissue

- Microbial products may themselves attract leukocytes.
- Injured tissue and host inflammatory pathways also generate mediators.
- Directional movement concentrates leukocytes where defense is required.



Injury generates a local focus for recruitment.

Chemoattractants have exogenous and endogenous origins

- Exogenous cues include bacterial N-formyl peptides and certain bacterial lipids.
- Endogenous cues include chemokines, complement fragments, and LTB₄.
- Both origins can contribute to the same acute inflammatory focus.

Exogenous

Compounds supplied by microbes.

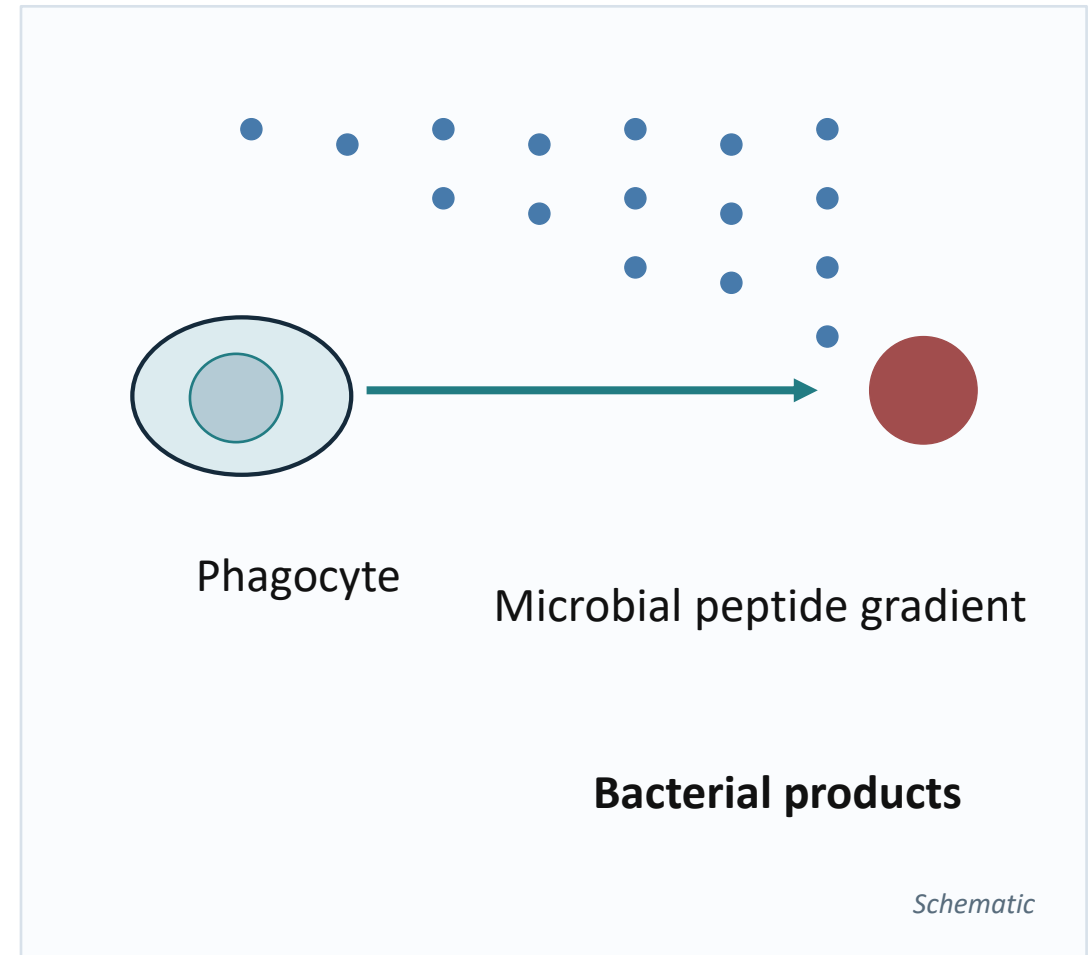
Endogenous

Host-generated inflammatory mediators.

Multiple cues may coexist at one injured site.

Bacterial N-formyl peptides provide microbial guidance

- Bacterial products contain peptides with terminal N-formylmethionine.
- These molecules function as exogenous chemoattractants.
- They attract leukocytes toward the microbial source.



The microbe itself can contribute a directional cue.

Certain bacterial lipids also serve as cues

- Not every chemotactic signal is a cytokine or peptide.
- Some bacterial lipids are identified as exogenous attractants.
- Origin, not molecular class alone, separates endogenous from exogenous.

Bacterial peptides

N-formylmethionine-containing products.

Bacterial lipids

Additional exogenous attractant class.

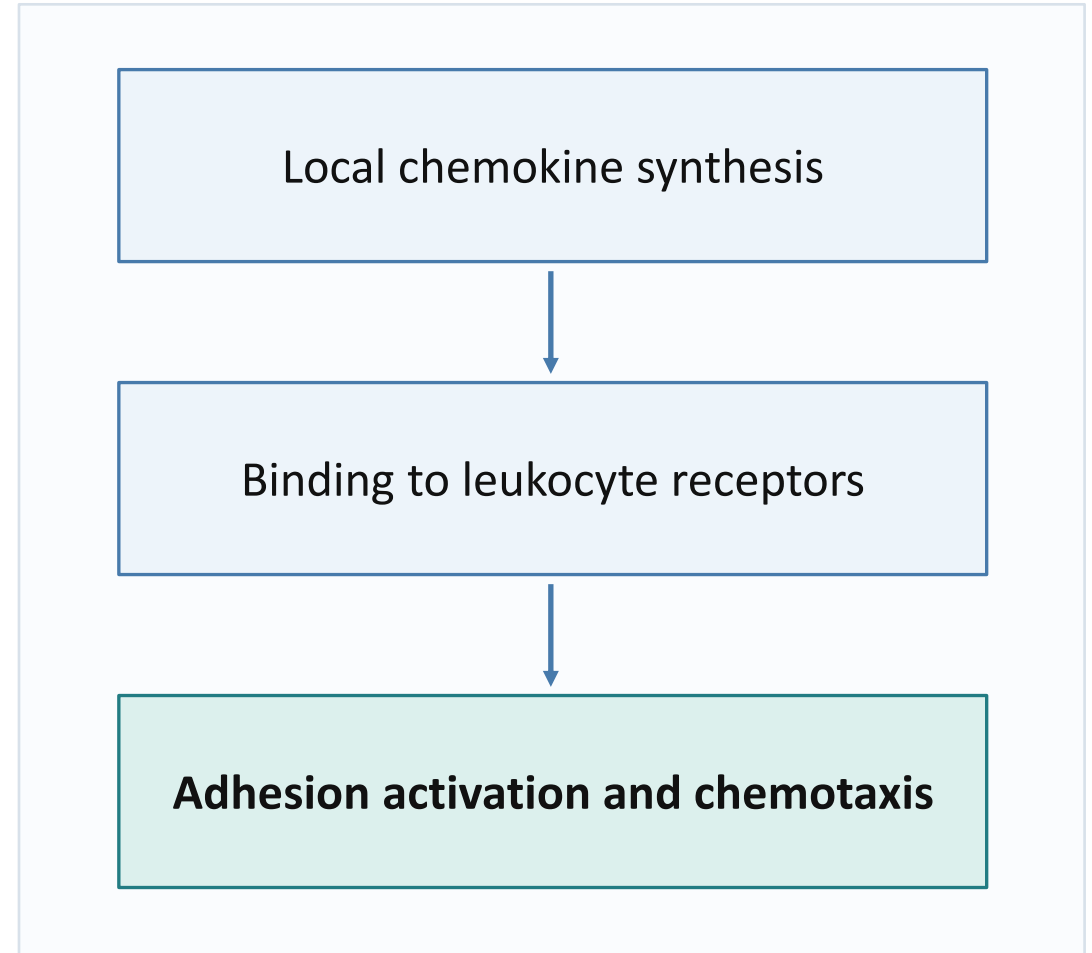
Exogenous attraction is not limited to proteins.

Three host mediator groups dominate the core example

Host class	Named example	Major teaching role
Chemokines	IL-8 / CXCL8	Leukocyte attraction
Complement	C5a	Chemotactic recruitment
AA metabolite	LTB4	Attraction and activation

Chemokines are cytokines specialized for cell guidance

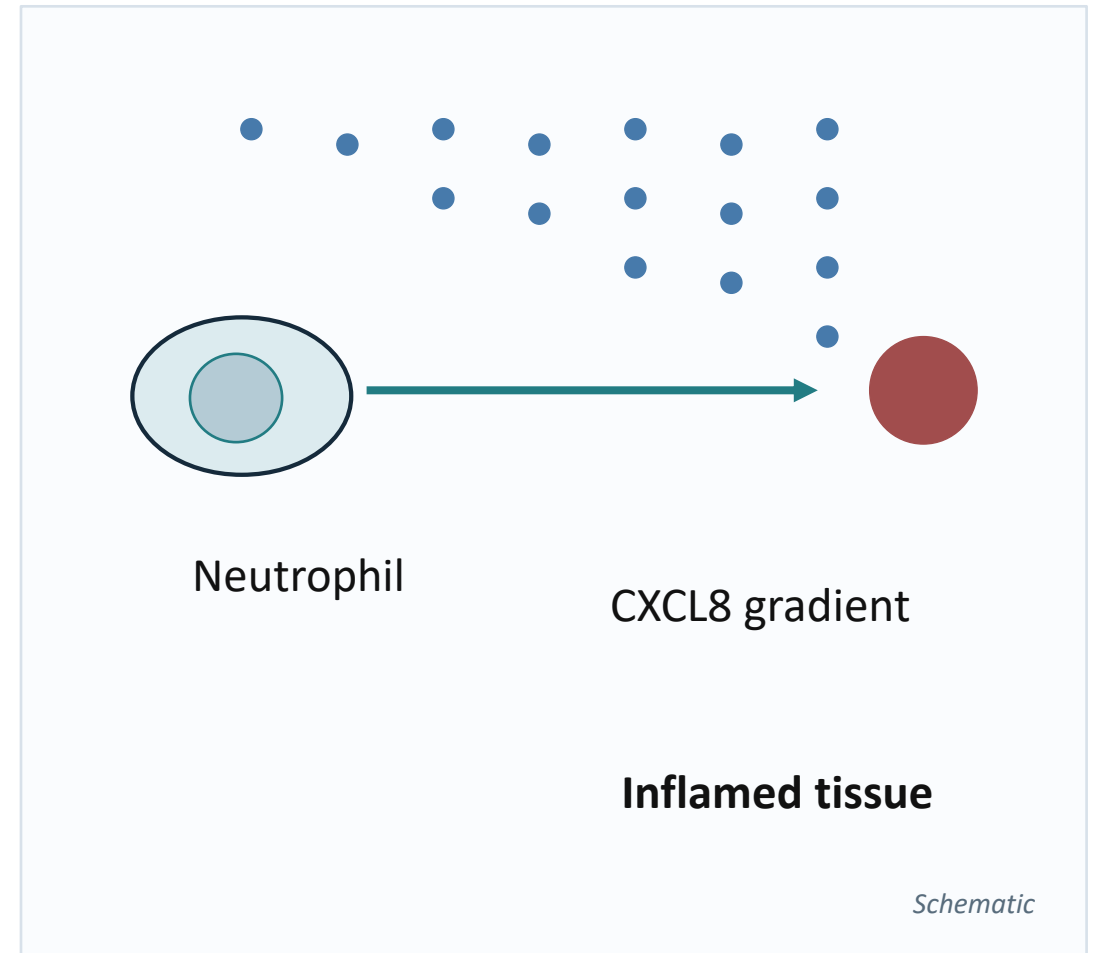
- Chemokines are small proteins with leukocyte-attracting actions.
- Different chemokines recruit different leukocyte subsets.
- Chemokines also activate leukocytes and promote integrin affinity.



Chemokines link cellular selectivity with directional recruitment.

CXCL8 is a major chemokine for neutrophils

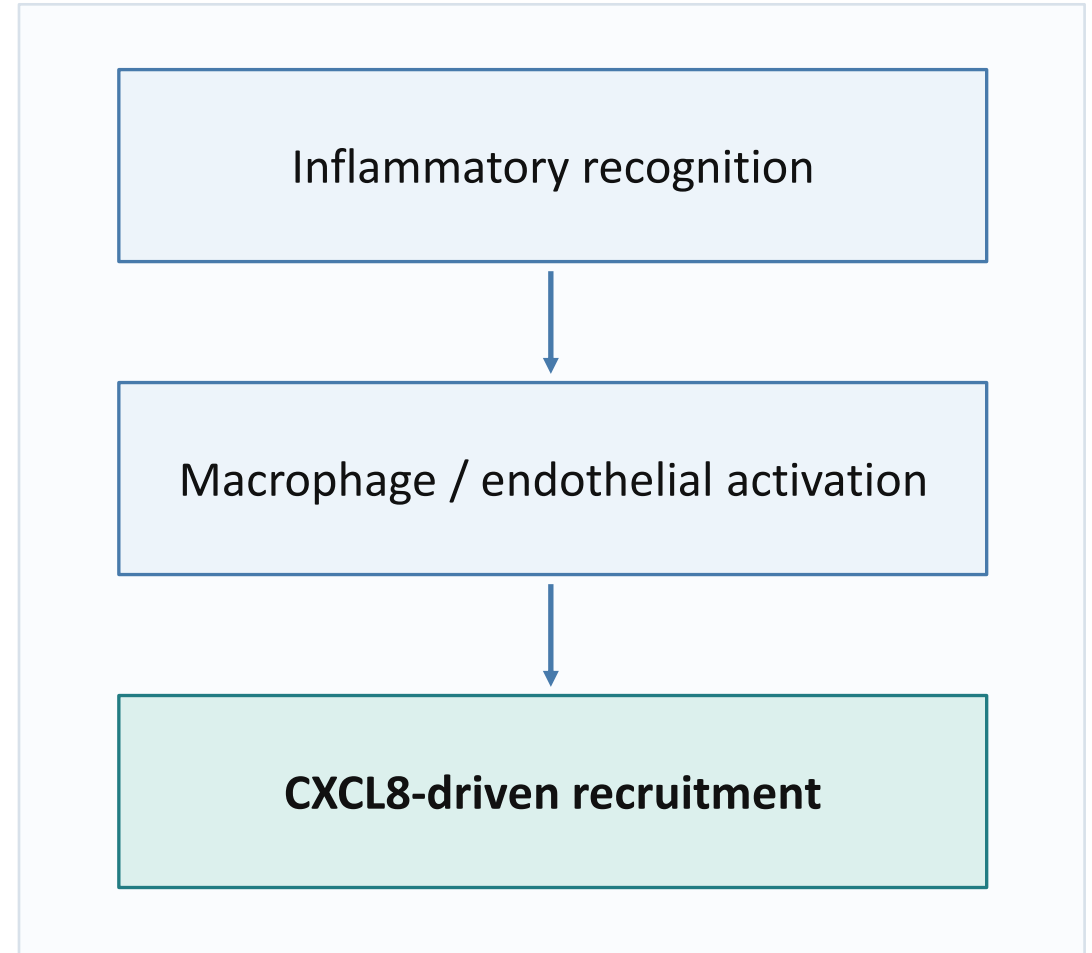
- IL-8 is also designated CXCL8 in the chemokine nomenclature.
- CXCL8 prominently activates and recruits neutrophils.
- Its effect on monocytes and eosinophils is comparatively limited.



The cell type recruited depends in part on the chemokine identity.

Activated macrophages can generate CXCL8

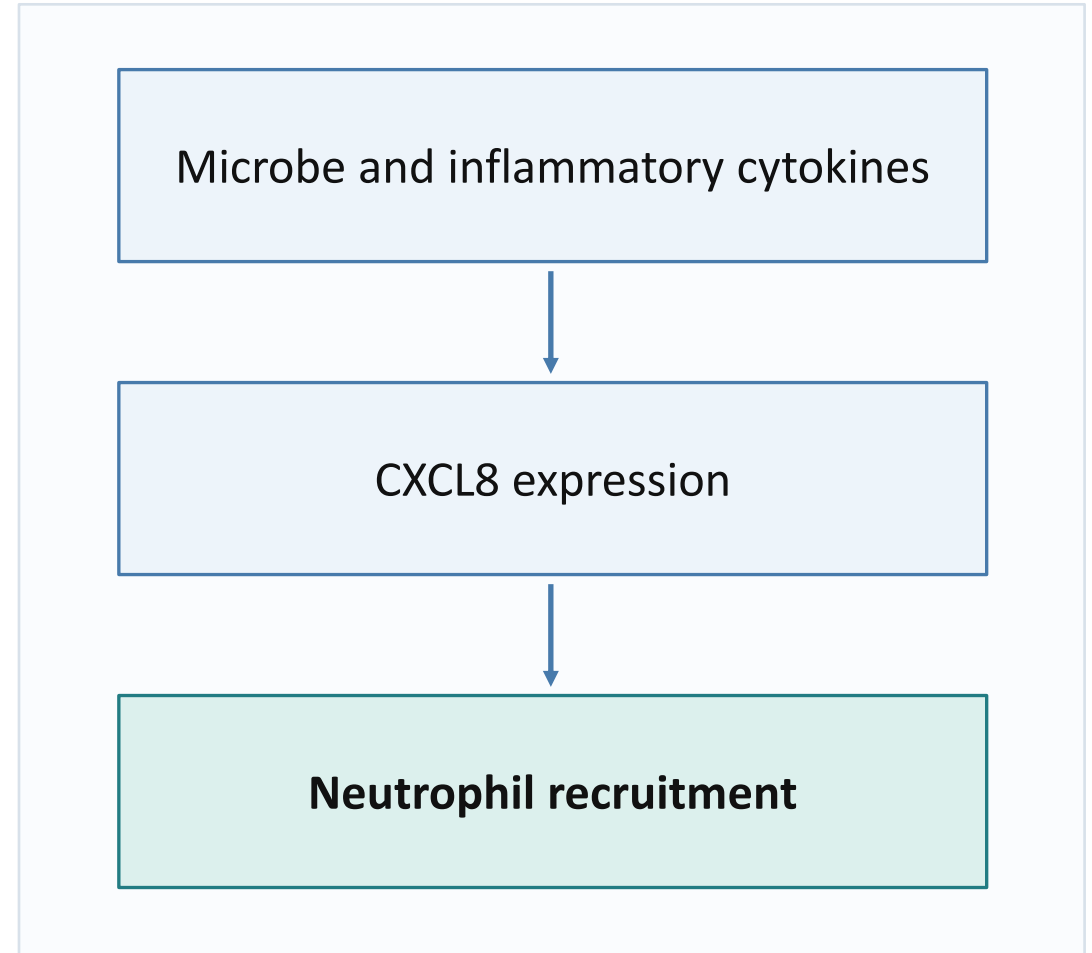
- Activated macrophages produce chemokines in response to inflammation.
- Endothelial cells and additional cell types can also secrete CXCL8.
- Locally produced chemokine signals help attract circulating neutrophils.



Sentinel and vascular cells help establish the chemotactic environment.

TNF and IL-1 amplify chemokine recruitment

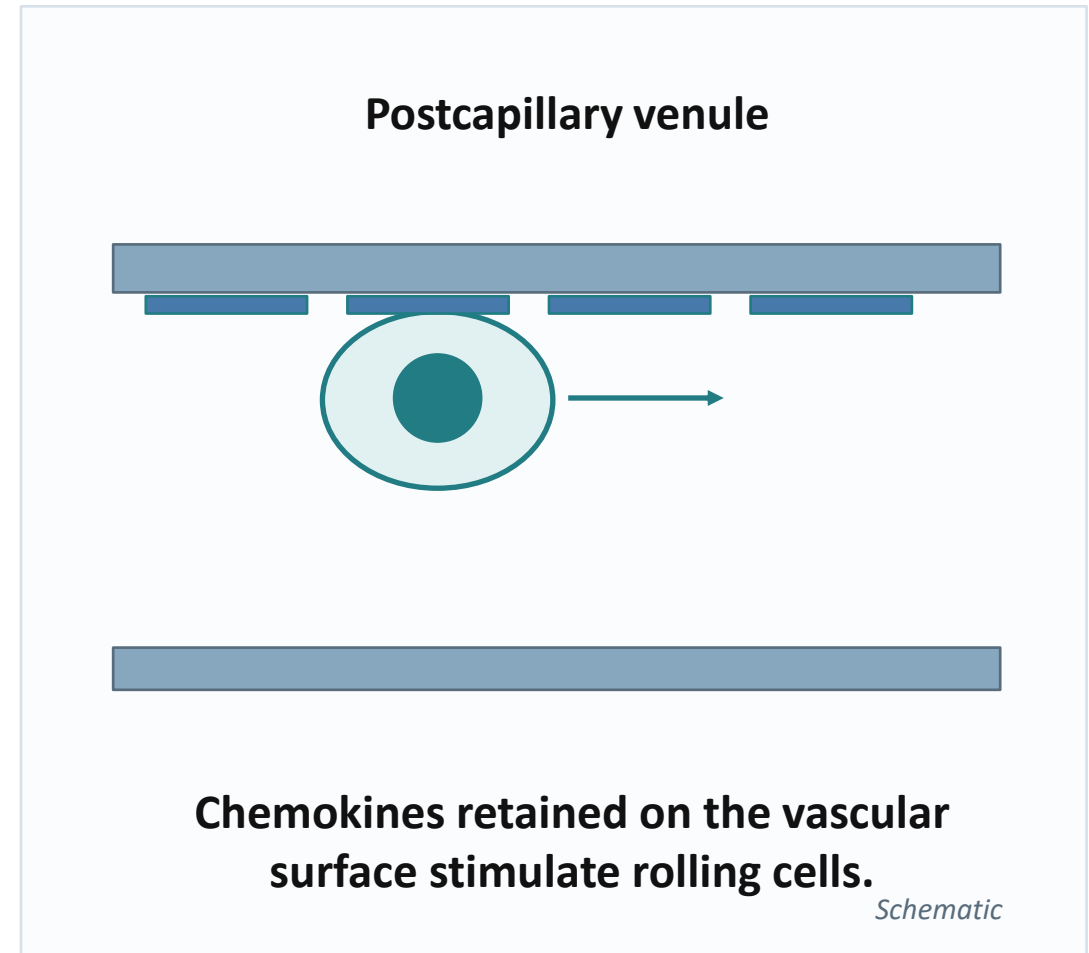
- Microbial products and inflammatory cytokines induce CXCL8 production.
- TNF and IL-1 are prominent cytokine inducers of CXCL8.
- An early tissue signal can thus stimulate further leukocyte entry.



Inflammatory signaling amplifies recruitment without changing the cell source.

Endothelial proteoglycans display chemokines

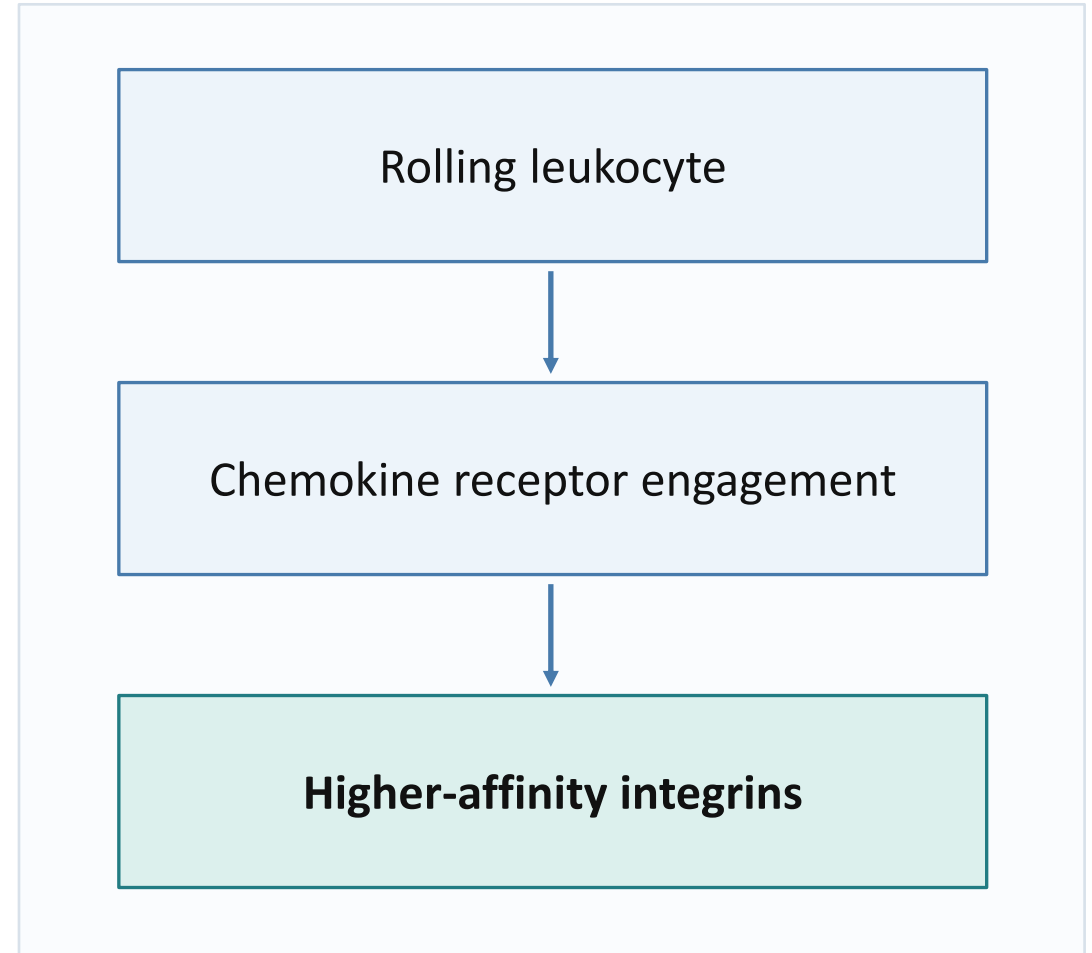
- Chemokines bind to proteoglycans on endothelial surfaces.
- This displays local chemokine signals to rolling leukocytes.
- Presentation of chemokines helps activate adhesive integrins.



Surface display makes a soluble signal locally accessible.

Chemokines produce a high-affinity integrin state

- Rolling leukocytes initially carry low-affinity integrins.
- Endothelium-bound chemokines activate the adherent leukocyte.
- Integrins shift toward higher affinity for endothelial ligands.



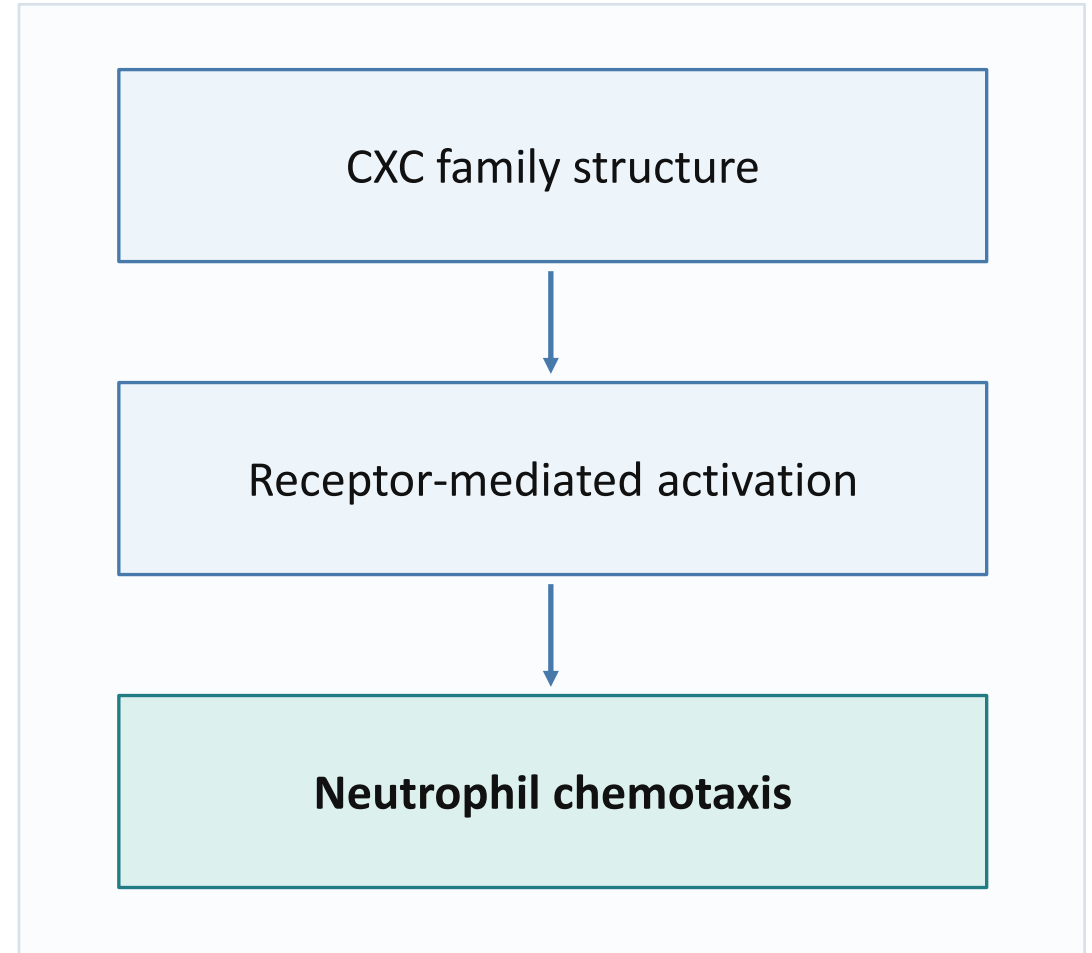
Chemokine sensing can convert loose contact into firm arrest.

Chemokine families help explain leukocyte selectivity

Class	Representative	Prominent context
CXC	CXCL8 (IL-8)	Neutrophil chemotaxis
CC	CCL2; CCL11	Monocytes; Eosinophils
C	XCL1	Lymphocytes
CX3C	CX3CL1	Monocytes and T cells

CXC chemokines commonly recruit neutrophils

- CXC chemokines have one amino acid between the first two conserved cysteines.
- A functional subset acts primarily on neutrophils.
- CXCL8 is the major example highlighted in acute inflammation.



Sequence classification can correlate with cell-type recruitment.

CC chemokines often attract mononuclear cells

- CC chemokines contain adjacent first two conserved cysteines.
- Examples include CCL2 (MCP-1) and CCL3 (MIP-1 α).
- Members overlap in activity but tend to attract monocytes, lymphocytes, and selected granulocytes.

CCL2 / MCP-1

Example linked to monocyte recruitment.

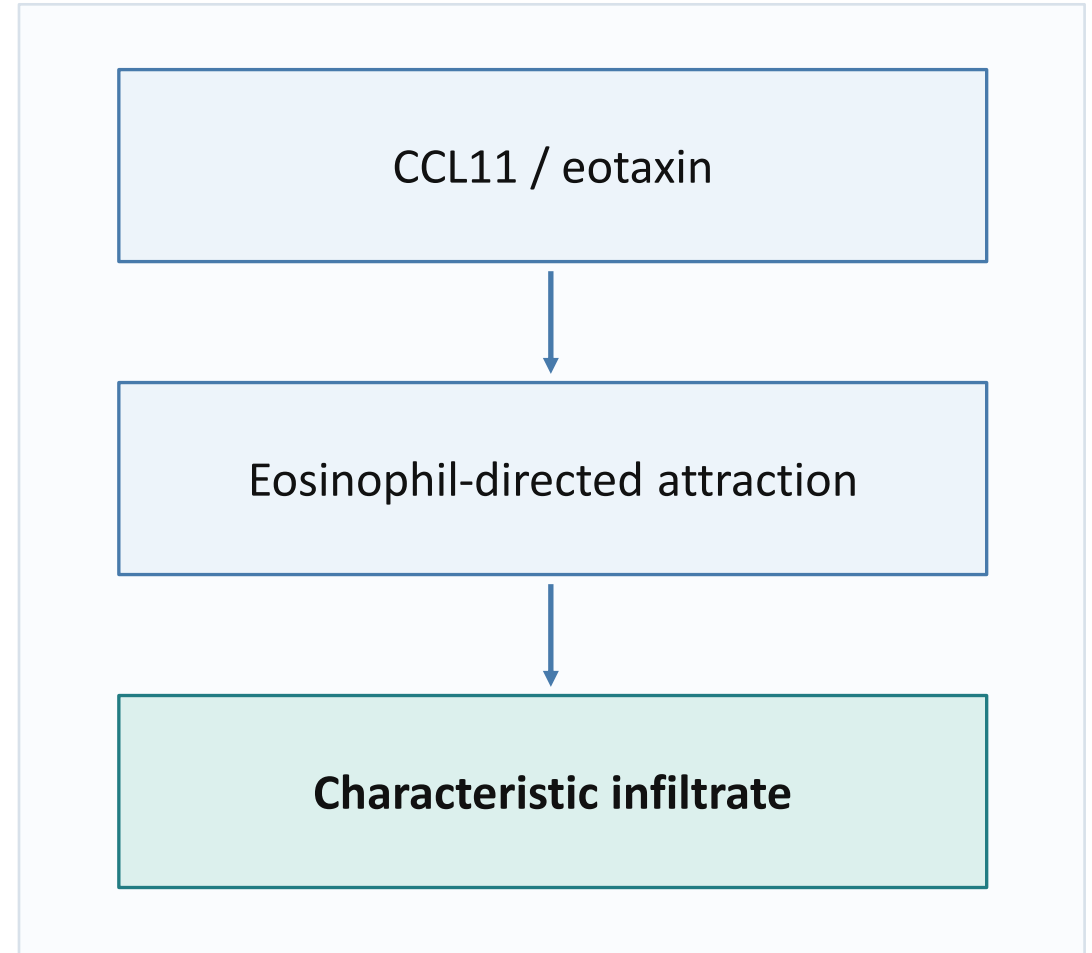
CCL3 / MIP-1 α

Member of a family with overlapping targets.

CC chemokines are generally less potent for neutrophils than CXC examples.

CCL11 has marked eosinophil selectivity

- Eotaxin is also called CCL11.
- Eotaxin as selectively recruiting eosinophils.
- The mediator helps explain eosinophil-rich allergic and helminthic responses.



A chemokine can bias the leukocyte composition at an injury site.

C and CX3C chemokines broaden the recruitment repertoire

- XCL1 is a C-family chemokine with relative lymphocyte specificity.
- CX3CL1 (fractalkine) exists in cell-bound and soluble forms.
- The bound form supports adhesion; The soluble form attracts monocytes and T cells.

XCL1 / lymphotactin

A C chemokine with lymphocyte preference.

CX3CL1 / fractalkine

Membrane-associated adhesion; Soluble chemoattraction.

Chemokine effects extend beyond neutrophil-dominant inflammation.

Not all chemokines are inflammation inducible

- Inflammatory chemokines are induced by microbes or injury signals.
- Homeostatic chemokines maintain organized cellular positioning in tissues.
- In lymphoid organs they help localize T and B lymphocytes to distinct regions.

Inflammatory

Inducible recruitment to a damaged site.

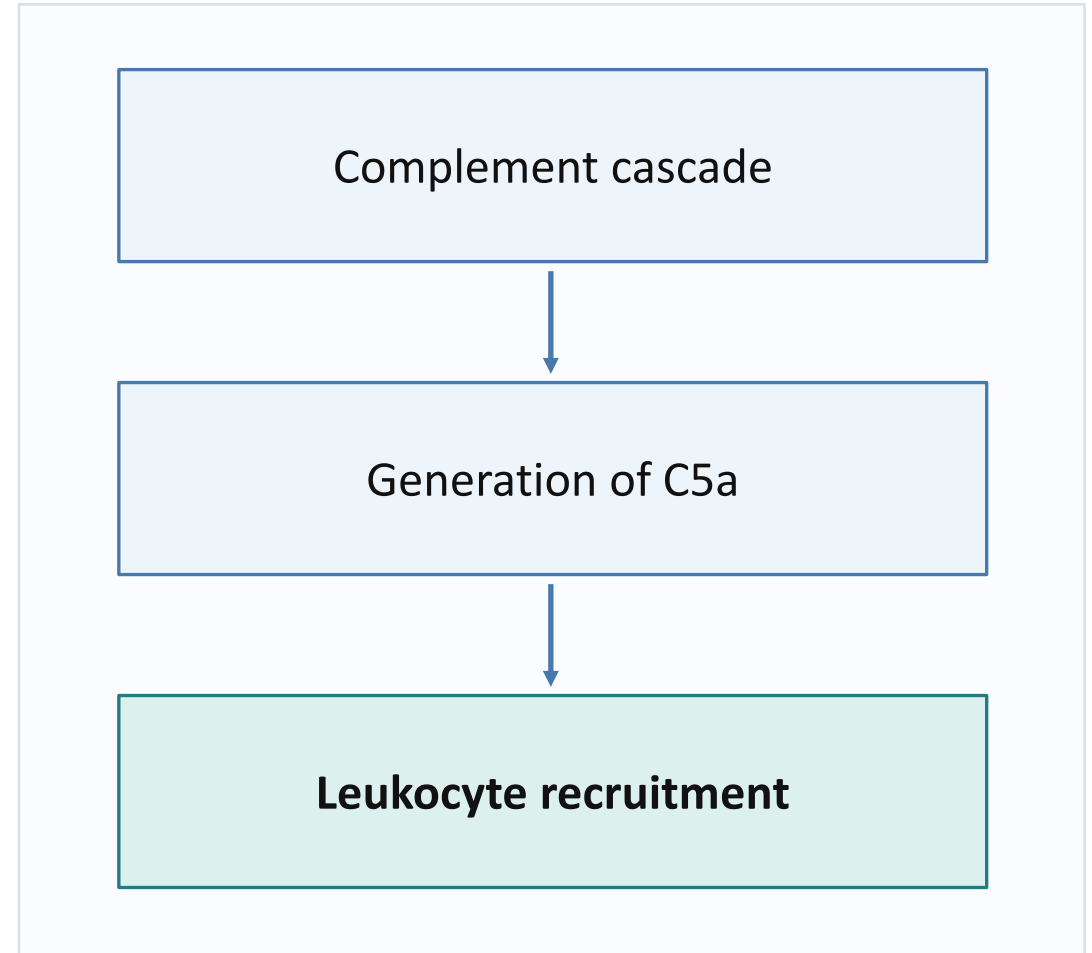
Homeostatic

Constitutive regulation of tissue architecture.

Chemotactic guidance is also a normal tissue-organizing mechanism.

C5a connects complement activation to chemotaxis

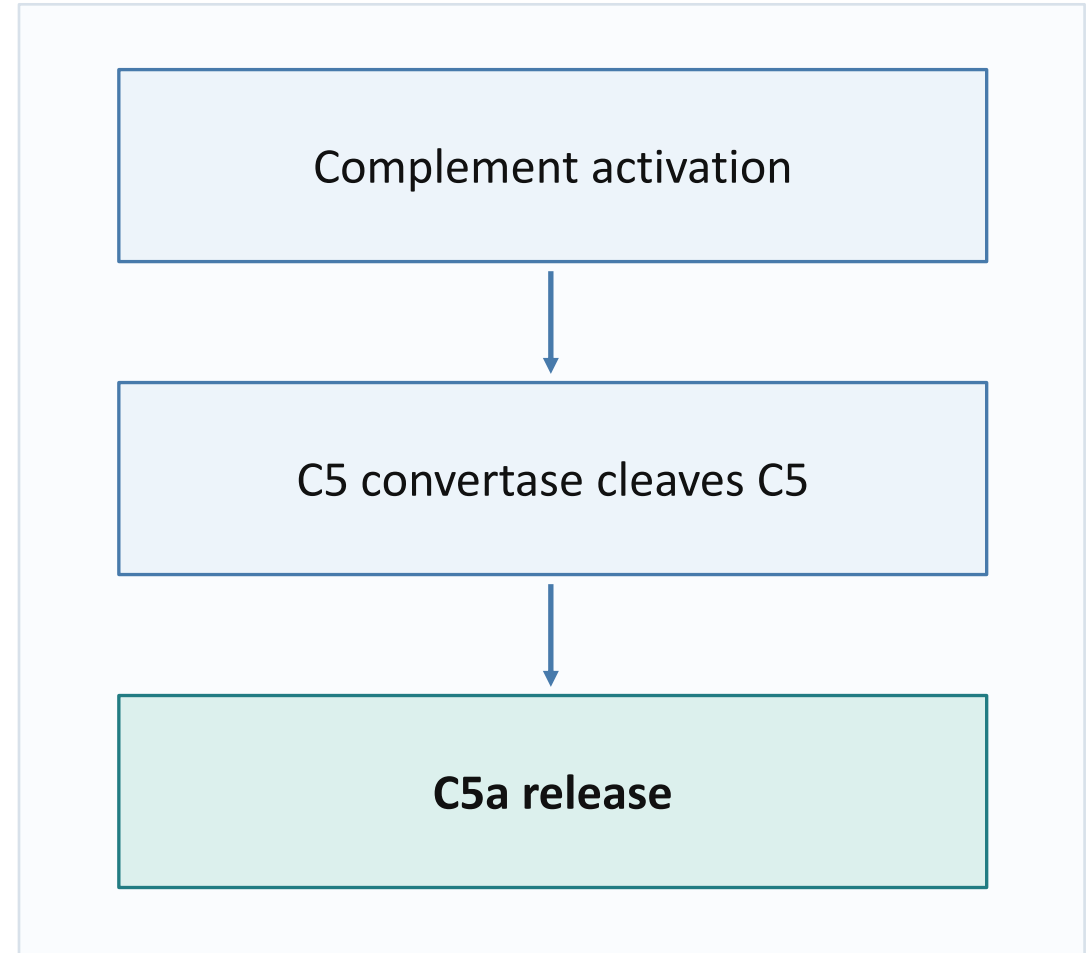
- Complement activation yields biologically active cleavage fragments.
- C5a is a major chemotactic complement-derived mediator.
- It also contributes to the inflammatory actions of complement.



Innate humoral activation can directly guide cellular migration.

C5 convertase generates C5a during complement activation

- All major complement activation pathways converge on common downstream steps.
- C5 convertase cleaves C5 into C5a and C5b.
- Released C5a can act on leukocytes at inflammatory sites.



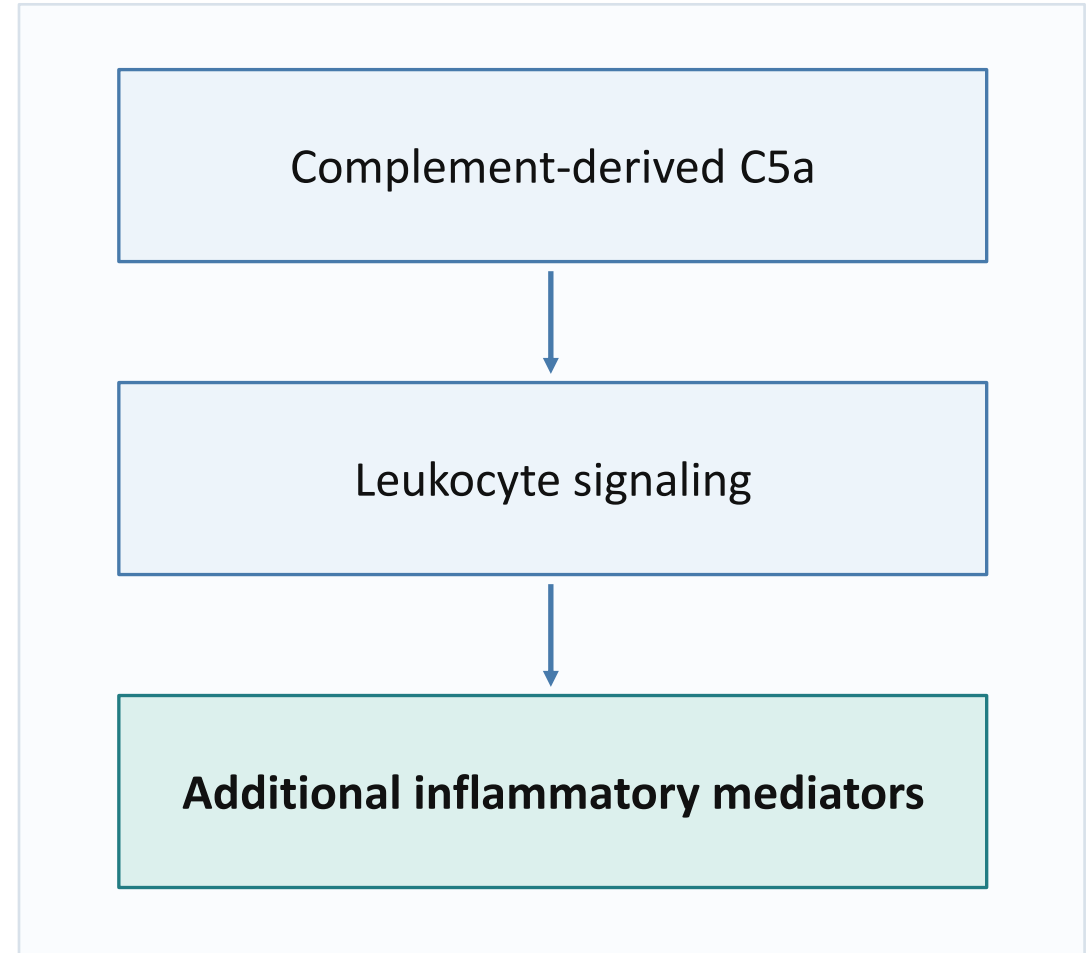
C5a formation arises from proteolytic complement activation.

C5a recruits multiple leukocyte populations

Cell population	C5a chemotactic action	Interpretive point
Neutrophils	Recognized target	Acute innate response
Monocytes	Recognized target	Later tissue macrophages
Eosinophils	Recognized target	Granulocyte recruitment
Basophils	Recognized target	Broad leukocyte activity

C5a also amplifies inflammatory mediator release

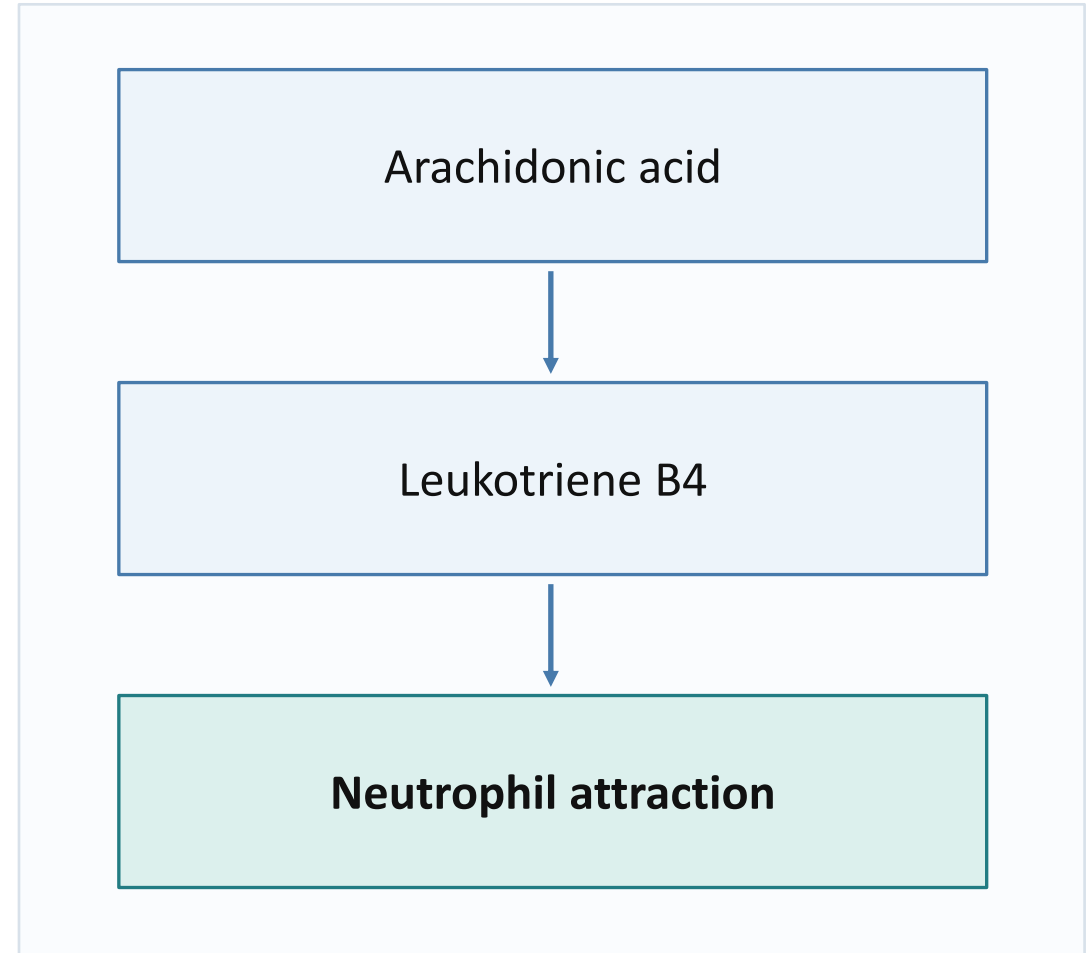
- C5a activates the lipoxygenase pathway in neutrophils and monocytes.
- This promotes additional inflammatory mediator production.
- The same complement fragment therefore attracts and activates recruited cells.



A chemoattractant can also amplify the downstream inflammatory program.

LTB4 is a lipid-derived chemoattractant

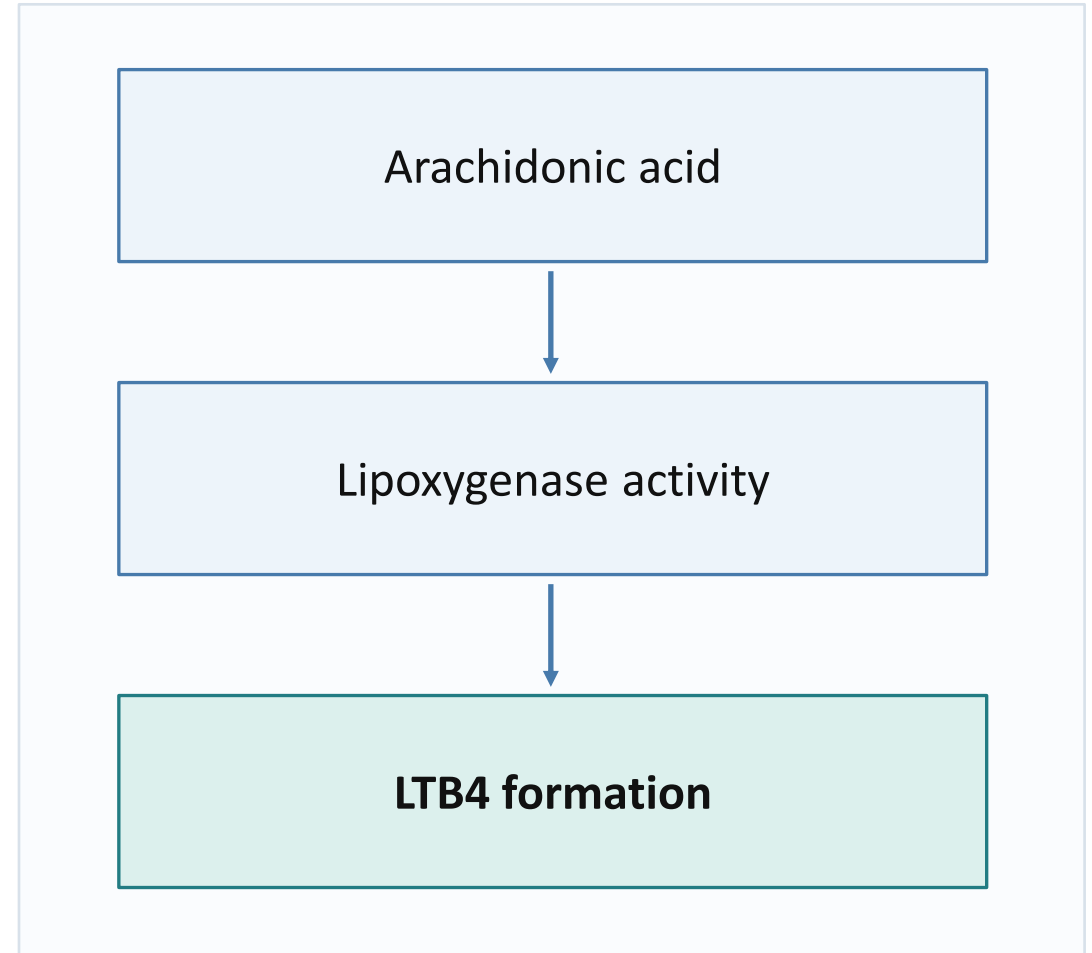
- Leukotriene B4 is derived from arachidonic acid metabolism.
- It is a potent chemoattractant for neutrophils.
- It also activates neutrophils after receptor engagement.



Chemoattractants include lipids as well as peptides and proteins.

The lipoxygenase pathway produces leukotrienes

- Lipoxygenase enzymes metabolize arachidonic acid.
- 5-lipoxygenase is especially prominent in neutrophils.
- LTB₄ is one product of the leukotriene-forming pathway.



Eicosanoid synthesis converts a membrane-derived substrate into inflammatory signals.

LTB4 attracts and activates neutrophils

- LTB4 promotes leukocyte chemotaxis.
- It also augments aggregation and adhesion to venular endothelium.
- Neutrophils may generate reactive oxygen species and release lysosomal enzymes.

Recruitment

Chemotaxis, aggregation, and adhesion.

Effector activation

ROS generation and lysosomal enzyme release.

Chemotactic signals can prime destructive effector responses.

LTB4 differs from cysteinyl leukotrienes

- LTB4 is strongly associated with leukocyte attraction and activation.
- LTC4, LTD4, and LTE4 primarily cause bronchospasm and increased venular permeability.
- Both mediator sets are leukotrienes but their emphasized actions differ.

LTB4

Neutrophil chemotaxis and activation.

LTC4 / LTD4 / LTE4

Smooth-muscle and vascular-permeability effects.

Do not treat all leukotrienes as mechanistically interchangeable.

Lipoxins counter neutrophil recruitment

- Lipoxins are also products of arachidonic acid metabolism.
- They suppress neutrophil chemotaxis and endothelial adhesion.
- Their activity illustrates the active limitation of inflammatory recruitment.

Leukotrienes

Promote acute leukocyte recruitment.

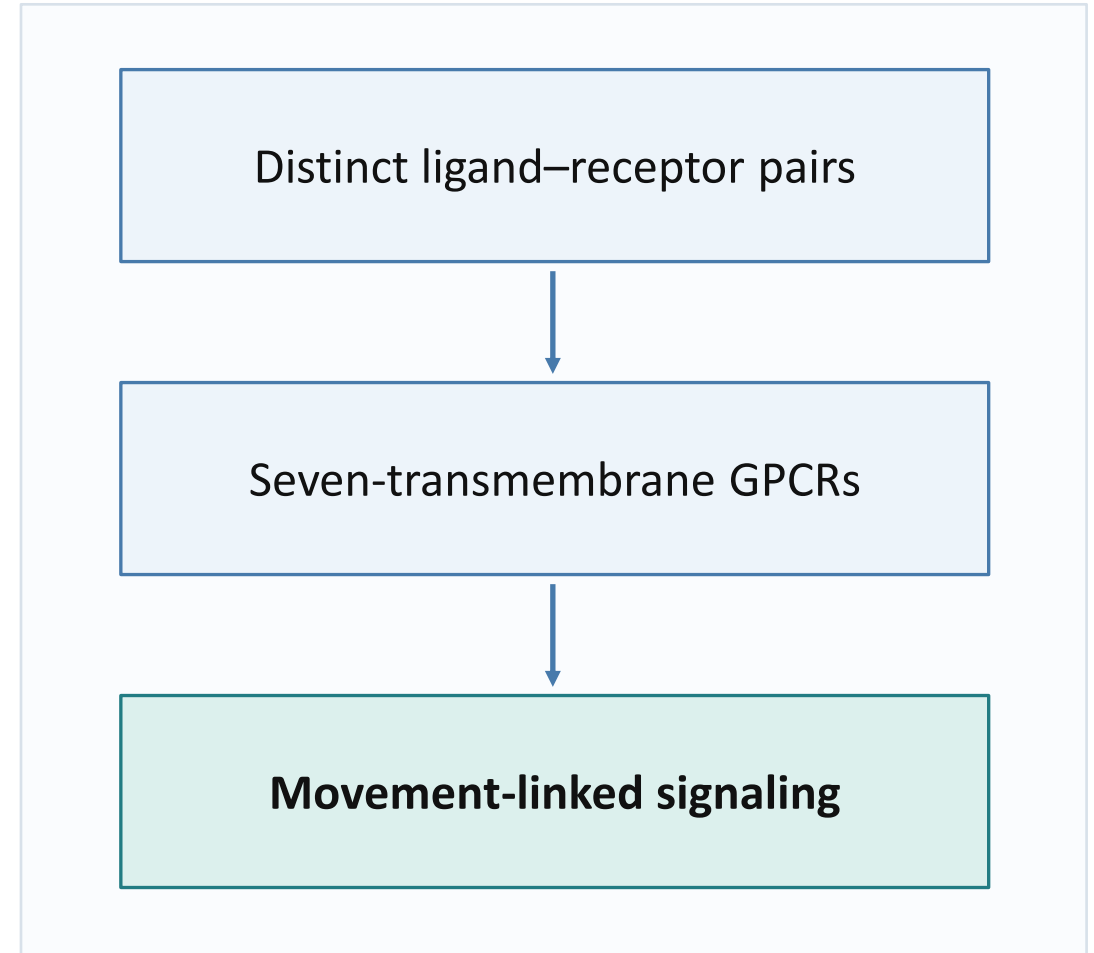
Lipoxins

Inhibit neutrophil chemotaxis and adhesion.

Related lipid mediator pathways can have opposing inflammatory effects.

Distinct chemoattractants converge on GPCR signaling

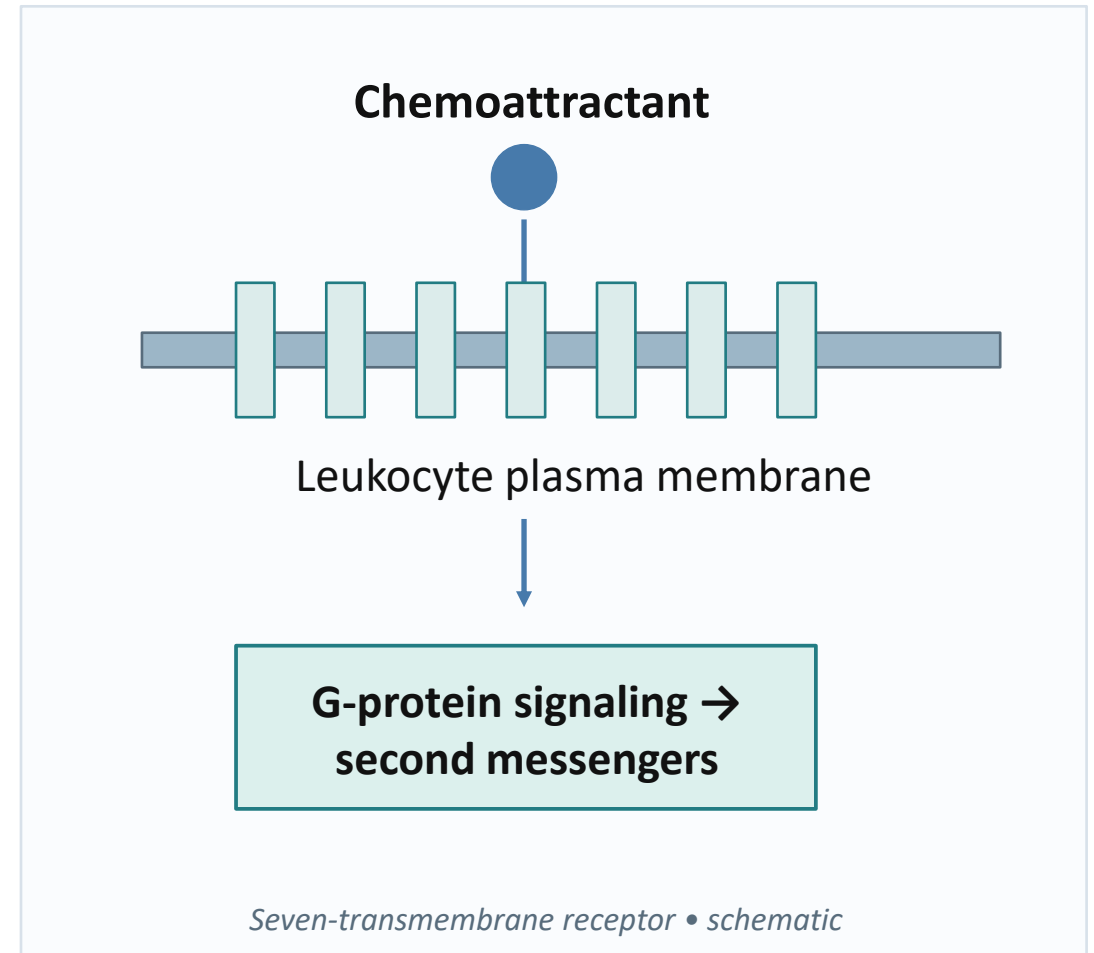
- Chemokines, C5a, LTB4, and bacterial products act through their specific receptors.
- The relevant chemotactic receptors belong to the seven-transmembrane GPCR class.
- Different ligands therefore converge on a shared signaling logic.



A common receptor architecture supports multiple chemotactic inputs.

Chemotactic receptors span the membrane seven times

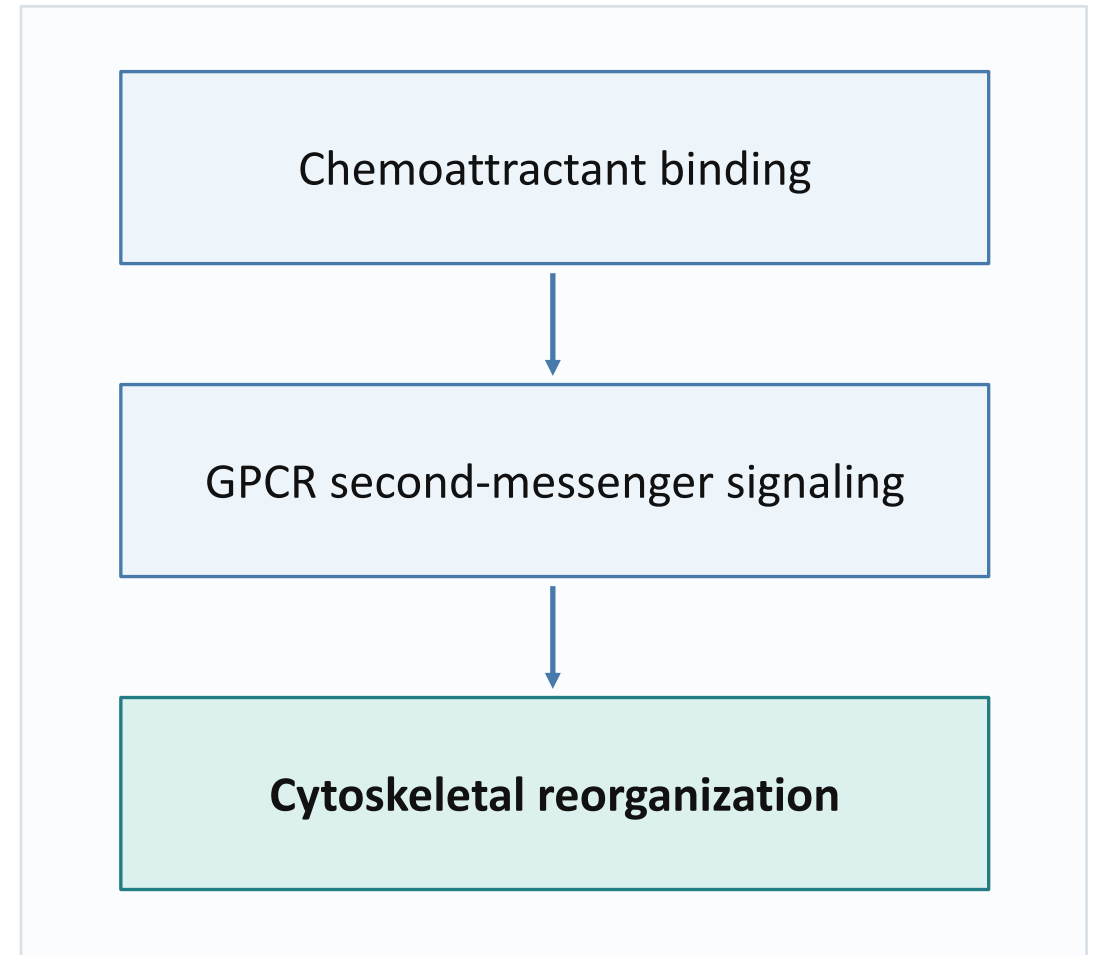
- The receptor resides in the leukocyte plasma membrane.
- The extracellular signal is translated into intracellular G-protein signaling.
- A seven-transmembrane GPCR is a receptor type, not seven separate receptors.



Ligand recognition is converted into changes inside the leukocyte.

Receptor activation engages intracellular second messengers

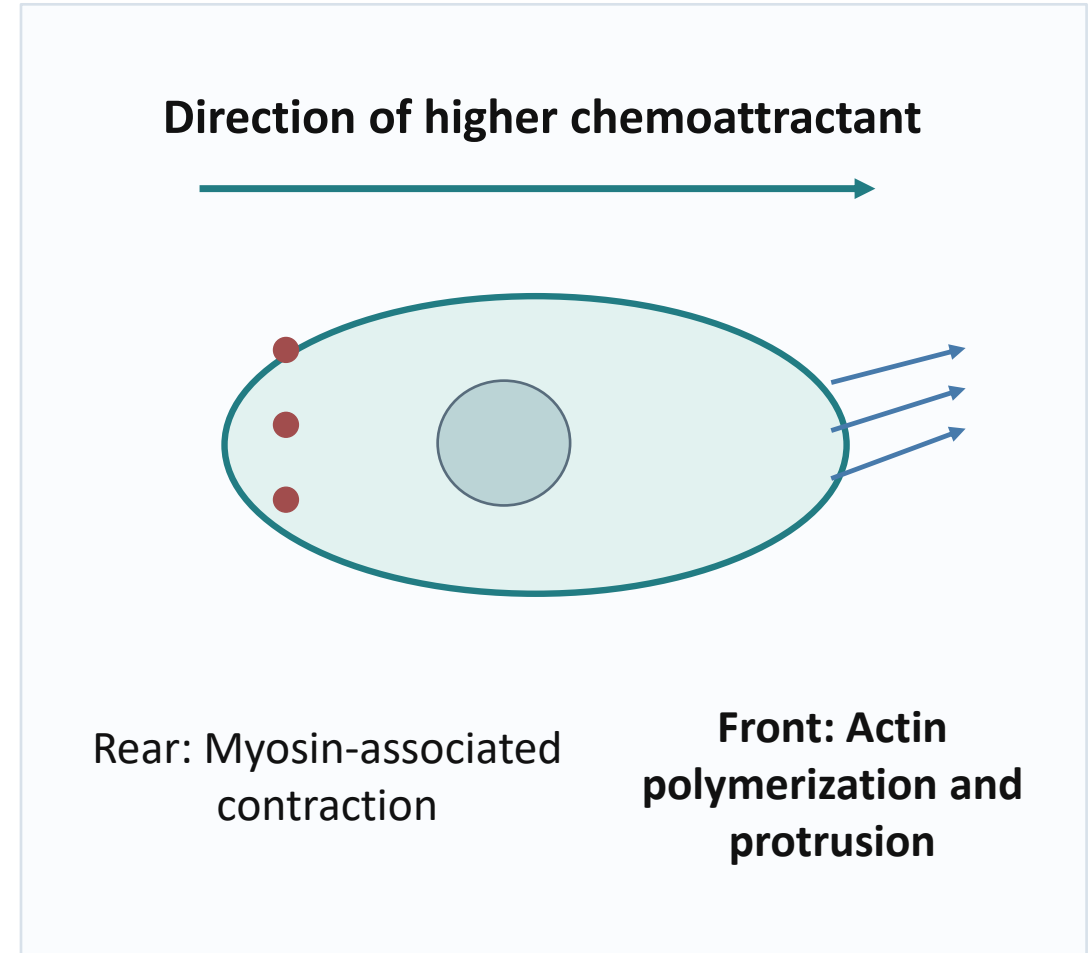
- Ligand binding activates its cognate surface receptor.
- Intracellular second messengers relay the received signal.
- These signals reorganize the actin–myosin cytoskeleton.



Chemotaxis is an actively signaled cellular response—not passive drift.

Actin polymerizes at the leading edge

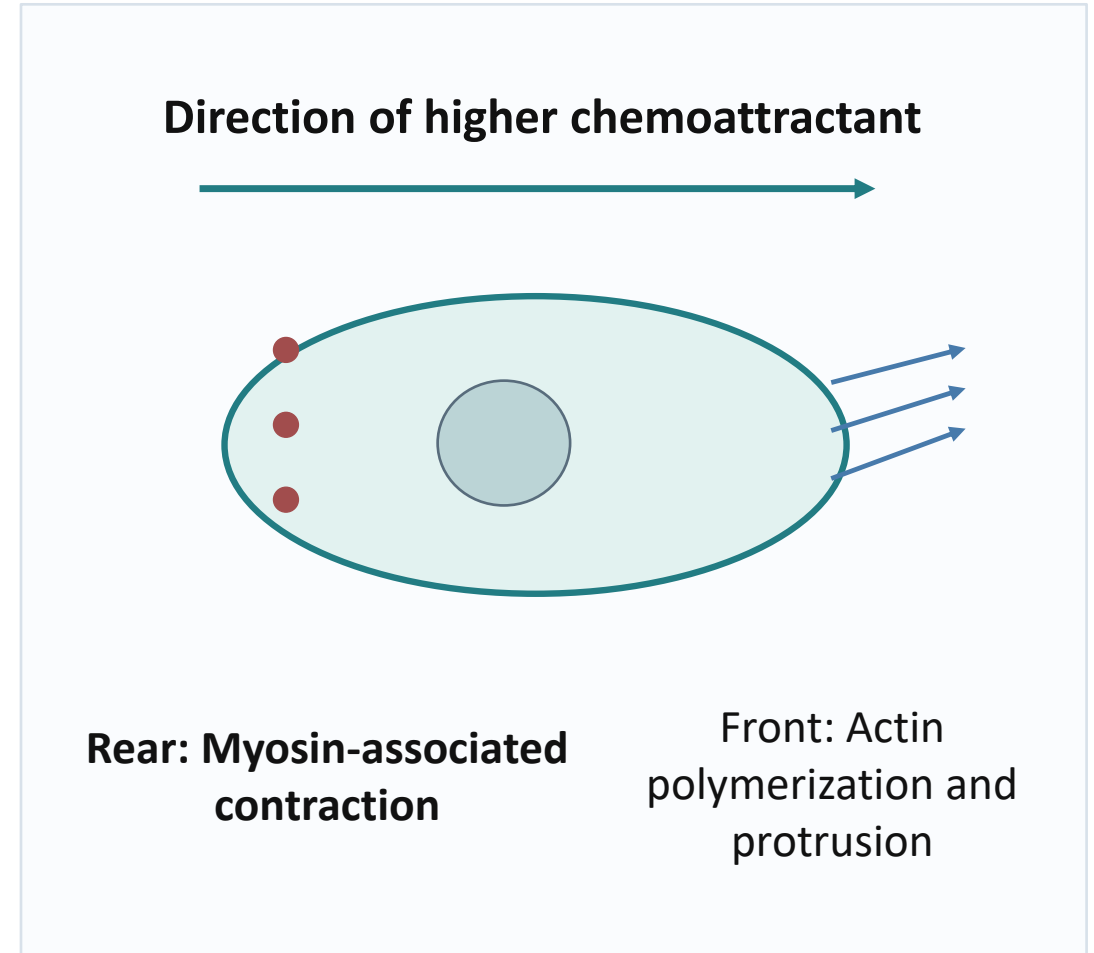
- A leukocyte develops a spatially defined front and rear during migration.
- Actin polymerization occurs at the leading edge.
- The extended front advances toward the stronger attractant cue.



Forward protrusion is driven by regulated cytoskeletal remodeling.

Myosin filaments localize toward the cell rear

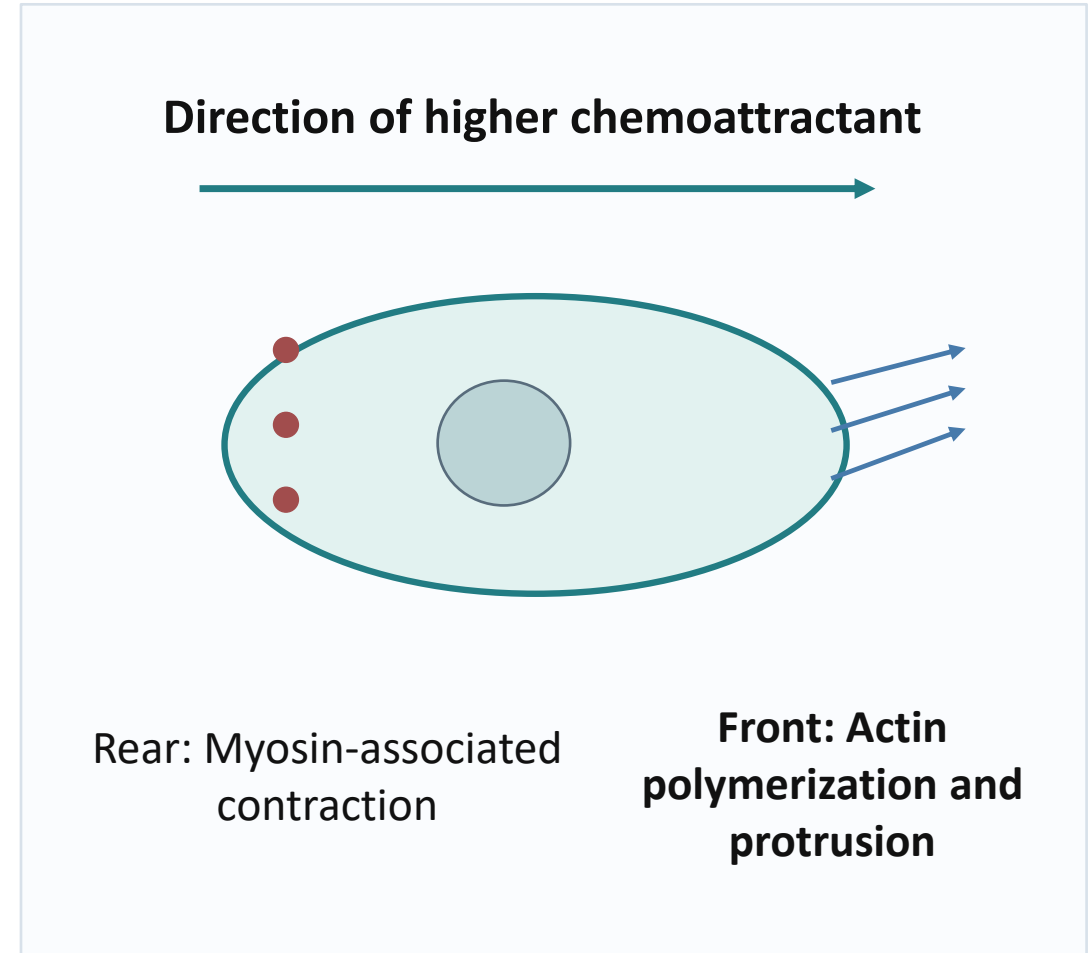
- Chemotactic receptor signaling also reorganizes myosin.
- Myosin filaments are localized predominantly toward the trailing region.
- This organization contributes to movement of the cell body.



Front-directed actin and rear-positioned myosin cooperate.

Front–rear polarity enables persistent directionality

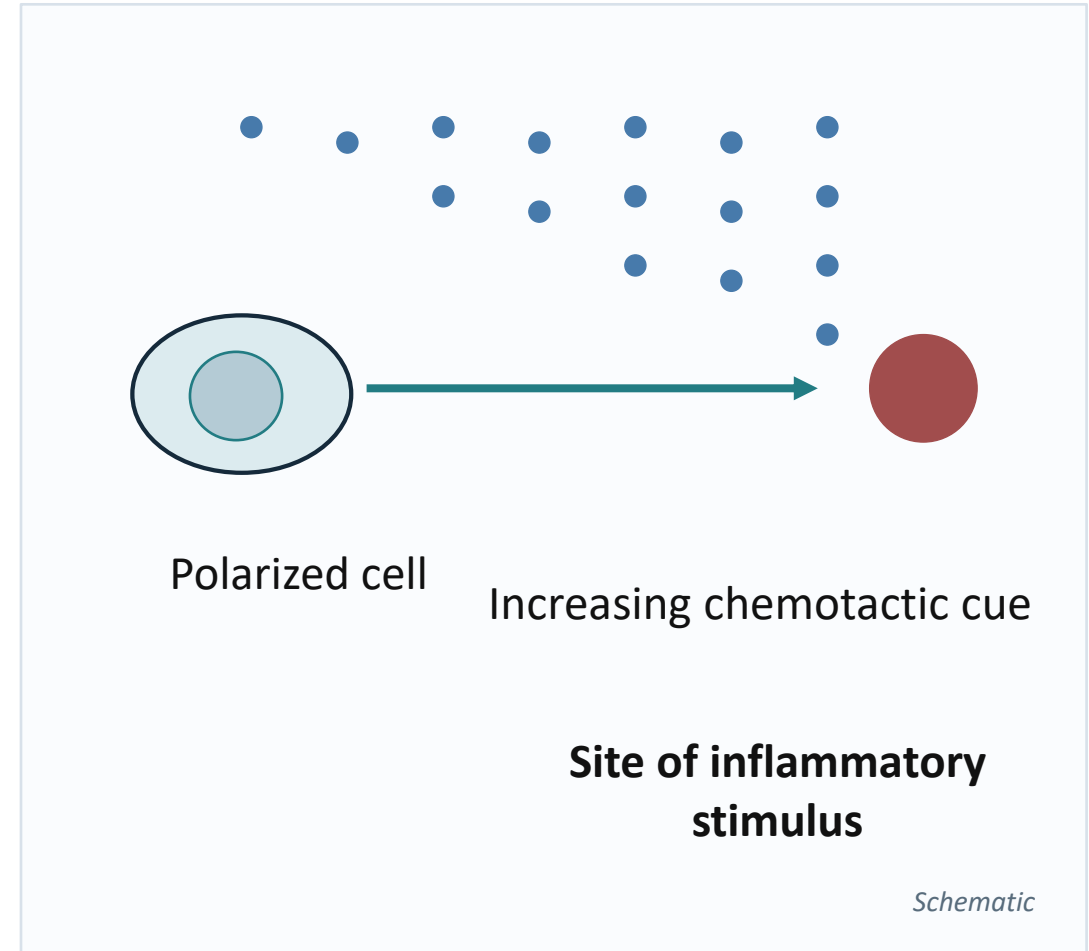
- Actin-rich protrusion develops at the front.
- Myosin-associated contraction is located toward the rear.
- The asymmetry permits net migration rather than undirected shape change.



Directional movement depends on spatial coordination of the cytoskeleton.

Filopodia extend toward the attractant source

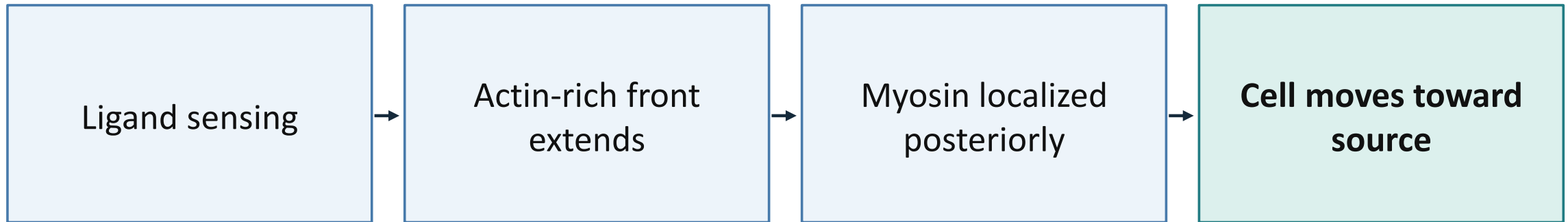
- The leading edge produces cytoplasmic extensions termed filopodia.
- These extensions lead leukocyte movement toward increasing signal.
- The cell body follows the advancing edge.



Protrusion pulls the cell toward the injury focus.

Motility requires repeated protrusion and translocation

Chemotactic migration is a coordinated sequence of receptor signals and cell-shape change.



The intracellular machinery translates a chemical gradient into locomotion.

Chemotaxis links two kinds of information

- The concentration gradient indicates where to move.
- GPCR-triggered cytoskeletal remodeling supplies the mechanism of movement.
- Both elements are needed to explain directional accumulation in tissues.

Directional information

Local differences in attractant signal.

Mechanical output

Actin extension and rearward myosin localization.

A gradient alone does not move a cell; Receptor-driven remodeling does.

Chemokines connect vascular adhesion with tissue guidance

- On endothelial surfaces, chemokines raise leukocyte integrin affinity.
- After vascular exit, chemokines guide movement through tissues.
- One signal class therefore participates at more than one recruitment stage.

At the endothelial interface

Converts integrins to a high-affinity state.

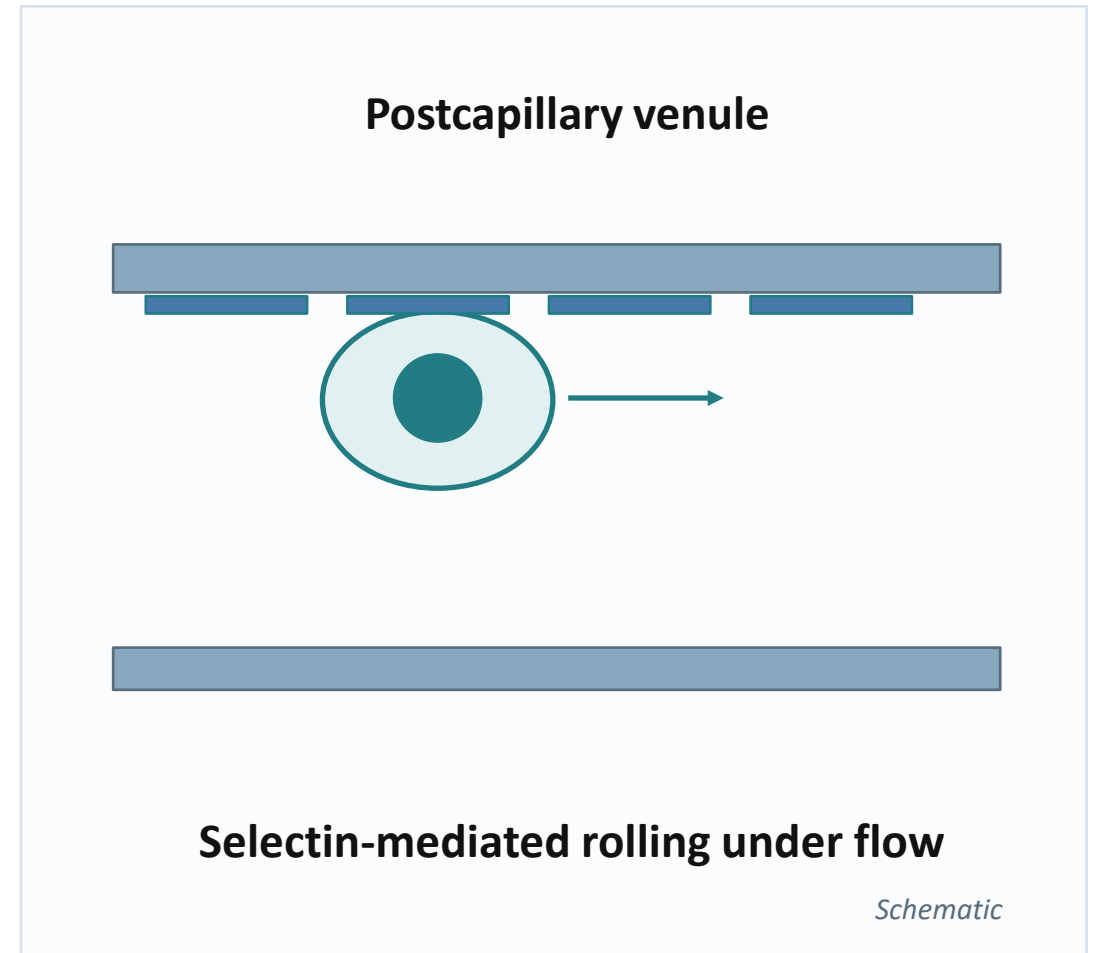
Inside interstitial tissue

Provides chemotactic guidance.

Location determines the dominant effect of the same signaling class.

Selectins support the first adhesive contacts

- P-selectin and E-selectin participate in leukocyte rolling.
- Rolling brings leukocytes into repeated contact with endothelial chemokines.
- Selectins are not the principal molecules of firm attachment.



Initial contact increases access to endothelial activation signals.

Integrin activation converts rolling into arrest

- Resting leukocyte integrins are generally in a lower-affinity state.
- Chemokine receptor activation increases integrin affinity.
- Interaction with endothelial ligands produces firm adhesion and spreading.

Lower-affinity integrins

Rolling leukocyte is not fully arrested.

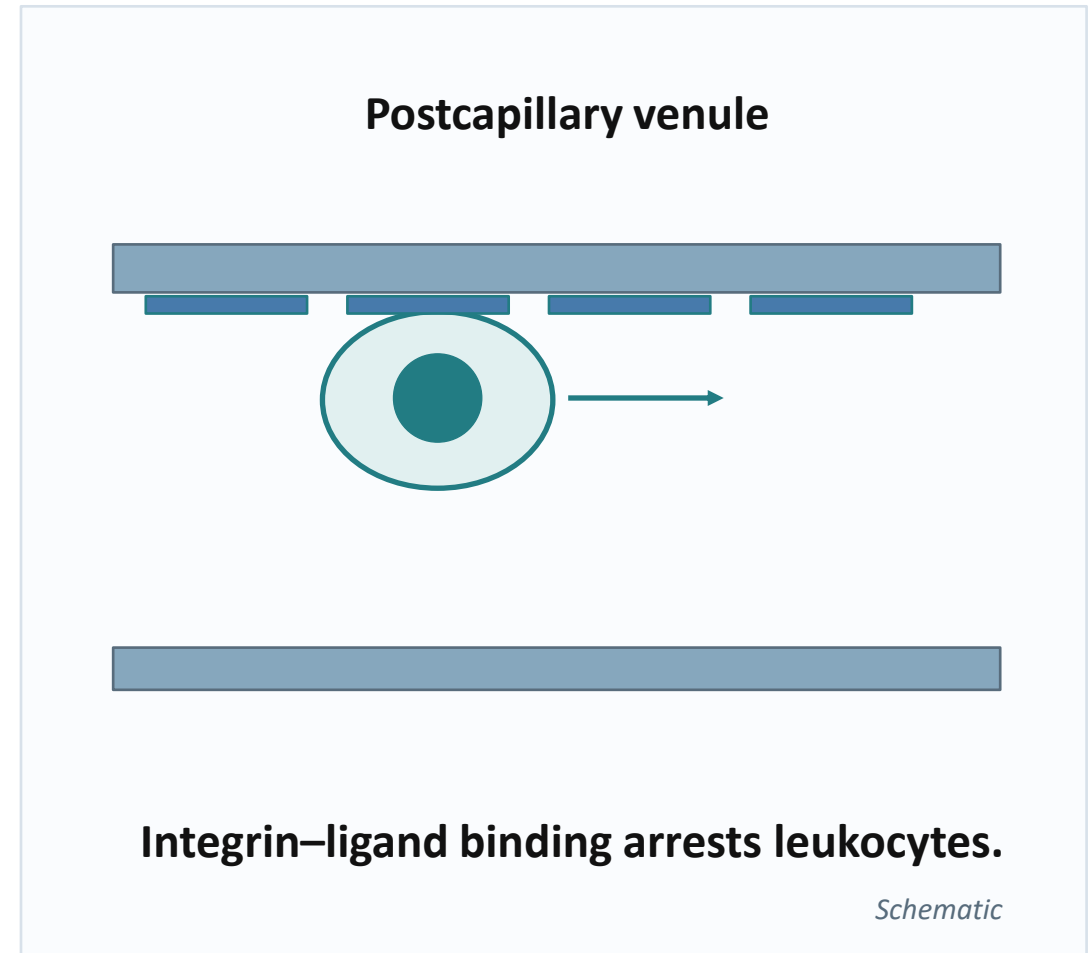
Higher-affinity integrins

Leukocyte binds firmly to activated endothelium.

Inside-out activation of adhesion receptors precedes transmigration.

ICAM-1 and VCAM-1 support stable adhesion

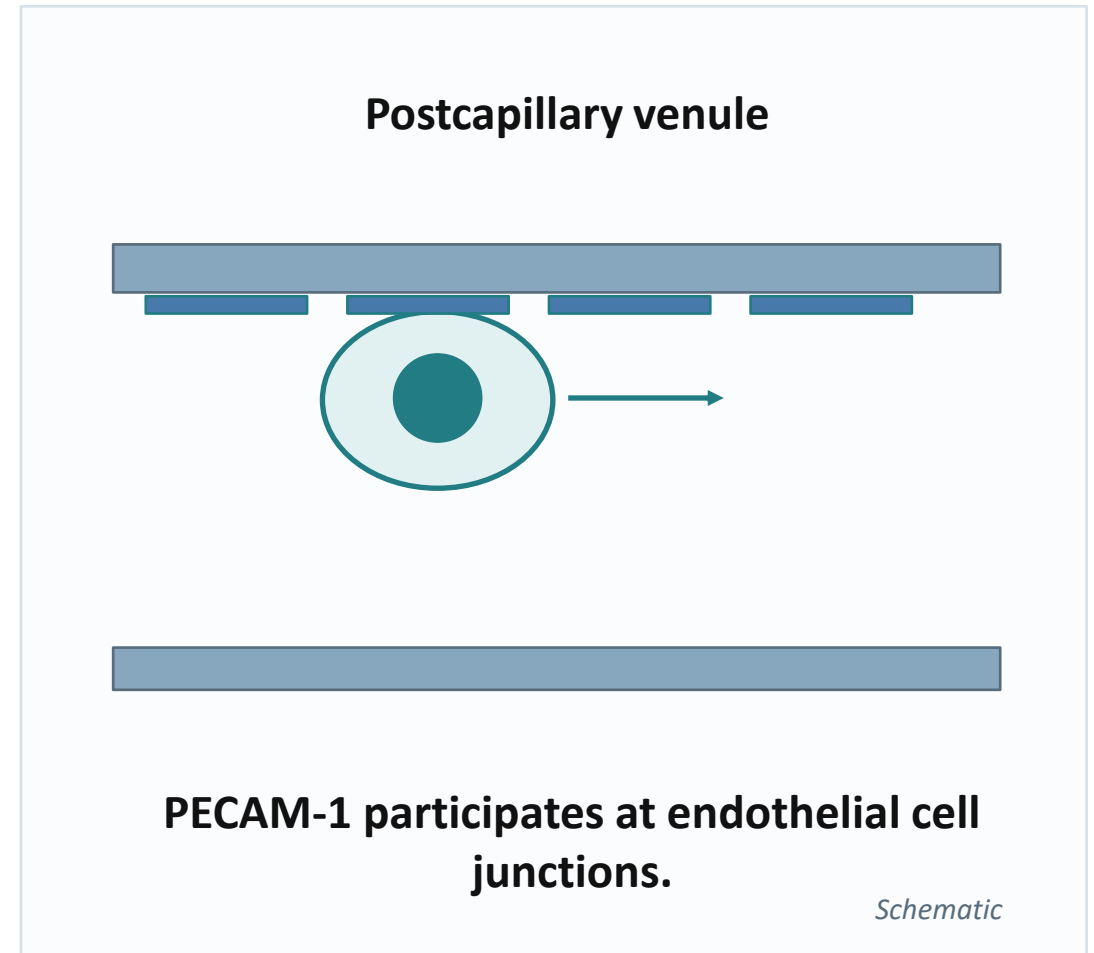
- Activated endothelium expresses integrin ligands including ICAM-1 and VCAM-1.
- Leukocyte LFA-1 and VLA-4 integrins bind their corresponding ligands.
- High receptor affinity and endothelial ligand display produce stable contact.



Firm adhesion is an interaction between two activated cellular surfaces.

CD31 is involved in endothelial transmigration

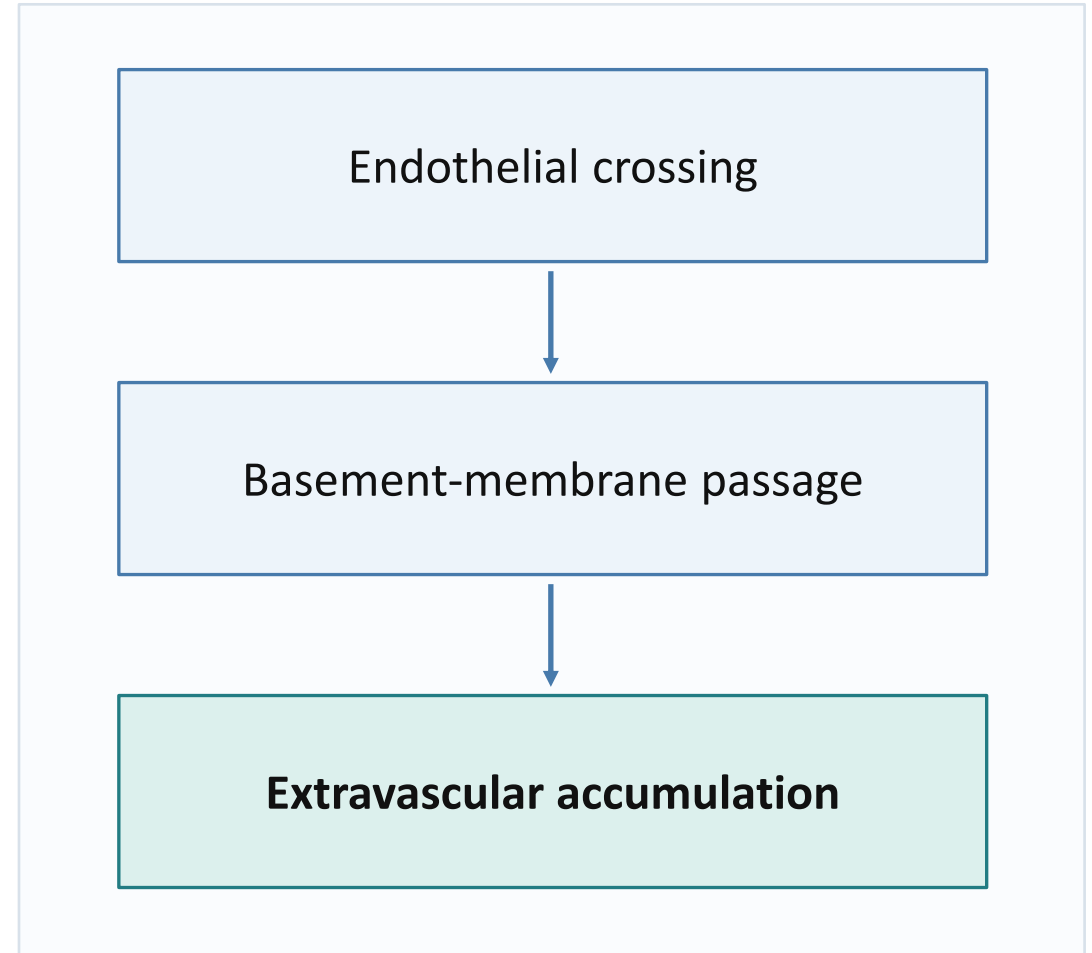
- Leukocytes pass mainly through postcapillary venules in typical acute inflammation.
- CD31 (PECAM-1) participates at endothelial intercellular junctions.
- This step is also termed diapedesis.



Diapedesis follows firm adhesion and precedes full tissue chemotaxis.

Leukocytes then cross the basement membrane

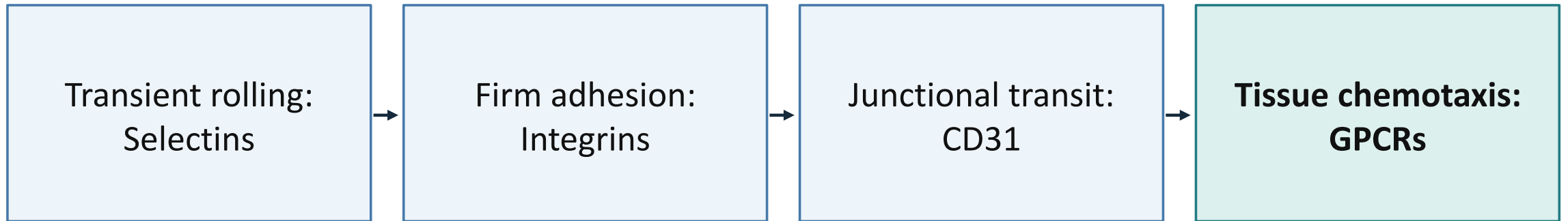
- Following endothelial transit, leukocytes must traverse the vessel basement membrane.
- Collagenase secretion is a proposed means of enabling this passage.
- The basement membrane is repaired after leukocyte transit.



The vascular wall presents sequential structural barriers.

Recruitment culminates in migration toward injury

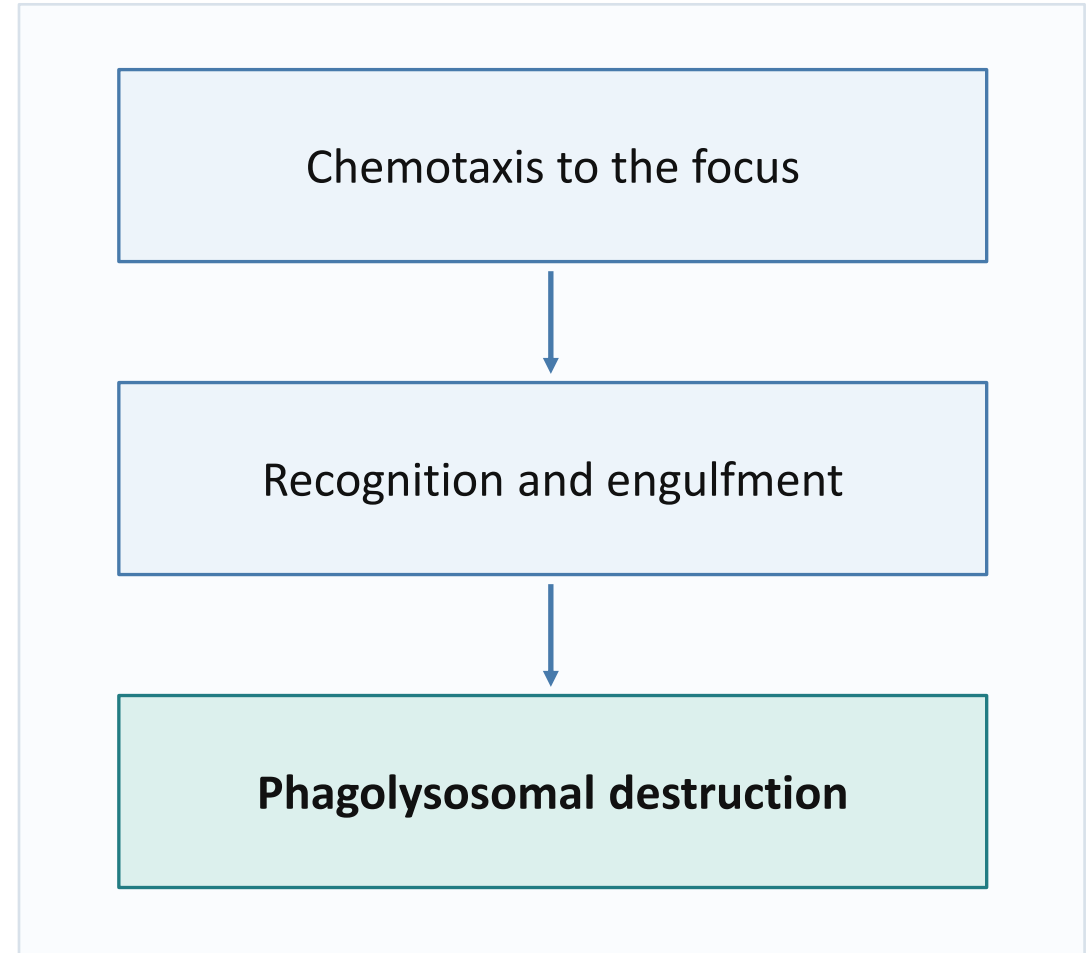
The dominant molecular mechanisms differ at successive stages of recruitment.



Recruitment delivers phagocytic cells to the site requiring defense.

Reaching the target is not the same as killing it

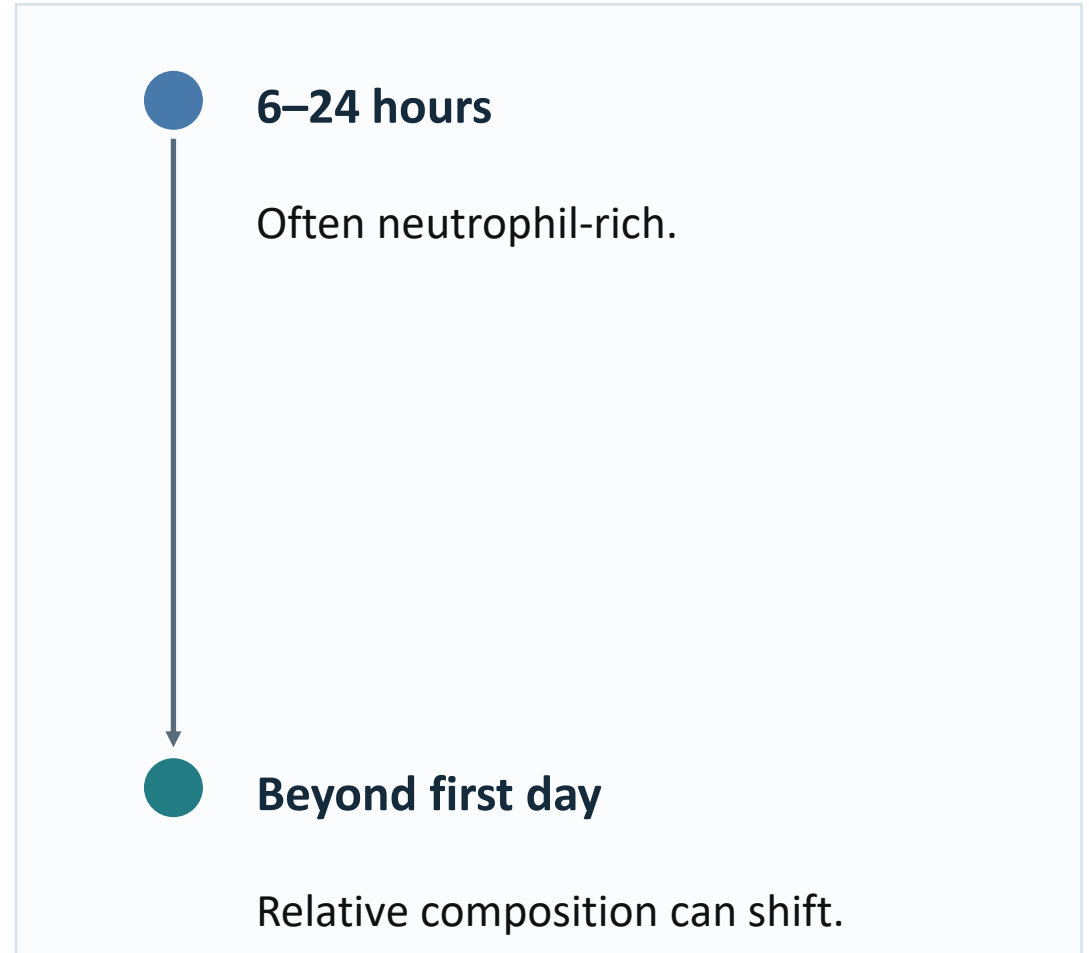
- Chemotaxis delivers leukocytes to infected or injured tissue.
- Recognition receptors then bind microbes and other offending material.
- Phagocytosis and intracellular destruction constitute subsequent effector functions.



Recruitment and microbe removal are sequential but distinct processes.

Neutrophils usually predominate early

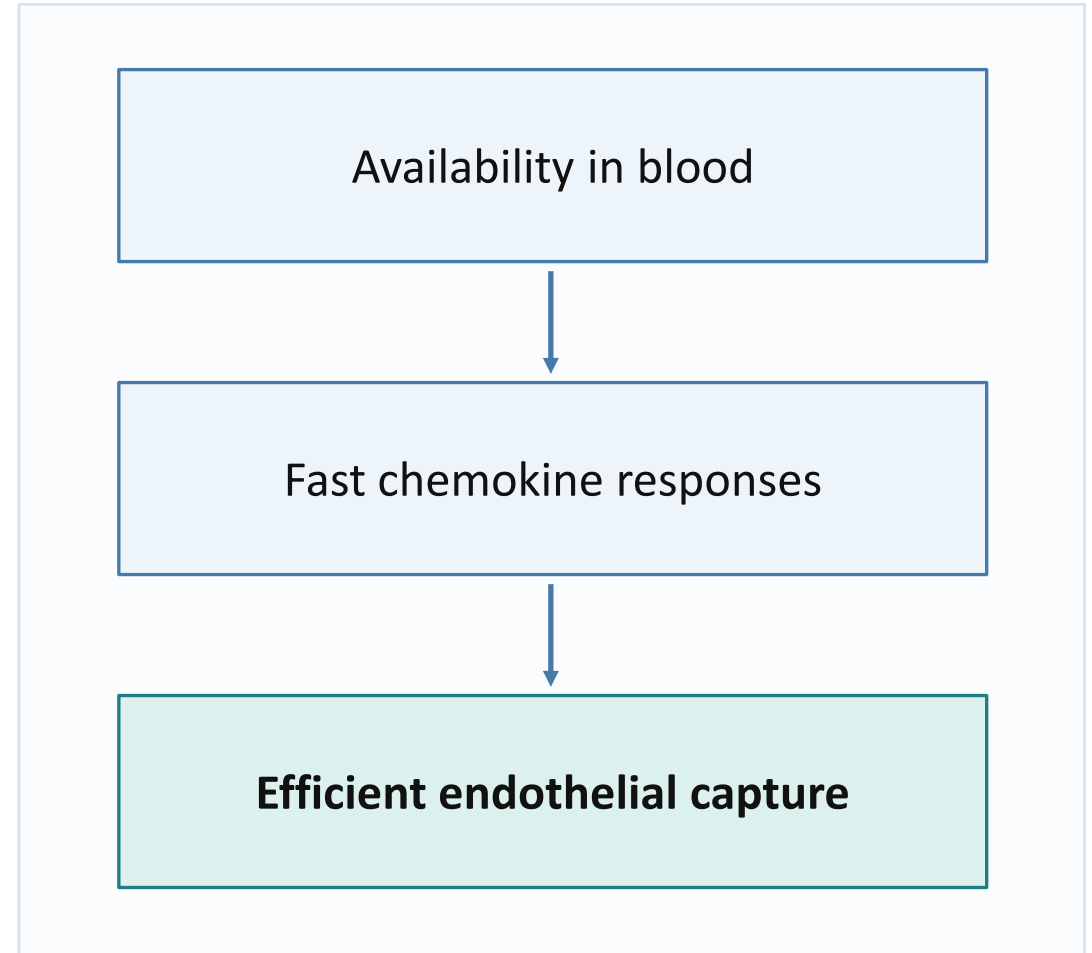
- Neutrophils are the usual major infiltrating cells during early acute inflammation.
- Robbins gives a typical predominance during the first 6–24 hours.
- This is a common pattern, not a universal rule.



Time and stimulus jointly shape infiltrate composition.

Several features favor rapid neutrophil recruitment

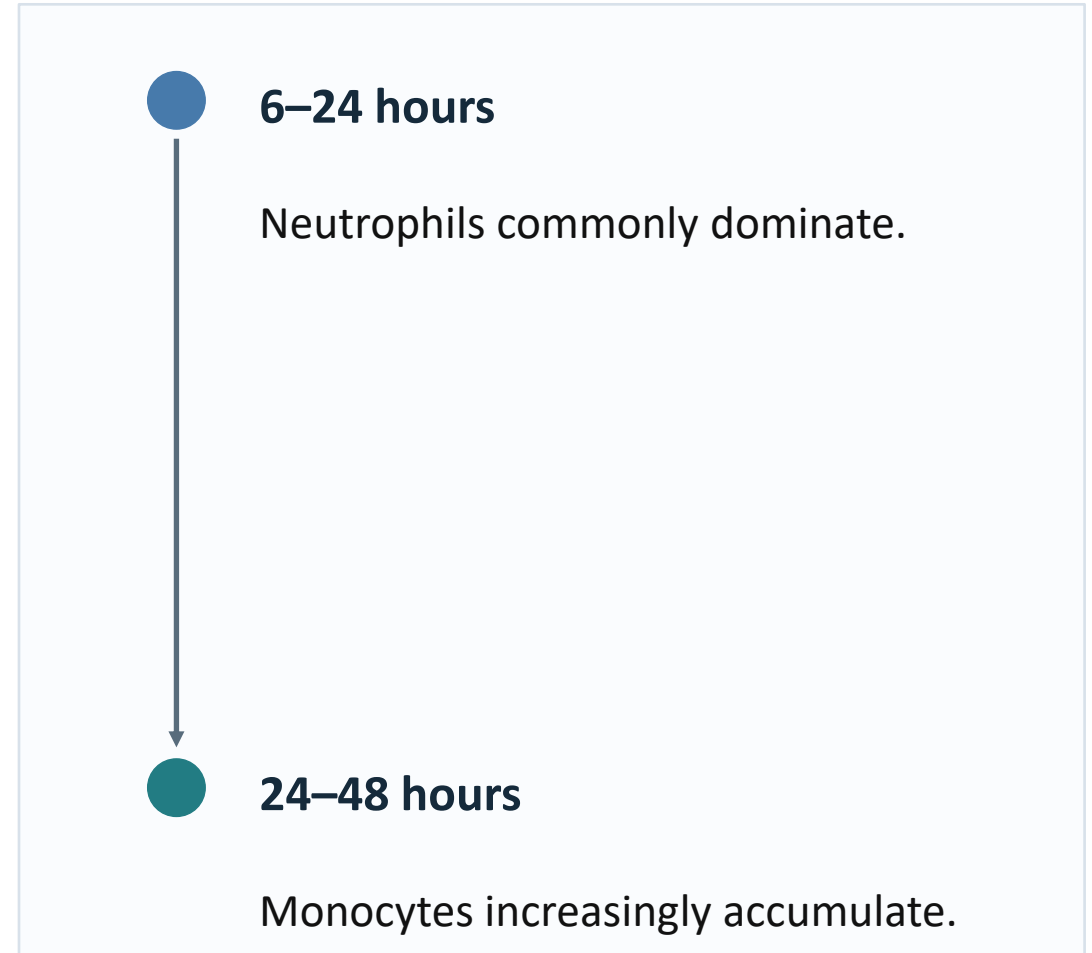
- Neutrophils are abundant in the circulation relative to other leukocytes.
- They respond rapidly to chemokines.
- They may adhere strongly to rapidly induced endothelial P- and E-selectin.



Early neutrophil predominance has multiple contributing reasons.

Monocytes gain prominence at 24–48 hours

- In many acute reactions, monocytes increasingly replace neutrophils after the first day.
- This transition generally within 24–48 hours.
- Recruited monocytes differentiate into tissue macrophages.



A later macrophage-rich field can reflect evolving inflammation.

Neutrophil apoptosis contributes to the cellular shift

- Extravascular neutrophils are relatively short-lived.
- Many undergo apoptosis within days after tissue entry.
- Short survival contributes to their replacement by other inflammatory cells.

Early phase

Rapid influx of neutrophils.

Later phase

Apoptosis reduces accumulated neutrophils.

Loss of existing cells matters as well as recruitment of new ones.

Macrophages can persist during prolonged inflammation

- Macrophages derived from monocytes generally survive longer in tissues.
- Macrophages may proliferate locally within an inflammatory focus.
- They can become dominant when an inflammatory reaction persists.

Neutrophil

Short-lived and rapidly acting.

Macrophage

Longer-lived, potentially locally proliferative.

Persistence of macrophages contributes to later tissue responses.

Cellular composition reflects both stimulus and duration

- The common neutrophil-to-monocyte sequence is a useful baseline.
- Different infections and immune responses can alter that pattern.
- Infiltrate interpretation requires attention to the suspected cause.

Temporal influence

Cell recruitment and survival vary with time.

Etiologic influence

Distinct stimuli recruit different leukocyte types.

An inflammatory differential is mechanistic evidence, not a precise timestamp.

Pseudomonas can sustain a neutrophilic infiltrate

- Certain bacterial infections maintain continuing neutrophil influx for days.
- Robbins specifically names *Pseudomonas* as an example.
- A persistent neutrophil-rich response can therefore outlast the earliest phase.

Clinical observation

Neutrophil-rich tissue several days after infection

Interpretation

Ongoing neutrophil recruitment may be stimulus-dependent.

Teaching vignette — illustrative, not patient data

Do not automatically label prolonged neutrophilic inflammation as atypical.

Some viral infections recruit lymphocytes early

- A lymphocytic infiltrate can be present early in certain viral infections.
- This deviates from the usual initial neutrophil-dominant pattern.
- Interpret the cells in the context of the infectious stimulus.

Clinical observation

Early inflammation with a lymphocytic infiltrate

Interpretation

A viral stimulus is one plausible explanation.

Teaching vignette — illustrative, not patient data

A cell type is informative but not by itself etiologically diagnostic.

Eosinophils may dominate allergic and helminthic responses

- Eosinophils can be the prominent leukocyte population in helminth infection.
- Allergic reactions can also generate eosinophil-rich inflammation.
- Chemokine selectivity, including eotaxin, helps explain this bias.

Clinical observation

Eosinophil-rich inflammatory focus

Interpretation

Consider allergy or helminthic infection among causes.

Teaching vignette — illustrative, not patient data

Stimulus-specific chemotaxis reshapes the inflammatory-cell distribution.

Hypersensitivity may favor mononuclear infiltrates

- Some hypersensitivity reactions favor activated lymphocytes.
- Macrophages and plasma cells may also predominate.
- The infiltrate therefore reflects the associated immune response.

Clinical observation

Lymphocytes, macrophages, plasma cells

Interpretation

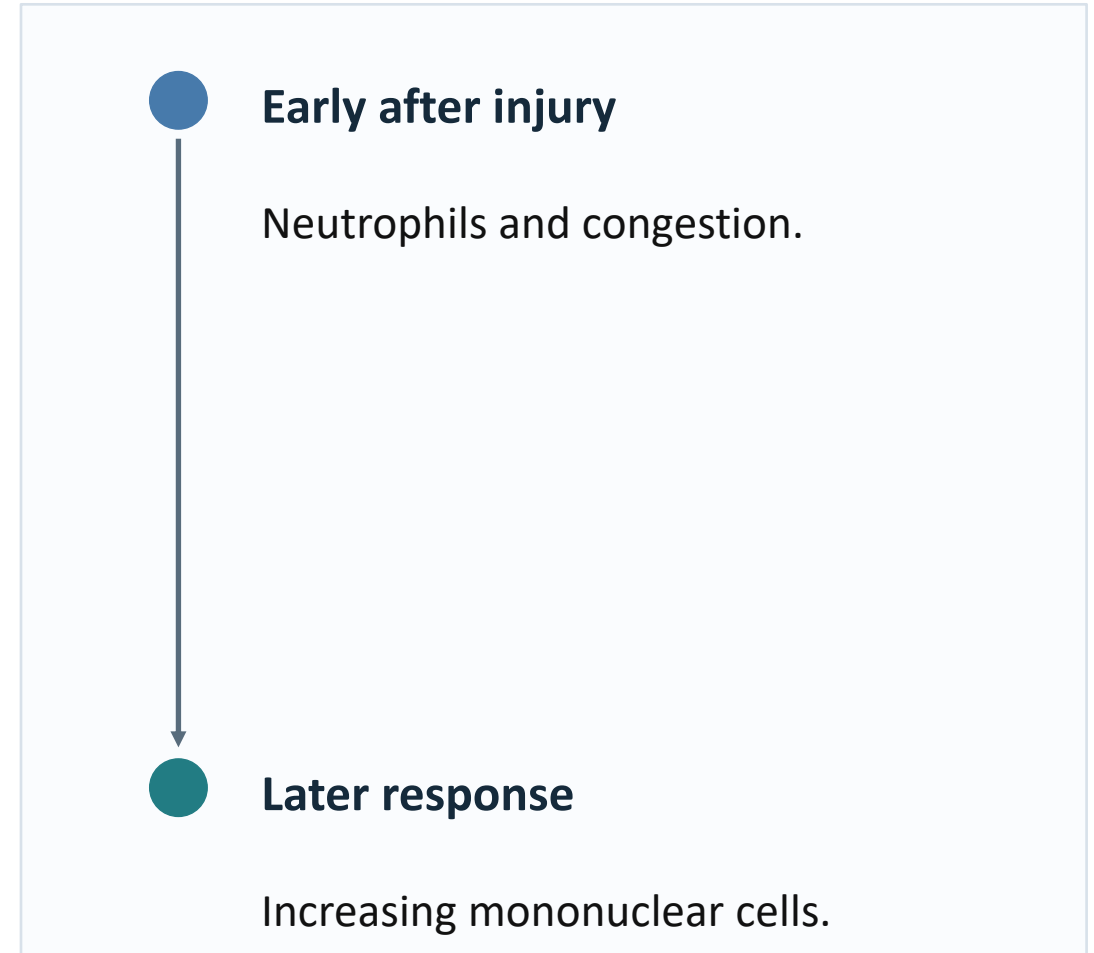
A hypersensitivity reaction is a relevant context.

Teaching vignette — illustrative, not patient data

Not all inflammatory reactions are neutrophil rich.

An infarct provides a temporal histopathology example

- An inflammatory reaction after myocardial ischemic necrosis.
- Early images show neutrophilic infiltrates and congested vessels.
- Later images show a mononuclear cellular infiltrate.



The example explains a sequence; It is not a diagnostic time calculator.

An infiltrate alone cannot precisely date an injury

- Robbins provides approximate kinetics for typical acute inflammation.
- Persistent bacterial stimulation and distinct immune responses produce exceptions.
- Interpret patterns together with morphology and clinical circumstances.

Helpful observation

Relative neutrophil versus monocyte predominance.

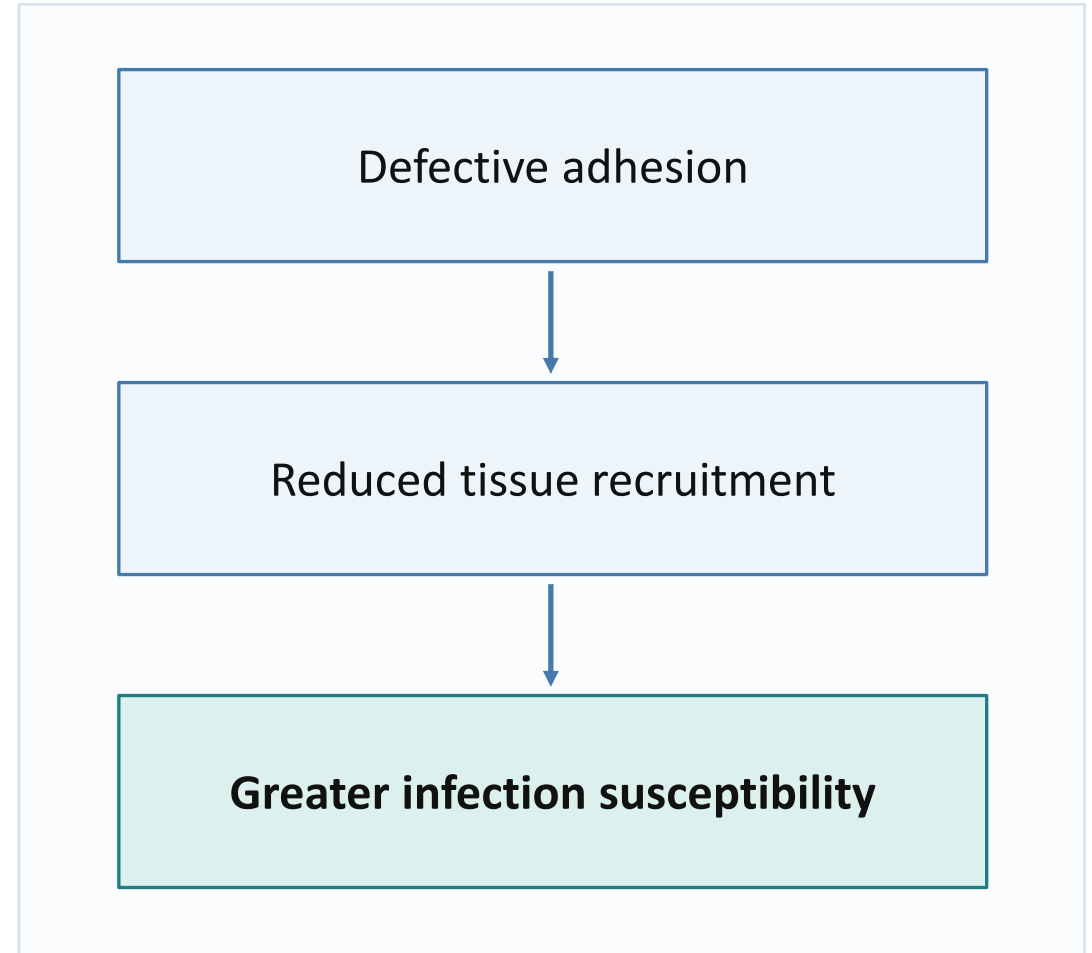
Important limitation

Overlapping stages and stimulus-specific differences.

Temporal interpretation should remain approximate.

Adhesion defects can impair antimicrobial recruitment

- Inherited deficits in leukocyte adhesion molecules predispose to bacterial infections.
- Defective endothelial attachment or migration limits leukocyte delivery.
- Normal chemotactic signals cannot substitute for missing earlier recruitment steps.



Multiple intact steps are required for effective host defense.

Blocking recruitment can compromise host defense

- Therapies targeting TNF or leukocyte integrins can limit harmful inflammation.
- They may also reduce leukocyte entry and antimicrobial responses.
- The intended anti-inflammatory benefit carries an infection-related trade-off.

Desired therapeutic effect

Reduced injurious inflammatory recruitment.

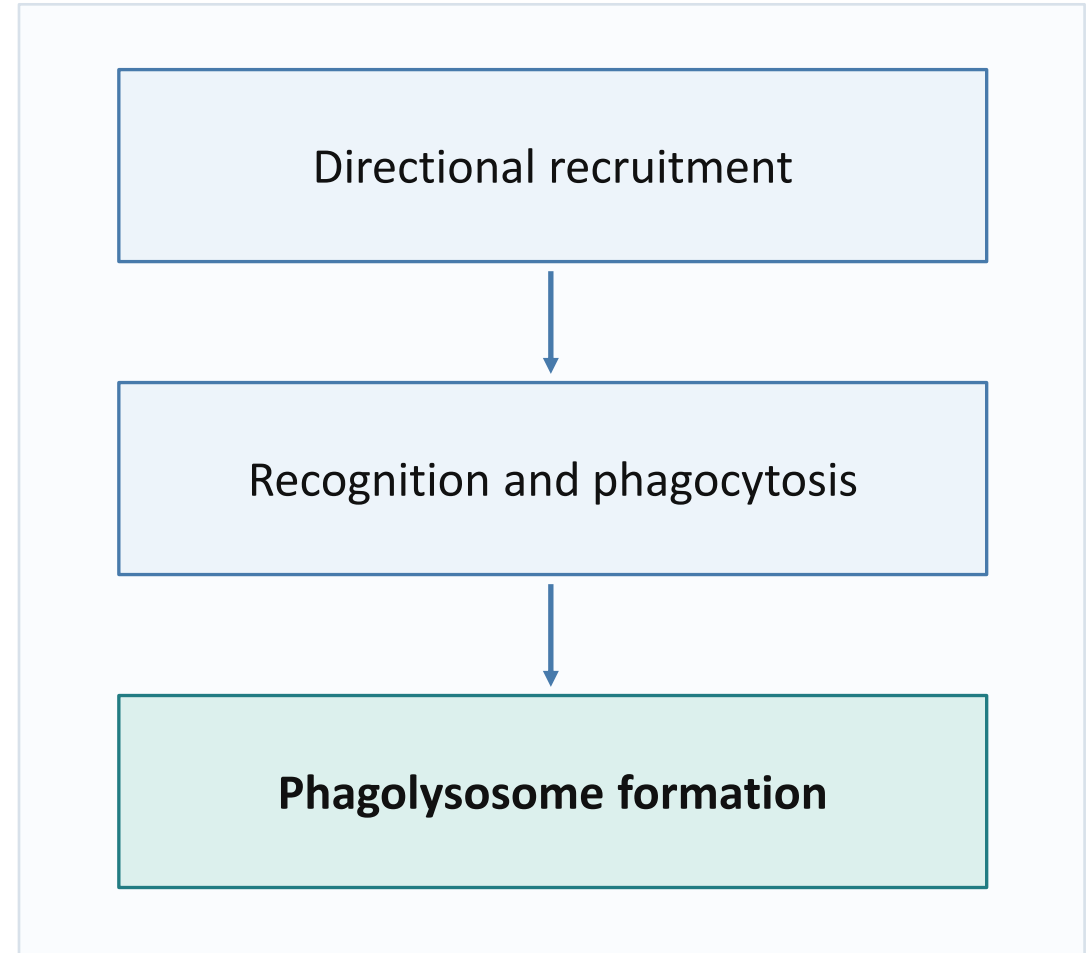
Potential consequence

Impaired defense against infectious microorganisms.

The physiologic role of recruitment explains a key treatment risk.

Chemotaxis precedes phagocytic recognition

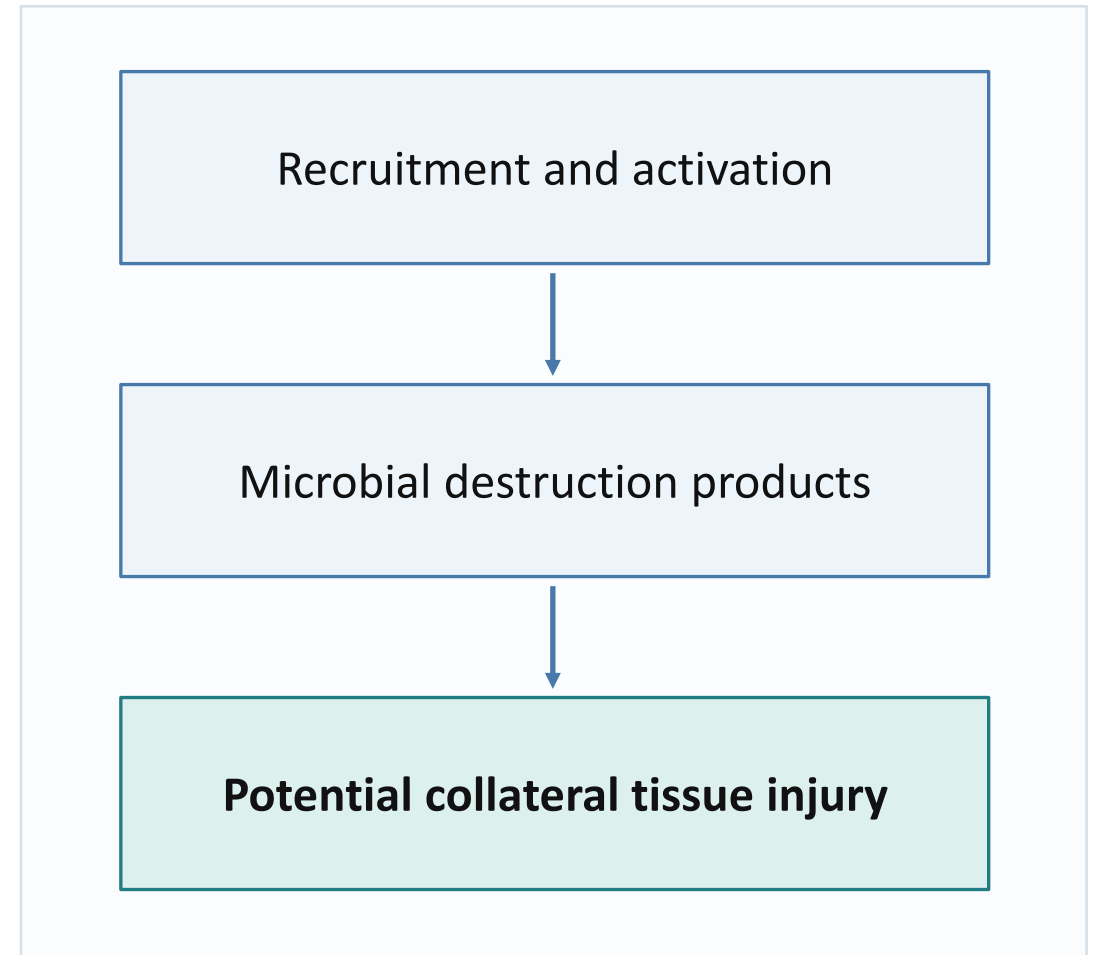
- Neutrophils and macrophages move toward offending material.
- Microbial recognition and attachment then initiate engulfment.
- An intracellular phagosome subsequently fuses with lysosomes.



Arrival at the lesion allows, but does not itself perform, microbial killing.

Recruited leukocytes may also damage host tissues

- Leukocyte activation releases products designed to destroy microbes.
- Reactive oxygen species and lysosomal enzymes can escape their normal compartments.
- The same defensive mechanisms may injure nearby viable host tissue.



Inflammatory recruitment is protective but not intrinsically harmless.

Inflammation contains mechanisms for shutting down recruitment

- Inflammatory mediators generally decay when stimuli subside.
- Neutrophil apoptosis limits their duration in tissues.
- Lipoxins actively oppose neutrophil chemotaxis and adhesion.

Positive phase

Chemoattractants recruit effector leukocytes.

Limiting phase

Mediator clearance, apoptosis, inhibitory lipids.

Resolution depends on diminished stimulus and active restraints.

Applied question: Identify the chemotactic signal

- At a bacterial focus, leukocyte chemotaxis is present without naming a single cytokine.
- Which exogenous and endogenous attractant families could explain this?
- Distinguish the origin of the molecules from their signaling receptor class.

Clinical observation

Bacterial peptides and host inflammatory mediators

Interpretation

N-formyl peptides; CXCL8, C5a, and LTB4 are source-listed cues.

Teaching vignette — illustrative, not patient data

The correct answer contains multiple independent attractant sources.

Applied question: Explain direction of movement

- A neutrophil responds to a higher attractant concentration at one edge.
- Which cytoskeletal changes generate migration toward this edge?
- Connect receptor signaling to actin and myosin position.

Clinical observation

Chemoattractant receptor signals

Interpretation

Leading-edge actin polymerization and posterior myosin localization.

Teaching vignette — illustrative, not patient data

Chemotaxis requires polarized motility rather than passive displacement.

Applied question: Interpret leukocyte predominance

- A tissue specimen shows a mainly eosinophilic inflammatory infiltrate.
- Which stimulus classes belong in the interpretation?
- Why is “more than 48 hours old” not a sufficient explanation?

Clinical observation

Eosinophil-rich infiltrate

Interpretation

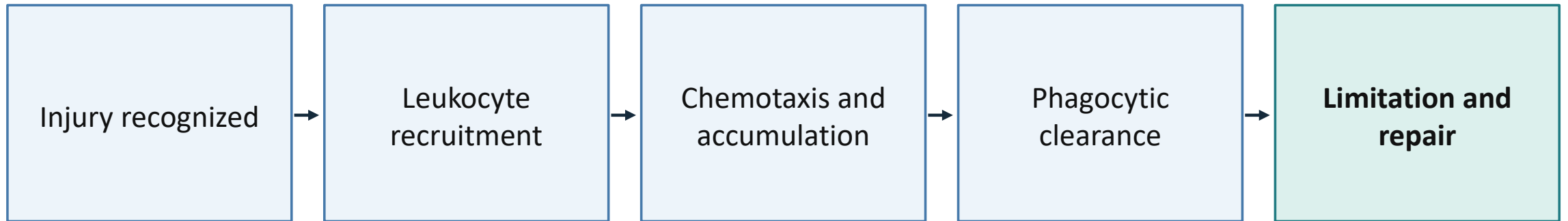
Allergic reactions or helminthic infection are relevant source-listed contexts.

Teaching vignette — illustrative, not patient data

Stimulus identity can override the stereotypic recruitment timeline.

The inflammatory response is an ordered functional chain

Cell migration is indispensable but occupies only part of acute inflammation.



Chemotactic cues couple tissue injury to the positioning of defensive cells.

Mechanism checklist for leukocyte chemotaxis

Level	Essential element	Functional outcome
Signal	Microbial products; CXCL8; C5a; LTB4	Attractive gradient
Recognition	Specific GPCRs	Second-messenger activation
Motility	Front actin; Rear myosin	Directional translocation
Context	Selectins; Integrins; CD31	Effective tissue delivery
Outcome	Recruitment of suitable cells	Local defense ± injury

Reference and scope

Robbins, Cotran & Kumar: Pathologic Basis of Disease, 11th edition (2026)

Chapter 3 — Inflammation and Repair

Core discussion: leukocyte chemotaxis, recruitment and evolving inflammatory infiltrates.

Supporting context: chemokines, complement, leukotrienes, lipoxins and phagocytosis.

Conceptual diagrams are original editable schematics; no histologic specimens are reproduced.