What is chronic pain
Like all signals from the body it's telling the person something. Like the bladder, hunger and emotions, pain is alerting us there is an injury or harm to the body.

While acute pain is helpful, like touching a prickle tells us to pull away, and pain in a scratch tells us to take care of the injury, chronic pain is different. Chronic pain can get better if short term chronic but it also can indicate the pain system itself is no longer working properly and is not a reliable signal of ongoing harm. The internal mechanism has gotten confused and complicated so to speak.

Let take a look at pain.

What are pain receptors
Pain receptors—also known as **nociceptors**—are your body's built-in alarm system. They're specialized sensory neurons that detect potentially harmful stimuli and alert your brain to danger. Let's break them down:

What Are Pain Receptors?

- **Nociceptors** are free nerve endings found in the skin, muscles, joints, bones, and internal organs.
- They respond to **noxious stimuli**—things that could cause tissue damage.
- Once activated, they send electrical signals through the nervous system to the brain, where the sensation is interpreted as pain.

Types of Pain Receptors
Each type is tuned to a different kind of threat:

Type	Stimulus detected	Example
Mechanical	Intense pressure, stretch, or cutting	Bruises, cuts, fractures
Thermal	Extreme temperatures (hot or cold)	Burns, frostbite
Chemical	Irritating chemicals or inflammatory substances	Acid exposure, inflammation
Polymodal	A mix of mechanical, thermal, and chemical	Complex injuries, widespread damage

Polymodal nociceptors are the most common—they're like the Swiss Army knife of pain detection.

Fast vs. Slow Pain
Pain signals travel via two types of nerve fibres:

- **Aδ fibres**: Thinly myelinated, fast-conducting. They carry sharp, immediate pain (like a prickle).
- **C fibres**: Unmyelinated, slow-conducting. They carry dull, aching, or burning pain that lingers.

Bonus Insight: TRP Channels
Nociceptors use **TRP channels** (transient receptor potential channels) to detect damage. These are specialized proteins that respond to heat, cold, and chemicals—kind of like molecular sensors.

CHRONIC PAIN
In chronic pain conditions, the pain receptors can become overactive or misfire, which is where things get tricky. The pain receptors don't just get "activated"; their behaviour changes, often due to a process called sensitization, where they become hypersensitive and fire more easily or continuously. This can be caused by receptors migrating to hide inside nerve cells, a weakening of the body's natural pain-dampening systems, and interactions with the immune system that cause inflammation to persist.

Changes in pain receptor behaviour:

- **Sensitization:**

With repeated or prolonged pain, pain receptors become more sensitive, lowering the threshold for what triggers a pain signal. Normally harmless stimuli can then be perceived as painful.

- **Receptor migration:**

A study found that in some chronic pain, pain receptors can move from the surface of a nerve cell to its inner chambers, making them unreachable by many common pain medications.

- **Weakened inhibitory signals:**

The body's natural ability to suppress pain signals can weaken in chronic pain. For example, inhibitory neurotransmitters like GABA may become less effective, allowing pain signals to pass through and intensify.

- **Sprouting and synaptic changes:**

The sympathetic nervous system can grow new connections (sprouting) into the central nervous system, creating more direct pathways for pain signals to travel.

- **Tonic activity:**

In chronic pain, certain brain cells involved in pain signaling can remain in a constant state of "tonic activity," continuously firing signals even without a new injury. This is similar to an

engine running at idle, which may explain why pain persists after the initial injury has healed.

- **Neurotransmitter and inflammatory mediator involvement:**

Various inflammatory factors and neurotransmitters play a key role. For instance, chemicals like tumour necrosis factor-α (TNF-α) and interleukin-1β can amplify pain signals by altering the excitability of neurons and their ability to communicate with each other.

What this means for chronic pain
This combination of changes can create a vicious cycle where the pain signal becomes self-perpetuating. Instead of a simple, temporary warning, chronic pain is a complex condition involving a combination of hypersensitive receptors, a disrupted nervous system, and the persistence of inflammatory signals.

Treatments
There's the pain and then there's coping with the pain

Coping with the pain
CBT and ACT to help cope with the thoughts and feelings that accompany the pain
Learning to pace and know your limits
Changing our relationship with the pain
Relaxation techniques e.g. meditations and breathing techniques
Changing old beliefs,
Building new coping strategies, e.g. types of distraction
Exercise
Socialising

Reducing the pain
Turning down the pain dial
Turning the pain switch off
Glove amnesia
Anaesthetic mist
Healing light
Replacing the feeling.
Bilateral stimulation
Hypnosis