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## Fever: When Will They Ever Learn?

*“Fever is generally considered harmful by physicians and is treated with antipyretics as it may lead to febrile seizures, stupor, dehydration, increased breathing, discomfort and tachycardia. It is a common practice to treat even low-grade fevers of 101° to 102°F with antipyretics. Home use of antipyretics upon the first signs of fever is also common. These behaviors have led to the ubiquitous use of aspirin, acetaminophen, nimesulide, and ibuprofen which control temperature by inhibiting prostaglandin synthesis in the hypothalamus.”<sup>1</sup>*

Paracetamol (or, acetaminophen, or Tylenol to Americans) was first used in medicine in 1893, but only became a commonly used drug in 1949.<sup>2</sup> Until 1971, no one had a clue how it worked, but that didn't matter. Doctors didn't seem to think that was important. Fever was “dangerous” so you stamped it out at all costs. Since 1972, scientists have been gradually starting to unravel some of the ways paracetamol suppresses various pathways in the brain and in the body, but as of 2008, their knowledge is incomplete, and part of the reason for that is that these same researchers still don't understand all the gears the body goes through to produce a fever, or why each gear is important, or the reason for the body getting into immune-system cruise as a result of fever. Most of these researchers just don't understand that fever is there as a beneficial adaptive response. When you don't know something as basic as that, but are intent on simply suppressing

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1 Torres, A.R. 2003 “Is fever suppression involved in the etiology of autism and neurodevelopmental disorders?” *BMC Pediatr*, 3: 9, September 2. Epub 2003, September 2. Review. PMID: 12952554.

2 Davies N.M. 2004. “Cyclooxygenase-3: axiom, dogma, anomaly, enigma or splice error? – not as easy as 1, 2, 3.” *J Pharm Pharmaceut Sci* ([www.ualberta.ca/~csps](http://www.ualberta.ca/~csps)) 7(2): 217–26. [http://www.ualberta.ca/~csps/JPPS7\(2\)/N.Davies/cyclooxygenase-3.htm](http://www.ualberta.ca/~csps/JPPS7(2)/N.Davies/cyclooxygenase-3.htm). Accessed 5 December 2007.

## FROM ONE PRICK TO ANOTHER

it because it can be done, you can be sure you are asking for trouble somewhere down the line.

In the late 1990s I was invited to participate in an afternoon's presentation at an Auckland medical education facility, ostensibly to speak about vaccination. My talk was sandwiched in between those of two other speakers, so to reduce any disruption of student concentration I was invited to attend the whole afternoon. The room had chairs and tables in a horseshoe shape, and I was seated near the rounded top of the  $\Omega$  hump, so to speak. The tutor was next to a whiteboard, by the two "heels". Within 15 minutes I decided I wasn't going to speak about vaccination only, because as the tutor's presentation progressed, I got angrier and angrier. How could paediatric staff be taught unscientific opinion?!

Come my turn, I said that I had some grave concerns about the accuracy of some of the "opinions" expressed by the previous speaker. The word opinion was used since I saw no references or "facts" put up on the whiteboard. This person was purely talking off the top of their head. Without sparing anyone's feelings or reputation, I launched into a literary review of the FACTS indicating that FEVER has a crucial role in fighting infections, and then into another literary review, showing paracetamol to be dangerous when suppressing a temperature. The article I started with was a 1995 medical article,<sup>3</sup> the conclusion of which says:

*There is little evidence to support the use of paracetamol to treat fever in patients without heart or lung disease, or to prevent febrile convulsions. Indeed, paracetamol may decrease the antibody response to infection, and increase morbidity and mortality in severe infection. It should be explained to parents that fever is usually a helpful response to infection, and that paracetamol should be used to reduce discomfort, but not to treat fever.*

The whiteboard rapidly filled with facts from this article, and other articles, showing that the use of paracetamol as an infection temperature reducer was not only unscientific, but highly dangerous, because, as intensive care unit specialist, Dr Shann, said:

*Immunity: Too many parents and health workers think that infection is bad, infection causes fever, and that therefore fever is bad. In fact, fever is often a beneficial host response to infection, and moderate fever improves immunity.*

Shann had discussed mammalian studies which showed increased death rates for

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3 Shann, F. 1995. "Paracetamol: use in children" *Australian Prescriber*, 18: 233–4. <http://www.australianprescriber.com/magazine/18/2/33/5/>

both virus and bacterial infections, increased viral shedding in flu patients, and reduced antibody levels when antipyretics were used. He then said that:

*Therefore, it may not be a good idea to give drugs that reduce temperature to patients with severe infection. This evidence suggests that aspirin and paracetamol increase mortality in severe infection, and that they may prolong the infection and reduce the antibody response in mild disease.*

By the time I'd finished, the board was covered with medical references, but as I looked around the room, it seemed as if the audience had shut off, in some mind-numbing, glazed-eyes "default" mode, which presumably said, "Listen to the teacher, not to some numbskull mother." So I quickly asked for questions. The first one was, "*What medical school did you go to?*" My reply was instant. "*Which medical articles on fever and infection have you read?*"

Looking through my 2007 telephone logbook, I have had about 12 conversations with people during the year, who were in hospital, and who were treated like scum by staff who thought they were criminally negligent because they didn't want their children treated with paracetamol for fever.

I had one conversation with an overseas mother whose child had been exposed to chickenpox and was taken to the doctor with a fever. The doctor thought it would be chickenpox, given the known exposure and time frame, and told the mother to treat with paracetamol. The doctor then had a brainwave, and gave this child an MMR shot because it would "save" the mother coming back in three weeks' time. The mother did as told, and for several days, the child's fever was treated as specified by the doctor. Not only did the child get chickenpox, but got measles as well, had seizures, and died.

In the child's post-mortem, neither the role of paracetamol, nor of MMR was considered relevant to the cause of death, which was specified as "chickenpox". I believe the role of both paracetamol and the MMR were very relevant as factors in this child's death, and that such a post mortem reveals the ignorance and contempt that many doctors have to this day, to the immunosuppressive role of fever reducers, or to any suggestion that a sick child should *never* be vaccinated.

When I settled down to read a 2007 article in *Pediatrics*,<sup>4</sup> these two parts of sentences leapt off the page:

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4 Curran, L.K. 2007. "Behaviors Associated With Fever in Children With Autism Spectrum Disorders" *Pediatrics*, 6: 120: e1386–e1392, December (doi:10.1542/peds.2007-0360). Published online 2007, November 30. <http://pediatrics.aappublications.org/cgi/content/full/120/6/e1386>

*“Understanding the role of fever, if any ...” and later, “... the functional significance of fever remains uncertain.”*

In 2007, no one in the department of Neurology and Developmental Medicine in Maryland, or any of the people in the Department of Epidemiology and Biostatistics, Pennsylvania, had a clue about the role of fever in infection? Why is that?

Okay, they were looking at it in the context of autistic children. This study was undertaken because, *“In the past few decades, parents and clinicians have reported that behaviors of children with ASD<sup>5</sup>s tend to improve, sometimes dramatically, during febrile episodes.”* The children’s improvement subsided afterwards, but the question remains to be answered, “WHY?”

Here again, we have a wonderful example of what “proof” is. Proof is whatever the doctor says it is, until they are proven incorrect. When a parent says, *“My autistic child improved dramatically during fever”*, it is anecdote. Even when clinicians agree, that knowledge is still “anecdote”, and it takes *decades* before a study of individuals is done, to confirm what parents have known for a very long time.

When the same parent says, “My child had absolutely no problems before any vaccines, had this reaction, was never the same again, and here’s the proof,” the eyes of the medical profession glaze over.

The only useful response from this study was that, *“more research is needed to prove conclusively fever-specific effects and elucidate their underlying biological mechanisms ...”*

However, I’m wondering if there’s more to the 2007 article than meets the eye.

The premise of another autism study,<sup>6</sup> conducted in 2003, was that: *“The blockage of fever with antipyretics interferes with normal immunological development in the brain, leading to neurodevelopment disorders such as autism in certain genetically and immunologically disposed individuals.”*

The article then goes on to say that *“The effects may occur in utero or at a very young age when the immune system is rapidly developing.”* Antipyretics might lead to neurodevelopment disorders if given when the immune system is rapidly developing? What about vaccines?

Such statements allow blame to be placed back on the mother to take the focus off all the talk about autism and vaccines. What these studies should show people, is how little doctors actually know.

There is another interesting point in the discussion, and that’s the fact that for once, someone has taken “anecdote” seriously, albeit just about a generation

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5 ASDs = Autism Spectrum Disorders.

6 Torres, A.R. 2003. “Is fever suppression involved in the etiology of autism and neurodevelopmental disorders?” *BMC Pediatr*, 3: 9, September2. Epub 2003, September 2. Review. PMID: 12952554.

after the anecdotes were first told. Let me tell you some “anecdotes” from the days when parents were not paranoid about measles, and when some young wives and mothers knew how to dose measles with vitamin A, vitamin C and other treatments which doctors said didn’t exist. We knew that contrary to vaccination-spin pamphlets, complications and deaths were very unlikely in healthy children treated correctly.

Like-minded parents used to get together and comment how, after measles, or even moderate fevers from other infections, children would make developmental milestone leaps, and it was not trickery of the imagination. This happened twice in our house. I have a habit of writing everything down, during and after infections, because I know it won’t be remembered in days or years to come. Also, I liked Plunket nurses<sup>7</sup> and doctors to know what I’d written before they filled in the next gap, even if they did sigh and roll their eyes before writing in their own words of wisdom!

After our older son’s bout of measles, he made leaps and bounds in language. His already good vocabulary suddenly increased in both numbers of words, and the fluency with which he strung them together. With our younger son, his development improvement was in a totally different area. He had been very clumsy and used to fall forwards a lot. After measles, not only did he stop falling over at all, but his overall co-ordination, including eye-hand co-ordination, was a lot less “random”.

Our friends noticed similar things, but all of them shrugged and said, “That’s just normal. All kids make strides of some sort after measles.”

Our GP, on hearing this, laughed somewhat like a donkey’s bray. Ten years later, I listened with interest, as an anthroposophical doctor talked about this phenomenon, and noted articles from anthroposophical medical journals on his table.

Is there something valid to these anecdotes from parents who saw their children’s overall health improve after a decent fever?

What say it’s not “just” autistic children who show temporary improvement during a fever? What if fever is a very powerful, positive neurodevelopmental tool required for all young children, which is needed to burn out (for the lack of a better term) “glitches” in the cranial system, or perhaps unknown epigenetic influences?

What say depriving children of infectious diseases, by using vaccines and using paracetamol for every other fever, is doing exactly the opposite to what the body needs, and is designed to do?

Why do doctors and hospitals make parents treat fever as if it’s something bad, to be brought down immediately, and to be feared?

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<sup>7</sup> Plunket nurses in those days, came to the homes of babies for many weeks, and then after a few months, parents would take their babies to the Plunket rooms every month.

## FROM ONE PRICK TO ANOTHER

Looking through clear files full of medical articles on (ab)use of paracetamol for infectious fever, I am amazed to see the number of times, and in such a broad variety of clinical situations,<sup>8</sup> that this phrase comes up:

*“Routine antipyretic therapy in children with infectious diseases has long been the source of controversy.”*

Controversy? Where? I know of no mother who frequents a doctor’s surgery who realizes there is any *controversy* around the use of paracetamol for infection. For decades now, a few medical people have had doubts, and made rumbling noises, but does their discontent achieve anything in reality? Is anyone researching what fever does in the body, not just in terms of infection outcome, but in the context of the overall health of children?

No. So, why is paracetamol even suggested?

The answer lies in some of the advertisements we have seen, and still see. For instance, the McNell Motrin advertisement used in American Newsweek in 2000,<sup>9</sup> told us that Motrin *“never surrenders”* and is *“For Moms who don’t fool around with fever.”*

In other words, to do nothing is fooling around, and fooling around equates to being a bad parent.

A recent advertisement<sup>10</sup> in New Zealand for paracetamol is a lot more subtle and takes the “intellectual pride” route. It says:

*“I wouldn’t put just anything in my body. That’s why I always think twice about what I do. Some decisions are hard to make. But in the end, you’ve got to do what’s right for you. Panadol. It’s my choice.”*

Which tells you nothing about Panadol<sup>®</sup>, but is pitched to make you think that if clever people who think twice, make the “choice” to take Panadol<sup>®</sup>, that would be the right thing for you to do as well. It’s the old ‘go with the (alleged) crowd’ trick. Do readers think about the fact that they aren’t told what those supposedly clever people even thought about in the first place?

Studies conducted overseas<sup>11</sup> and in New Zealand<sup>12</sup> have shown that children

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8 Brandts C.H. 1997. “Effect of paracetamol on parasite clearance time in Plasmodium falciparum malaria.” *Lancet*, 350(9079): 704–9, September 6. PMID: 9291905.

9 Newsweek pullout, sent to me from America. McNell ©McN-PPC, Inc. 2000.

10 Paracetamol advertisement by GlaxoSmithKline, *Sunday Star Times Magazine*, 2007, April 8.

11 Riece, K. et al. 2007. “A matched patient-sibling study on the usage of paracetamol and the subsequent development of allergy and asthma.” *Pediatr Allergy Immunol*, 18(2): 128–34, March. PMID: 17338785.

12 Cohet C. et al. 2004. “Infections, medication use, and the prevalence of symptoms of asthma, rhinitis, and eczema in childhood.” *J Epidemiol Community Health*, 58(10): 852–7, October. PMID: 15365112.

who were given paracetamol early in life have a 25% higher risk<sup>13</sup>, of having asthma symptoms. Antibiotic use in infancy has been found to have the same association. It would seem logical to assume that both paracetamol and antibiotics have a negative impact on the immune system in the long term. What does paracetamol do in the immune system, during fever, or to the immune system afterwards? I can't find any answers in the medical literature.

It's vital that the fever/paracetamol/immune system issues are resolved, for the sake of both parents' and children's health.

No doubt until then, I will continue to be sent stories like this one from an overseas blogger who had finished reading Chapter 39 in our first book,<sup>14</sup> *Just a Little Prick*, and felt compelled to tell their story. He gave permission for me to publish their experience with fever.

*One morning when Savannah was barely one, while playing around with us in bed, she suddenly went slack and inert. Controlled panic ensued. I drove, in pyjamas and stockinged feet, at breakneck speed to get her to the hospital, about 8 minutes away. Several white-clad professionals immediately went to work on her. She was given some kind of fever-reducing injection (I probably don't want to know what it was). I think her fever had spiked to 105 °F or so. When I asked if this might cause brain damage, I was told that only an EEG could tell. So we subjected Savannah to the machine, with wires stuck to her scalp. She "turned out" to be just fine, for which "intelligence" we had to fork out aplenty. We were advised to bathe Savannah in water as cold as she could stand. We did. Next day, we took her to a pediatrician someone recommended.*

*He diagnosed Roseola.*

*He became visibly angry when we told him what we had been sprung for the EEG. Then he told us the truth. "Children are capable of withstanding temperature spikes like that with no damage. My hardest job is to convince parents to DO NOTHING when their children develop high fevers. They can handle it."*

How many doctors do you know, who would have told the parents that children can handle fever?

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13 Massey University. 2004. "Paracetamol or antibiotic use early in life may increase the subsequent risk of asthma." September 16. [http://masseynews.massey.ac.nz/2004/Press\\_Releases/09\\_16\\_04.htm](http://masseynews.massey.ac.nz/2004/Press_Releases/09_16_04.htm). Accessed 6 December 2007.

14 *Just a Little Prick*. "The Fever-Pitch Bandwagon," p. 259.

# 48 Bringing Chickenpox to the Boil

Avid readers of dramatic novels from yesteryear will recall stories from the days when fevered patients were watched over by family, and the oldies in the group just “knew” that a proper fever would “break” with a sweat. When that happened, they knew that the prognosis would be good. Of course, such sentiments today would be greeted with alarm, or scepticism, by those who consider illness should never be endured.

Isn't that why acetaminophen (in all their different brand names) is reached for, at the first sign of a fever?

In 2001, a headline<sup>1</sup> made me look twice. “*Sweat has the power to fight off disease.*” We were told that sweat contains a versatile antibiotic that may be on the front line against disease-causing bacteria and that: “The researchers said dermcidin probably plays a key role in the innate immune responses of the skin”. A news roundup from the *British Medical Journal* told us<sup>2</sup> that dermcidin killed *escherichia coli*, *enterococcus faecalis*, *staphylococcus aureus* and *Candida albicans*. It was active at high salt concentrations and the acidity range of human sweat. In concentrations of 1–10 µg/ml, it killed all of the *staph aureus* colonies in only four hours. Unsurprisingly, the scientists didn't know how dermcidin worked.

Up until the late 1990s the skin was simply thought to be a “barrier” with no active participation in the immune system. The original 2001 paper<sup>3</sup> said that during some inflammatory skin disorders and wound healing, skin cells functioning within a salty sweat with a pH of 4–6.8, produced many effective pharmacologically active substances, such as immunoglobulin A, interleukin 1, 6 and 8, tumour

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1 Associated Press. 2001. “Sweat has the power to fight off disease.” *The New Zealand Herald*, November 9, p. A13.

2 Josefson, D. 2001. “Bacteria killer found in sweat” *BMJ*, 323: 1206, November 24. <http://bmj.bmjournals.com/cgi/content/full/323/7232/1206/c>

3 Schitteck, B. 2001., “Dermcidin: a novel human antibiotic peptide secreted by sweat glands.” *Nat Immunol*, 2(12): 1133–7, December. PMID: 11694882.



necrosis factor, transforming growth factor  $\beta$  receptor, epidermal growth factor, and a prolactin-inducible protein.

As time has gone on, other researchers have taken a closer look at skin, and have found that the neutrophil,<sup>4</sup> which is the professional phagocyte of fundamental importance for defence against micro-organisms, provides instant help, not only in microbial infection,<sup>5</sup> but to the growth factors when the skin is broken and there is a risk of infection. Another article<sup>6</sup> says that mast cells, macrophages and skin cells produce antimicrobial peptides. These are called cathelicidin, which disrupts bacterial cell walls, modifies the host cells inflammation, and provides additional immune defence. At the heart of this all, is our friendly neutrophil:

*“These studies clearly illuminate the importance of neutrophil recruitment in cutaneous defense against bacterial infection. ... Recent advances in understanding of innate immune defense systems have suggested that these ancient evolutionary immune mechanisms may be important to human disease yet previously underappreciated.”* (Underlining mine)

The article looked at whether just skin and mast cells were involved, or whether neutrophils were also important. Using mice, they found that mice with few neutrophils developed much worse tissue death (necrosis) and had 3,000 times the amount of bacteria on the skin than mice with active neutrophils. The skin cells worked hard and could produce some cathelicidin on their own, but didn't have the killing power of the skin cells plus neutrophils. The article's conclusion said that life-threatening necrotizing skin and soft-tissue infections can develop in patients with depressed neutrophils, but that numerous examples exist of patients with increased frequency of skin infections who have no “*demonstrable defect<sup>7</sup> in leukocyte recruitment or function.*”

Many countries have recently been bombarded with stories<sup>8</sup> about chickenpox resulting in death or serious bacterial infection.

The *New Zealand Herald* article cited above talked about a 14-year-old student, Luchan Li, who “*died of heart failure as a result of a blood infection, also known as septic shock. The illness was possibly connected to a case of chickenpox Luchan had two weeks earlier, but no one knows for certain.*”

Is it a coincidence that this article was published before the proposed introduction of the chickenpox vaccine in this country?

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4 Neutrophil; See Chapter 70 (on Vitamin C and sepsis).

5 Borregaard, N. et al. 2005. “Neutrophils and keratinocytes in innate immunity – cooperative actions to provide antimicrobial defense at the right time and place.” *J Leukoc Biol*, 77(4): 439–43, April. Epub 2004, December 6. Review. PMID: 15582983.

6 Braff, M.H. et al. 2005. “Keratinocyte production of cathelicidin provides direct activity against bacterial skin pathogens.” *Infect Immun*, 74(10): 6771–81, October. PMID: 16177355.

7 Demonstrable defect = Did the researchers check to see if the patient had enough vitamin C for the leucocyte system to work? Not as far as I can see.

8 Vass, B. 2007. “Mystery bug claims teen's life” *The New Zealand Herald*, November 20. [http://www.nzherald.co.nz/category/story.cfm?c\\_id=204&objectid=10477164](http://www.nzherald.co.nz/category/story.cfm?c_id=204&objectid=10477164) Accessed 21 November 2007.

At the same time, the *Daily Mail* in England ran a very emotive article about a little girl called Isobel: “*Within days, the virus had taken hold of her body, leading to toxic shock syndrome – a rare type of blood poisoning caused by bacteria – and necrotising fasciitis, a bacterial infection that rapidly eats away at the flesh.*”

The article went on to say that it is “thought” that dozens of other chickenpox children have the same complications.

Isobel’s mother said that “*if she’d had a big dose of antibiotics at the start, none of this would have happened.*” Just maybe Isobel didn’t have enough vitamin C to operate her leucocyte system to get rid of the bacteria. And did Isobel’s mother use the English version of acetaminophen? The second child in the article, Christopher, who died from chickenpox, was given that drug.

Before antibiotics were used in medical practice, when rickets was still rife and scurvy relatively common, chickenpox was known to have a much higher rate of Group A streptococcal (GAS) infection complications than that seen today. Group A streptococcus also causes scarlet fever, and rheumatic fever, which in most developed countries, started declining in 1850<sup>9</sup>, well before antibiotics were marketed. As a marker of group A streptococcus severity, scarlet fever has exhibited at least four cycles of varying severity followed by remission, believed to have been due largely to virulence variation. A very good article<sup>10</sup> on the web states, “*...reports of fatal infection with invasive strep A bacteria have been increasingly recognized in the United States since 1987. Researchers do not know why the new strain of strep A is on the increase or why it targets certain otherwise healthy people.*” Older textbooks and papers all mention the need to be careful when GAS infections follow chickenpox. For thirty years after the introduction of penicillin, there were no reports of serious GAS complications after chickenpox. But those years follow hard on the heels of the “conquest” of rickets, which up to the 1930s had affected nearly 50% of wealthy parents’ children in London. There are still some alive who remember the blackstrap molasses and cod liver oil morning routines of the times. Both “malnutrition” and “bad” nutrition can result in infections becoming far more serious.

After the Depression era in the 1930’s, food was a lot more basic than it is today, with minimal additives, and very little “junk” food to be found. Nutrition was far better in a general sense than it is now. Because of the huge increase of empty calories in family diets today, many children may now be at greater risk of secondary bacterial infections after chickenpox.

Properly fed, healthy children, whose parents know what to do, and what not

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9 McKeown, T and Lowe C.R. 1974. “An Introduction to Social Medicine.” ISBN 0 632 09310 2. Pgs 12–13.

10 Directors of Health Promotion and Education. “Group A Streptococcus.” Accessed on 26 January 2008. <http://www.dhpe.org/infect/strepa.html> This article is a very good ABC on the various very different infections with a single bacterial group can cause.

to do, will rarely get any complications to chickenpox. As was the case for our children, well-managed chickenpox should not even lead to any scarring. So let's ask some questions here, with chickenpox in mind. *What is the function of fever?*

Here's a really simple statement<sup>11</sup> from twenty years ago: "... *elevated body temperature enhances the inflammatory response and function of the immune system at the same time that it reduces the replication of microbes and tumor cells.*"

Not so simple is this sentence. "*Fever also appears to be a prominent component of cytokine therapy and attends the use of several biologic response modifiers.*" Fever switches on the chemical messengers and processes which call on the body immune system to respond and "modify" or deal with the infection.

If fever is a key to an immune-system process, without a fever, how effective is the body going to be in fighting viruses, or bacteria? With viruses like chickenpox, which are known to have an affinity with *group A streptococcus*, which can infect the pox rash and so have access to the body, what do we want the immune system to do? It's pretty obvious isn't it?

We *want* to allow the body temperature to rise to the level it needs so that all the on-switches can be thrown.

We *want* the body to send out all those little chemical messengers which get the antiviral side of things going.

We *want* the messengers to call the neutrophils to join the skin cells in producing cathelicidin, and to work with the whole array of anti-viral and antibacterial components<sup>12</sup> in "sweat" to stop *group A streptococcus* in its tracks.

As a 1991 article<sup>13</sup> says: "... *temperature elevation ... enhances the processes involved in initial antigen recognition and support for immunological specific response to challenge.*"

We want the body to recognize the virus, ring the bell and sound the red alert (fever) to fight, don't we? Why, then, turn the fever off with acetaminophen products? Doesn't that defy logic?

Another article<sup>14</sup> of that era said: "*There is considerable in-vitro evidence that a variety of human immunological defences function better at febrile temperatures than at normal ones ... Studies have clearly shown that fever helps laboratory*

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11 Dinarello, C.A. et al. 1988. "New concepts on the pathogenesis of fever." *Rev Infect Dis*, 10(1):168-89, January-February. Review. PMID: 2451266.

12 Dorschner, R.A. et al. 2001. "Cutaneous injury induces the release of cathelicidin anti-microbial peptides active against group A streptococcus." *J Invest Dermatol*, 117(1):91-7. PMID: 11442754. <http://www.nature.com/jid/journal/v117/n1/pdf/5601121a.pdf> (Pox from chickenpox qualifies as cutaneous injury.)

13 Roberts. N.J. Jr. 1991. "Impact of temperature elevation on immunologic defenses." *Rev Infect Dis*, 13(3): 462-72, May-June. Review. PMID: 1866550.

14 Kramer, M.S. et al. 1991 "Risks and benefits of paracetamol antipyresis in young children with fever of presumed viral origin." *Lancet*, 337(8741): 591-4, March 9. PMID: 1671951.

*animals to survive an infection whereas antipyresis<sup>15</sup> increases mortality.”*

A 1998 article<sup>16</sup> said: “*The elevation of body temperature by a few degrees may improve the efficiency of macrophages in killing invading bacteria, whereas it impairs the replication of many microorganisms, giving the immune system an adaptive advantage. There is a simultaneous switch from the burning of glucose, an excellent substrate for bacterial growth, to metabolism based on proteolysis and lipolysis. The host organism is anorectic (doesn’t want to eat) minimizing the availability of glucose, and somnolent, reducing the demand by muscles for energy substrate. During the febrile response, the liver produced proteins known as acute phase reactants ... the net effect ... is to give the host organism an adaptive advantage over the invader.*” (Underlining mine.)

I could bombard you with article after article showing not only that fever in infections is beneficial, but also that when you use paracetamol products, you *increase* the likelihood of dying and you *increase* the likelihood of complications. Pubmed is littered with articles from around the world saying this. The World Health Organization surprised me by having two articles on its website decrying the use of paracetamol for bringing down fevers.

Treating fevers is dicing with more severe infection, and a greater likelihood of death, because fever is a key immune response to get the immune system working properly.

You mess with fever, and you mess with lots of things. It stands to reason. Do you need to know what the medical profession does not *yet know about fever in its totality*, to see that?

Back to chickenpox. Tucked away in a small corner of the *New Zealand Herald* in 2001 was a warning:<sup>17</sup> “*GPs warned over chickenpox drug.*” Doctors were warned about treating chickenpox with ibuprofen to reduce fever because of a higher rate of necrotizing fasciitis<sup>18</sup>. There was no mention of paracetamol in the warning, yet, since both perform the same function, there is reason to argue that paracetamol might do the same as ibuprofen. In USA, the link between the use of non-steroidal anti-inflammatories and chickenpox reached the ears of doctors,<sup>19,20</sup> but not, it seems, the public.

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15 *Antipyresis* = reducing fever; bringing a temperature back down to normal. Anti and “pyresis” = bonfire.

16 Saper, C.B. 1998. “Neurobiological basis of fever.” *Ann NY Acad Sci*, 856: 90–4, September 29. Review. PMID: 9917869.

17 (No author named.). 2001. “GPs warned over chickenpox drug.” *New Zealand Herald*, February 1, p. A5.

18 *Necrotising fasciitis* = many bacteria can cause flesh-eating disease, but Group A *Streptococcus* is the most common of these.

19 Gonzalez, B.E. et al. 2005. “Severe Staphylococcal sepsis in adolescents in the era of community-acquired methicillin-resistant *Staphylococcus aureus*.” *Pediatrics*, 115(3): 642–8, March. PMID: 15741366.

20 Barton, L.L. 2005. “Nonsteroidal anti-inflammatory drugs and invasive staphylococcal infections: the cart or the horse?” *Pediatrics*, 115(6): 1790 and author reply p. 1791; June. No abstract available. PMID: 15930253.

There was a flurry of articles suggesting it was dangerous to use anti-febrile drugs with chickenpox; there was also an article by a group of doctors, who in defiance of all logic and known immunological impacts of drugs used to reduce fever, decided that there was no association. They<sup>21</sup> decreed that when parents used drugs to “treat high fever and severe illness”, drug use was merely the identifying factor of who was at high risk for secondary bacterial infection! That interesting little word “coincidental” again.

Doctors<sup>22</sup> will say that the resurgence of streptococcal infections “highlights the wisdom of recommending widespread use of the varicella vaccine to prevent this kind of infection”. Why worry about GAS, when a vaccine will prevent both chickenpox and GAS. On the surface, this looks logical.

I see the increase in these infections as evidence of a total lack of common sense about how to prevent complications. I see the association between non-steroidal anti-febrile drugs and GAS as a predictable outcome of the loss of home nursing skills and handed-down generational wisdom. I see the increase in secondary bacterial infections as something which can stem from parental lack of understanding that messing around with fever, and using symptom-suppressing/immune-suppressing drugs can restrict the ability of the immune system to fight the virus. It also reduces the ability of the leucocyte system of neutrophils, macrophages and phagocytes to fight bacterial toxins from secondary bacterial infections.

As pointed out in Chapter 70, if you don't have enough vitamin C in your system, then the neutrophils won't be recognized by the macrophages, and you might be in big trouble, because if that happens, the result could be toxic shock/sepsis taking hold very quickly. Even if you have enough vitamin C, if the amount of GAS toxin is such that the glucose transporters (which are part of the vitamin C shuttle service which takes ascorbate from A to B) are blocked, that can result in a GAS infection which threatens to run out of control. The quickest way to restore the immune function in a case of sepsis is by giving vitamin C intravenously. The body can fight sepsis by itself, but it's a bit more of a lottery as to whether it will succeed if it doesn't have the tools to do the job.

“Health” is not a one-pronged fork. Lots of things have to be working well, for the body to do what it is programmed to do.

Get smart with your computer, and the whole thing can crash. That analogy applies to the processes of fighting infections. So the next time you read a historical novel where the family is relieved to see the break out of a fevered sweat, you will have an idea why. The anecdote of the old wives wins out yet again. Everyone knew

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21 Lesko, S.M. et al. 2001. “Invasive group A streptococcal infection and nonsteroidal antiinflammatory drug use among children with primary varicella.” *Pediatrics*, 107(5): 1108–15, May. PMID: 11331694.

22 Stevenson, M. 1997. “Gas infections and varicella have a long standing relationship”. *Infectious Diseases in Children*, August. <http://www.idinchildren.com/199708/frameset.asp?article=gasinfct.asp>

that to beat the sickness lottery, a big sweat was usually a plus. Now we know why. A big sweat is part of the beneficial natural defense your skin immune system uses to fight any bacterial flora on/in the skin, such as group A *streptococcus*.

A big sweat shows that the immune system is working properly. A fever and a sweat in any infection, if you do not have heart or lung disease,<sup>23</sup> is the right thing<sup>24</sup> to allow to happen.

In the “olden days”, they didn’t clean a patient during an infectious sweat, and after the sweat broke, they let them sleep. My grandma would change the sheets, but she knew that there would be no shower until after the patient had recovered. She just “knew” that was the right way to treat infections.

TLC,<sup>25</sup> drinks, maybe cool cloths to the wrists and face, and a gentle breeze from a slow fan is all that is needed.

Yet it’s amazing how often you find out that some well-meaning parent sees a sweat and does exactly the wrong thing by “cleaning” the child up with some new and improved antibacterial soap, all in the name of making the person more comfortable!

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23 Shann, F. 1995. “Paracetamol: use in children.” *Australian Prescriber*, 18: 233–4. <http://www.australianprescriber.com/magazine/18/2/33/5/>. Accessed 6 December 2007.

24 Eichenwalk, H.F. 2003. “Fever and antipyresis.” *Bulletin of the World Health Organization*, 81(5). [http://www.scielosp.org/scielo.php?script=sci\\_arttext&pid=S0042-96862003000500012](http://www.scielosp.org/scielo.php?script=sci_arttext&pid=S0042-96862003000500012). Accessed 6 December 2007.

25 TLC = Tender loving care.