

Osteomalacia and Rickets

Impaired mineralization of bone matrix

Bone development • matrix mineralization • skeletal fragility

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Learning outcomes

- Distinguish undermineralized bone from reduced bone mass.
- Explain vitamin D metabolism and calcium–phosphate feedback.
- Relate growth-plate abnormalities to the deformities of rickets.
- Recognize adult osteomalacia and its skeletal consequences.
- Integrate etiologies, morphology, and laboratory reasoning.

The same defect has two age-dependent expressions

Rickets — growing skeleton

- Unmineralized matrix at the growth plate.
- Abnormal endochondral ossification.
- Distortion of developing bones under load.

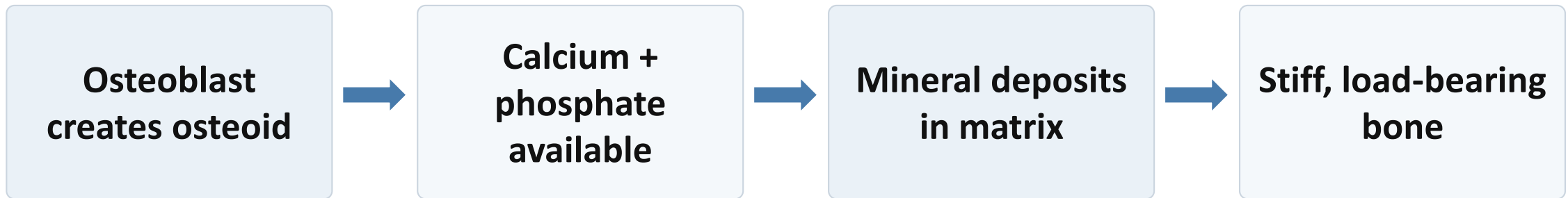
Osteomalacia — mature skeleton

- New remodeling osteoid mineralizes poorly.
- Growth plates are closed.
- Fractures and microfractures of weakened bone.

The defining lesion is defective mineralization, not deficient osteoid production.

Mineralization requires a matrix and a mineral supply

An osteoblast-produced organic scaffold becomes mechanically competent when mineral is deposited.



- 01 Insufficient calcium or phosphate disrupts the deposition step.
- 02 Matrix may be present in excess but remain unmineralized.
- 03 Consequences depend on whether the bone is growing or remodeling.

Growth plate versus adult remodeling

Endochondral growth

- Cartilage expands at the epiphyseal plate.
- Cartilage becomes provisionally calcified.
- Bone replaces cartilage at the metaphysis.

Remodeling in adult bone

- Old bone is removed and replaced.
- Osteoblasts lay down fresh osteoid.
- The new osteoid subsequently mineralizes.

A mineralization block affects growth-plate architecture only before plate closure.

Osteomalacia is not synonymous with osteoporosis

Osteomalacia

- Mineralization of newly formed bone is impaired.
- An excess of unmineralized osteoid persists.
- Reduced mechanical strength predisposes to fracture.

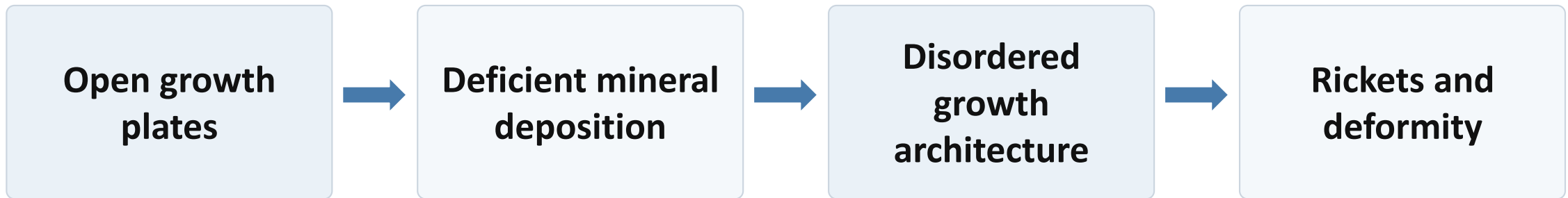
Osteoporosis

- Amount of bone tissue is reduced.
- Remaining bone has normal mineralization.
- Fracture risk rises through loss of bone mass.

The disorders may coexist, but they are not mechanistically interchangeable.

The developmental window determines the phenotype

Bone growth and bone remodeling both require mineral, but they use it in different anatomic settings.



- 01 Epiphyseal cartilage continues to proliferate.
- 02 Insufficient provisional calcification impairs orderly replacement.
- 03 Mechanical loads deform the structurally weak skeleton.

In a skeletally mature adult, the corresponding lesion is osteomalacia.

Rickets and osteomalacia: Anatomic comparison

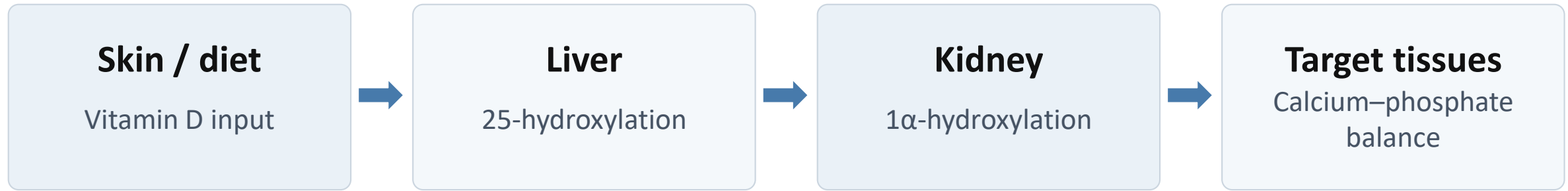
Feature	Rickets	Osteomalacia
Skeletal maturity	Growth plates open	Growth plates closed
Primary setting	Growth-plate ossification	Bone remodeling
Matrix finding	Excess unmineralized osteoid	Persistent, thick osteoid seams
Architecture	Distorted costochondral / metaphyseal regions	Overall contour may remain normal
Mechanical result	Developmental bowing and deformity	Microfractures and overt fractures

Calcium and phosphate are both required

- The mineral phase depends on adequate calcium and phosphate availability.
- Deficiency of either limits effective mineral deposition in osteoid.
- Vitamin D and parathyroid hormone coordinate mineral homeostasis.
- Defective phosphate handling can impair bone even when serum calcium recovers.

A normal calcium result does not prove that skeletal mineralization is normal.

Vitamin D metabolism links skin, liver, and kidney



25(OH)D is the major circulating metabolite; 1,25(OH)₂D is the active hormone.

PTH ↑ kidney activation | FGF23 ↓ kidney activation

UVB-dependent vitamin D production

- Skin 7-dehydrocholesterol undergoes a photochemical conversion.
- UVB exposure produces cholecalciferol (vitamin D₃).
- Reduced UVB exposure can limit endogenous vitamin D supply.
- Melanin absorbs UV light and can reduce production under similar exposure conditions.

The ultraviolet-to-vitamin D step is upstream of liver and kidney activation.

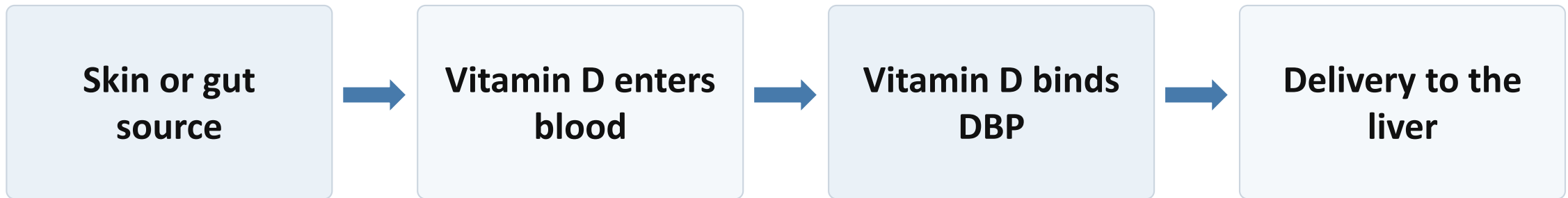
Dietary vitamin D requires intestinal absorption

- Diet and supplements can provide vitamin D independently of skin synthesis.
- Dietary uptake depends on absorption of fat-soluble compounds.
- Malabsorption can reduce effective vitamin D entry despite oral intake.
- A low dietary supply amplifies the effect of limited sunlight exposure.

The nutritional pathway and cutaneous pathway converge in the bloodstream.

Vitamin D-binding protein carries the precursor

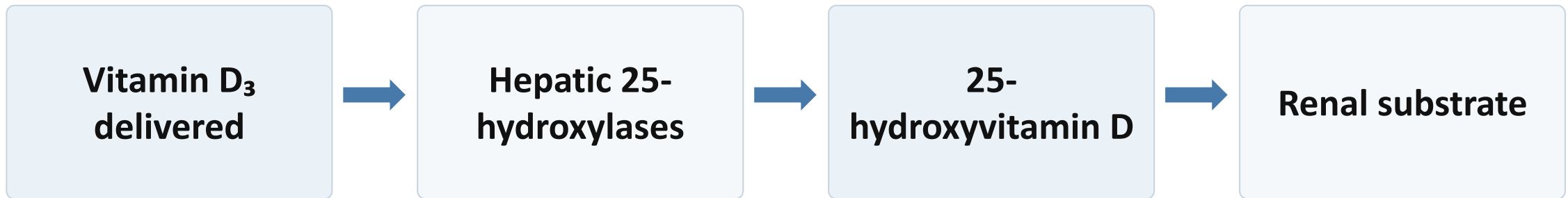
Vitamin D derived from skin and food joins the same transport pathway.



- 01 Vitamin D circulates bound to vitamin D-binding protein (DBP).
- 02 Transport is followed by biochemical activation in two organs.
- 03 Source of entry does not change the subsequent hepatic step.

Hepatic 25-hydroxylation creates 25(OH)D

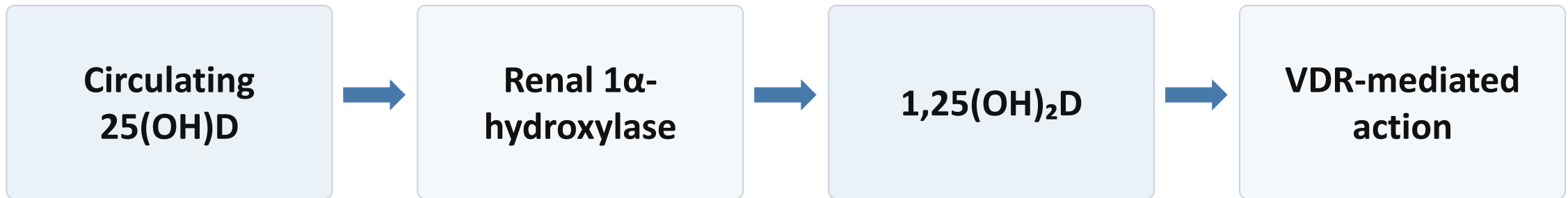
The liver converts vitamin D into its major circulating 25-hydroxy metabolite.



- 01 CYP family hydroxylases participate in the first activation step.
- 02 25(OH)D is the circulating species discussed in deficiency testing.
- 03 Substrate supply can be low even when kidney enzymes are intact.

Renal 1α -hydroxylation produces the active hormone

The kidney converts 25(OH)D to 1,25-dihydroxyvitamin D.



- 01 The renal enzyme is under endocrine and mineral-feedback control.
- 02 Kidney disorders can reduce the final activation step.
- 03 A normal precursor supply alone cannot guarantee sufficient active hormone.

The four controls of renal 1α -hydroxylase

Signals increasing activity

- Hypocalcemia induces PTH secretion.
- PTH enhances renal 1α -hydroxylase.
- Hypophosphatemia upregulates enzyme expression.

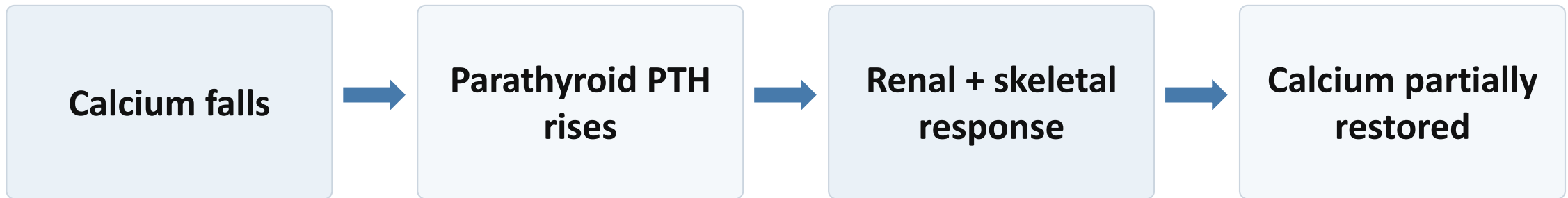
Signals decreasing activity

- $1,25(\text{OH})_2\text{D}$ restrains its own synthesis.
- FGF23 inhibits enzyme expression.
- The balance depends on mineral status.

Feedback coordinates hormone activation with calcium–phosphate availability.

Calcium decline triggers a compensatory PTH response

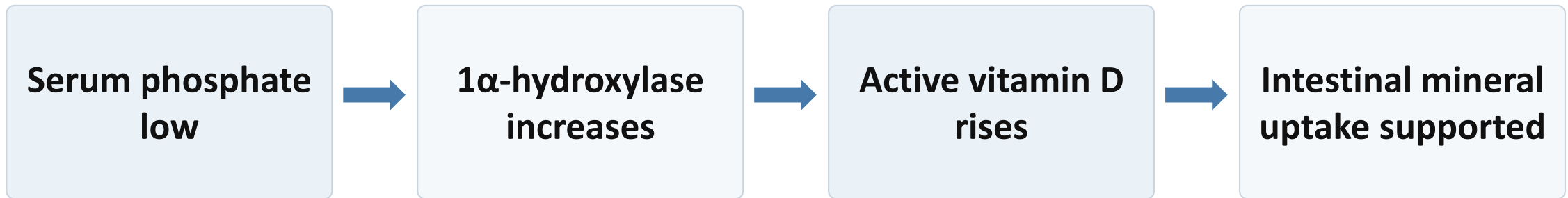
The body prioritizes maintenance of extracellular ionized calcium.



- 01 PTH enhances renal activation of vitamin D.
- 02 PTH promotes bone resorption indirectly through osteoblast RANKL.
- 03 PTH decreases renal calcium loss but increases phosphate loss.

Hypophosphatemia also activates renal vitamin D synthesis

Low phosphate is itself an input to renal 1α -hydroxylase regulation.



- 01 This is a compensatory response to reduced phosphate availability.
- 02 Concurrent PTH-mediated phosphaturia can perpetuate depletion.
- 03 The final mineralization outcome depends on actual mineral supply.

FGF23: Endocrine brake on active vitamin D

Bone-derived signal

- Osteocytes and osteoblasts produce FGF23.
- FGF23 participates in phosphate regulation.
- Pathologic excess may be hereditary or tumor-associated.

Kidney-directed effects

- FGF23 suppresses renal 1α -hydroxylase.
- Phosphate excretion increases through renal mechanisms.
- Reduced phosphate supply may produce rickets or osteomalacia.

FGF23 excess provides a non-nutritional route to defective mineralization.

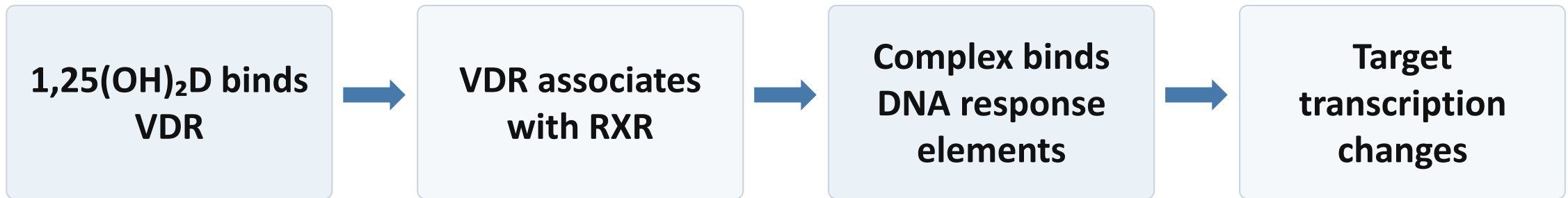
Active vitamin D regulates its own formation

- $1,25(\text{OH})_2\text{D}$ is the active hormonal form of vitamin D.
- Increasing active hormone inhibits expression of renal 1α -hydroxylase.
- Feedback constrains further active-hormone formation.
- It is one of several simultaneous inputs to renal enzyme activity.

The vitamin D pathway is regulated—not simply a one-way conversion chain.

The VDR–RXR complex regulates target genes

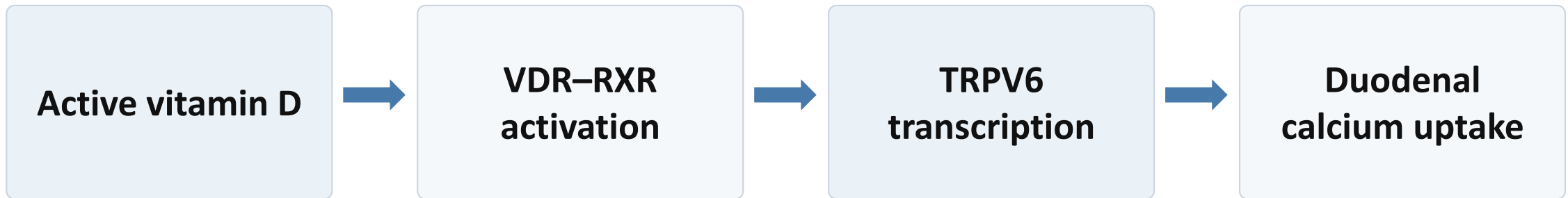
Active vitamin D acts through a nuclear receptor transcriptional mechanism.



- 01** The vitamin D receptor is widely distributed among tissues.
- 02** Intestinal, renal, and skeletal target genes alter mineral handling.
- 03** These effects connect endocrine signaling to bone mineralization.

Intestinal TRPV6 supports calcium absorption

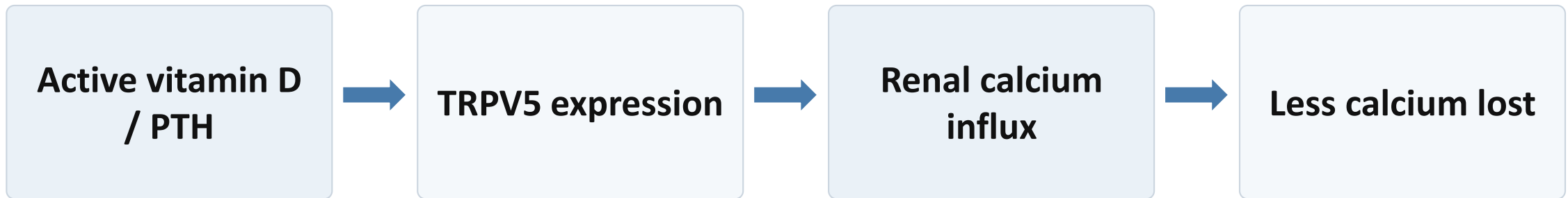
Vitamin D signaling strengthens intestinal calcium transport.



- 01 TRPV6 encodes a calcium transport channel.
- 02 Reduced active vitamin D can impair calcium absorption.
- 03 The skeletal effect is partly mediated by reduced mineral availability.

Renal TRPV5 supports calcium retention

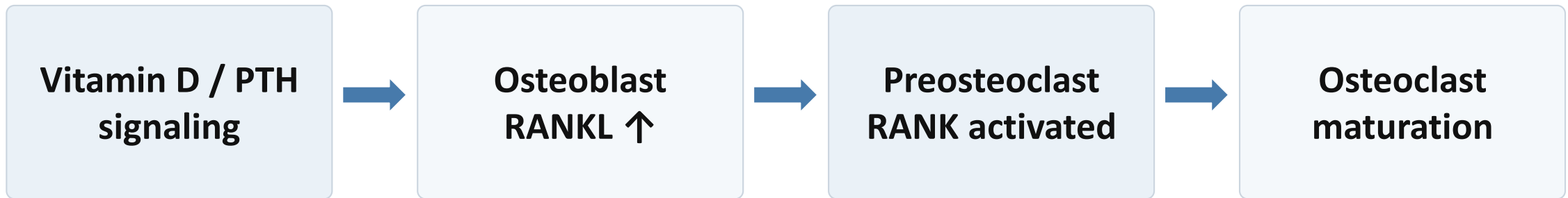
Vitamin D and PTH modify calcium reabsorption in the distal nephron.



- 01 TRPV5 mediates calcium entry in distal tubular cells.
- 02 Both vitamin D and PTH participate in its regulation.
- 03 Calcium conservation helps defend plasma calcium during deficiency.

RANKL links hormone signals to bone resorption

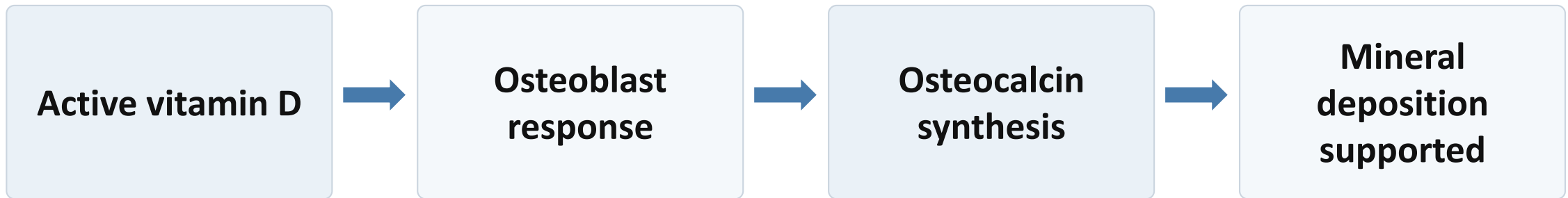
Bone-resorbing osteoclasts respond indirectly to osteoblast signals.



- 01 Osteoclasts resorb mineralized bone and release calcium and phosphate.
- 02 This can support serum calcium during inadequate intestinal uptake.
- 03 Chronic compensation may consume existing bone reserves.

Osteocalcin links osteoblast activity to mineralization

Vitamin D signaling also influences the bone-forming compartment.



- 01 Osteoid is first synthesized as an organic matrix.
- 02 Osteocalcin is a calcium-binding protein involved in bone deposition.
- 03 Deficient substrate availability can still limit final mineralization.

Calcium homeostasis can mask phosphate depletion

What is being defended?

- Ionized extracellular calcium is tightly regulated.
- PTH increases calcium conservation and skeletal release.
- Plasma calcium can return toward normal.

What may remain abnormal?

- PTH enhances phosphate excretion.
- Hypophosphatemia can persist despite calcium recovery.
- New osteoid may then mineralize inadequately.

Assess the full feedback sequence rather than interpreting calcium in isolation.

Causes converge on insufficient mineralization

Upstream defect	Why mineralization fails
Low vitamin D supply	Lower delivery of vitamin D precursor
Inadequate dietary calcium	Reduced mineral substrate
Intestinal malabsorption	Impaired absorption of fat-soluble vitamin D
Renal disease	Less active $1,25(\text{OH})_2\text{D}$ synthesis
Phosphate depletion / wasting	Insufficient phosphate for matrix mineralization
Inherited pathway defects	Vitamin D response or mineral handling altered

Low sunlight exposure restricts endogenous supply

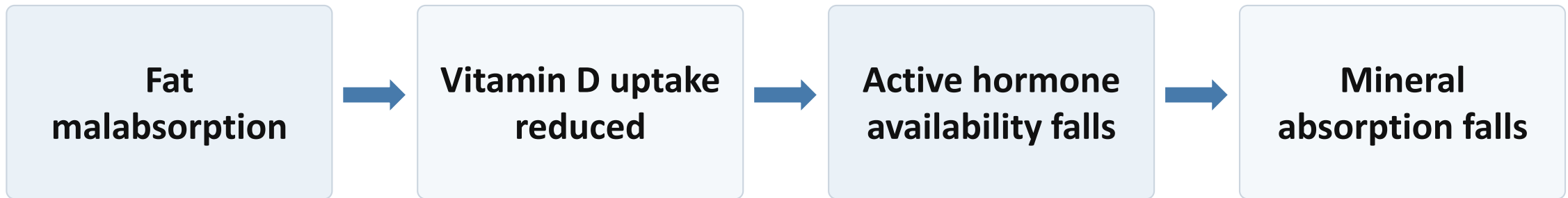
- Less cutaneous UVB exposure means less cholecalciferol generation.
- Northern latitudes and extensive skin covering as contexts.
- The same mechanism can occur at low or high latitudes.
- Clinical susceptibility depends on diet and other patient factors.

Nutritional deficiency reduces the available substrate

- Dietary insufficiency of vitamin D or calcium can impair mineralization.
- Vitamin D availability and calcium availability are related but distinct.
- A diet may fail to compensate for limited cutaneous vitamin D synthesis.
- The downstream lesion is mineralization failure, not absent matrix production.

Fat malabsorption interrupts the nutritional pathway

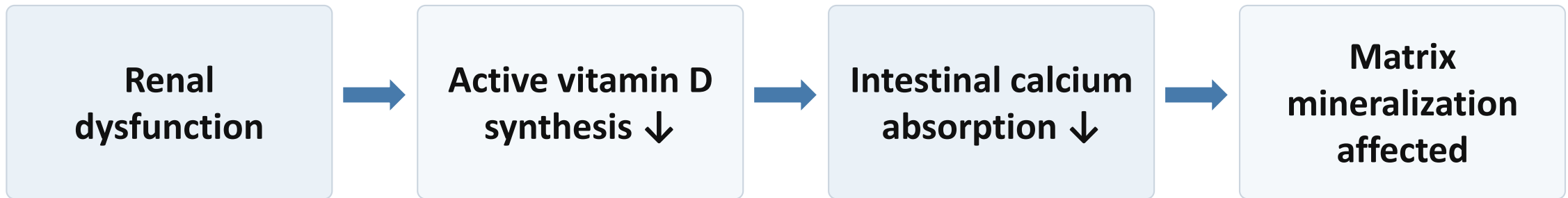
Vitamin D is fat-soluble: Intestinal absorption is an upstream checkpoint.



- 01 The defect begins before liver and renal hydroxylation.
- 02 Available vitamin D substrate may become inadequate.
- 03 The mechanism is relevant to secondary osteomalacia.

Renal disease alters both vitamin D and phosphate

Chronic kidney dysfunction can produce overlapping mineral and bone lesions.



- 01 Renal disease can induce secondary hyperparathyroidism.
- 02 Phosphate excretion and systemic acid–base balance may also be altered.
- 03 Renal osteodystrophy encompasses more than osteomalacia alone.

Phosphate depletion can be sufficient to impair bone

- Phosphate is required alongside calcium for skeletal mineral deposition.
- Excessive renal phosphate loss can lower available phosphate.
- PTH-driven phosphaturia aggravates deficiency-associated mineralization failure.
- An FGF23-mediated disorder offers another route to phosphate depletion.

Inherited defects disrupt different steps

Vitamin D pathway

- Inherited defects may impair vitamin D metabolism.
- Some variants disrupt vitamin D receptor function.
- Target tissues fail to respond normally to active hormone.

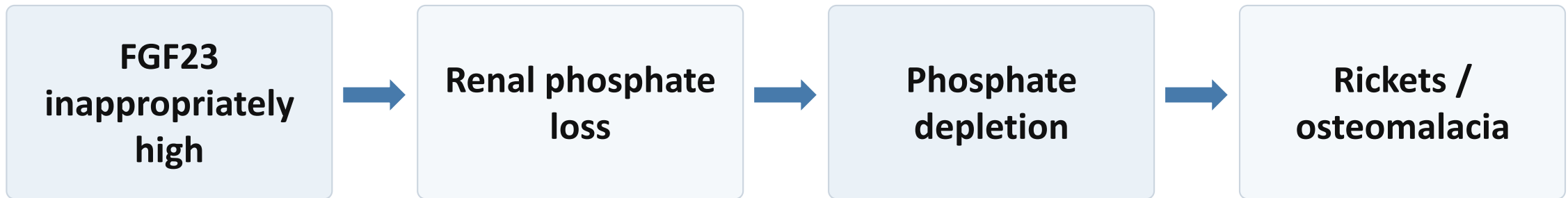
Phosphate pathway

- Inherited abnormalities can cause excessive FGF23 activity.
- Renal phosphate loss limits available substrate.
- Affected children may develop hypophosphatemic rickets.

Inherited rickets is not invariably caused by dietary deficiency.

Excess FGF23 creates hypophosphatemic disease

Overactive bone-to-kidney signaling diverts mineral physiology away from deposition.



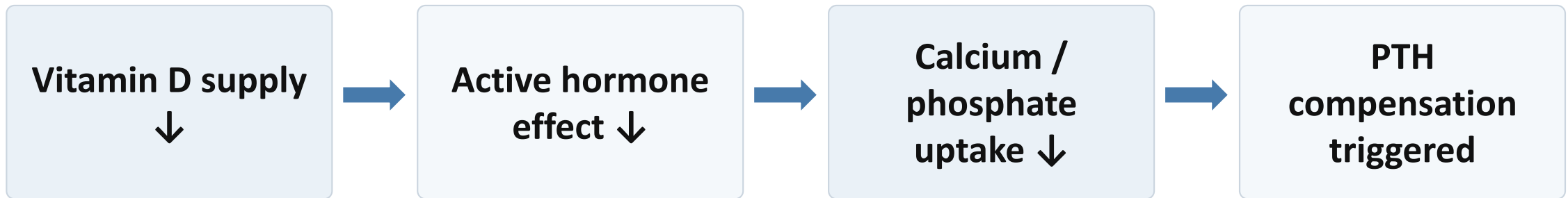
- 01 The textbook recognizes inherited and tumor-associated forms.
- 02 FGF23 also suppresses renal activation of vitamin D.
- 03 These effects reinforce inadequate mineral deposition.

Tumor-associated phosphate wasting is a distinct mechanism

- Certain tumors are associated with inappropriately high FGF23 production.
- Phosphate loss can lead to osteomalacia in adults.
- The endocrine mechanism differs from limited UVB or dietary vitamin D.
- A careful etiologic evaluation distinguishes nutrient deficiency from phosphate wasting.

Vitamin D deficiency starts an endocrine cascade

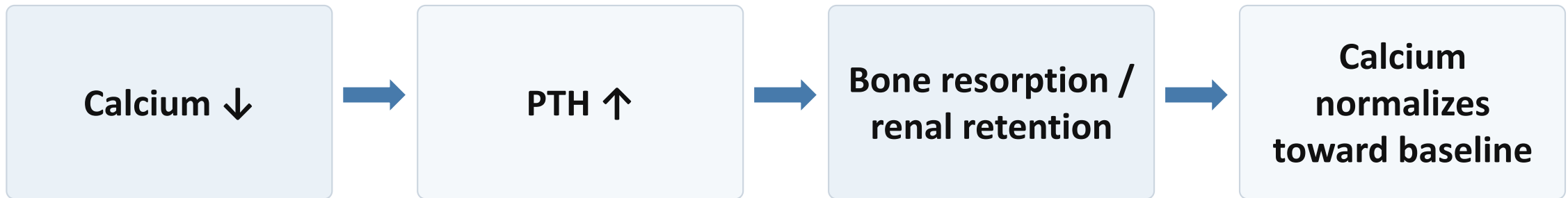
Inadequate vitamin D availability reduces mineral uptake from the gut.



- 01 Calcium falls initially in the causal model.
- 02 The parathyroid response is a homeostatic consequence.
- 03 Phosphate can remain inadequate for mineralization.

PTH compensation preserves calcium at a skeletal cost

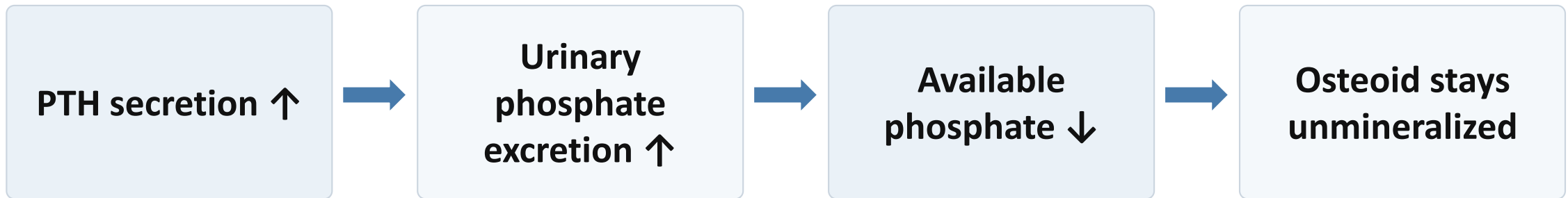
Secondary hyperparathyroidism is a response to calcium stress.



- 01 PTH increases renal calcium conservation.
- 02 RANKL-mediated bone resorption releases stored calcium.
- 03 The concurrent phosphate loss creates a competing problem.

Phosphaturia explains persistent undermineralization

Defending calcium concentration does not assure adequate phosphate concentration.



- 01 Hypophosphatemia may persist despite near-normal calcium.
- 02 Excess unmineralized matrix accumulates where bone is newly formed.
- 03 The final phenotype reflects skeletal maturity.

This is the mechanistic link between endocrine compensation and osteoid accumulation.

Why serum calcium may be misleading

Compensated state

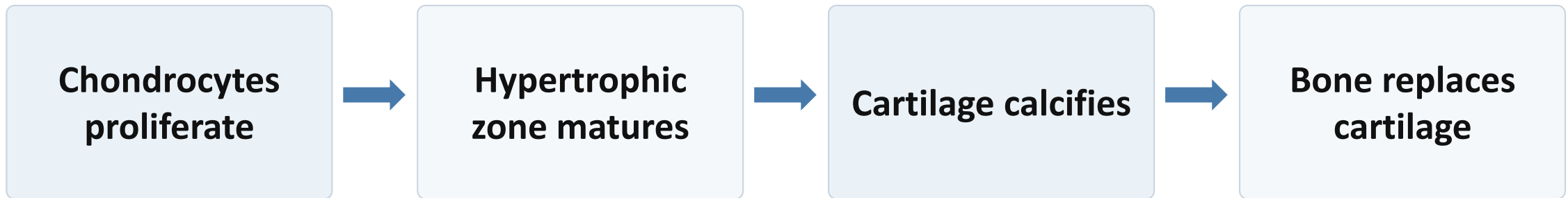
- Calcium is mobilized from bone.
- Renal calcium excretion decreases.
- The measured circulating calcium may be near normal.

Uncompensated skeletal effect

- Phosphate continues to be lost in urine.
- Cartilage/osteoid mineralization remains defective.
- Weak bone and growth-plate abnormalities continue.

Normal endochondral ossification has an ordered sequence

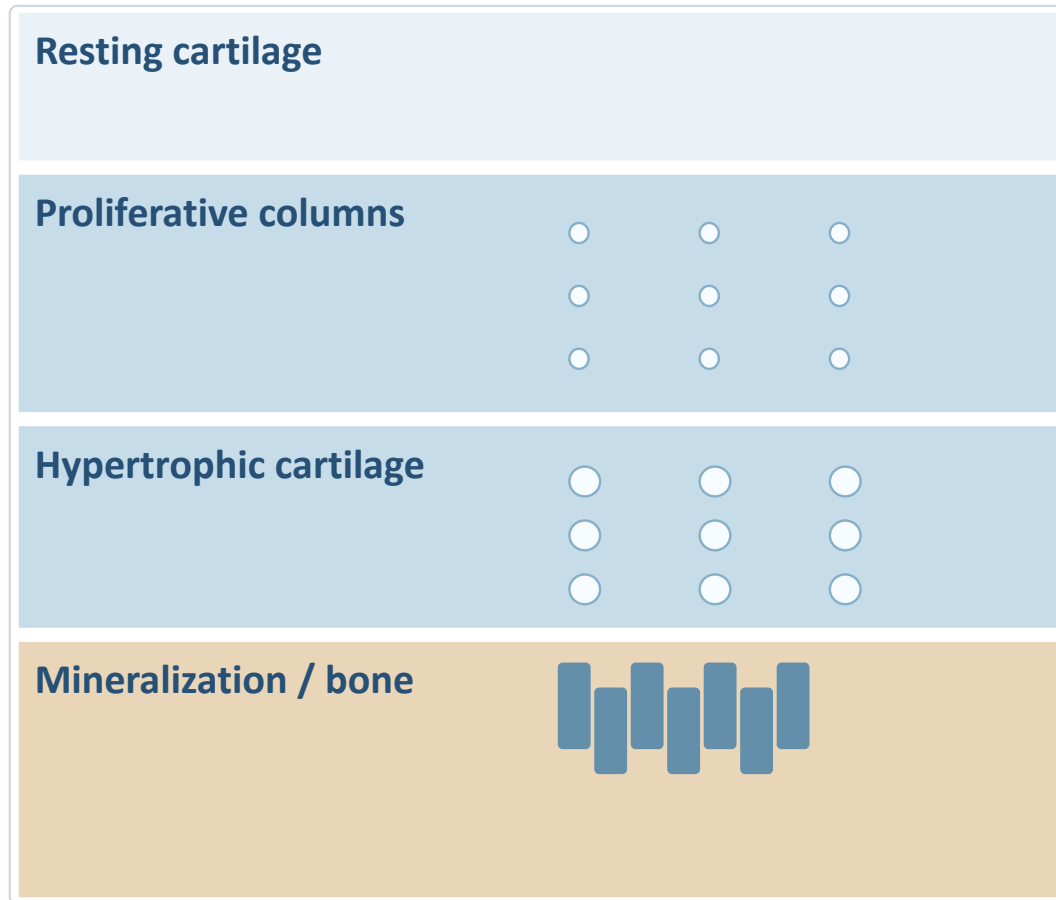
Growth plate cartilage is a temporary scaffold for newly formed long bone.



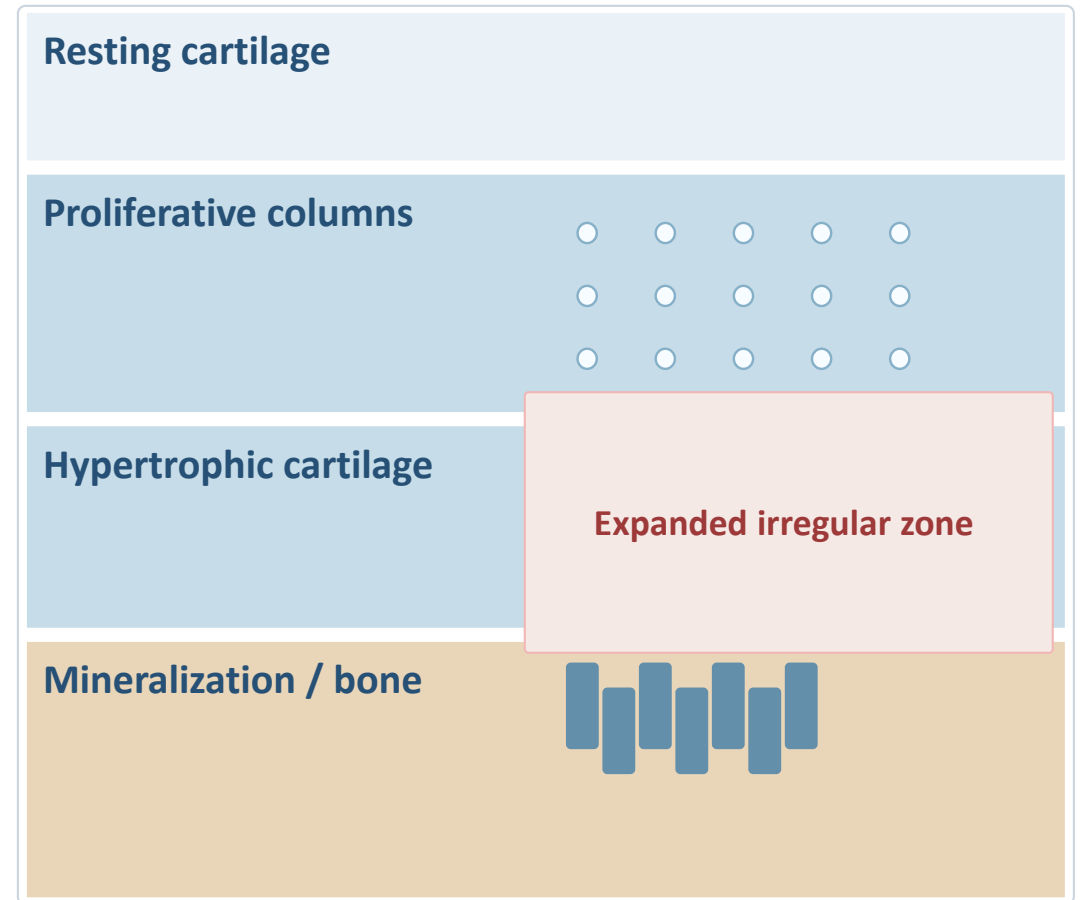
- 01 Cell arrangement is ordered in longitudinal columns.
- 02 Provisional cartilage mineralization precedes replacement by bone.
- 03 Loss of this sequence is fundamental to rickets.

Architecture of a normal and rachitic growth plate

Original schematic — not a histology image



Orderly growth plate



Rachitic growth plate

Rickets begins with failed provisional calcification

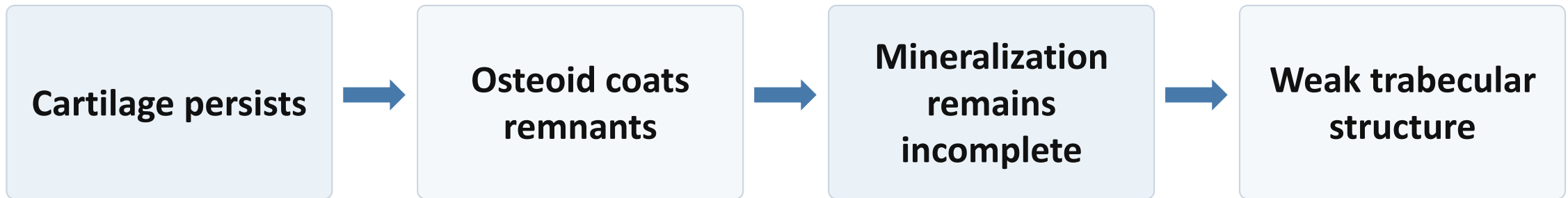
- Epiphyseal cartilage continues growing but fails to calcify normally.
- Chondrocytes fail to complete orderly maturation and disintegration.
- The growth-plate region expands instead of transitioning cleanly to bone.
- This initiates the histologic disorganization of rickets.

Cartilage remnants extend into the marrow space

- Irregular masses of incompletely removed cartilage persist.
- These remnants can project into the adjacent marrow cavity.
- They mark failure of orderly cartilage-to-bone replacement.
- The abnormal substrate distorts developing metaphyseal architecture.

Osteoid is deposited on an inadequate scaffold

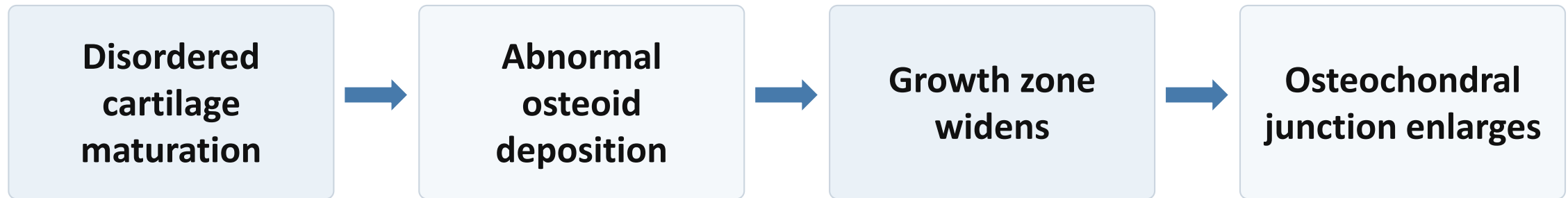
Osteoblast deposition continues despite defective mineralization of cartilage remnants.



- 01 The lesion combines abnormal residual cartilage and unmineralized osteoid.
- 02 Bone production is not the same as production of mineralized bone.
- 03 Accumulated poorly mineralized matrix contributes to deformation.

The osteochondral junction expands laterally

Architectural disorganization becomes a macroscopic metaphyseal abnormality.



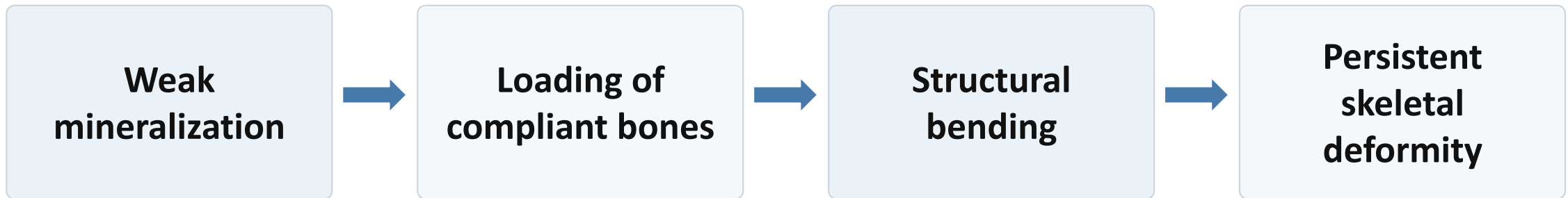
- 01 Normal cartilage palisades are lost.
- 02 Irregular osteochondral boundaries replace an organized junction.
- 03 Gross widening reflects accumulated poorly mineralized tissue.

Microfracture repair adds fibrovascular tissue

- Weak mineralized tissue develops microfractures under mechanical stress.
- Capillaries and fibroblasts proliferate in disorganized zones.
- This reparative response further disturbs growth-plate architecture.
- The pathologic process is mechanical plus metabolic—not purely inflammatory.

The developing skeleton deforms under mechanical loads

The distribution of deformity changes as an affected child grows.



- 01 Infants load the skull and chest disproportionately.
- 02 Walking transfers major load to pelvis, spine, and lower limbs.
- 03 Rickets expresses the interaction of tissue weakness and mechanical stress.

Age changes the sites of visible rickets

Before walking

- Head and chest experience important compressive forces.
- Occipital and parietal bone changes may be evident.
- Costochondral junctions may become prominent.

After walking

- Lower limbs bear greater longitudinal load.
- Tibial bowing may develop.
- Spinal and pelvic deformities become more conspicuous.

Craniotabes reflects a softened infant skull

- Softened occipital bone may flatten under pressure.
- Parietal regions can be depressed by external force.
- Elastic recoil allows the buckled bone to snap back.
- This physical sign reflects reduced skeletal rigidity.

Frontal bossing reflects accumulation of osteoid

- Excess poorly mineralized osteoid accumulates during skull growth.
- Frontal prominence can produce a squared head contour.
- The change is a gross expression of altered mineralization during development.
- Extent varies with disease severity and duration.

The rachitic rosary marks widened costochondral junctions

- Cartilage/osteoid accumulates at rib costochondral junctions.
- The junctions become enlarged and visibly prominent.
- Serial prominences along the chest wall constitute the rachitic rosary.
- The morphology follows directly from abnormal growth-plate mineralization.

Pigeon breast arises from rib and chest-wall deformation

- Poorly mineralized ribs are more susceptible to respiratory-muscle forces.
- Weak metaphyseal rib regions can bend inward.
- The sternum may consequently protrude anteriorly.
- The resulting pattern is described as pigeon breast deformity.

Bowing of the legs follows weight bearing

- Tibial bone is exposed to substantial mechanical load after ambulation begins.
- Insufficient mineralization decreases resistance to bending.
- Progressive deformation can produce bowed lower limbs.
- The shape reflects both metabolic tissue weakness and habitual stress.

Spinal and pelvic stress causes further deformity

- Ambulation shifts load to axial and appendicular regions.
- Spinal deformation may include lumbar lordosis.
- The pelvis can be affected by the same mechanical weakness.
- Severity depends on timing, duration, and anatomic stress.

The visible lesions of childhood rickets

Each manifestation localizes mechanical stress onto poorly mineralized developing tissue.

Skull: Craniotabes

Compliant cranial bone buckles under pressure

Frontal bossing

Excess osteoid during skull growth

Rachitic rosary

Costochondral junction enlargement

Pigeon breast

Rib deformation with anterior sternal protrusion

Bowed tibiae

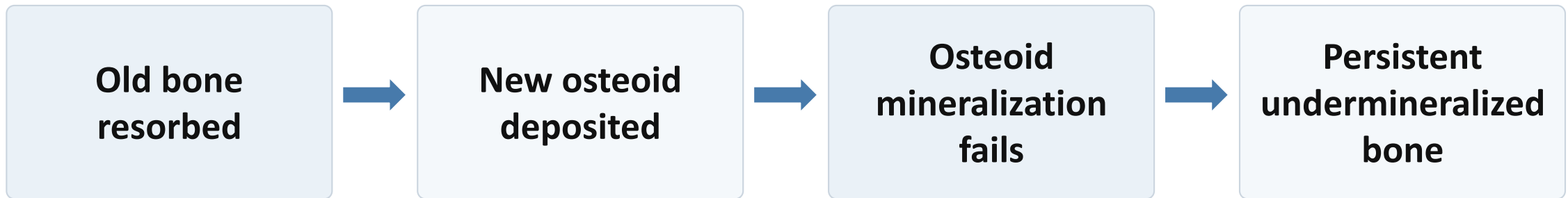
Weight-bearing load bends weak long bones

Lumbar lordosis

Axial skeletal deformation after ambulation

Osteomalacia is a remodeling-stage mineralization defect

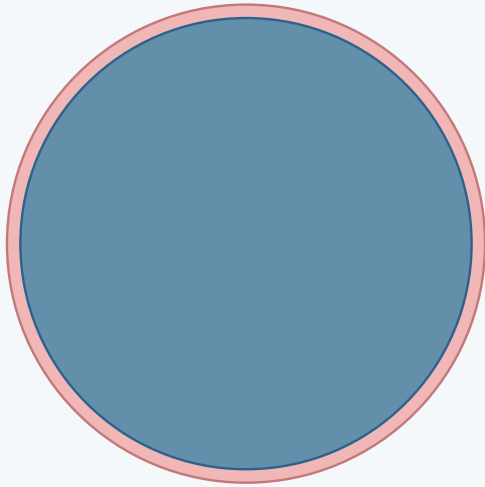
The adult skeleton has no open growth plates, but remodeling remains continuous.



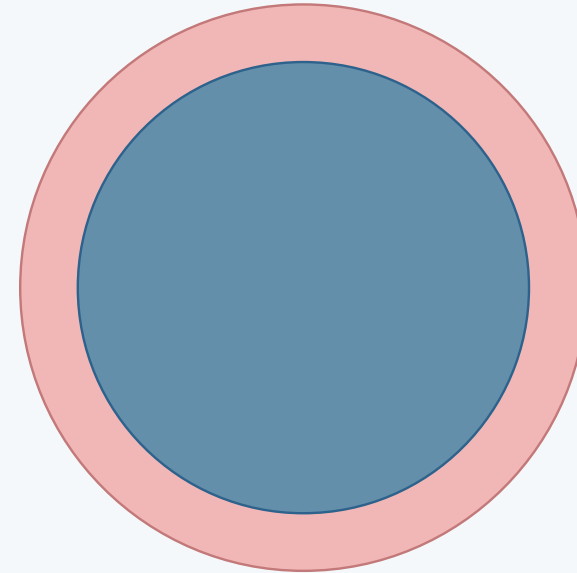
- 01 Defective mineralization occurs in newly synthesized matrix.
- 02 Unmineralized osteoid is abnormally retained along trabeculae.
- 03 Overall skeletal contours need not change.

Osteomalacia features widened unmineralized osteoid seams

Simplified trabecular cross-sections: Mineralized matrix versus unmineralized osteoid



Normally mineralized trabecula



Osteomalacic trabecula

 Mineralized

 Osteoid

Histology: Osteoid persists around mineralized trabeculae

- New osteoid is normally a transient stage before mineral deposition.
- In osteomalacia, osteoid seams become abnormally prominent.
- On H&E, unmineralized osteoid is pink around more basophilic mineralized trabeculae.
- Assessment of a true specimen requires histologic context and appropriate tissue processing.

The femoral neck is a site of skeletal fragility

- Poorly mineralized remodeled bone has impaired mechanical competence.
- Microfractures or overt fractures may affect femoral necks.
- The problem is matrix quality, even without gross bony contour distortion.
- Fracture risk follows the failure of new osteoid to mineralize.

Vertebral bodies may develop microfractures

- The axial skeleton undergoes continual loading and remodeling.
- Under-mineralized trabecular tissue is susceptible to microfailure.
- Vertebral bodies are specifically identified as vulnerable sites.
- Repeated subclinical damage can precede a major fracture.

Adult contour may be preserved despite weakness

Morphologic appearance

- Long-bone contour may remain essentially normal.
- Trabeculae carry excessive unmineralized osteoid.
- Gross inspection may not convey tissue quality.

Mechanical consequence

- Weakness predisposes to microfractures.
- Femoral necks and vertebrae are vulnerable.
- Fractures can occur without a rachitic shape change.

Differential diagnosis: Defects of quantity or mineralization

Disorder	Primary abnormality	Characteristic setting
Osteomalacia	Failure to mineralize newly formed osteoid	Adult bone remodeling
Rickets	Failure to mineralize developing bone and growth plate	Skeletally immature patient
Osteoporosis	Reduced bone mass with normal mineralization	Adult skeletal fragility
Hyperparathyroid bone disease	PTH-driven osteoclast resorption	Primary or secondary PTH excess
Renal osteodystrophy	Mixed patterns of turnover and mineralization	Chronic kidney dysfunction

Renal osteodystrophy is broader than osteomalacia

High-turnover processes

- Secondary hyperparathyroidism raises remodeling activity.
- Resorption may predominate over formation.
- Associated bone loss can coexist with mineral disturbance.

Low-turnover processes

- Adynamic bone has little cellular turnover.
- Osteomalacia can be present in some low-turnover cases.
- Mixed patterns also occur in renal disease.

Do not use “renal osteodystrophy” as a synonym for a single mineralization defect.

Physiology guides etiologic investigation

Upstream defect	Why mineralization fails
Cutaneous vitamin D synthesis	UVB exposure and skin-related context
Dietary vitamin D / calcium	Intake and nutritional context
Intestinal absorption	Malabsorption and fat handling
Renal activation	Kidney function and vitamin D metabolism
Renal phosphate handling	Phosphate availability / wasting
Endocrine regulators	PTH–FGF23–vitamin D feedback

25(OH)D and 1,25(OH)₂D answer different questions

- 25(OH)D is generated in the liver and circulates as a major metabolite.
- 1,25(OH)₂D is the active hormone generated in the kidney.
- The level of active hormone is subject to PTH, phosphate, FGF23, and feedback.
- The source defines vitamin D deficiency using a circulating 25(OH)D measurement.

Robbins (11th ed.) states vitamin D deficiency at 25(OH)D < 20 ng/mL; Laboratory standards may vary.

Mechanism-based interpretation of a deficiency pattern

Initial deficit

- Vitamin D availability is reduced.
- Intestinal calcium and phosphate uptake fall.
- Serum calcium may decline initially.

Compensated physiology

- PTH increases to defend extracellular calcium.
- Renal phosphate loss can persist.
- Insufficient phosphate limits osteoid mineralization.

These are directional pathophysiologic relationships, not a universal diagnostic laboratory profile.

Teaching case: A child with progressive leg bowing

TEACHING SCENARIO

A walking child develops progressive tibial bowing and visible costochondral prominences. A history suggests limited sunlight exposure and low dietary vitamin D.

Which tissue defect best unifies the skeletal findings?

- A. Failure of provisional growth-plate calcification
- B. Normally mineralized but quantitatively depleted bone
- C. Excessive cartilage-to-bone conversion
- D. Primary mature-bone remodeling without plate involvement

Case interpretation: Growth-plate failure

A — impaired provisional calcification at an open growth plate

REASON 1

Persistent cartilage and osteoid disorganize the metaphyseal junction.

REASON 2

Costochondral enlargement explains the rachitic rosary.

REASON 3

Weight bearing bends inadequately mineralized long bones.

Age, growth-plate status, and loading explain the pattern of rickets.

Teaching case: Adult fractures without gross bowing

TEACHING SCENARIO

An adult with a condition affecting intestinal fat absorption develops skeletal fragility, including a femoral-neck fracture. Bone remodeling continues, but new matrix mineralizes inadequately.

Which pathologic lesion is most consistent with the scenario?

- A. Persistent widened seams of unmineralized osteoid
- B. Overgrowth of epiphyseal growth-plate cartilage
- C. Preserved mineralization with primary loss of bone mass only
- D. Brown tumors caused by autonomous PTH secretion

Case interpretation: Osteomalacic osteoid

A — persistent unmineralized osteoid in the adult skeleton

REASON 1

Fat malabsorption can compromise vitamin D uptake.

REASON 2

New osteoid is produced but its mineral deposition is inadequate.

REASON 3

Fracture susceptibility does not require distortion of skeletal contours.

Bone quantity and bone mineral quality should be considered separately.

Integrated pathogenesis: From supply to skeletal phenotype

- Low vitamin D, inadequate calcium/phosphate, renal disease, or endocrine dysfunction compromises mineral availability.
- PTH compensation may normalize calcium while perpetuating phosphate loss.
- Open growth plates develop disordered endochondral ossification and deformity.
- Closed growth plates leave an adult remodeling defect with osteoid accumulation and fractures.

The common denominator is impaired mineralization of newly formed matrix.

Final synthesis: Two phenotypes, one mineralization problem

Rickets

- Open growth plate is the key anatomic context.
- Persisting cartilage and osteoid disrupt bone formation.
- Skull, chest, limbs, and spine deform under stress.

Osteomalacia

- New adult remodeling osteoid remains undermineralized.
- Trabeculae have prominent unmineralized seams.
- Vertebral and femoral-neck fractures are characteristic risks.

Ask first: Is the defect in bone mass or in mineralization?

Core textbook references

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

Chapter 26 — Bones, Joints, and Soft Tissue Tumors: “Osteomalacia and Rickets,” p. 1072.

Chapter 9 — Environmental and Nutritional Diseases: Vitamin D metabolism, deficiency, rickets, and osteomalacia, pp. 400–403.

Original native PowerPoint schematics paraphrase these source mechanisms; no patient specimens, microscopy, or diagnostic images are represented.