[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/" \t "_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.

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