## PHGY 212 - Physiology

# SENSORY PHYSIOLOGY
## Somatic Senses


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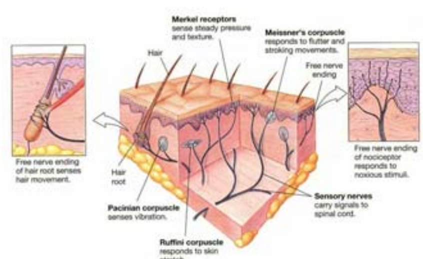
## Somatosensory Modalities

There are four somatosensory modalities:

Touch-Pressure
Proprioception
Temperature
Nociception

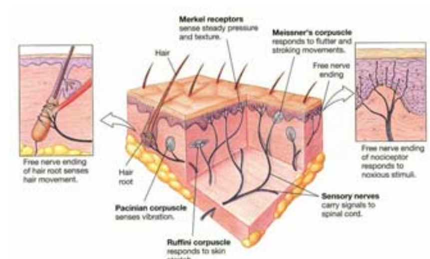
## Somatosensory Receptors

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



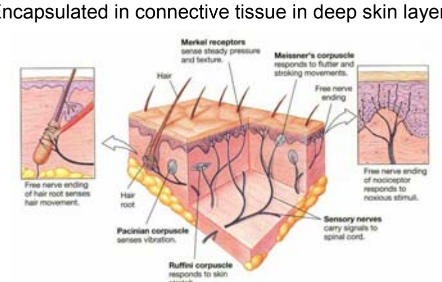

## Touch-Pressure Receptors

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



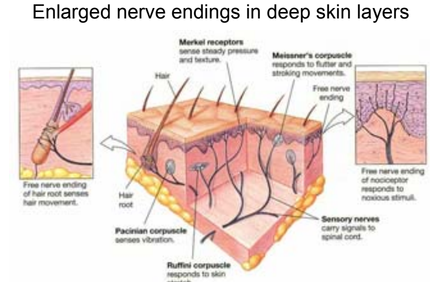

## Touch-Pressure Receptors

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



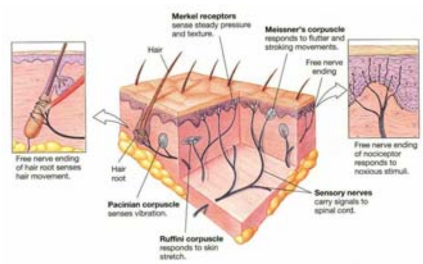

## Touch-Pressure Receptors

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



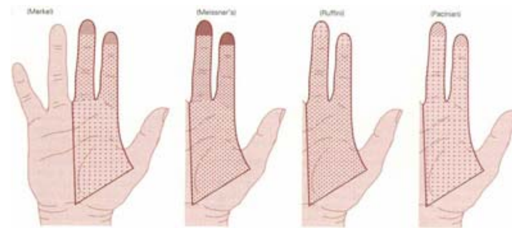
## Touch-Pressure Receptors

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

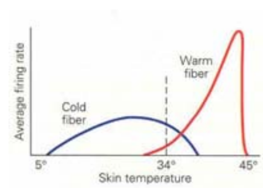
**Aδ fibers:** small, myelinated, slow fibers (12 – 30 m/s)
*crude touch; cold; fast & sharp pain*

**C fibers:** small, unmyelinated, very slow fibers (0.5 – 2 m/s)
*temperature; slow & dull pain*

## Sensing Temperature

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

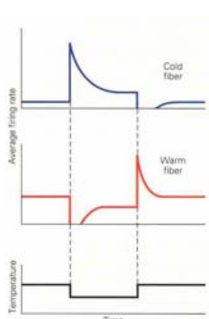
Skin thermoreceptors play a role in temperature regulation, which is controlled by centers in hypothalamus.




## Sensing Temperature

Thermoreceptors are sensitive to 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

Nociceptors are free nerve endings sensitive to a variety of molecules released with tissue injury.

Chemical mediators include:

1) $K^+$, histamine, bradykinin & prostaglandins
from site of injury;

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

3) Substance P
from the primary sensory neurons.

## Sensing Pain

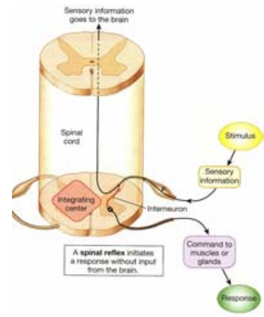
**Fast & sharp pain:** transmitted via Aδ fibers from the activation of *thermal nociceptors* (>45° or <5° C) or *mechanical nociceptors* (intense pressure).

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

## Spinal Reflexes

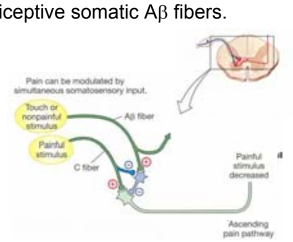
Painful stimuli are carried by ascending pathways to the cortex, where they become conscious sensation.

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



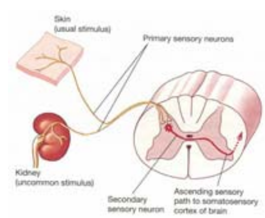

## Gating Theory of Pain Modulation

Our perception of pain is subject to modulation. 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.




## Referred Pain

Visceral pain is often poorly localized and often felt in somatic areas distant from the painful stimulus. This **referred pain** is due to the *convergence* of nociceptive fibers (of same **dermatome**) onto a single ascending tract.




## Analgesia

Analgesic drugs range from aspirin to opiates.

**Aspirin** inhibits the synthesis of *prostaglandins* and thus slows the transmission of pain signals from the site of injury.

**Opiates** (*endogenous opioids: endorphins & enkephalins*) act directly on opioid receptors in the brain, which activate descending pathways that inhibit incoming pain signals.

## Reading

**Silverthorn (2nd edition)**
pages 291 – 294

**Silverthorn (1st edition)**
pages 273 – 276