
LA FIBROMYALGIE

VOYAGE DANS LE CERVEAU

2^{ème} partie

EFFETS DU STRESS CHRONIQUE

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THE ANATOMY OF ANXIETY

TIME Diagram by Joe Lertola.
Text by Alice Park

WHAT TRIGGERS IT ...

When the senses pick up a threat—a loud noise, a scary sight, a creepy feeling—the information takes two different routes through the brain

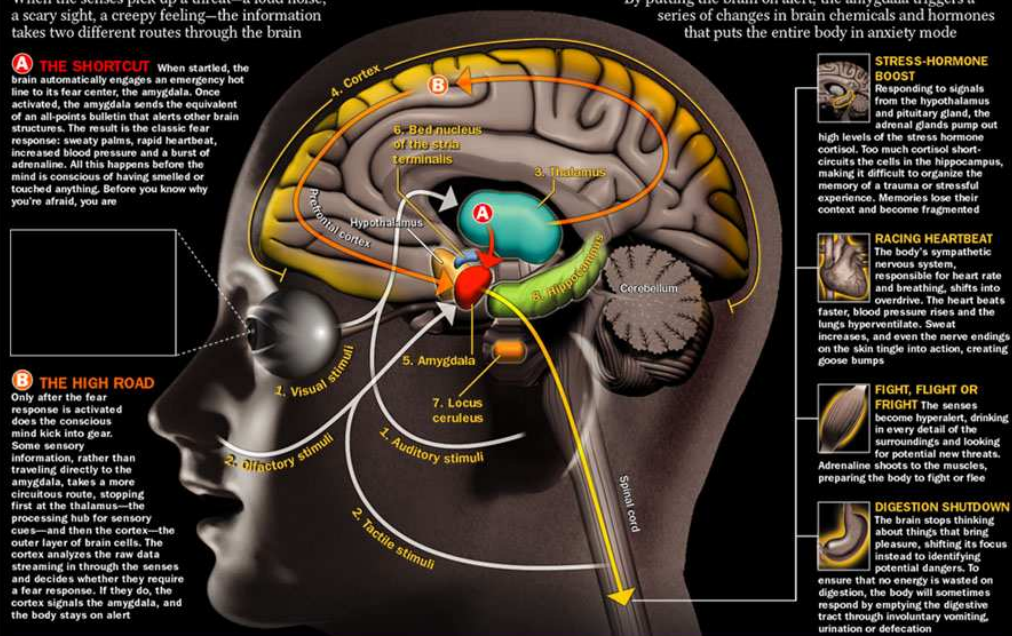
A THE SHORTCUT When startled, the brain automatically engages an emergency hot line to its fear center, the amygdala. Once activated, the amygdala sends the equivalent of an all-points bulletin that alerts other brain structures. The result is the classic fear response: sweaty palms, rapid heartbeat, increased blood pressure and a burst of adrenaline. All this happens before the mind is conscious of having smelled or touched anything. Before you know why you're afraid, you are

B THE HIGH ROAD

Only after the fear response is activated does the conscious mind kick into gear. Some sensory information, rather than traveling directly to the amygdala, takes a more circuitous route, stopping first at the thalamus—the processing hub for sensory cues—and then the cortex—the outer layer of brain cells. The cortex analyzes the raw data streaming in through the senses and decides whether they require a fear response. If they do, the cortex signals the amygdala, and the body stays on alert

... AND HOW THE BODY RESPONDS

By putting the brain on alert, the amygdala triggers a series of changes in brain chemicals and hormones that puts the entire body in anxiety mode



1. Auditory and visual stimuli
Sights and sounds are processed first by the thalamus, which filters the incoming cues and shunts them either directly to the amygdala or to the appropriate parts of the cortex

2. Olfactory and tactile stimuli
Smells and touch sensations bypass the thalamus altogether, taking a shortcut directly to the amygdala. Smells, therefore, often evoke stronger memories or feelings than do sights or sounds

3. Thalamus
The hub for sights and sounds, the thalamus breaks down incoming visual cues by size, shape and color, and auditory cues by volume and dissonance, and then signals the appropriate parts of the cortex

4. Cortex
It gives raw sights and sounds meaning, enabling the brain to become conscious of what it is seeing or hearing. One region, the prefrontal cortex, may be vital to turning off the anxiety response once a threat has passed

5. Amygdala
The emotional core of the brain, the amygdala has the primary role of triggering the fear response. Information that passes through the amygdala is tagged with emotional significance

6. Bed nucleus of the stria terminalis
Unlike the amygdala, which sets off an immediate burst of fear, the BNST perpetuates the fear response, causing the longer-term unease typical of anxiety

7. Locus ceruleus
It receives signals from the amygdala and is responsible for initiating many of the classic anxiety responses: rapid heartbeat, increased blood pressure, sweating and pupil dilation

8. Hippocampus
This is the memory center, vital to storing the raw information coming in from the senses, along with the emotional baggage attached to the data during their trip through the amygdala

Source: Dennis S. Charney M.D., National Institute of Mental Health

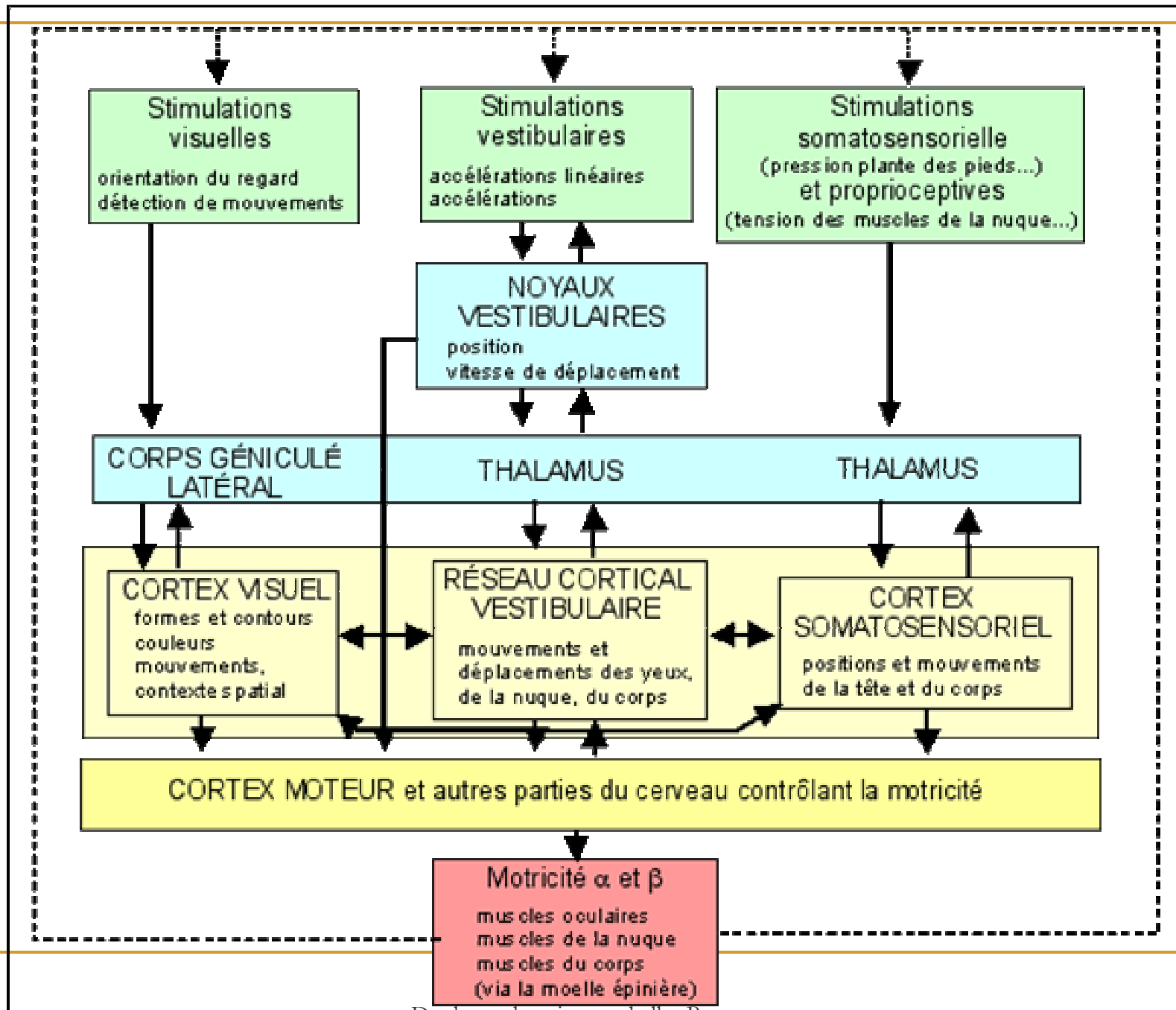
STRESS-HORMONE BOOST
Responding to signals from the hypothalamus and pituitary gland, the adrenal glands pump out high levels of the stress hormone cortisol. Too much cortisol short-circuits the cells in the hippocampus, making it difficult to organize the memory of a trauma or stressful experience. Memories lose their context and become fragmented

RACING HEARTBEAT
The body's sympathetic nervous system, responsible for heart rate and breathing, shifts into overdrive. The heart beats faster, blood pressure rises and the lungs hyperventilate. Sweat increases, and even the nerve endings on the skin tingle into action, creating goose bumps

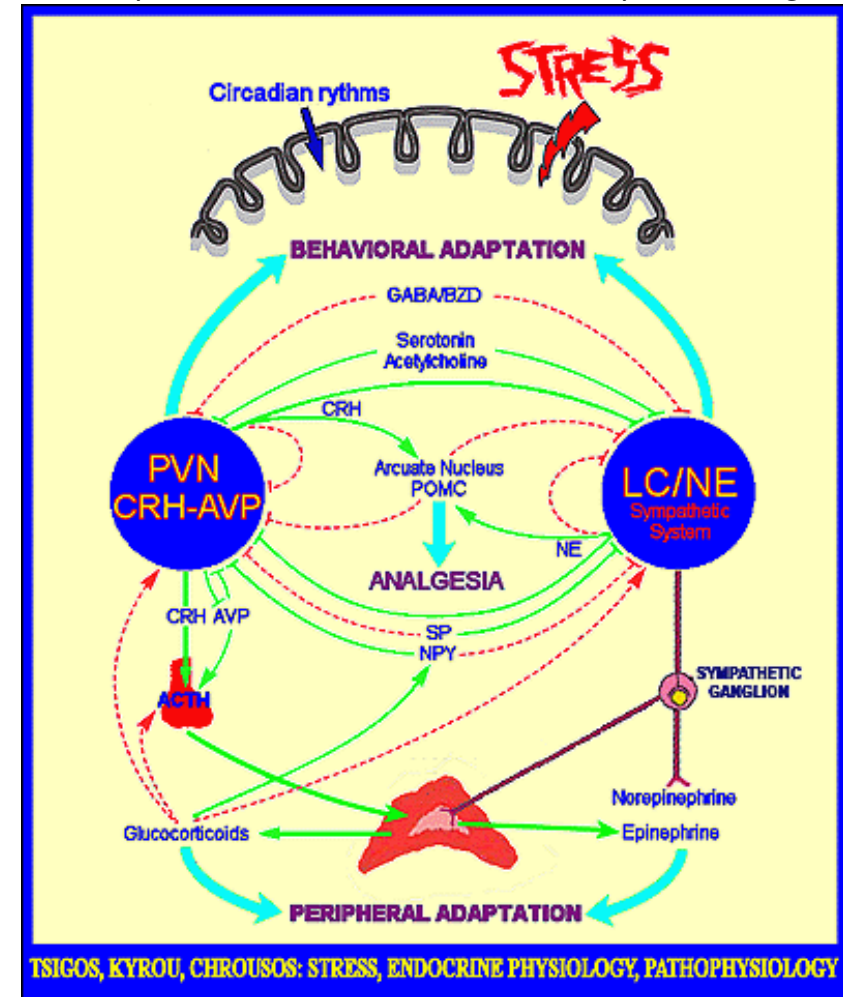
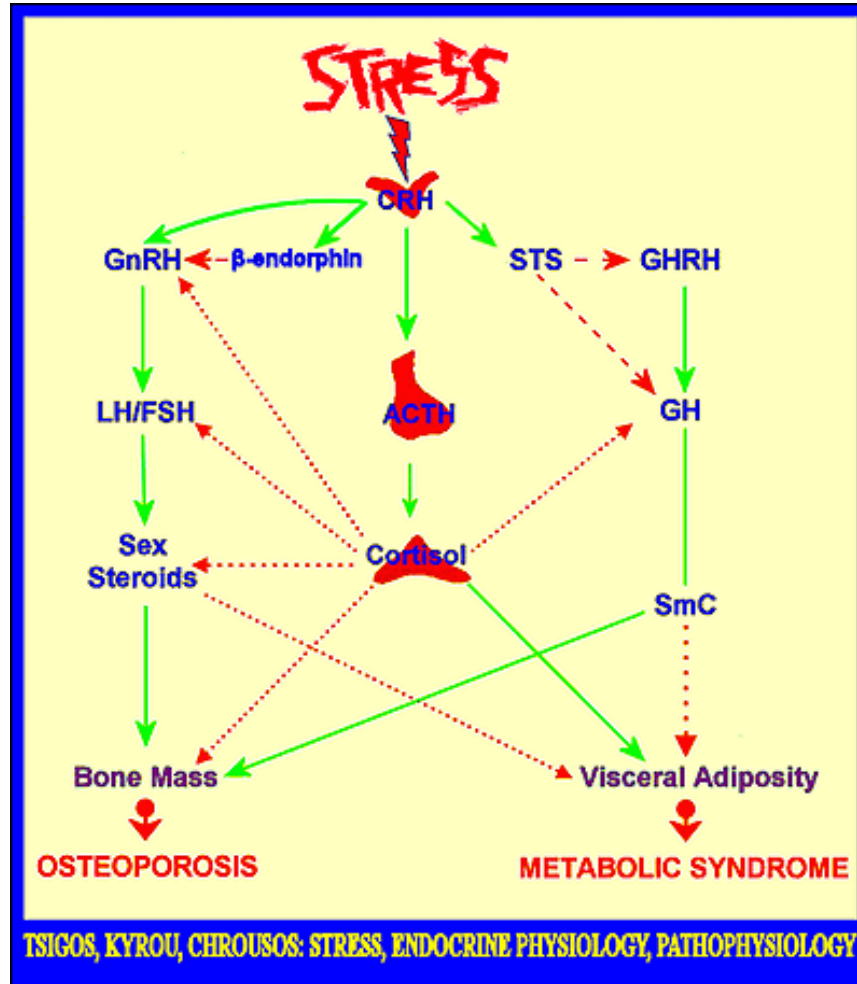
FIGHT, FLIGHT OR FRIGHT
The senses become hyperalert, drinking in every detail of the surroundings and looking for potential new threats. Adrenaline shoots to the muscles, preparing the body to fight or flee

DIGESTION SHUTDOWN
The brain stops thinking about things that bring pleasure, shifting its focus instead to identifying potential dangers. To ensure that no energy is wasted on digestion, the body will sometimes respond by emptying the digestive tract through involuntary vomiting, urination or defecation

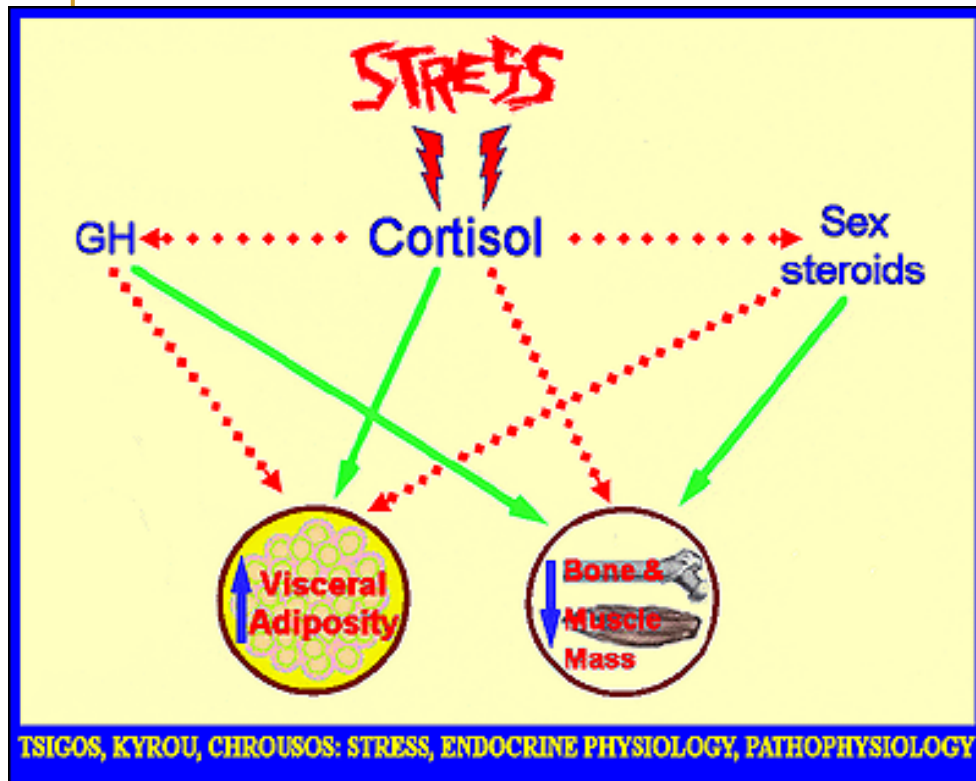
Dans le stress chronique (qu'il soit physiologique ou psychologique), l'hippocampe est saturé de cortisol et ne peut plus assurer son rôle de régulateur dans la circuiterie limbique.
Le cortisol, en excès, va modifier le fonctionnement neuronal.



CRH: corticotropin-releasing hormone, LC/NE systeme sympathique: locus ceruleus/norepinephrine-système sympathique, POMC: proopiomelanocortin, AVP: arginine vasopressin, GABA: acide γ -aminobutyrique, BZD: benzodiazepine, ACTH: corticotrophin, NPY: neuropeptide Y, SP: substance P. Activation est représentée en vert et l'inhibition en pointillé rouge.

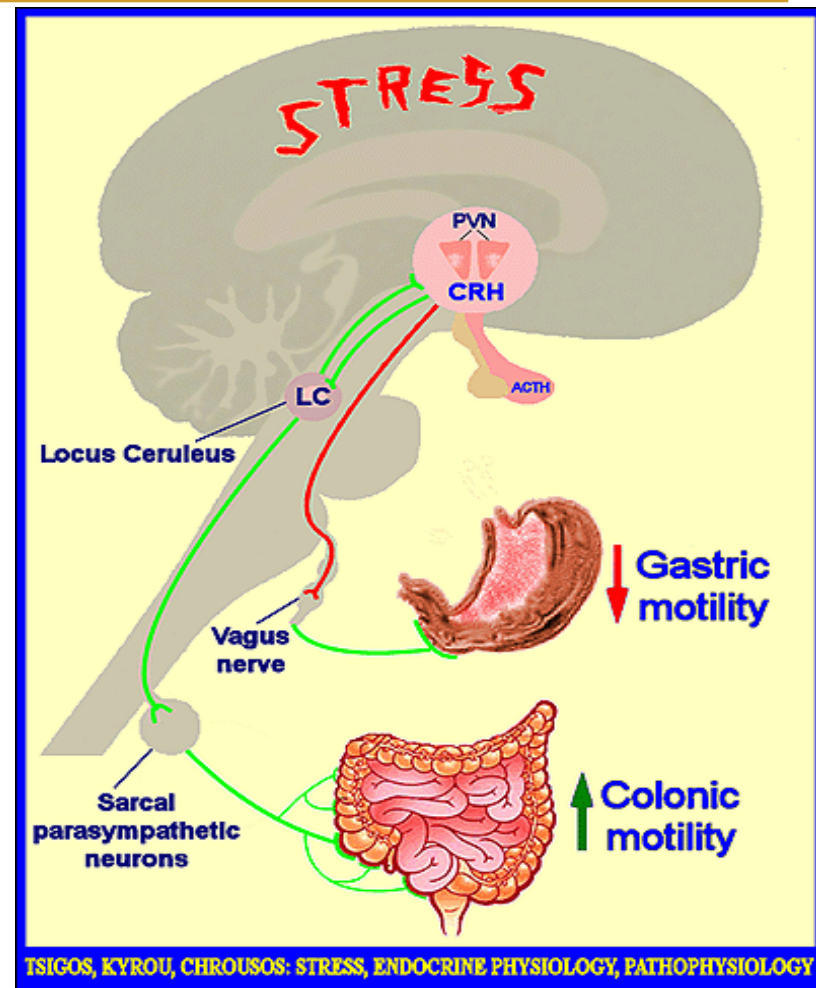


Les composantes principales périphériques et centrales du stress : rapports étroits entre ces composantes d'une part et le cerveau d'autre part.



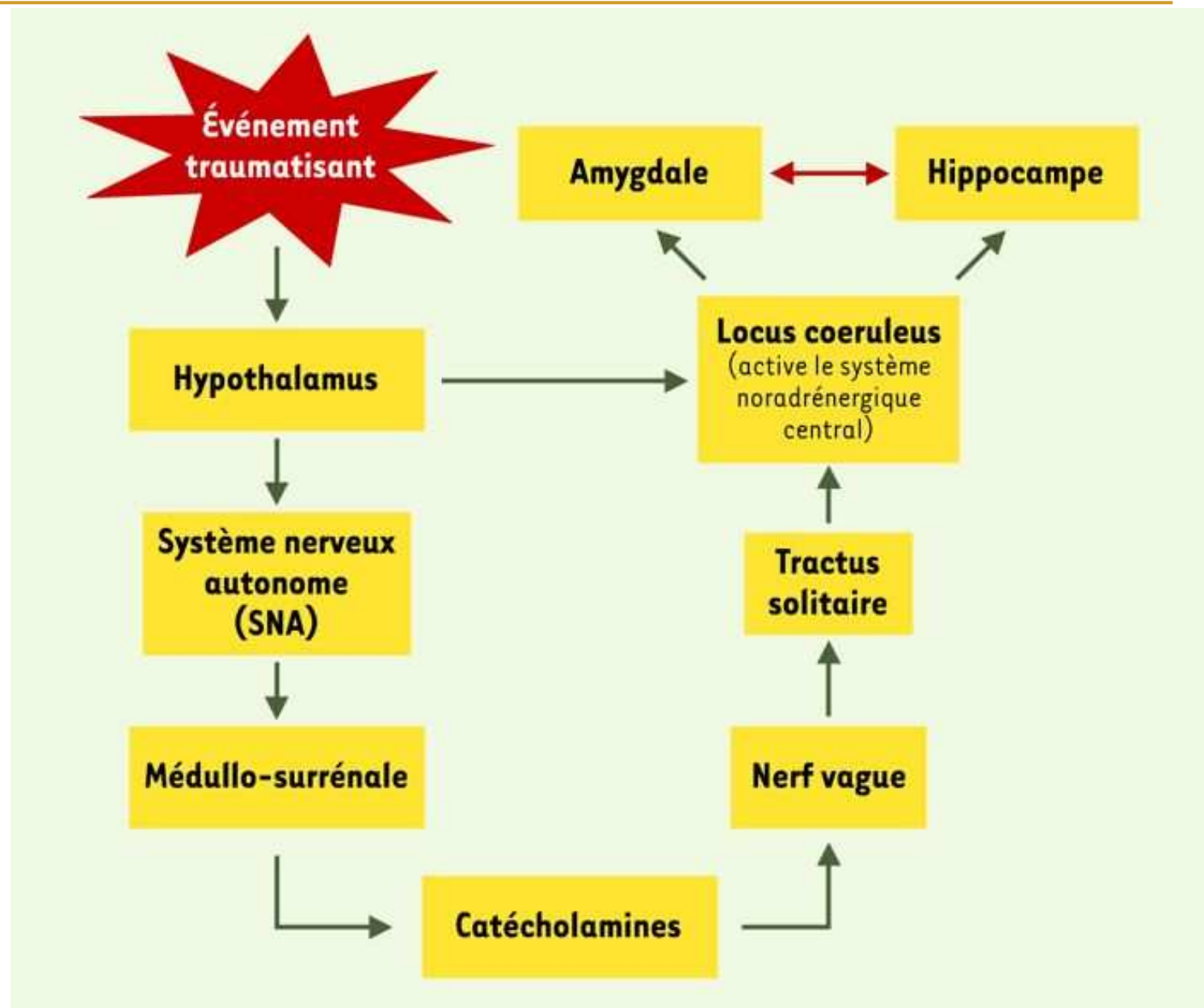
Le stress chronique :

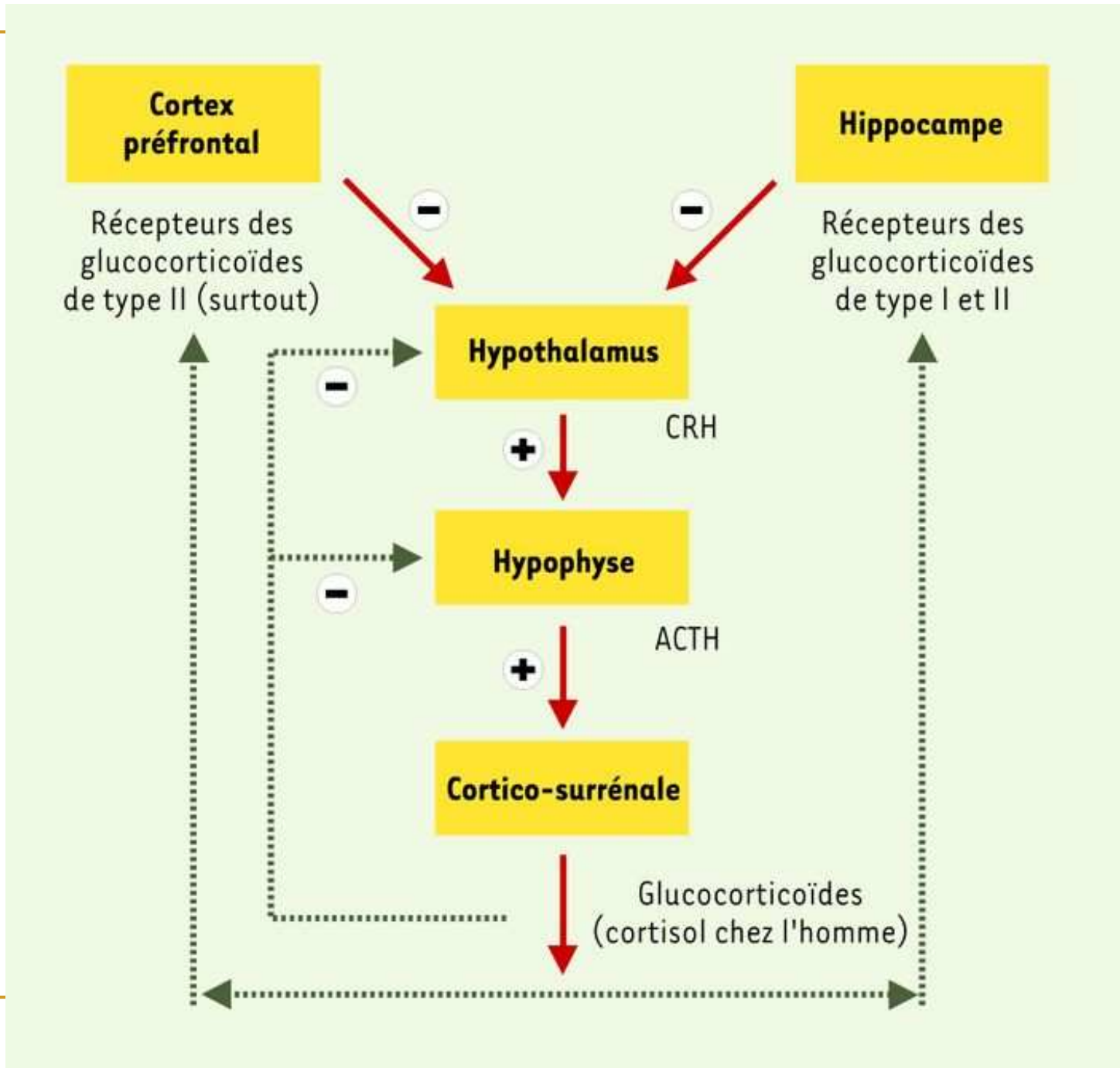
- augmente la quantité du tissu adipeux
- fait « fondre » les muscles et entraîne l'ostéoporose

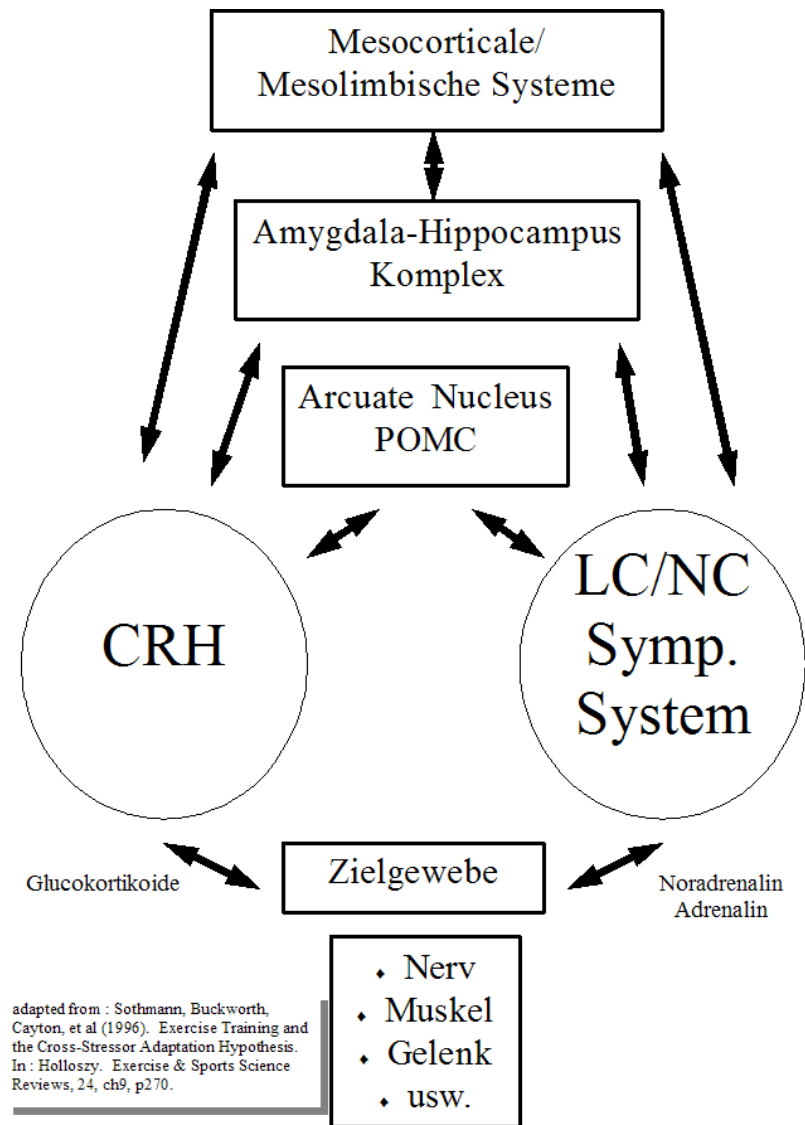
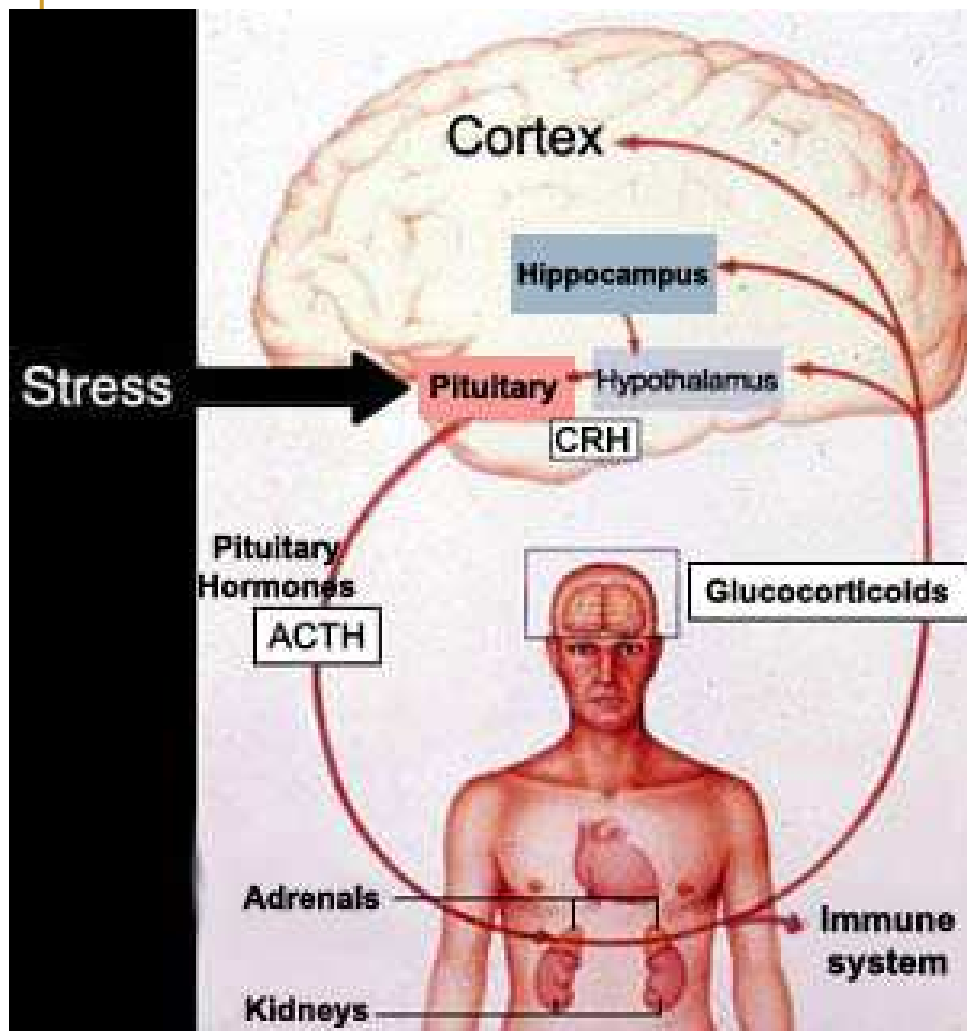


Effets du stress chronique sur la fonction gastro-intestinale

CRH: corticotropin-releasing hormone, ACTH: corticotrophin, PVN: paraventricular nucleus, LC: locus ceruleus.

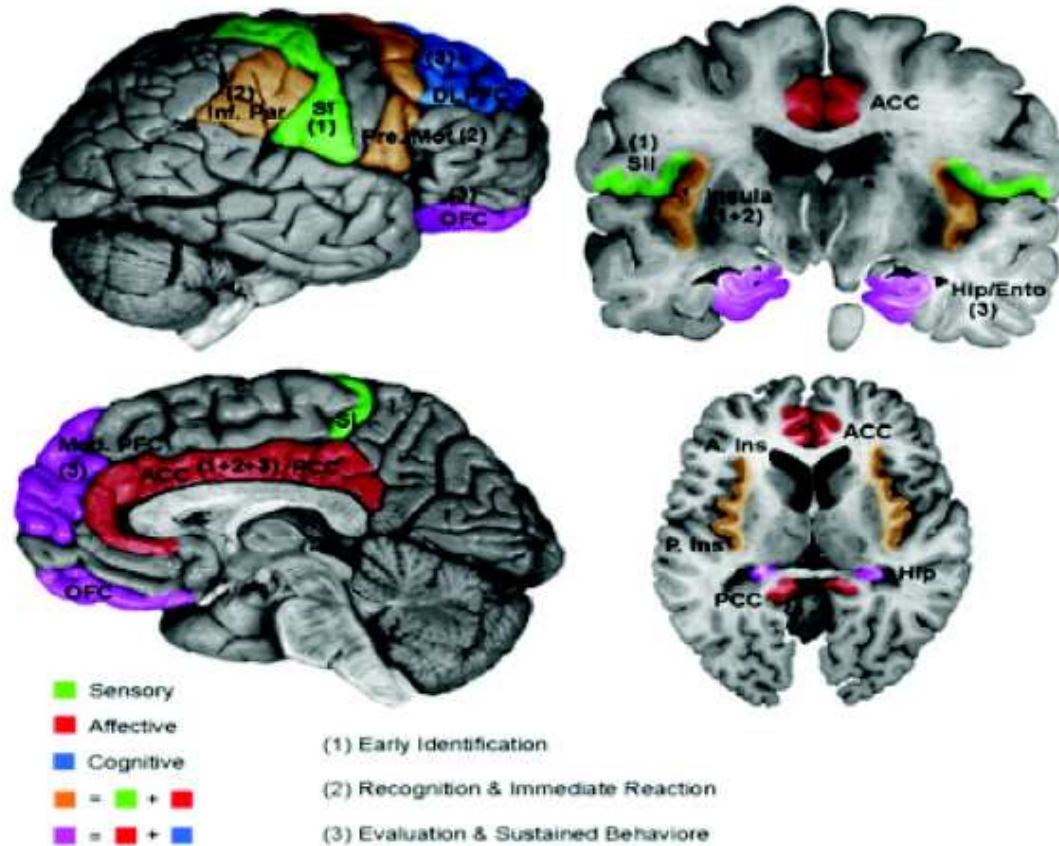




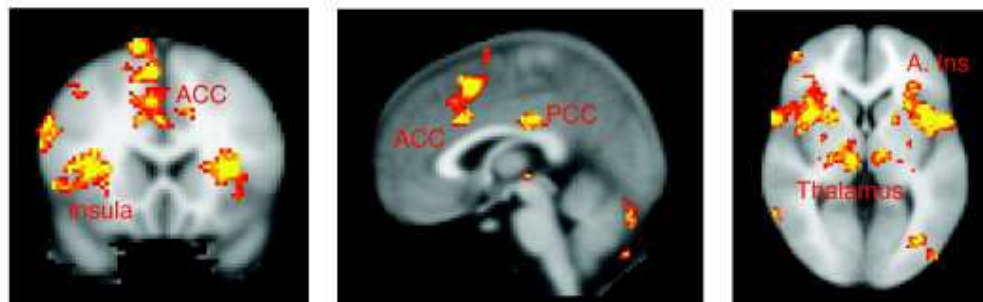


Functional measures

A. Brain areas functionally related to pain processing.



B. Example of functional MRI response to painful stimulation.



L'hippocampe régule le stress.

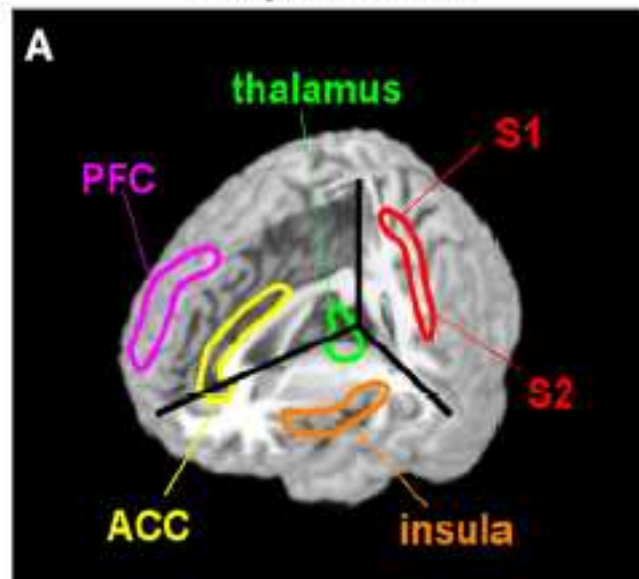
L'amygdale est activée (potentialisation à long terme) : le patient devient hyper réactif et hypersensible.

Le cortex cingulaire antérieure fait les liens entre les structures limbiques et le cortex préfrontal.

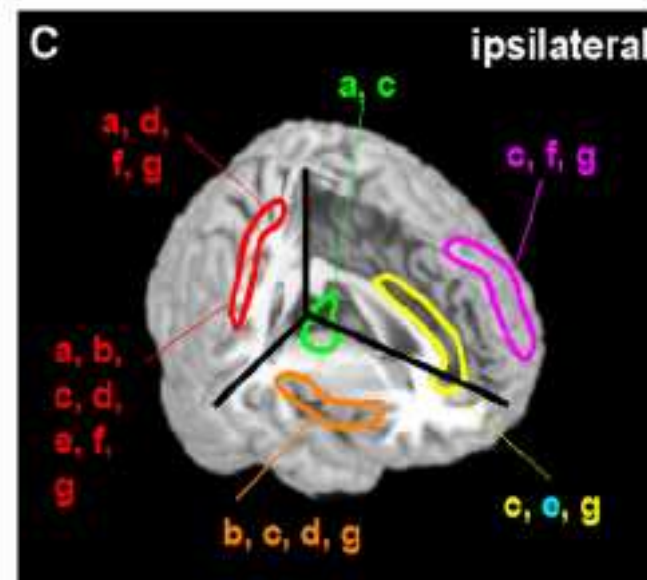
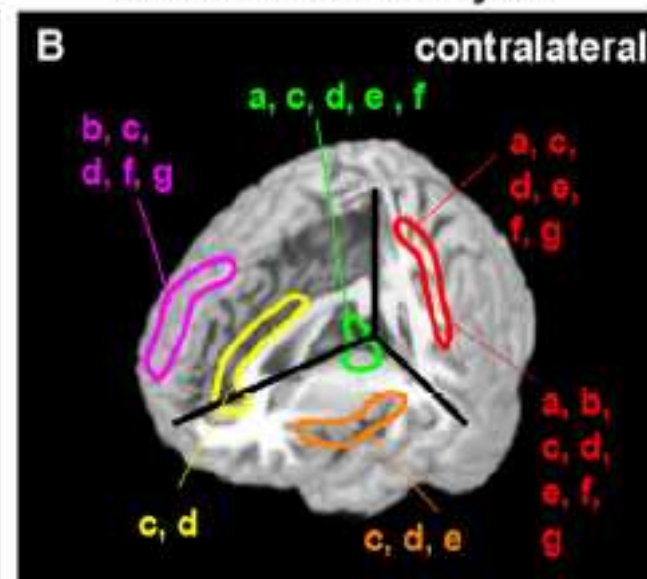
Le dysfonctionnement du cortex cingulaire antérieur fait que le patient n'arrive plus à contrôler ses émotions.

Le cortex préfrontal fonctionne au ralenti.

The pain matrix



Brush-evoked allodynia

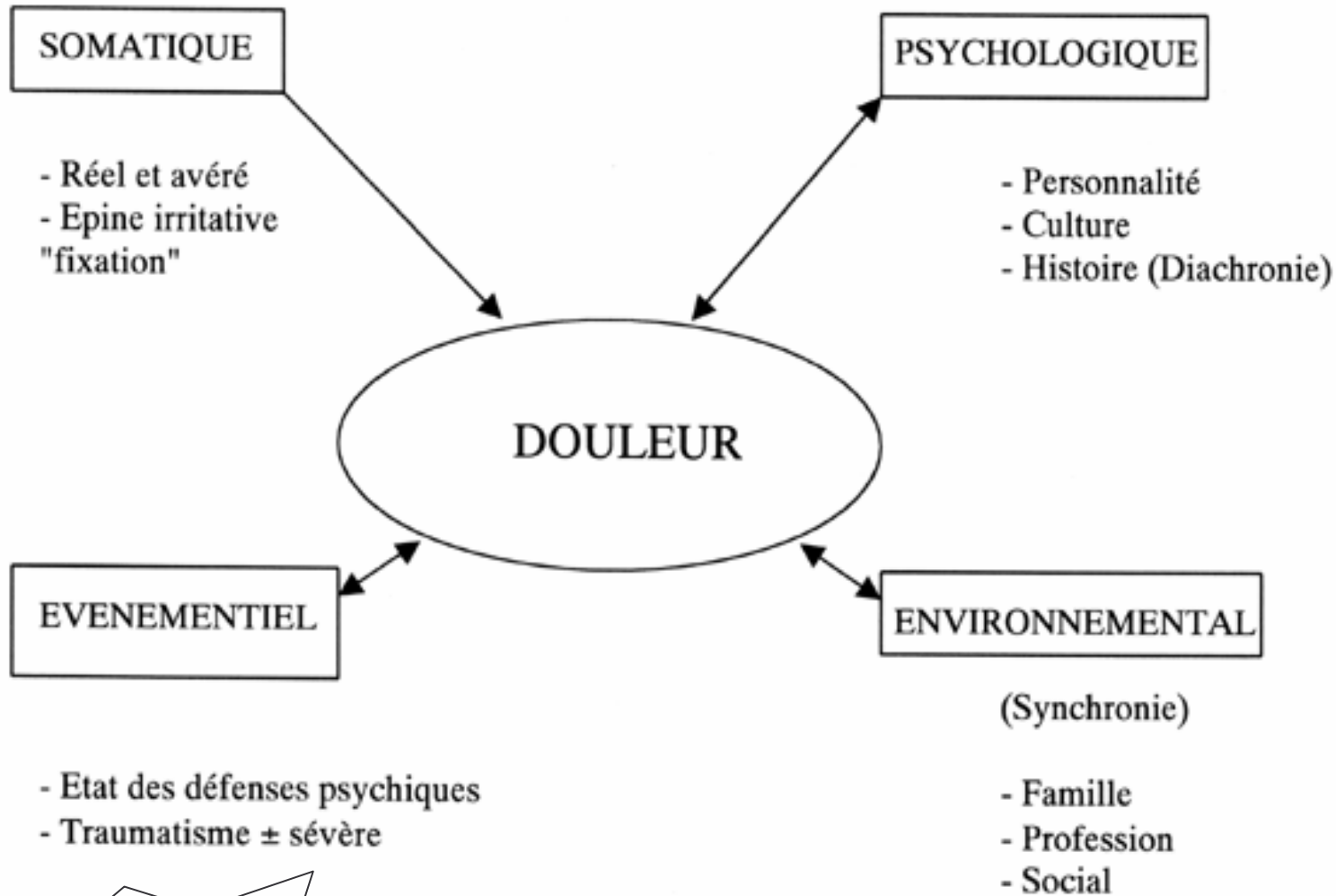


References

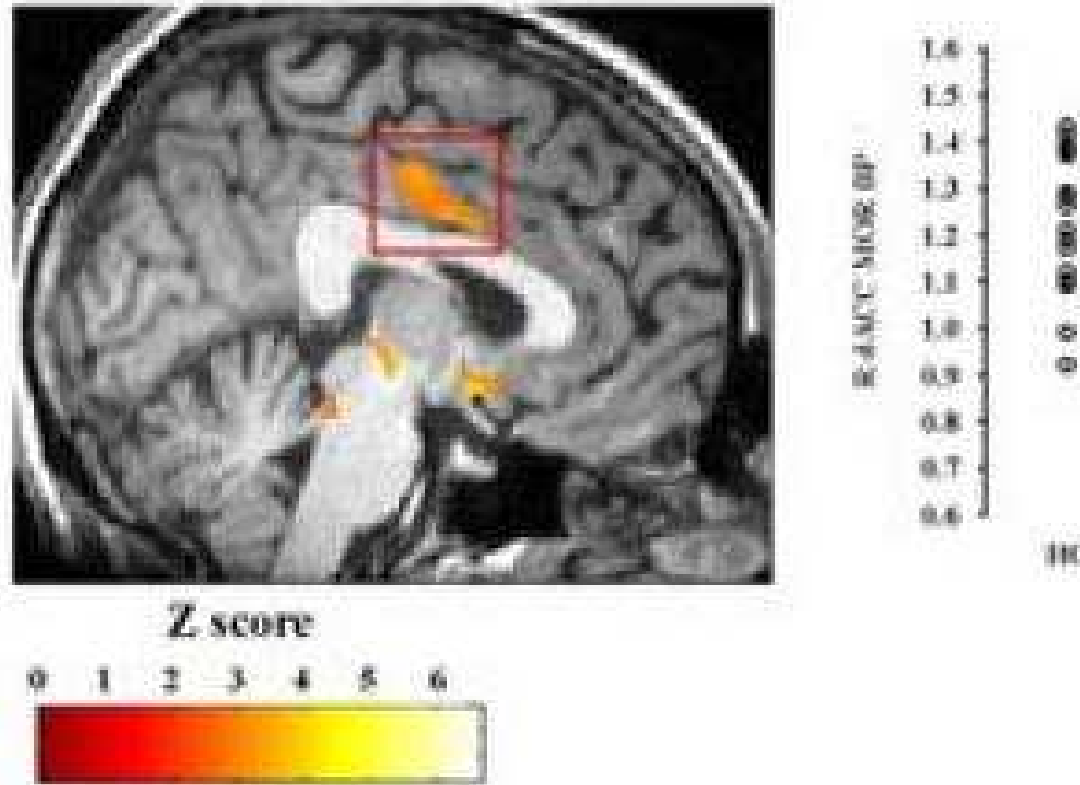
- a: Petrovic et al., 1999
- b: Witting et al., 2006
- c: Becerra et al., 2006
- d: Schweinhardt et al., 2006
- e: Peyron et al., 1998
- f: Ducreux et al., 2006
- g: Peyron et al., 2004

Ce sur quoi se focalisent patient et médecin!

Le médecin sait bien que cela intervient, mais pas le patient !



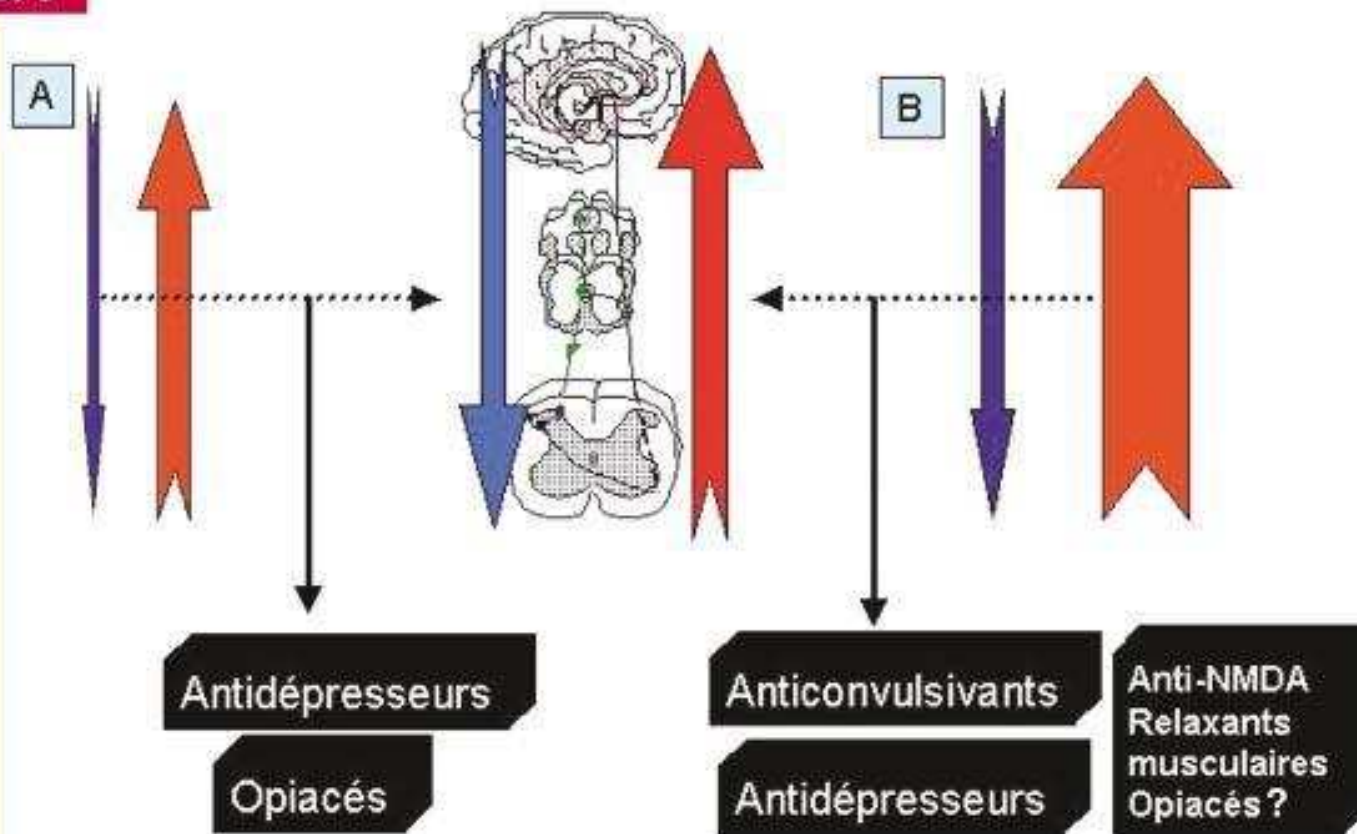
Le grand oublié!



Les patients souffrant de la fibromyalgie n'ont pas la même disponibilité en récepteurs opioïdes μ dans les régions cérébrales qui répondent aux stimulus douloureux-spécialement dans : le noyau acumbens, le cortex cingulaire et l'amygdale.

Mode d'action des médicaments utilisés dans le traitement du syndrome fibromyalgique

Figure 5



Pour augmenter l'activité du CIDN dans le modèle A, il faut augmenter les activités sérotoninergique (5-HT), noradrénergique (NA) et enképhalinergique (ENK). Les antidépresseurs et les opiacés permettent cette action. Dans le modèle B, il faut diminuer l'activité des afférences nociceptives. Les anticonvulsivants, certains antidépresseurs et les antagonistes des récepteurs NMDA pourraient réaliser cette action. Source : Arsenault et Marchand

Les rôles de l'amygdale : émotions, comportement et système nerveux autonome

