

Gardasil®: What you don't know.

The spiel on Radio, and the ads on TV, tell you that Gardasil® will eliminate 70% of cervical cancer caused by human papilloma viruses types 16 and 18, as well as genital warts.

You are told your daughters will benefit from this vaccine “breakthrough”, but they must be jabbed **before** they become sexually active, because the first time your daughter has sex, HPV viruses explode out of nowhere to suddenly threaten your daughter with cancer.

Think about that for a moment. You are being asked to believe that babies and children exist in sterile little bubbles, while these viruses are running rampant in their parents, older siblings and the wider community, causing warts and all sorts of other infections?

Thomas R Broker, President of the International Papillomavirus Society, made this comment¹ at a talk: ***“Papillomavirus is in, effectively, all the vertebrates: snakes, amphibians, birds, and almost all the mammals. This virus coevolved with the vertebrate kingdom, and it’s just part of what it is to be alive. It’s a virus that’s extraordinarily successful at persisting and passing itself down to the next generation not just in people but in any animal you’ve ever seen. So it’s something we just have to deal with.”***

FACTS: HPV viruses:

- 1) are transmitted from mothers to babies,²
- 2) are found in oral and genital mucosa of infants³ during their first three years of life,
- 3) have turned up in HPV DNA oral swabs in 87%⁴ of newborn babies and 57% of children,
- 4) have been found in hyperplastic tonsils and adenoids⁵ in Greek children,
- 5) have been found in the mouths⁶ of Japanese children aged 3–5 years.
- 6) Caesarean delivery⁷ is not “protective” against oral HPV infection. Half of the children HPV-positive infants in this study were born by caesarean delivery.

¹ DEBORAH ARRINDELL, THOMAS R. BROKER, NEAL A. HALSEY, GREGORY ZIMET, 2006. “PREVENTING HPV, EASY AS 1, 2, 3 SHOTS? ENSURING ACCESS TO THE NEW ANTI-CANCER VACCINES.” JANUARY 27, http://www.americanprogress.org/kf/hpv_event_transcript.pdf Pg 23.

² Puranen, M. et al. 1996. “Vertical transmission of human papillomavirus from infected mothers to their newborn babies and persistence of the virus into childhood.” *Am J Obstet Gynecol*, 174(2): 694–9, February. PMID: 8623809.

³ Rintala, M.A.M. et al. 2005. “High-risk types of human papillomavirus (HPV) DNA in oral and genital mucosa of infants during their first 3 years of life: experience from the Finnish HPV Family Study.” *Clin Infect Dis*, 41(12): 1728–33, December 15. PMID:16288396.

⁴ Syrjanen, S. et al. 2000. “Human papillomavirus infections in children: the potential role of maternal transmission.” *Crit Rev Oral Biol Med*, 11(2): 259–74. PMID: 12002819.

⁵ Mammas, I.N. et al. 2006. “Human papilloma virus in hyperplastic tonsillar and adenoid tissues in children.” *Pediatr Infect Dis J*, 25(12): 1158–62, December. PMID: 11174573.

⁶ Kohima, A. et al. 2003. “Human papillomaviruses in the normal oral cavity of children in Japan.” *Oral Oncol*, 39(8): 821–8, December. PMID: 13679205.

⁷ Summersgill, K.F. et al. 2001. “Human Papillomavirus in the oral cavities of children and adolescents.” *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*, 91(1): 62–9, January. PMID: 11174573.

- 7) There is a very large transfer of the virus amongst children⁸ and this study concluded that HPV-16 DNA in childrens' mouths was a transient event, and that the virus is most probably acquired from their peers.
- 8) In a 1994 study which found perinatal transmission of HP viruses 16 and 18 in 55% of babies, the authors⁹ cautioned that, ***"Information on the persistence of perinatally acquired human papillomavirus is required before rational vaccination programmes can be considered."***
- 9) Persistent HPV 16 and HPV 18 infection¹⁰ was found in infants in 1995, which led to those authors saying: ***"the observation that infection with high cancer risk genital HPVs may occur in early life and persist is of considerable importance for HPV vaccine strategies."***
- 10) In 1996¹¹ different researchers found the same thing, and listed studies which found HP16 viruses in children whose mothers ***did not*** have evidence of HP16.
- 11) Again, in 1998 researchers¹² said: ***"Thus the traditional view that cervical cancer associated HPV infections are primarily sexually transmitted needs to be re-assessed...These facts are pertinent to those developing prophylactic vaccines to prevent high-risk HPV infections and cervical carcinoma."***
- 12) And what do we read¹³ 2000? ***"The mode of in utero transmission remains unknown ... The understanding of viral transmission routes is important, particularly because several vaccination programs are being planned worldwide."***

You mean, they don't understand viral transmission routes? And that even before the Gardasil vaccine was even trialled, the vaccination programmes were planned? Interesting.

Do they know how many types of HPV viruses exist; and which cause cancer, and factor in all variables?

In 2000¹⁴, researchers took 33 skin samples from 13 individuals, found 20 previously described HPV viruses, and 30 completely novel virus types never before typed. A document on

⁸ Mant, C. et al. 2003. "Buccal exposure to human papillomavirus type 16 is a common yet transitory event of childhood." *J Med Virol*, 71(4): 593-8, December. PMID: 14556274.

⁹ Pakarian, F. et al. 1994. "Cancer associated human papillomaviruses: perinatal transmission and persistence." *Br J Obstet Gynaecol*. Jun;101(6):524-7. PMID: 8018641.

¹⁰ Cason, J. et al., 1995. "Perinatal infection and persistence of human papillomavirus types 16 and 18 in infants." *J Med Virol*, 47(3): 209-18, November. PMID: 8551271.

¹¹ Kaye, J.N. et al. 1996. "Human papillomavirus type 16 in infants: use of DNA sequence analyses to determine the source of infection." *J Gen Virol*. Jun;77 (Pt 6):1139-43. PMID: 8683198.
<http://vir.sgmjournals.org/cgi/reprint/77/6/1139.pdf>

¹² Cason, J. et al. 1998. "Transmission of cervical cancer-associated human papilloma viruses from mother to child." *Intervirology*, 41(4-5):213-8. PMID:10213899>

¹³ Syrjanen, S. et al. 2000. "Human papillomavirus infections in children: the potential role of maternal transmission." *Crit Rev Oral Biol Med*, 11(2): 259-74. PMID: 12002819.

¹⁴ Antonsson, A. et al. 2000. "The ubiquity and impressive genomic diversity of human skin papillomaviruses suggest a commensalic nature of these viruses." *J Virol*, 74(24): 11636-41, December. PMID: 11090162.

the FDA website¹⁵, shows that every time researchers have looked for new types in the past, they have found them. In this document, Dr Thomas Broker mentioned before, said: ***“So, we know there are a huge number of these viruses, perhaps millions, and every time they turned around to look at someone, they found a new type.”*** He went on to say: ***“We also know that in the developed world, herpes viruses which cause clinical problems are mainly a problem for people whose immune systems are suppressed somehow.”***

Cervical cancer is mainly a problem in people whose immune systems are suppressed somehow!!

What lifestyle influences operate in women who have a high HP viral load¹⁶ which their immune system won't get rid of? Do they smoke?¹⁷

A Pubmed search using the words “cervical cancer selenium” or “cervical cancer folic acid”, show studies like this one¹⁸ which found that: ***“... cervical dysplasia gradually decreased in the group supplemented with oral folate but remained unchanged in the group given the placebo.”***

Medical articles¹⁹ and newspaper articles²⁰ since the early 1990s have shown that smoking, and a diet low in crucial dietary micronutrients are two key factors which result in the development and progression of any cancer, including cervical cancer.

So why aren't you told about that?

Why are you not also told that: *“Although most women will at some time be infected with HPV, very few will progress to invasive disease.”*²¹ And why are researchers not concentrating on finding out why most people don't get cancer?

¹⁵ Minutes vaccine cell substrate meeting. <http://www.fda.gov/cber/minutes/0910evolv.txt>.

¹⁶ Song, S.H. et al. 2006. “Risk factors for the progression or persistence of untreated mild dysplasia of the uterine cervix.” *Int J. Gynecol Cancer*, 16(4): 1608–13, July–August. PMID: 16884373.

¹⁷ McIntyre-Seltman, K. et al. 2005. “Smoking Is a Risk Factor for Cervical Intraepithelial Neoplasia Grade 3 among Oncogenic Human Papillomavirus DNA-Positive Women with Equivocal or Mildly Abnormal Cytology.” *Cancer Epidemiol Biomarkers Prev*, 14(5): 1165–70, May. PMID: 15894667.

¹⁸ Ziegler, R.G. 1986. “Epidemiologic studies of vitamins and cancer of the lung, esophagus, and cervix.” *Adv Exp Med Biol*, 206: 11–26. PMID: 3591517.

¹⁹ Batieha, A.M. et al. 1993. “Serum micronutrients and the subsequent risk of cervical cancer in a population-based nested case-control study.” *Cancer Epidemiol Biomarkers Prev*, 2(4): 335–9, July–August. PMID: 8348056.

²⁰ *The Press*. 1995. “Diet and cervical cancer.” May 11. “... the association between low vitamin A intake and high risk of dysplasia was the strongest link found ... these results are consistent with previous studies ... The studies' key message is to increase the variety and intake of fruits, vegetables, wholegrain cereals and breads. This will increase dietary intake of fibre, the antioxidant vitamins betacarotene, vitamin E and vitamin C, folate and other protective substances found in these foods.”

When it comes to Gardasil®, the only information it is considered necessary for you to act on, is a belief is that potentially cancer causing HPV viruses are ONLY found in sexually active young people. And you don't want your daughter to get cancer, do you?

Will the vaccine work, you ask?

Merck says, "Yes", but you can see why you haven't been told this²²:

"We don't know, but I frankly do strongly suspect that when we do eradicate or minimize the HPV 16 and 18, that their very, very close relatives will fill in. Nature abhors the vacuum and these ecological niches are going to be vacant when HPV 16 and 18 and 6 and 11 are minimized, and I'm deeply concerned that there'll be backfill of those ecological niches by these very, very similar types. I think it's imperative to expand the coverage in the vaccines. We don't know, however, because the studies have never been done, whether a cocktail with 14 types would be equally effective against all 14 or whether they might actually conflict with each other. We simply don't know. We don't suspect that there's much cross protection of one type to any other even similar type. So far the evidence doesn't suggest that."

The information here is only the tip of the iceberg with regards to what you've not been told, about human papillomaviruses, and their possible effects in your child's life.

And I've not even started to show what you weren't told about the vaccine!

"Buyer" beware. Gardasil isn't "free". It will be paid for out of your taxes, so it would pay for you to fully investigate, before you consider any option to vaccinate. A full review of HPV viruses and Gardasil can be read in our new book, "***From One Prick To Another***" at www.beyondconformity.org.nz.

²¹ Woodman, C.B.J. et al. 2007. "The natural history of cervical HPV infection: unresolved issues." *Nature*, 7: 12. Reviews, Cancer, January. <http://www.nature.com/nrc/journal/v7/n1/pdf/nrc2050.pdf>

²² DEBORAH ARRINDELL, THOMAS R. BROKER, NEAL A. HALSEY, GREGORY ZIMET, 2006 "PREVENTING HPV, EASY AS 1, 2, 3 SHOTS? ENSURING ACCESS TO THE NEW ANTI-CANCER VACCINES." JANUARY 27, http://www.americanprogress.org/kf/hpv_event_transcript.pdf Pg 15.