

Title: **Are dental X-rays causing Alzheimer's disease?**
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Good afternoon. My name is Caroline Rodgers. I am a researcher and writer specializing in public health issues.

I am sure that everyone here would agree that Alzheimer's disease has turned the prospect of aging into a scary and uncertain future that will rob many of us of our memories, our dignity and even our lives. I am here because I envision a world in which Alzheimer's is once again a rarity and our senior years are a true Golden Age.

Last year my article proposing that dental X-rays are causing Alzheimer's disease was published in the Journal of Medical Hypotheses¹. I also made a poster presentation on the subject at the 2011 Alzheimer's Association International Conference².

The technical explanation of my hypothesis is:

Ionizing radiation from dental X-rays shortens the telomeres of microglia, which are critical to maintaining neuronal health. This reduces the lifespan of microglia, stranding neurons. Stranded neurons subsequently die, causing irreversible dementia.

More simply stated:

Head exposure to low-dose ionizing radiation is causing us to outlive the brain cells designed to support our neurons all lifelong.

NOTE: Dental X-rays are the only form of ionizing radiation that virtually all Americans are routinely exposed to at regular intervals throughout their entire lives, starting in early childhood. Although low-dose ionizing radiation amounts have been compared to background radiation exposure received during long airline flights, the amount of whole-body ionizing radiation received cannot be fairly compared to the amount beamed directly into the head.

It is not realistic to believe that decades of dental X-ray exposure would be without consequence for all people. The question is not, "Why should we consider whether dental X-rays are causing Alzheimer's," but rather, "Why didn't we think of this sooner?"

Hypothesis foundation:

- 1) Alzheimer's prevalence data
- 2) Population-based dental care information
- 3) Scientific studies¹⁻⁴

Dental care & dementia: A sampling of countries

INDIA

67% have never visited a dentist⁵

Dementia prevalence estimated at 1/5 -1/4 that of Europe's⁶

CHINA

30% to 43% adults have never visited a dentist⁷

Dementia prevalence about half of Europe's⁶

UNITED STATES

1% have never visited a dentist⁸

13% of people 65 and older have AD⁹

Let's test this hypothesis against the facts

FACT: The emergence of AD symptoms is delayed 10 or more years following the presence of AD brain pathology.

Microglial telomere shortening would have a delayed effect on neurons because it reduces microglial lifespan, not function.

FACT: AD mortality increased rapidly after 1979, making it the sixth leading cause of death by 2000.

Since AD symptoms are delayed by 10 or more years, it is worthwhile to look at health trends taking place in the decades before the surge in AD mortality. This was a time of major changes in the nation's dental health habits.

1940 it wasn't until the '40s that X-ray machines were common in America's dental offices¹⁰. However, nearly two decades later, in 1954 . . .

1957 only 37% had visited a dentist within the year, while 18% reported never visiting a dentist¹¹.

2008 59% had visited a dentist within the year, with only 1% never having visited a dentist⁸.

2010 The national average that had been to a dentist or dental clinic within the year was 69.7%⁹

FACT: The hippocampus is one of the first brain regions to suffer AD-related damage.

It contains both microglia and neural progenitor cells which —unlike other mature brain cells — keep dividing, making them more susceptible to radiation-induced damage.

FACT: Men die sooner than women following an AD diagnosis¹².

Older men have shorter telomeres than women the same age¹³, so they would have less time if their microglia telomeres were prematurely shortened.

FACT: Virtually all people with Down syndrome have AD brain pathology by age 40 – but there is a wide variance in the onset of dementia^{14,15}.

People with Down syndrome lose telomere length faster than the general population, but just like others, there is variation in newborn telomere length^{16,13}. Also, people with Down syndrome are subject to many genetic dental anomalies that could entail additional X-ray exposure.

FACT: AD prevalence is higher in urban areas¹⁷.

City dwellers make more dental visits¹⁸.

FACT: AD is increasing most rapidly in developing countries – especially Latin America⁶.

In the last few decades, many countries started providing free dental care to all citizens, such as Cuba in 1976, Venezuela in 1999 and Brazil in 2004,.

FACT: AD does not respond to anti-inflammatory or cholesterol-lowering drugs — even though AD is associated with brain inflammation and high cholesterol^{19,20}.

Neither treatment can help neurons that have lost their support system.

FACT: Mentally stimulating activities initially delay AD, yet ultimately accelerate it²¹.

Additional brain growth would eventually overwhelm microglia struggling to support existing neuronal networks.

If dental X-rays are causing Alzheimer's, it raises new questions and concerns, such as:

- ✓ Could diligent dental care explain the increase in non-familial early-onset AD?
- ✓ At what price, a smile: What are the long-term risks for orthodontia patients exposed to cone-beam CT scanners that create 3-D images – at much higher radiation levels?
- ✓ Could head X-rays after sports injuries contribute to early-onset dementia?
- ✓ If dental X-rays pose risks, could brain imaging utilizing ionizing radiation to monitor AD accelerate disease progression?
- ✓ Do plateaus in declining cognition relate to intervals between X-ray exposures?
- ✓ Could the ratio of dental professionals to population be used to create accurate algorithms to predict dementia trends?

What are the prospects for Alzheimer's prevention, treatment or cure?

If dental X-rays are causing Alzheimer's disease, future cases can be decreased by eliminating or strictly limiting dental X-rays. Dentists can also turn to alternative imaging technologies already developed, yet not in common use. Interventions for people already exposed to dental X-rays yet without AD symptoms could include developing ways to safely transplant self-donated bone marrow or dental stem cells to replenish microglia populations. Other possibilities would be to develop ways to preserve or even lengthen microglia telomere length or to selectively remove permanently senescent microglia cells to stimulate replacement microglia that would actively provide neuroprotection. Sadly, there is no evident cure for people who have already lost their microglial support system to the point where enough neurons have died to cause symptoms.

Closing comments

I don't know the answer to the questions I have raised in this short talk. I do, however, know that we have to look in new places with open minds to solve the puzzle of AD's emergence as a major killer and to restore health, dignity and luster to our "golden years."

REFERENCES

- 1) Rodgers, CC. Dental X-ray exposure and Alzheimer's disease: a hypothetical etiological association. *Med Hypotheses*. 2011;77(1):29-34. Epub 2011 Mar 31.
- 2) Alzheimer's Association International Conference (AAIC) 2011. (PA-382)
- 3) Streit WJ, Braak H, Xue QS, et al. Dystrophic (senescent) rather than activated microglial cells are associated with tau pathology and likely precede neurodegeneration in Alzheimer's disease. *Acta Neuropathol*. 2009;118(4):475-85. Epub 2009 Jun 10.
- 4) Xue QS, Streit WJ. Microglial pathology in Down syndrome. *Acta Neuropathol*. 2011;122(4):455-66. Epub 2011 Aug 17..
- 5) Kalm M, Lannering B, et al. Irradiation-induced loss of microglia in the young brain. *J Neuroimmunol*. 2009;206(1-2):70-5. Epub 2008 Dec 13.

- 6) Grodstein F, van Oijen M, Irizarry MC, et al. Shorter telomeres may mark early risk of dementia: preliminary analysis of 62 participants from the nurses' health study. *PLoS One*. 2008;3(2):e.1590.
- 7) Indo-Asian News Service. 67 percent Indians have never visited a dentist: Survey. Aug 22 2009. http://www.thaindian.com/newsportal/health1/67-percent-indians-have-never-visited-a-dentist-survey_100236506.html Accessed Jul 9 2010.
- 8) Llibre Rodriguez JJ, Ferri CP, Acosta D, et al. Prevalence of dementia in Latin America, India, and China: a population-based cross-sectional survey. *Lancet*. 2008;372(9637):464-74. Epub 2008 Jul 25.
- 9) Zhu L, Peterson PE, Wang HY, et al. Oral health knowledge, attitudes and behaviour of adults in China. *Int Dent J* 2005;55(4):231-41.
- 10) The Kaiser Family Foundation, statehealthfacts.org. Data source: The National Oral Health Surveillance System, The Centers for Disease Control and Prevention (CDC), based on the Behavioral Risk Factor Surveillance System (BRFSS). <http://www.statehealthfacts.org/comparebar.jsp?ind=108&cat=2&sub=30&yr=138&typ=2&sort=a&o=a> Accessed 1-20-2012.
- 11) S Pleis JR, Lucas JW, Ward BW. Summary health statistics for U.S. adults: National Health Interview Survey, 2008. *Vital Health Stat* . 2009;10(242):1-157.
- 12) Alzheimer's Association. 2011 Alzheimer's Disease Facts and Figures. http://www.alz.org/downloads/Facts_Figures_2011.pdf Accessed Jan 13 2012.
- 13) Frommer HH. The History of Dental Radiology. *Tex Dent J*. 2002;119(5):416-21, 423.
- 14) U.S. National Health Survey. Dental care interval and frequency of visits. United States July 1957-June 1959. Washington, U.S. Dept. of Health, Education, and Welfare, Public Health Services, Division of Public Health Methods, 1960 .
- 15) Larson EB, Shadlen MF, Wang LI, et al. Survival after Initial Diagnosis of Alzheimer Disease. *Ann Intern Med* 2004;140(7):501-9 .
- 16) Benetos A, Okuda K, Lajemi M, et al. Telomere length as an indicator of biological aging: the gender effect and relation with pulse pressure and pulse wave velocity. *Hypertension* 2001;37(2 Part 2):381-5.
- 17) Karlinsky H. Alzheimer's disease in Down's syndrome. A review. *J Am Geriatr Soc*. 1986;34(10):728-34.
- 18) Stanton LR, Coetsee RH. Down's syndrome and dementia. *Advances in Psychiatric Treatment*. 2004;10:50-8. <http://apt.rcpsych.org/content/10/1/50.full.pdf+html> Accessed Jan 15 2012.
- 19) de Arruda Cardoso Smith M, Borsatto-Galera B, Feller RI, et al. Telomeres on chromosome 21 and aging in lymphocytes and gingival fibroblasts from individuals with Down syndrome. *J Oral Sci*. 2004;46(3):171-7.
- 20) World Health Organization. Mental Health and Substance Abuse. Facts and Figures, Alzheimer's Disease: the Brain Killer. http://www.searo.who.int/en/Section1174/Section1199/Section1567/Section1823_8066.htm Last update Aug 18 2006. Accessed Jul 7 2010.
- 21) Fos P, Hutchison L. (2003) The State of Rural Oral Health: A literature review. Rural Healthy People 2010: A companion document to Healthy People 2010. Vol. 2. College Station, TX: The Texas A&M University System Health Science Center, School of Rural public Health, Southwest Rural Health Research Center.
- 22) McGuinness B, O'Hare J, Craig D, et al. Statins for the treatment of dementia. *Cochrane Database Syst Rev*.2010: Aug 4;(8):CD007514.
- 23) ADAPT Research Group, Martin BK, Szekeley C, et al. Cognitive function over time in the Alzheimer's Disease Anti-inflammatory Prevention Trial (ADAPT): results of a randomized, controlled trial of naproxen and celecoxib. *Arch Neurol* 2008;65(7):896-905. Epub 2008 May 12.
- 24) Wilson RS, Barnes LL, Aggarwal NT, et al. Cognitive activity and the cognitive morbidity of Alzheimer disease. *Neurology* 2010;75(11):990-6. Epub 2010 Sep 1.