[https://www.smithsonianmag.com/videos/category/science/a-coconut-octopus-uses-tools-to-snatch-a-crab/](https://www.smithsonianmag.com/videos/category/science/a-coconut-octopus-uses-tools-to-snatch-a-crab/%22%20%5Ct%20%22_blank)

Rheumatic fever

<https://en.wikipedia.org/wiki/Group_A_streptococcal_infection>

<https://en.wikipedia.org/wiki/Antibody-dependent_cell-mediated_cytotoxicity>

<https://en.wikipedia.org/wiki/Cephalosporin>
<https://en.wikipedia.org/wiki/Peptidoglycan>

Adverse effects

Why fever

How mechanistically ?
<http://jeb.biologists.org/content/218/18/2961>

<https://www.quora.com/What-is-the-mechanism-by-which-the-body-raises-the-temperature-to-cause-a-fever>

White blood cells called monocyte-macrophages release proteins called
pyrogens when the cells encounter pathogenic microorganisms. The
pyrogens act on the hypothalamus, causing it to reset the body's
"thermostat" upward.

<https://books.google.com/books?id=-EBJms4f-zMC&pg=PA124&lpg=PA124&dq=pyrogens+act+on+the+hypothalamus&source=bl&ots=v0JrvCJ10H&sig=a4oub7xFLR1z2PQV1KnVBNpkxUQ&hl=en&sa=X&ved=0ahUKEwjLke-DkMHYAhXiYt8KHV9yABwQ6AEIdDAK#v=onepage&q=pyrogens%20act%20on%20the%20hypothalamus&f=false>

<https://www.ncbi.nlm.nih.gov/pubmed/?term=pyrogen+mechanism+of+action+neuron>

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3033975/>

<https://www.ncbi.nlm.nih.gov/pubmed/21177120>

thermosensitive neurons localized in the anterior hypothalamus
/preoptic area (AH/POA) to regulate core body temperature. Several
studies have demonstrated that some chemokines are also implicated in
the generation of a febrile response: central injections of IL-8/CXCL8
[40], macrophage inflammatory protein-1 alpha and beta (MIP-1α/CCL3
and MIP-1β /CCL4) [41], and RANTES/CCL5 [42] administered into the
AH/POA, evoke a febrile response characteristic of endogenous
pyrogens.

How activate cells ?

<https://www.ncbi.nlm.nih.gov/pubmed/17054999>

<http://www.sciencedirect.com/science/article/pii/036192309290005I>

"There have been many reports suggesting that IL-6 may be a central
mediator of fever in many species (6, I 1,18,19,2 I)."

IL6

Interleukin 6 (IL-6) is an interleukin that acts as both a
pro-inflammatory cytokine and an anti-inflammatory myokine. In humans,
it is encoded by the IL6 gene.[5]

<https://en.wikipedia.org/wiki/Interleukin_6>

Interleukin 6 is secreted by T cells and macrophages to stimulate
immune response, e.g. during infection and after trauma, especially
burns or other tissue damage leading to inflammation. IL-6 also plays
a role in fighting infection, as IL-6 has been shown in mice to be
required for resistance against bacterium Streptococcus pneumoniae.[6]

<https://en.wikipedia.org/wiki/Interleukin-6_receptor>

IL6  receptor neuron firing

<http://www.jneurosci.org/content/20/23/8637>

However, chronic exposure of neurons to IL-6 increases Ca2+ influx in
response to NMDA and causes neurodegenerative changes (Campbell et
al., 1993; Qiu et al., 1998).

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3491449/>

Back to pubmed:
<https://www.ncbi.nlm.nih.gov/pubmed/?term=hypothalamic+neuron+response+to+pyyrogen>
hypothalamic neuron responses to pyrogen.

<https://www.ncbi.nlm.nih.gov/pubmed/15733324>

Yet, little is known about the electrical responses by which PGE2
regulates the firing rates of VMPO neurons. We have hypothesized that
these PGE2 dependent changes in firing rate are not the result of a
change in the frequency of synaptic input to these neurons, but a
selective effect on specific electrical properties of VMPO neurons. To
characterize these responses, whole-cell recordings were made from
VMPO neurons in tissue slices from male Sprague-Dawley rats, in
response to changes in temperature and PGE2.

google
TRP channels hypothalamus

TRP channels hypothalamus PGE2

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3051412/>

The neurophysiological mechanism underlying the thermosensitivity of
warm-sensitive neurons in the POA continues to be investigated. The
suggestion that a heat-induced membrane depolarization allows
warm-sensitive neurons to reach their discharge threshold potential
and then determines their discharge frequency (55) contrasts with the
concept that a warming-dependent facilitation of the rate of rise of a
depolarizing (pacemaker) prepotential in warm-sensitive neurons
shortens the intervals between action potentials and thereby increases
their firing rates (56). In the latter case, a transient, outward
hyperpolarizing K+ current (A-type potassium current) helps maintain a
hyperpolarized membrane for a brief time after an action potential and
the heat-induced increase in the inactivation rate of the A-type
potassium current allows the prepotential to depolarize at a faster
rate (56). Although TRPV4 channels have not been found in POA neuronal
cell bodies, other ion channels that could contribute to the
thermosensitivity of warm-sensitive neurons, such as
hyperpolarization-activated cyclic nucleotide-gated channels and
background potassium leak channels are localized in the cell bodies of
many POA neurons, but are also distributed ubiquitously in the brain
(13, 57). Identification of the molecule(s) responsible for the
thermosensitivity of POA neurons await further investigation and the
identification of specific anatomical markers for thermosensitive
neurons would be a major discovery in this field.

