C FIBER

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1. Core Definition

The **C fiber** is a designation used in neurophysiology to describe a class of nerve fibers that are characterized fundamentally by the absence of a myelin sheath. These fibers possess the smallest diameter among all peripheral nerve axons, a structural attribute that directly dictates their primary functional characteristic: extremely slow conduction velocity. While C fibers are located throughout the peripheral nervous system, they are particularly critical components of the **somatic sensory system**, where they function almost exclusively as **afferent fibers**, meaning they conduct nerve impulses inward from the periphery of the body toward the central nervous system (CNS). They are essential for transmitting slow, persistent, and often non-discriminative sensory information, including chronic pain, temperature, and certain types of affective touch.

In the standard classification schemes used to categorize nerve axons based on structure and speed, C fibers represent the slowest group. Their lack of the fatty insulating layer known as **myelin** prevents the saltatory conduction characteristic of faster A-group fibers. This structural deficit necessitates continuous depolarization along the entire length of the axon, leading to significantly reduced conduction efficiency. Functionally, this slowness is crucial; C fibers are responsible for the delayed, persistent sensation known clinically as "second pain," contrasting sharply with the immediate, sharp pain signaled by the faster A-delta fibers. Understanding the role and morphology of the C fiber is paramount to grasping the complex neural pathways underpinning chronic pain perception and autonomic regulation.

2. Classification and Structural Characteristics

C fibers are structurally defined by their small caliber and their unmyelinated status. The diameter of these axons typically ranges from 0.4 to 1.2 micrometers, making them the narrowest fibers in the peripheral nervous system. Unlike myelinated axons, which are individually wrapped by Schwann cells forming the myelin sheath, multiple C fibers are often bundled together and ensheathed by the cytoplasm of a single **Schwann cell** in grooves, but without the multiple concentric wraps that define myelin. This arrangement, known as a Remak bundle, provides structural support but fails to enhance electrical insulation or conduction speed.

Nerve fibers are classified using two primary systems: the Erlanger and Gasser scheme (based on velocity) and the Lloyd and Hunt scheme (primarily used for sensory afferents). In the Erlanger and Gasser classification, C fibers constitute Group C, characterized by the lowest velocity. In the Lloyd and Hunt scheme, which classifies sensory neurons originating in muscles and skin, C fibers

correspond to **Group IV afferents**. This specific classification highlights their origin--sensory endings in the skin, deep tissues, and viscera--and their relay through the dorsal root ganglia (DRG) before synapsing in the dorsal horn of the spinal cord. The anatomical location of their cell bodies within the DRG distinguishes them as primary afferent neurons (PANs).

The structural characteristic of being unmyelinated necessitates a higher capacitance and lower resistance across the axonal membrane compared to myelinated fibers. This physical reality dictates that a greater expenditure of energy and time is required to generate the necessary current to propagate an action potential sequentially down the fiber. While structurally simple, the sheer volume of C fibers--often constituting over 50% of the nerve fibers in a peripheral nerve bundle--underscores their vital importance in sensory processing and communication between the body and the CNS.

3. Conduction Properties and Velocity

The conduction velocity of C fibers is notably slow, ranging generally between 0.7 and 2.3 meters per second (m/s), as evidenced by physiological measurements. This speed is negligible when compared to the fastest A-alpha motor fibers, which can reach speeds exceeding 100 m/s, or even the fast sensory A-beta fibers (30-70 m/s). The sluggish conduction rate is a direct mechanical consequence of the absence of myelin, which prevents the process of **saltatory conduction**--the rapid leaping of the action potential from one Node of Ranvier to the next.

In an unmyelinated C fiber, the action potential must be generated at every infinitesimal segment along the axon. This continuous process involves sequential depolarization and repolarization, demanding maximum time and energy. Crucially, the speed of propagation is inversely proportional to the time constant of the membrane and the resistance of the cytoplasm. Because C fibers have a high membrane time constant (due to poor insulation) and high internal resistance (due to small diameter), their speed is severely limited. This slow conduction velocity means that the sensory information they carry is inherently delayed in reaching the brain, a factor critical for the perception of certain stimuli.

This characteristic slowness is not a defect but an integrated functional design, essential for transmitting signals related to prolonged or sustained somatic states. For instance, in the case of painful stimuli, the initial sharp sensation is transmitted rapidly by A-delta fibers. The lingering, dull, or throbbing pain that follows--the one that persists and signals tissue damage--is transmitted by the C fibers. This temporal disparity allows the CNS to differentiate between immediate threat detection (A-delta) and sustained tissue damage monitoring (C fiber).

4. Functional Role in Sensory Transmission

C fibers are responsible for conveying a wide range of essential, though often poorly localized,

sensory information. Their primary functional roles include nociception (pain), thermoreception (temperature), and certain forms of non-discriminative mechanical sensation. They are typically associated with **polymodal receptors**, meaning a single fiber can respond to multiple types of intense stimuli--mechanical deformation, excessive heat or cold, and chemical irritants released during tissue injury.

In the realm of nociception, C fibers are the principal mediators of **slow pain**, also termed second pain. This sensation is characterized as dull, aching, burning, or throbbing. Because C fibers adapt slowly and have a high threshold for firing, they are ideally suited to signal continuous damage or inflammation. Furthermore, these fibers release critical neuropeptides, such as Substance P and calcitonin gene-related peptide (CGRP), at their central terminals in the spinal cord and peripherally at the site of injury. The peripheral release contributes significantly to neurogenic inflammation, characterized by localized swelling and redness.

Beyond pain, specialized populations of C fibers also mediate crucial affective and homeostatic signals. **C-tactile (CT) fibers**, found predominantly in the hairy (non-glabrous) skin, respond optimally to slow, gentle stroking. These fibers are not involved in discriminative touch (which is mediated by fast A-beta fibers) but rather in conveying pleasant, emotionally rewarding, or affective touch. These signals bypass traditional somatosensory cortical pathways and project instead to areas associated with emotion and social bonding, such as the insular cortex, highlighting their significance in social behavior and well-being.

5. Specific Subtypes and Receptor Associations

The broad category of C fibers is functionally diverse, reflecting the specialized receptors they innervate. While all share the unmyelinated structure, their molecular profiles and projection targets vary significantly, enabling specialized roles. These subtypes are often identified using specific chemical markers or receptor expression profiles found on their terminals in the skin or viscera.

The most abundant subtype are the **polymodal nociceptors (PNs)**, which express various transient receptor potential (TRP) channels, notably **TRPV1**, the receptor activated by heat and capsaicin. These fibers are the core pathway for inflammatory and thermal pain. Another important class includes silent nociceptors, which are normally unresponsive to mechanical stimuli but become activated (sensitized) following inflammation or injury, contributing substantially to hypersensitivity states like hyperalgesia.

A separate, non-nociceptive population consists of the aforementioned **C-tactile fibers**. These fibers typically have lower mechanical thresholds than nociceptive C fibers and are critical for conveying signals important for emotional regulation and comfort. Additionally, C fibers are integral to the autonomic nervous system. Postganglionic sympathetic efferents, which regulate involuntary functions such as blood vessel diameter, heart rate, and glandular secretion, are predominantly

unmyelinated C fibers, reflecting their need for continuous, slow, modulatory output rather than rapid signaling.

6. Clinical Significance and Pathology

Due to their extensive presence and critical role in nociception and autonomic function, C fibers are centrally implicated in numerous clinical conditions, particularly those involving chronic pain and small fiber neuropathies. Damage to C fibers, often resulting from metabolic disorders like diabetes, autoimmune diseases, or exposure to toxins, leads to conditions grouped as **small fiber neuropathy (SFN)**. Symptoms of SFN often include burning pain, allodynia (pain from non-painful stimuli), and autonomic dysfunction (e.g., orthostatic intolerance).

The plasticity of C fibers following injury is a major driver of chronic pain states. When peripheral tissue is damaged, C fibers become sensitized--their firing threshold drops, and their responsiveness increases--a process known as **peripheral sensitization**. This can lead to exaggerated pain responses (hyperalgesia) to subsequent stimuli. Furthermore, sustained C fiber activity leads to changes in the central nervous system (central sensitization), permanently altering the pain processing pathways in the spinal cord and brain.

The characteristics of C fibers also influence pharmacological treatment. Local anesthetics, which block sodium channels, often preferentially affect smaller, unmyelinated fibers first. However, their physical location and the nature of the signal they transmit mean that analgesic drugs targeting chronic pain must often address their prolonged neurotransmitter release and central signaling patterns, rather than just their initial activation. Their involvement in both sensory and autonomic pathways makes them a complex target for therapeutic intervention in conditions ranging from fibromyalgia to complex regional pain syndrome.

7. Further Reading

Myelin (Wikipedia)
Nociceptor (Wikipedia)
C Fibers (ScienceDirect)
Schwann Cell and Remak Bundles (Wikipedia)
The Sensory System: Sensory Receptors, Afferent Nerves, and Their Central Connections (NCBI Bookshelf)