PHGY 210,2,4 - Physiology

SENSORY PHYSIOLOGY

Somatic Senses

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SENSORY PHYSIOLOGY

Somatic Senses

Reading

Rhoades & Pflanzer (4th edition)

Chapter 8: *Somatosensory Pathways* (p. 260-268)

Somatosensory Modalities

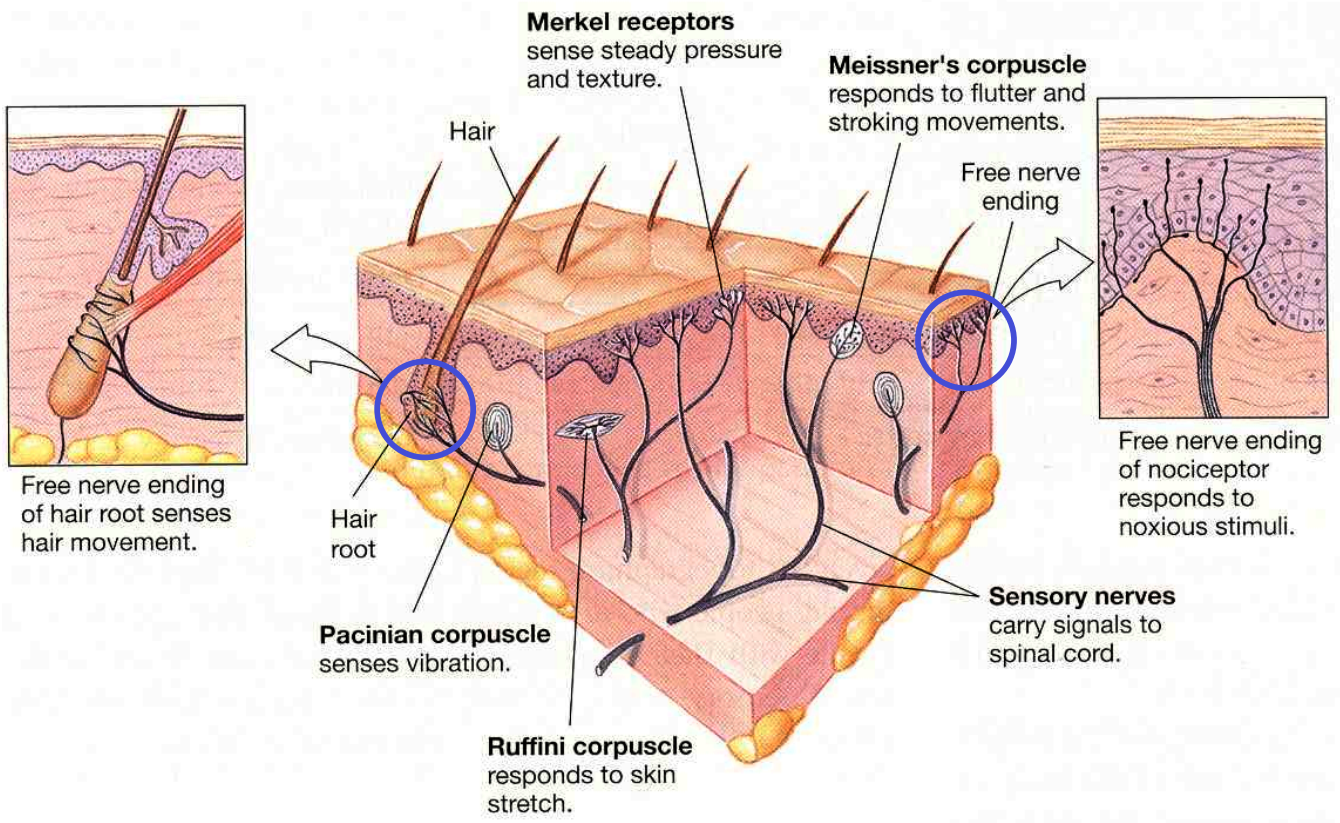
There are four somatosensory modalities:

Touch / Pressure
Proprioception
Temperature
Nociception

Natural stimuli are multifaceted,
i.e., they usually activate multiple receptor types.

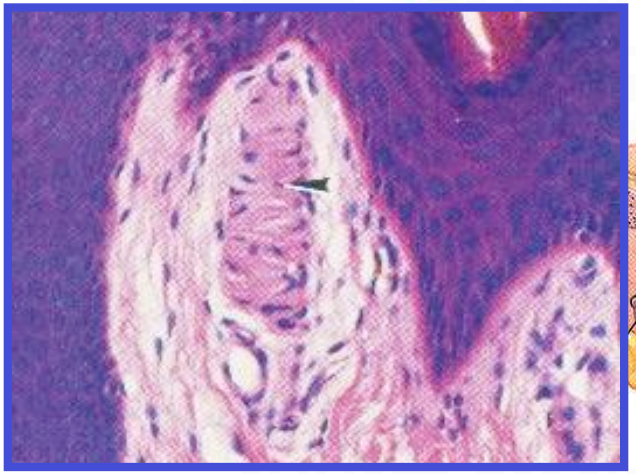
Somatosensory Receptors

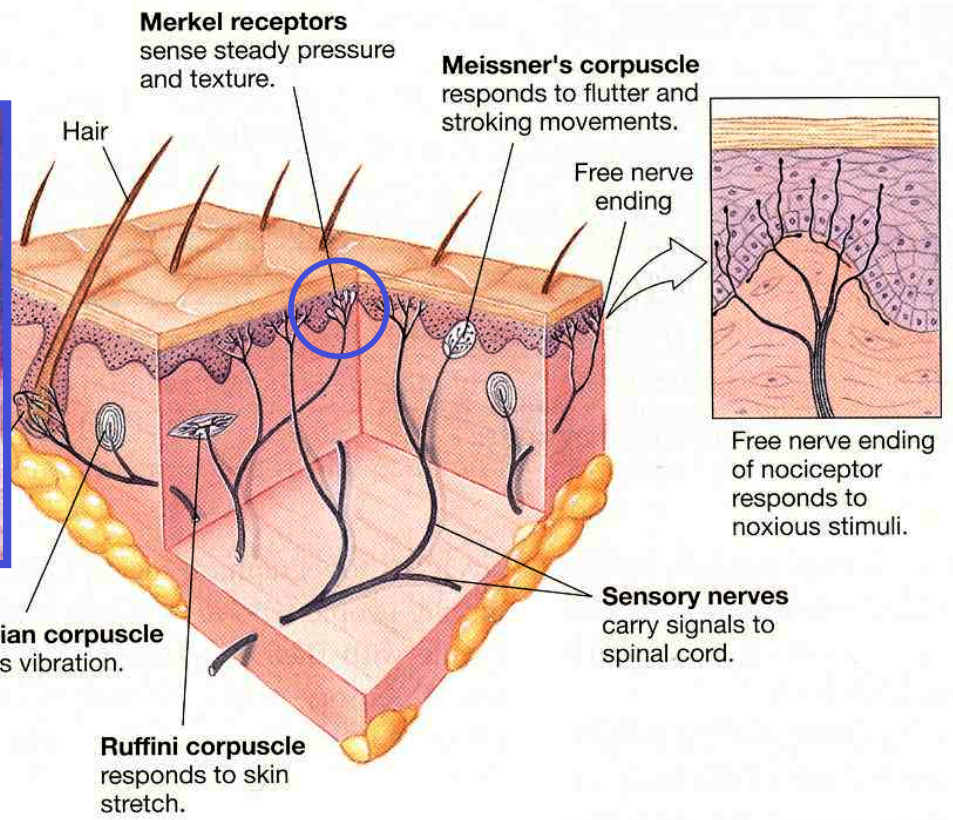
Free nerve endings (*coarse touch, pain, temperature*)
Around hair roots and under skin surface



Touch-Pressure Receptors

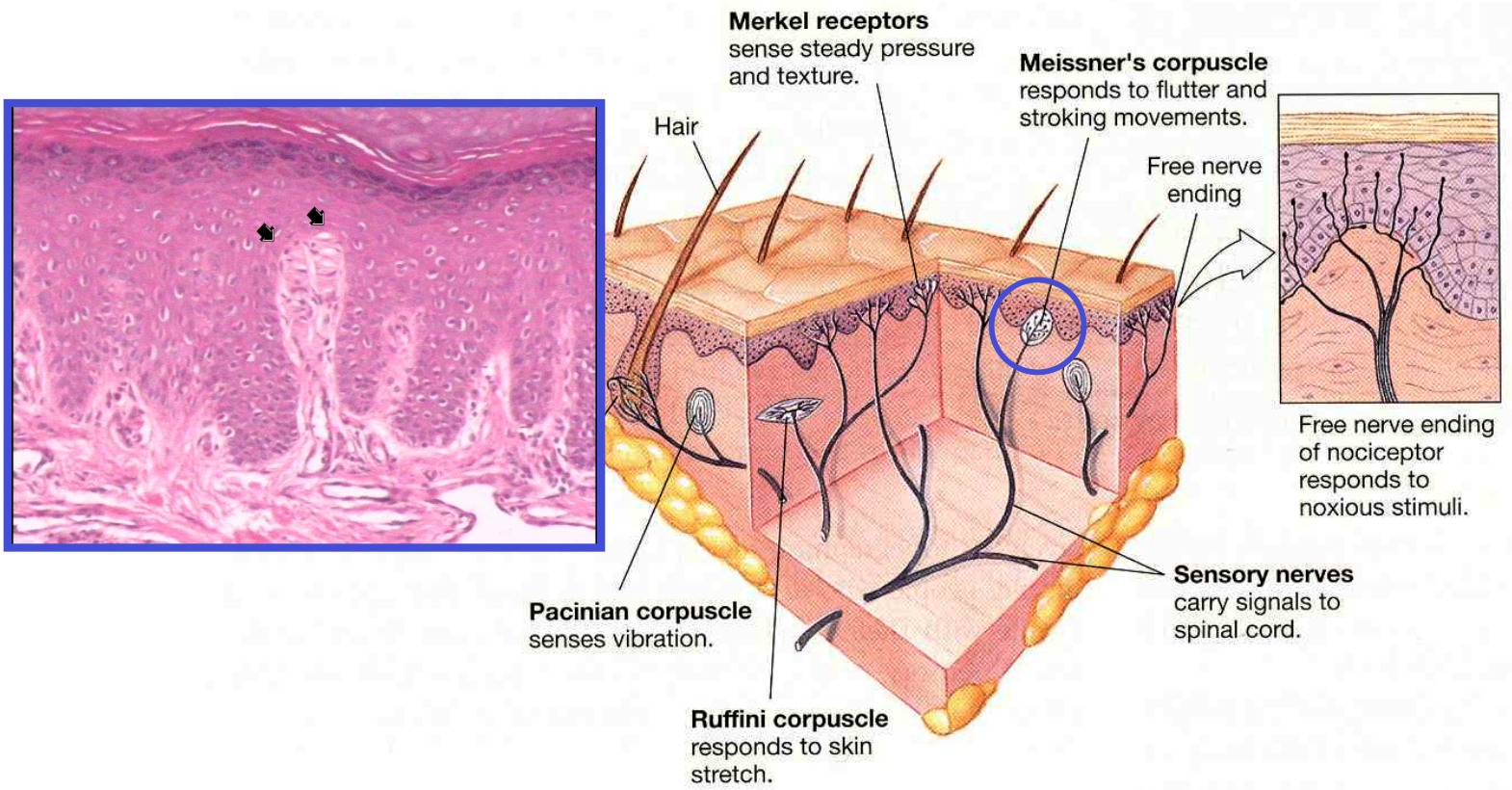
Merkel receptors (*skin pressure*)
Enlarged nerve endings in superficial skin layers





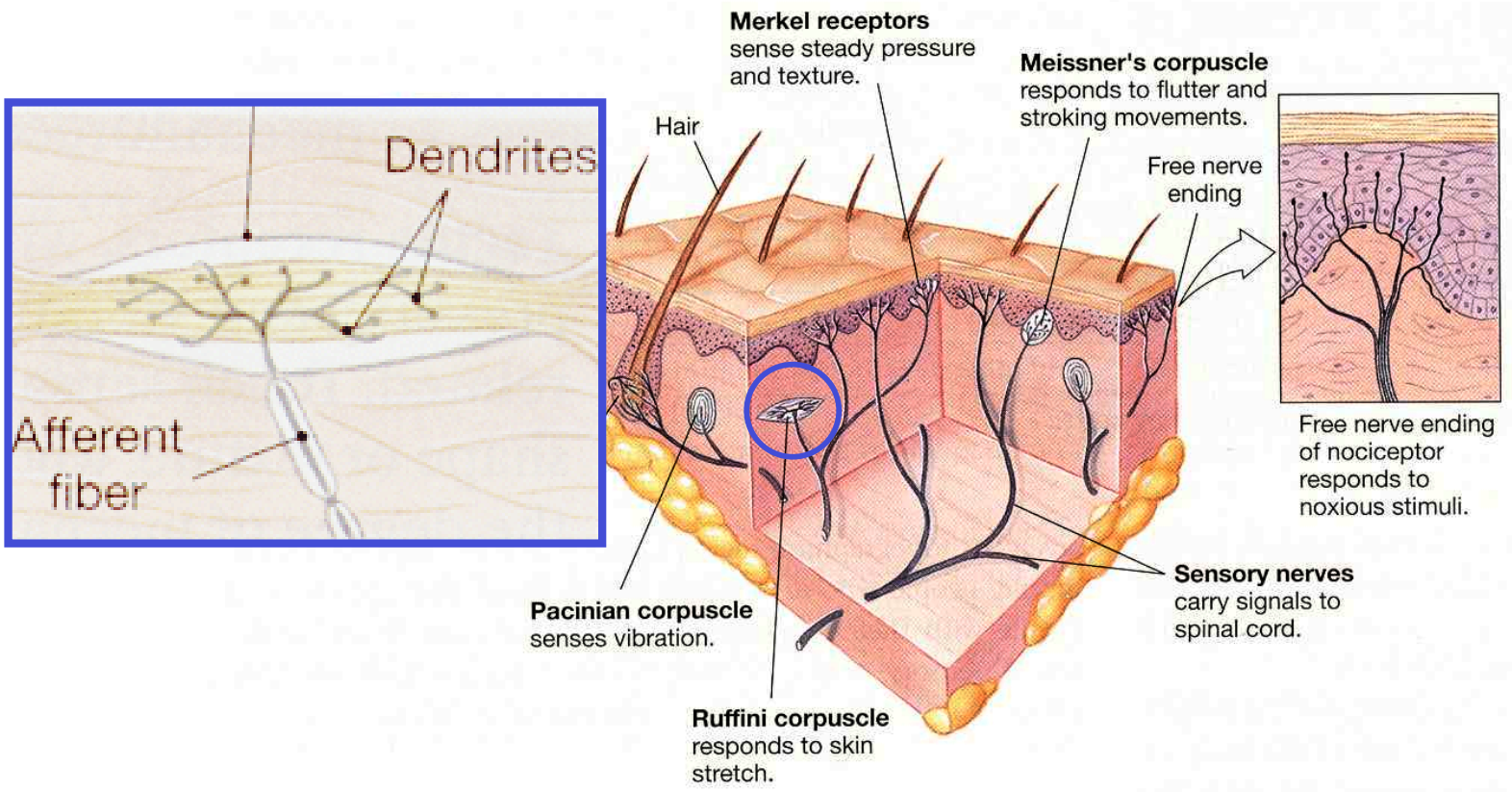
Touch-Pressure Receptors

Meissner's corpuscle (*flutter*)
Encapsulated in connective tissue in superficial skin layers



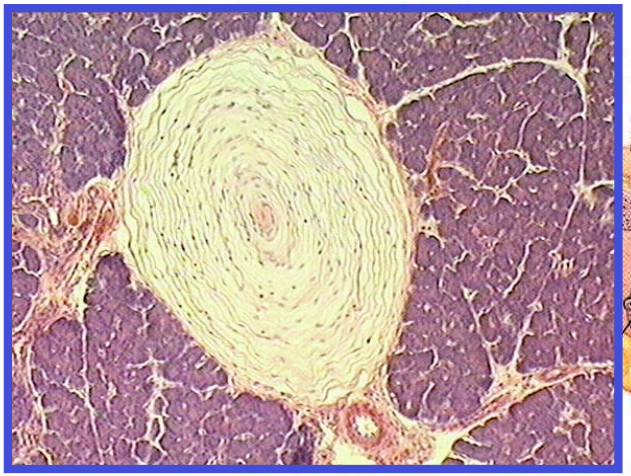
Touch-Pressure Receptors

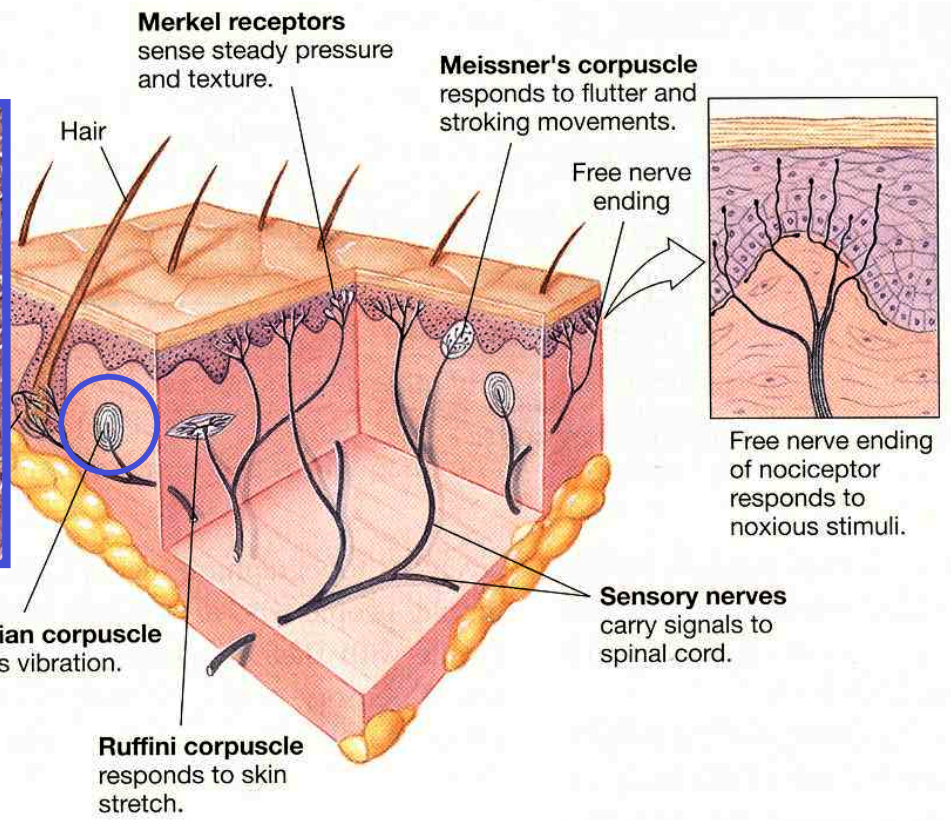
Ruffini corpuscle (*skin stretch*)
Enlarged nerve endings in deep skin layers



Touch-Pressure Receptors

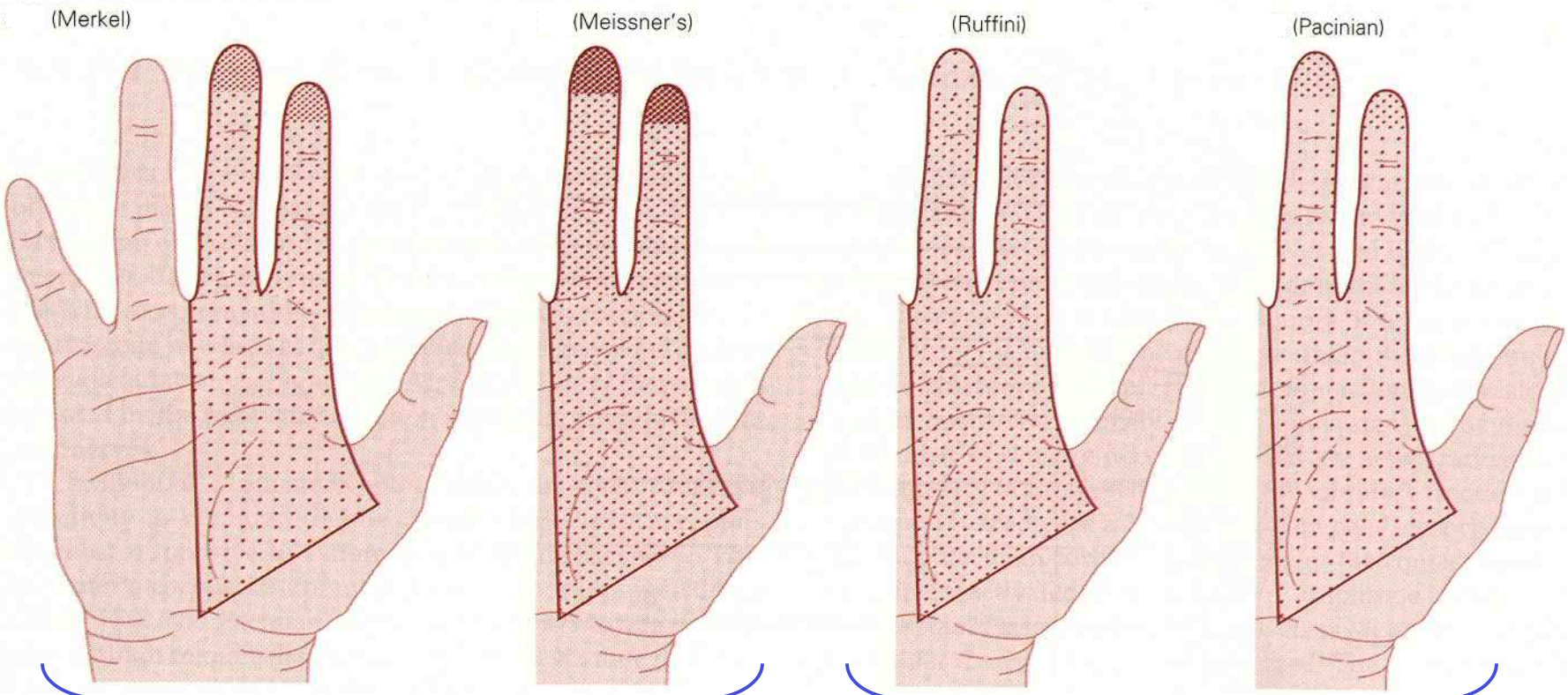
Pacinian corpuscle (*vibration*)
Encapsulated in connective tissue in deep skin layers





Touch-Pressure Receptors

Touch-pressure receptors are distributed neither identically nor uniformly.



Primary Sensory Fibers

Aβ fibers: large, myelinated, fast fibers (30 – 70 m/s)

fine touch; pressure; proprioception

(Specialized nerve endings)

Fine touch/pressure: Dorsal column

Aδ fibers: small, myelinated, slow fibers (12 – 30 m/s)

cold; fast & sharp pain; coarse touch

(Free nerve endings)

C fibers: small, unmyelinated, very slow fibers (0.5 – 2 m/s)

temperature; slow & dull pain; coarse touch

(Free nerve endings)

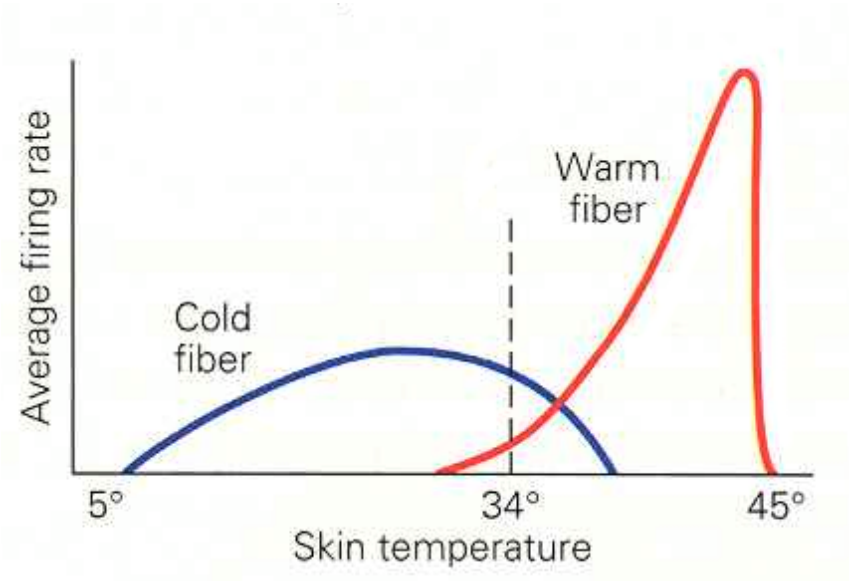
Pain/temperature: Lateral spinothalamic tract

Coarse touch: Ventral spinothalamic tract

Sensing Temperature

Thermoreceptors are free nerve endings with small receptive fields (~1-mm) scattered across the body.

Skin thermoreceptors play a role in **homeostasis**, which is controlled by centers in hypothalamus.

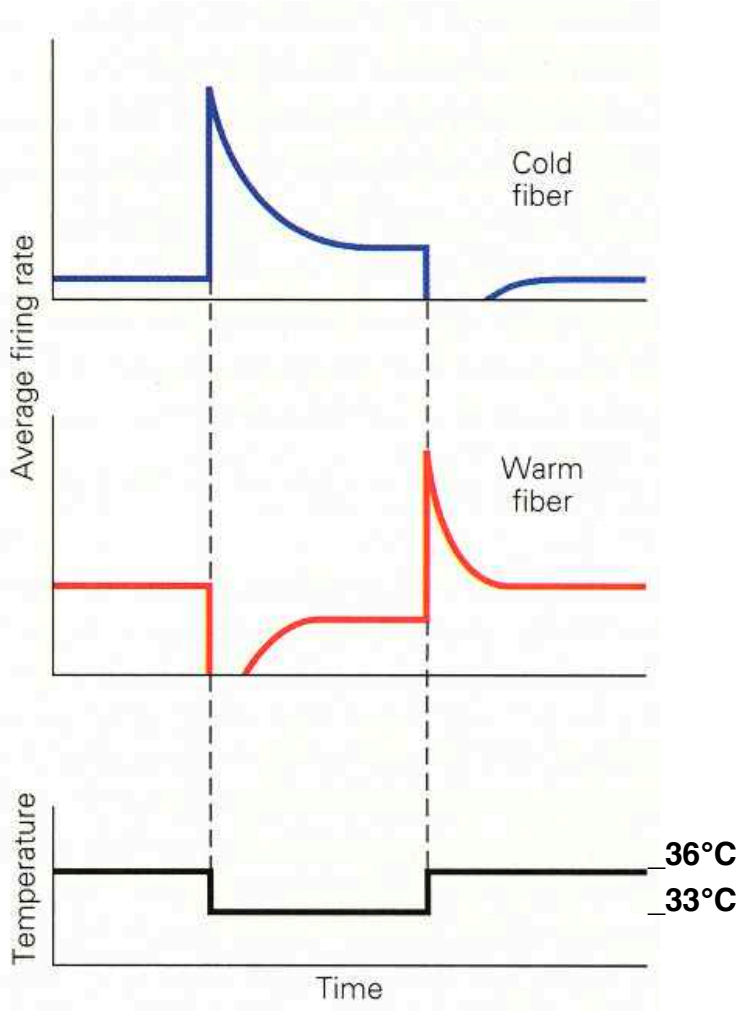


Sensing Temperature

Thermoreceptors are sensitive to (fast) changes in temperature, not to absolute temperature.

Thermoreceptors adapt only between 20° and 40° C.

Stimuli outside this range activate *nocireceptors* because of the high probability of tissue damage.



Sensing Pain

Pain is the consequence of **tissue injury**:
Nociceptors are free nerve endings sensitive to a variety of molecules released with tissue injury.

Chemical mediators include:

1) K^+, histamine, *bradykinin* & *prostaglandins*
   from damaged tissue at site of injury;

2) ATP & 5-HT (serotonin)
   from platelets activated by injury;

3) Substance P
   from the primary sensory neurons (C fibers).

Sensing Pain

Fast & sharp pain: transmitted via Aδ fibers from the activation of *thermal nociceptors* (>45° or <5° C) or *mechanical nociceptors* (intense pressure).

Detection of injured site / Good stimulus localization

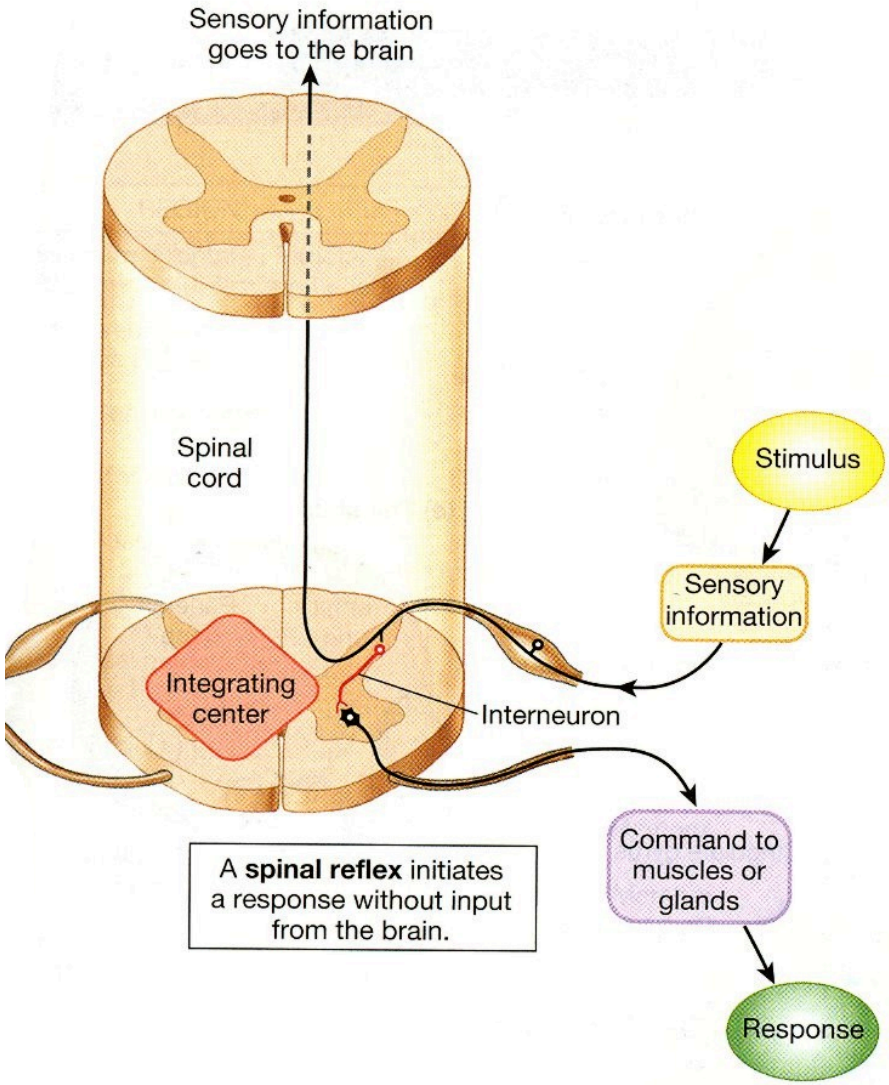
Slow & dull pain: transmitted via C fibers from the activation of *polymodal nociceptors* (high-intensity mechanical, thermal or chemical stimuli).

Protection of injured site / Poor stimulus localization

Sensing Pain

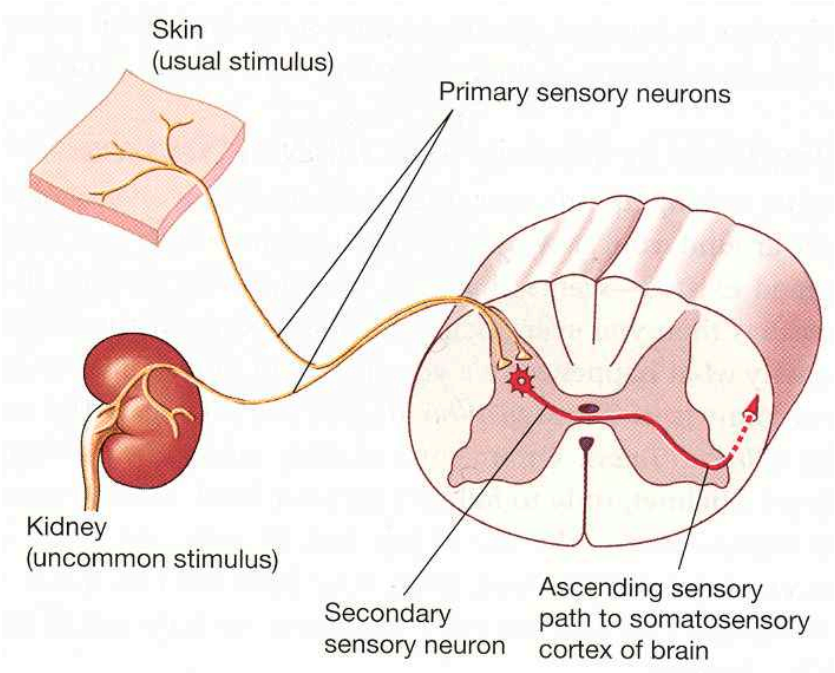
Painful stimuli are carried by ascending pathways to the cortex, where they become *conscious sensation*.

Not all nociceptive responses rely on cortical circuits. *Subconscious withdrawal reflexes* can occur within the spinal cord. These are called **spinal reflexes**.



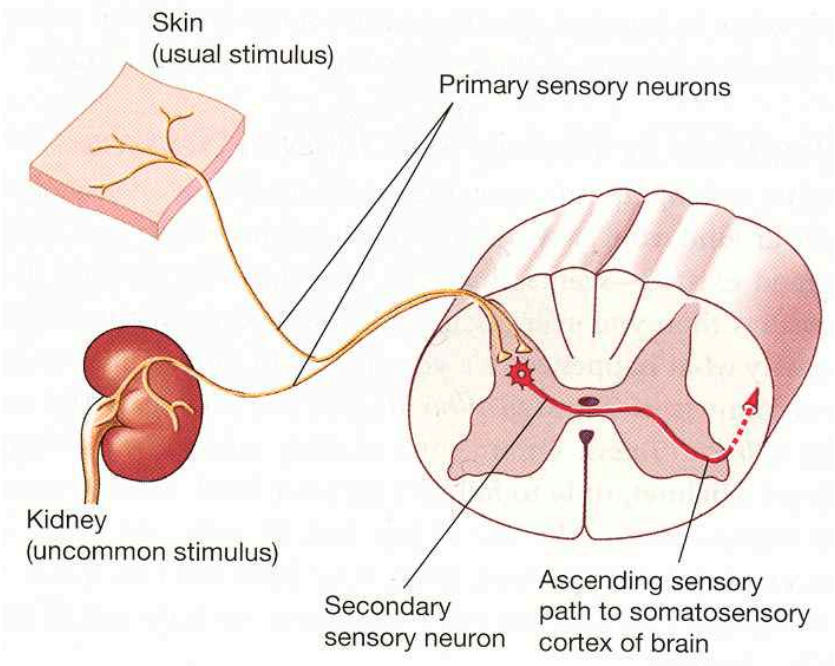
Sensing Pain

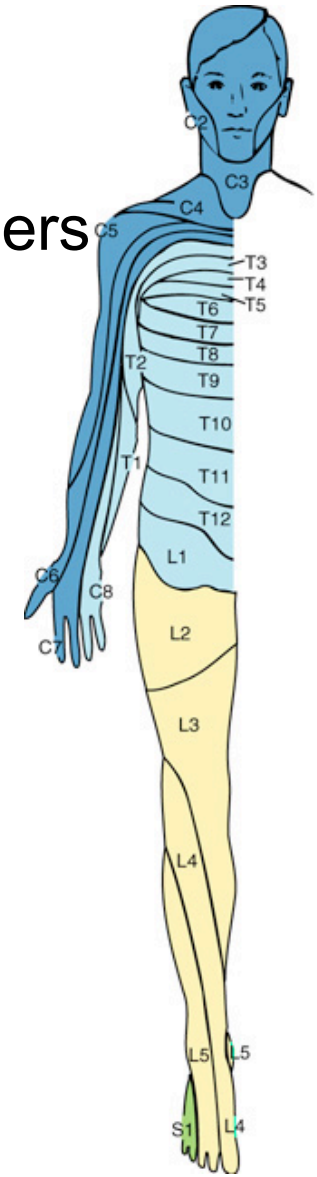
Somatic pain tends to be well localized, constant pain that is described as sharp, aching, throbbing, or gnawing. **Visceral pain** is often poorly localized and often felt in somatic areas distant from the painful stimulus.

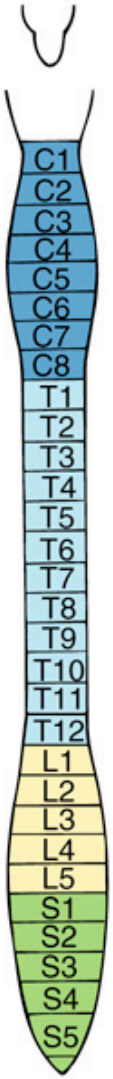


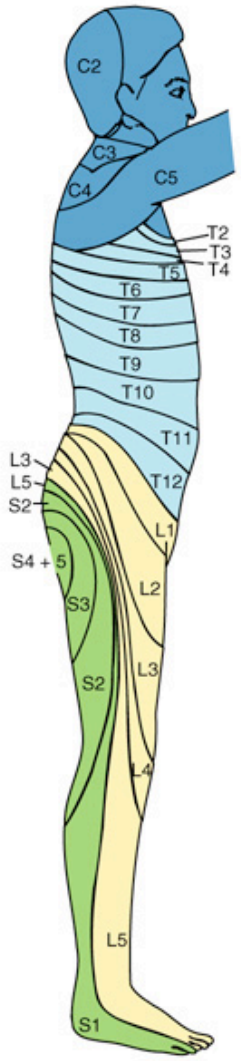
Sensing Pain

Referred pain is due to the ***convergence*** of nociceptive fibers onto a single ascending tract.









Sensing Pain

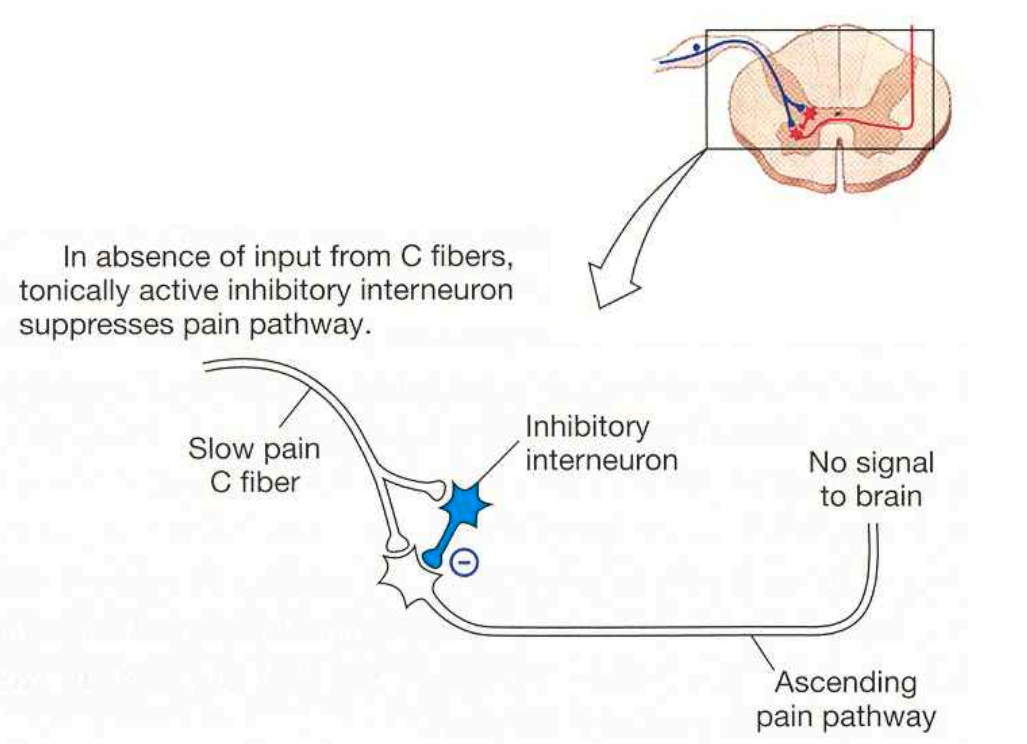
Neuropathic pain is usually produced by damage to the peripheral nervous systems. In contrast to nociceptive pain, it is described as burning, electric, tingling, or shooting.

Neuropathic pain is characterized by chronic pain experience: *allodynia* and *hyperalgesia*.

Allodynia: pain resulting from a stimulus that ordinarily does not elicit a painful response.
Hyperalgesia: increased sensitivity to a normally painful stimuli.

Gating Theory of Pain Modulation

Our perception of pain is subject to modulation.



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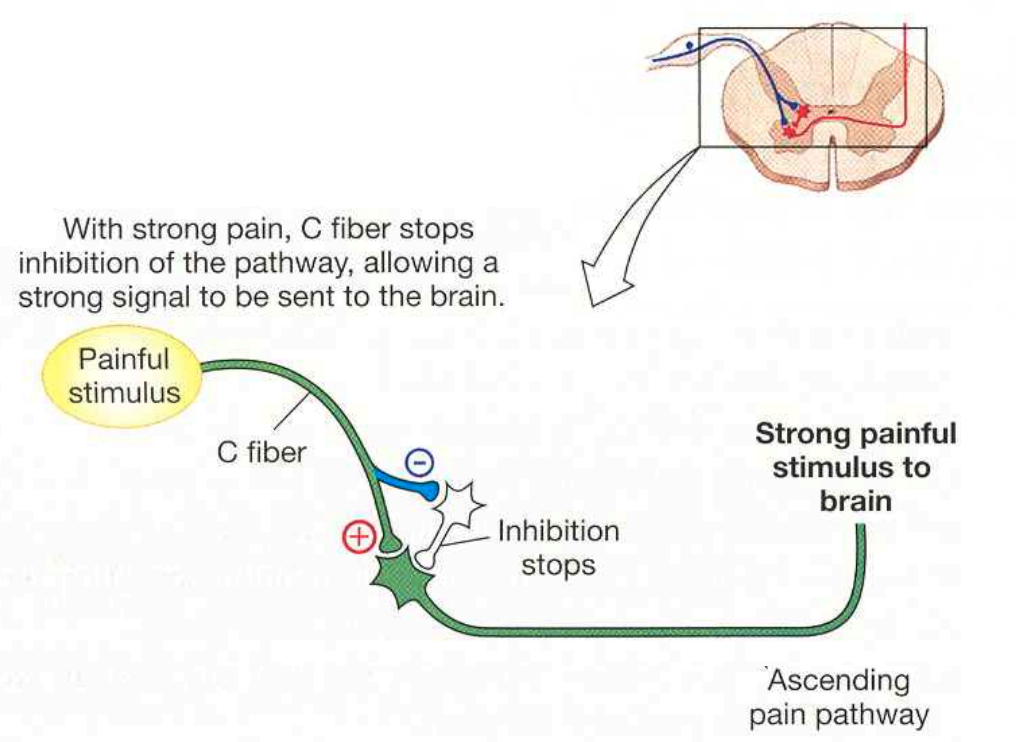
In the gating theory of pain modulation, inhibition of the ascending pain pathway can be enhanced by the activation of non-nociceptive somatic Aβ fibers.



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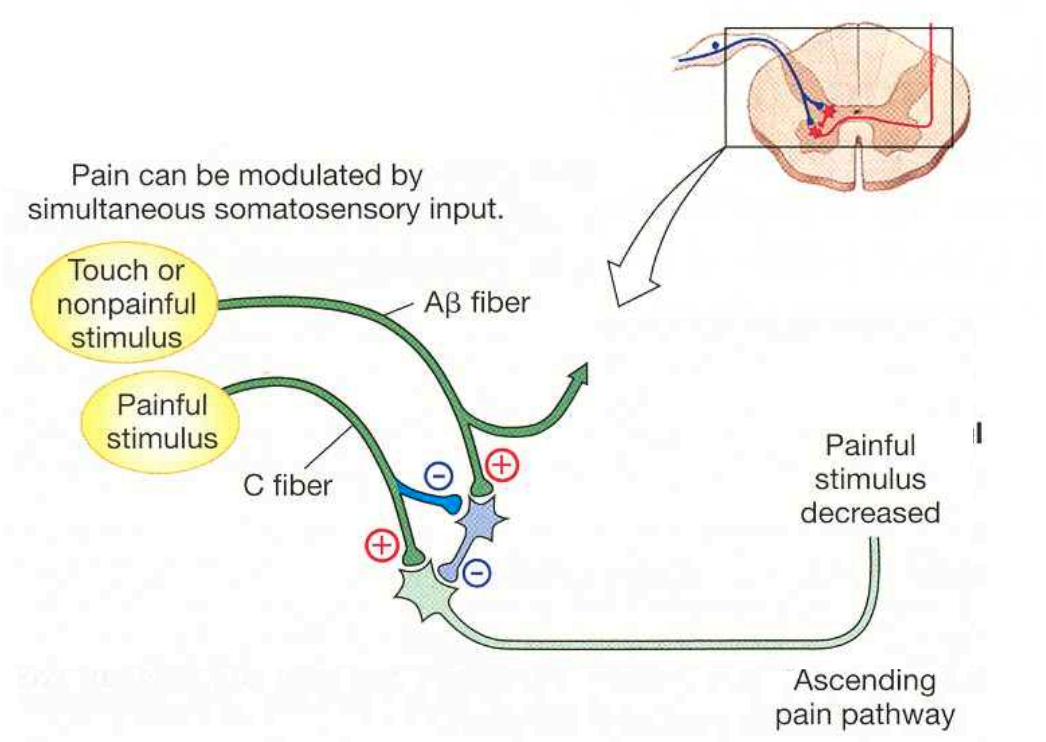
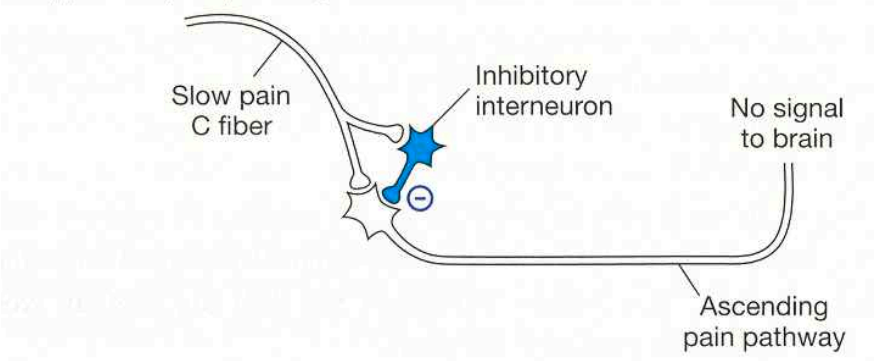


Analgesia

Analgesic drugs range from NSAIDs to opiates.

NSAIDs (e.g. Aspirin) block the synthesis of *prostaglandins* by inhibiting cyclooxigenases (COX), which then slows the transmission of pain signals from the site of injury.

Opiates (*endogenous opioids: endorphins & enkephalins*) act directly on opioid receptors within descending neural pathways, whose action is to inhibit incoming pain signals.



Sensory Receptors

simple receptors	***complex receptors***	***special receptors***
free nerve endings: *under skin surface, around hair roots*	***modified nerve endings:*** *Merkel, Meissner, Ruffini, Pacinian*	***specialized cells*** *(e.g., photoreceptors, hair cells)*
somatic senses: *coarse touch, pain, temperature*	***somatic senses:*** *fine touch / pressure*	***special senses:*** *vision, audition, taste, olfaction, equilibrium*
distal part of ***1°sensory neurons*** *"A(delta) or C fibers"*	*distal part of* ***1°sensory neurons*** *"A(beta) fibers"*	*distinct cells from* ***1°sensory neurons***