HAWAIʻI PACIFIC HEALTH | KAPIʻOLANI PALI MOMI STRAUB WILCOX

Tuesday, February 4, 2019 at 8:30 AM
Conference Room 329

House Committee on Health

To: Representative John Mizuno, Chair
Representative Bertrand Kobayashi, Vice Chair

From: Michael Robinson
Vice President, Government Relations & Community Affairs

Re: Testimony in Support of HB 2507
Relating to Health

My name is Michael Robinson, Vice President, Government Relations & Community Affairs at Hawaiʻi Pacific Health. Hawaiʻi Pacific Health is a not-for-profit health care system comprised of its four medical centers – Kapiʻolani, Pali Momi, Straub and Wilcox and over 70 locations statewide with a mission of creating a healthier Hawaiʻi.

I write in **support of HB 2507** which prohibits the sale or furnishing to tobacco and tobacco products, including electronic smoking devices, to persons under the age of 25.

Tobacco use remains the leading cause of preventable disease and death in the United States and in Hawaiʻi. Tobacco use is a serious public health problem in terms of the human suffering and loss of life it causes, as well as the financial burden it imposes on society and our healthcare system. All tobacco products, including electronic smoking devices, severely contribute to the injurious health burdens impacting our state. Annually, $526,000,000 in health care costs are directly attributed to smoking in the State.

Smoking continues to be a serious problem despite efforts to reverse the smoking epidemic. Many are turning to the use of electronic smoking devices as a substitute to cigarettes. The popularity of electronic cigarettes among youth is concerning, as these products contain nicotine. Research conducted by the University of Hawaii Cancer Center has shown that the use of electronic smoking devices among school-age children has risen dramatically in the last few years.

E-cigarette use or vaping among youth and young adults has become a national public health concern. E-cigarettes are now the most popularly used tobacco product among youth and young adults, surpassing cigarettes. While smoking rates in Hawaii have decreased through the years, electronic smoking device (ESD) use has rapidly increased, threatening significant public health gains through our Tobacco 21 law and tobacco youth

access laws. This is particularly concerning because e-cigarettes provide a new way to deliver the addictive drug nicotine. No matter how it is delivered, nicotine exposure can lead to addiction and harm the developing brain. Studies are also finding that ESDs can lead to smoking cigarettes for new users, including kids. Marketing strategies by the tobacco industry and electronic smoking device industry have significantly increased the introduction and marketing of flavored non-cigarette tobacco products, especially ESDs. Increasing the minimum age of persons who may purchase tobacco products and electronic smoking devices to 25 ensures that a greater percentage of our population would be saved from the harmful effects of smoking and nicotine addiction.

Thank you for the opportunity to testify.

HB-2507
Submitted on: 2/3/2020 10:23:11 AM
Testimony for HLT on 2/4/2020 8:30:00 AM

Submitted By	Organization	Testifier Position	Present at Hearing
Michael Zehner	Hawaii Smokers Alliance	Oppose	No

Comments:

If you are going to raise the age, then make it equal to the voting age just to be fair.

HPCA
HAWAI'I PRIMARY CARE ASSOCIATION

Testimony to the House Committee on Health
Tuesday, February 4, 2020; 8:30 a.m.
State Capitol, Conference Room 329

RE: HOUSE BILL NO. 2507, RELATING TO HEALTH.

Chair Mizuno, Vice Chair Kobayashi, and Members of the Joint Committee:

The Hawaii Primary Care Association (HPCA) is a 501(c)(3) organization established to advocate for, expand access to, and sustain high quality care through the statewide network of Community Health Centers throughout the State of Hawaii. The HPCA **SUPPORTS** House Bill No. 2507, RELATING TO HEALTH.

The bill, as received by your Committee, would:

(1) Raise the age to purchase or possess a tobacco product or electronic smoking device from twenty-one to twenty-five years of age;

(2) Raise the age in which a seller of tobacco products or electronic smoking devices must check purchaser identification from twenty-seven to thirty-five years of age; and

(3) Increases the fine for violators under the age of twenty-five years of age from $10 to $50 for the first offense, and subsequent offenses from $50 to $100.

By way of background, the HPCA represents Hawaii Federally-Qualified Health Centers (FQHCs). FQHCs provide desperately needed medical services at the frontlines in rural and underserved communities. Long considered champions for creating a more sustainable, integrated, and wellness-oriented system of health, FQHCs provide a more efficient, more effective and more comprehensive system of healthcare.

FQHCs have long seen first-hand how tobacco has literally destroyed the lives of our patients and their families. Because of the ubiquity of cigarettes, chewing tobacco, and now electronic smoking devices, the impacts of tobacco affect our citizenry on a generational basis with people experimenting at even earlier ages.

ADD 1003 BISHOP STREET PAUAHI TOWER SUITE 1810 HONOLULU, HI 96813 TEL 808.536.8442 WEB HAWAIIPCA.NET

It is for this reason that the HPCA joins the American Cancer Society of Hawaii, the Hawaii Public Health Institute, and other advocates in strong support of all efforts to rid the marketplace of these products.

Thank you for the opportunity to testify. Should you have any questions, please do not hesitate to contact Public Affairs and Policy Director Erik K. Abe at 536-8442, or eabe@hawaiipca.net.

Date: February 2, 2020

To: Representative John M. Mizuno, Chair
Representative Bertrand Kobayashi, Vice Chair
Members of the Health Committee

Re: Support for HB 2507, Relating to Health

Hrg: February 4, 2020 at 8:30 am at Conference Room 329

The Coalition for a Tobacco-Free Hawai'i, a program of the Hawai'i Public Health Institute[i] **Supports HB 2507,** which would raise the minimum age to purchase tobacco products to 25.

Raising the age of sale of tobacco products to 25 will prevent the initiation of tobacco use among youth and young adults.
According to the US Surgeon General's report in 2012, 99% of all adult smokers start smoking before the age of 26, and progression from occasional to daily smoking usually occurs by age 26.[ii] The data strongly suggests that if someone is not a regular tobacco user by 25 years of age, it is highly unlikely they will become one. Brain development continues until age 25, in particular the parts of the brain responsible for decision-making and impulse control. Delaying the age that youth and young adults begin using tobacco will reduce the risk that they will become regular smokers as they get older, leading to lower prevalence rates and saving millions of dollars in health care costs.[iii] Risk for smoking-caused diseases increases depending on how long the person smokes, and smokers who start at a young age are among the heaviest users.[iv] Tobacco use causes $132 billion in health care costs in the US each year[v], including $526 million the State of Hawai'i.[vi] The measure is expected to reduce these health risks and costs.

Studies of tobacco industry documents also suggest that young adults are targeted in tobacco promotions, such as marketing tobacco in bar settings and with alcohol consumption. These tactics can encourage young adults to start smoking or transition from occasional to regular smokers.[vii]

Institute of Medicine Report
A scientific report issued by the Institute of Medicine (IOM) concludes that increasing the age of sale for tobacco products to 25 will have a positive impact on public health. Raising the minimum legal age to purchase tobacco products would add 5.7 million more years of life to the next generation of American adults, 330,000 fewer premature

deaths, and 59,000 fewer deaths from lung cancer by 2100.[viii] Predicted smoking prevalence would decrease by 16% with the minimum age set at 25.

The Coalition supports HB 2507 and asks the committee to pass this measure. Thank you for the opportunity to provide testimony.

Mahalo,

Jessica Yamauchi, MA
Executive Director

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[i] The Coalition for a Tobacco-Free Hawai'i (Coalition) is a program of the Hawai'i Public Health Institute (HIPHI) that is dedicated to reducing tobacco use through education, policy, and advocacy. With more than two decades of history in Hawai'i, the Coalition has led several campaigns on enacting smoke-free environments, including being the first state in the nation to prohibit the sale of tobacco and electronic smoking devices to purchasers under 21 years of age.

The Hawai'i Public Health Institute is a hub for building healthy communities, providing issue-based advocacy, education, and technical assistance through partnerships with government, academia, foundations, business, and community-based organizations.

[ii] U.S. Department of Health and Human Services. Preventing Tobacco Use Among Youth and Young Adults: A Report of the Surgeon General. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, 2012.

[iii] https://www.tobaccofreekids.org/assets/factsheets/0127.pdf

[iv] https://www.tobaccofreekids.org/assets/factsheets/0127.pdf

[v] Campaign for Tobacco-Free Kids , Toll of Tobacco in the USA

[vi] Campaign for Tobacco-Free Kids, The Toll of Tobacco in Hawaii.

[vii] Ling, P. M., & Glantz, S. A. (2002). Why and how the tobacco industry sells cigarettes to young adults: evidence from industry documents. American journal of public health, 92(6), 908–916. doi:10.2105/ajph.92.6.908

[viii] Committee on the Public Health Implications of Raising the Minimum Age for Purchasing Tobacco Products; Board on Population Health and Public Health Practice; Institute of Medicine; Bonnie RJ, Stratton K, Kwan LY, editors. Public Health Implications of Raising the Minimum Age of Legal Access to Tobacco Products. Washington (DC): National Academies Press (US); 2015 Jul 23. Available from: https://www.ncbi.nlm.nih.gov/books/NBK310412/ doi: 10.17226/18997

HAWAII FOOD INDUSTRY ASSOCIATION

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TO:
Committee on Health
Rep. John M. Mizuno, Chair
Rep. Bertrand Kobayashi, Vice Chair

FROM: HAWAII FOOD INDUSTRY ASSOCIATION
Lauren Zirbel, Executive Director

DATE: February 4, 2020
TIME: 8:30am
PLACE: Conference Room 329

RE: HB2507 Relating to Health

Position: Oppose

The Hawaii Food Industry Association is comprised of two hundred member companies representing retailers, suppliers, producers, and distributors of food and beverage related products in the State of Hawaii.

State and Federal laws already prohibit the purchase of tobacco products by those under 21 years of age. There is no legal precedent at either the state or federal level for prohibiting adults under the age of 25 from using certain products. Tobacco products are Federally regulated. Creating state laws that conflict with Federal laws on these products is unnecessary and will create enforcement difficulties. We ask that this measure be held and we thank you for the opportunity to testify.

HB-2507
Submitted on: 2/1/2020 11:34:35 AM
Testimony for HLT on 2/4/2020 8:30:00 AM

Submitted By	Organization	Testifier Position	Present at Hearing
Chris Cooper	Individual	Oppose	No

Comments:

E-vapor flavor doesn't harm anyone. Don't ban the flavors people like.

HB-2507
Submitted on: 2/1/2020 11:47:16 AM
Testimony for HLT on 2/4/2020 8:30:00 AM

Submitted By	Organization	Testifier Position	Present at Hearing
Cindy Nettles	Individual	Oppose	No

Comments:

HB-2507
Submitted on: 2/1/2020 1:46:02 PM
Testimony for HLT on 2/4/2020 8:30:00 AM

Submitted By	Organization	Testifier Position	Present at Hearing
Alex Abe	Individual	Oppose	No

Comments:

I'm Alex and I srongly oppose this bill!

HB-2507
Submitted on: 2/1/2020 5:50:41 PM
Testimony for HLT on 2/4/2020 8:30:00 AM

Submitted By	Organization	Testifier Position	Present at Hearing
Kathy Kim	Individual	Oppose	No

Comments:

HB-2507
Submitted on: 2/2/2020 10:26:13 AM
Testimony for HLT on 2/4/2020 8:30:00 AM

Submitted By	Organization	Testifier Position	Present at Hearing
Anthony Orozco	Individual	Oppose	No

Comments:

HB-2507
Submitted on: 2/2/2020 1:23:30 PM
Testimony for HLT on 2/4/2020 8:30:00 AM

Submitted By	Organization	Testifier Position	Present at Hearing
Kimo Cruz	Individual	Oppose	Yes

Comments:

HB-2507
Submitted on: 2/2/2020 10:46:32 PM
Testimony for HLT on 2/4/2020 8:30:00 AM

Submitted By	Organization	Testifier Position	Present at Hearing
Bryan Mih	Individual	Support	No

Comments:

Dear Representatives:

As a pediatrician and medical director of the Kapi'olani Smokefree Families Program, I strongly support this bill.

We have known of the adverse effects of tobacco smoking for a long time, and that almost all smokers start their use when they are young. The brain has been found to continue to develop until the mid-20s. Nicotine is a highly addictive drug that impacts the developing brain.

The U.S Surgeon General's Report in 2016 reported that nicotine has the following effects:

- Adversely affects brain development until mid-20s.
- Disrupts the development of brain circuits that control attention and learning
- Makes it harder to control impulses.
- Leads to permanent lowering of impulse control
- Learning and cognitive deficits
- Mood disorders
- Affects the development of the brain's reward system, making the brain more susceptible to addiction to other drugs such as cocaine and methamphetamine

A ban on tobacco and nicotine use until age 25 years is reasonable to reduce the adverse health effects. Big Tobacco is a multi-billion-dollar industry that seeks to trade people's health for the industry's profit margins.

Once young people are addicted to nicotine, it is extremely difficult to quit. By reducing access to these products in Hawaii, we have the chance to improve the health of many, especially of our young people.

On behalf of the health and well-being of the people of Hawaii, I urge you to support this bill.

Mahalo for your consideration and support of this important measure.

Sincerely,

Bryan Mih, MD, MPH, FAAP

Pediatrician

HB-2507
Submitted on: 2/3/2020 2:46:31 AM
Testimony for HLT on 2/4/2020 8:30:00 AM

Submitted By	Organization	Testifier Position	Present at Hearing
Jennifer Azuma Chrupalyk	Individual	Support	No

Comments:

LATE

It is little, my lad, but it's terribly bad,
The vile old Cigarette.
And without any joking, there's danger in smoking
The vile old Cigarette
It adds to expenses and lessens the senses,
It only brings grief and regret;
Then let us endeavor to shun it forever,
The vile old Cigarette

(Anti-Cigarette League (1912))

That was written over 100 years ago! What happened?

"The cigarette today is the most vilified product available legally in the United States, blamed for causing the premature deaths of more than 400,000 Americans a year, banned from most public buildings, besieged in the courts, and subject to increasing restrictions on advertising, promotion and sales. Nonetheless, one out of four adults continues to smoke, a figure that has remained virtually unchanged since 1989."
(Wall Street Journal, June 23, 1997)

Again, what happened?

State Cigarette Prohibition Laws

(In order of adoption)

1. Washington state: Sale and manufacture of cigarettes banned in 1893; repealed 1895; reenacted 1907; sale manufacture and possession banned 1909; repealed 1911.

2. North Dakota: Sale banned 1895; repealed 1925.

3. Iowa: Sale and manufacture banned 1896; repealed 1921.

4. Tennessee: sale and giving away of cigarettes banned 1897; repealed 1919

(The U.S. Supreme court upheld this ban in Austin v State of Tennessee, 179 US343 (19 Nov 1900) and resolved the issue of whether commerce clause preempts state cigarette ban – it did NOT preempt the ban.)

5. Oklahoma: sale and giving away of cigarettes banned 1901; repealed 1919.
6. Indiana: Sale, manufacture, and possession banned 1905; repealed 1909.
7. Wisconsin: Sale, manufacture, and giving away of cigarettes banned 1905; repealed 1915.
8. Arkansas: Sale and manufacture banned 1907; repealed 1921.
9. Illinois: Sale and manufacture banned 1907; law declared unconstitutional by Illinois Supreme Court six months after enactment, but formally repealed until 1967.
10. Nebraska: Sale, manufacture, and giving away of cigarettes banned 1909; repealed 1919.
11. Kansas: Sale banned 1909; law amended to ban advertising and possession as well as sale in 1917; repealed 1927.
12. Minnesota: Sale and manufacture banned 1909; repealed 1913.
13. South Dakota: Sale, manufacture and giving away of cigarettes banned 1909; repealed 1917.
14. Idaho: Sale of cigarettes banned and then legalized by the same session of the legislature, 1921.
15. Utah: sale and advertising banned, 1921; repealed 1923.

What happened? Why did we cave in to the Tobacco Industry?

"It has been said that the true measure of any society can be found in how it treats its most vulnerable members"

I would say that some of the most vulnerable and victimized members of our current society are those that are affected by the scourge of the tobacco industry.

We have a great concern about teenage brains being affected by nicotine either from combustible cigarettes or vaping (which may have even higher nicotine than cigarettes) and is currently causing an epidemic in teenagers.

However, it is **pregnant women, their unborn(fetuses) and newborn babies** (SIDS deaths from nicotine) that are the MOST vulnerable and largely unacknowledged victims of the tobacco industry. (See Health Risk to "Cigarette Babies" is Neglected; Theodore Slotkin)

We know full well how horrible combustible cigarettes are. Robert Proctor, Ph.D. has said that the cigarette is the deadliest object of modern civilization.

We know the many ways it kills people, the many ways it harms and disables people, and we know how addictive and habituating cigarettes are.

We also know that as legislators **we have the power** to stop this monstrous habit.

Our country has recently shown some progress, but it is not enough. We no longer allow children to smoke. We first had a federal ban on selling cigarettes to minors (age 18) in 2009. Our state, Hawaii, was the first, effective 2016, to make 21 the minimum age of sale of combustible cigarettes **and** vaping devices. Other states followed, and the federal government recently enacted the 21 age of minimum sale for both smoking and vaping at the end of 2019.

My hope is that our state will be the first to raise the minimum age of both smoking and vaping to 25 as proposed in this bill.

The most vulnerable and innocent population of those damaged by nicotine in cigarette smoke or in nicotine by vaping are fetuses and newborn babies.

Of importance is that Hawaii was the first state to ban the pesticide chlorpyrifos which causes severe damage to fetal brains. A neuroscientist at Duke University,

Theodore Slotkin has shown that nicotine and chlorpyrifos have similar severe effects on the brain alone or what is worse, in combination.

A report of his work states:

"The medical community, government have neglected unequivocal scientific evidence that nicotine from maternal smoking causes possibly 100,000 fetal deaths each year as well as massive numbers of crib deaths, according to a Duke University Medical Center pharmacologist (Dr. Theodore Slotkin).

Also neglected are the severe neurological problems in "cigarette babies" of smoking pregnant women.

This neglect comes despite the widespread, chronic use of nicotine by one-fourth of pregnant women *(it is fortunately much less in Hawaii but still a serious problem that needs to be addressed).*

"Animal studies have revealed that nicotine, and hence smoking during pregnancy, inflicts serious damage on the fetus even at nicotine levels too low to cause the accepted sign of damage – low birth weight, said Slotkin."

"Maternal smoking during pregnancy kills between tens of thousands and possibly over a hundred thousand babies each year in utero" he said in an interview. "It also results in tens of thousands of admissions to intensive care units after birth and kills or brain-damages more during the birth process. Smoking is also responsible for one-third to two-thirds of all cases of Sudden Infant Death Syndrome (SIDS)."

"And none of these figures takes into account the enormous increase in learning disabilities, attention deficit and hyperactivity and other behavioral problems (*e.g. autism spectrum disorders)* that we know are part of the of the outcome of maternal smoking."

"To me, the denouement of our neglect of cigarette smoking as a source of damage to children came during the recent Congressional hearings on tobacco company policies and the potential for the FDA to regulate cigarettes as a nicotine delivery device *(it should be noted that the most popular vaping devices deliver a higher dose to the mother and fetus than combustible cigarettes*) Not once in those hearings did the issue of maternal smoking or children's health get raised.

And yet to me and to other people in the field, **this is potentially the most damaging event in our society caused by tobacco abuse."**

Slotkin hopes the media will give tobacco its proper attention as a substance hazardous to children, both unborn and born.

"Given that this is not a case where someone is simply damaging their own bodies and is aware of it, but instead is damaging someone else's entire future, I fail to understand why there aren't articles, headlines, magazine covers, or presidential statements targeting cigarette smoking *(and vaping)* in pregnancy. It's something that we stand a reasonable chance of influencing with publicity."

Slotkin has also published a paper entitled "Developmental Cholinotoxicants: Nicotine and Chlorpyrifos" where he showed the mechanism and extent of brain damage in fetuses from these two chemicals is similar.

We were the first state to have banned chlorpyrifos to protect babies and children's brains, but the number of unborn and born babies damaged by nicotine is likely to be massively greater than the number damaged by chlorpyrifos because of the continued intermittent exposure day in and day out from smoking (or vaping). The effects are as if the mother is spraying her fetuses' brain with a pesticide 20 times per day!

We cannot ban cigarettes just for pregnant women, or for all women of childbearing age. We must ban cigarettes for all individuals in the age range of procreation and childbearing, and eventually for all ages of addicted lifelong smokers.

We can get a start by taking the Institute of Medicines recommendation to raise the minimum age of sale of cigarettes to 25. We could be a beacon for the rest of the country. We were the first to go to 21. We were the first to recognize and ban the related neurotoxicant chlorpyrifos. We should be the first to protect the developing brains of those in their early twenties **and** the pregnant women and their babies of women in this age group which often has unintended and unrecognized pregnancies. Under 25 is the age when the first child is often born, and the age when health care during pregnancy may not be a priority because of socioeconomic factors.

How can young men insist on their "right to smoke" when that nonexistent "right" results in the deaths and disability of thousands of unborn and born babies and often death and disability to the mothers as well?

As was cited in the New England Journal article on January 24, 2013: "Because smoking has become a stigmatized behavior concentrated among persons of low social status, it risks becoming invisible to those who set health policies and *research* priorities. Yet, the need for greater attention to the policies known to reduce the prevalence of smoking *(such as raising the minimum age of sale further to 21 and hopefully now to 25)* remains urgent. As former Australian Health Minister Nicola Roton has said, "**We are killing people by not acting.** *NEJM, January 24, 2013"*

Let us help stop this needless slaughter of the innocent from prenatal exposure to nicotine from either combustible cigarettes or vaping, or too often, both. Stop mothers from finding their babies dead in their cribs from their smoking during their pregnancy. Stop the madness of allowing cigarettes and vaping devices that provide even higher levels of nicotine to be sold in our state.

A recent paper (2015) by Melissa Suter and colleagues asked, "Is There Evidence of Potential Harm of Electronic Cigarette Use in Pregnancy?"

They stated, "As the prevalence and incident use of electronic cigarettes continues to increase among reproductive age women and understanding of their risks during pregnancy becomes a pressing need in the public health arena". After reviewing the recent research, they conclude "Based on the evidence currently available, we summarily conclude that NO amount of nicotine is known to be safe during pregnancy."

We have sadly become complacent as our poorer brothers and sisters, and their sons and daughters continue to die, to suffer lifelong disabilities, and to suffer limited lives or early deaths.

We must stop the "Merchants of Death". In the foreword to the book of that name written in 1988 by Larry White about the American tobacco industry, C. Everett Koop, the U.S. Surgeon General praises the book for helping America to reach its goal of eradicating the cigarette by the year 2000. Sadly, in 1998 the Master Settlement Agreement, while reaping a windfall of cash for the states,

abandoned the goal of eradication and locked the states into a "profit-sharing" agreement that ensured that Big Tobacco and cigarettes would not only survive but thrive, and it is still thriving and people are still becoming addicted and dying.

We must stop profit-sharing as a state with the "merchants of death" and stop becoming as states, through our settlement monies and even worse our tobacco excise taxes merchants of death ourselves.

We can take the next step as a state and take the Institutes of Medicines suggestion and raise the minimum age of sale of combustible cigarettes to 25 and acknowledge the advice of others to include nicotine vaping devices. We must stop not only the old "merchants of death" through combustible cigarettes, but preemptively stop the new "merchants of death" (the vaping industry) before they kill more babies and mothers under the guise of protecting them. It should be noted that the glossy full-page ads for vaping devices, while warning that these devices contain nicotine an addicting substance do NOT have any warnings for unsuspecting pregnant women who might think they are safe.

Income inequality and social justice

Our House majority caucus wants to address income inequality this session. It should be noted that currently cigarettes are predominantly used by the lower economic strata of our society in our state. Those groups need all the assistance they can reasonably receive to succeed. They should not have to tolerate the brain and other organ damage to all the children of smoking mothers that we know is inevitably occurring.

My father and many of his generation were chain smokers, and he at times smoked two to three packs per day. Fortunately, he stopped shortly after he reached the age of forty. The New England Journal of Medicine articles on smoking in the January 13, 2013 issue point out convincingly that those individuals who quit by the age of 30-35 had an almost normal life expectancy, and those who quit between 35 and 44 lost only about one year of life expectancy compared to the ten years lost by a life-long heavy smoker.

Thus, a large group of current smokers can be saved if we make it difficult for them to buy cigarettes. Unfortunately, if they substitute vaping for smoking, the

nicotine alone will continue to kill them, albeit at slower rates. They would still incur the unneeded costs of that vaping.

My father quit smoking cold turkey in his early forties shortly after the birth of his seventh child and he is now 101 years old with no lethal health problems and no lung or brain disabilities. My mother, who smoked much less heavily, but whose vaping persisted into her sixties, eventually died of smoking caused emphysema at the age of 81.

At current levels of almost $10.00 per pack of cigarettes in Hawaii (and there is pressure to increase that cost even higher) a pack per day smoker would spend $3600. per year. I suspect their few chain smokers any more as three packs a day would cost over $10,000. per year. If those pack-a-day smokers stop, they would get to keep all that money tax free. For those in our minimum age brackets that is a huge sum, and one that we as a society are very unlikely to grant them otherwise.

The excise tax collected on cigarettes by the state of Hawaii in 2019 was $112,500,000. Using the figure of $3.20 of excise tax per pack the total cost of cigarettes to our adult Hawaii smokers was $351,562,000. That is a lot of money that would stay with those smokers if they no longer smoked. If those figures did not decrease (and they would likely decrease unless we succumbed to punitively raising the excise taxes even higher) our state would have to make up most of that $112 million from other sources, and that is why I am proposing that we consider starting or joining a lottery to make up that amount which currently supports important programs as well as providing $70 million to our general fund (but only a few million to smoking cessation efforts). It would also provide our small retailers with a replacement product to sell to replace cigarettes (a product that while considered regressive is less regressive in my view than cigarette excise taxes, and also a product that does not kill people and make the retailers "merchants of death). The Master Tobacco Settlement of 1998 money is based on national cigarette sale levels and would decrease very little from the current figure of about $30 million dollars even if all cigarette sales ceased in Hawaii.

Smoking related health care costs were in 2019 an estimated $526 million per year. If smoking were ended in Hawaii that figure would go down slowly due to delayed cancer and other diseases related to past smoking. While the rate of lung

cancer decreased by a record more than 2% last year if should be noted that lung cancer is still the most common type of cancer and in Hawaii that almost is almost all caused by cigarettes. If vaping replaced smoking it would have its own large health costs, albeit less than the costs of continued smoking.

Smoking related losses in productivity were estimated to be $387.3 million per year in 2019 but that figure would decrease over time, lacking new smokers. Heart attacks, strokes and smoking related infections would be considerably less with time, as long as vaping of nicotine, which can cause heart attacks, strokes, and loss of limbs, was also banned.

Ending smoking would be significantly beneficial economically and of course would be hugely significant to the long-term health of state residents. Everyone would of course die eventually, but far fewer would die horrible deaths from lung and other smoking related cancers or die gasping for breath from COPD.

Our teenager and young adult brains would be protected from smoking and hopefully from vaping which would in the absence of smoking be of no benefit.

Pregnant women who might otherwise smoke or vape would be protected, as would the brains of ALL their fetuses. The fraction of SIDS (or SUID) deaths attributed to smoking would disappear. A paper last year confirmed that over a quarter of all SIDS deaths (deaths often long after normal births) were due to brain injury caused by nicotine in the prenatal period. I contacted the principal author of that paper and she confirmed that the damage was from nicotine and although the nicotine in her study came from combustible cigarettes it is likely that the nicotine from vaping would likely continue to cause or possibly even increase the number of SIDS deaths due to the higher nicotine content in vaping devices.

We have a suicide problem in our state. But smoking is essentially slow suicide and may contribute to problems that would precipitate a fast suicide. In my view, if we don't act as legislators to stop this curse, we are accessories to those deaths.

Stop the "merchants of death". Let us finally put a stake in the heart of the tobacco industry and make Hawaii a beacon of hope for the rest of the nation and the world.

What are the objections to this course?

We have those who claim that ending smoking is infringing on their civil liberties. No one has a constitutional right to smoke, and they have no right in my view to a deadly privilege that puts the rest of society at risk.

To those who say that bans do not work, that there would be massive smoking as in prohibition I say it is highly unlikely in our remote state. The agencies responsible for controlling smoking tell me that there is little smuggling now and they would expect little smuggling to develop.

What is long forgotten is that at the end of the 19th and beginning of the 20th century there were 15 states that totally banned cigarettes and those bans were allowed by state courts and even the United States Supreme Court. There is no constitutional right to smoke (or to vape). Those bans failed because of massive efforts and money from the tobacco industry which gradually overcame them. Even in most states that did not ban them the minimum age of sale was often 21 until, again, there was massive spending and bribing of legislatures. Sadly, that massive spending and bribery continues today.

When we look at the past it is frightening how pernicious and powerful the tobacco industry was and still is. Some books that recount that ugly history are "Golden Holocaust" by Robert Proctor published in 2012, "Cigarette Wars: The triumph of "The Little White Soldier" published by Cassandra Tate in 1999, and "Merchants of Death: The American Tobacco Industry" published by Larry C. White in 1988 with a foreword by C. Everett Koop, M.D. the U.S. Surgeon General.

"Merchants of Death" provides excellent documentation of the ongoing efforts of the tobacco manufactures to evade justice in the courts of law and in this country's legislative bodies. We should consider its title in understanding how the "merchants of death" are still thriving, and how we as legislators have become enablers and arguably participants in the ongoing slaughter of our own constituents.

Some ethical retailers such as CVS and Target have stopped selling these death dealing products. The Pope and the Vatican finally stopped selling cigarettes in 2018 saying "the Holy See cannot contribute to an activity that clearly damages the health of people". Our small retailers still come to our Capitol to protest our legislative attempts to save people's lives from this ongoing abomination of a business that eventually and foreseeably kills half of its customers.

Even back in 1988, smoking was identified as today as THE preventable cause of death in our society. That is still, sadly, the case today over 30 years later. C. Everett Koop, M.D. the U.S. Surgeon General served from 1982 to 1989 and issued a report in 1986 which stated that secondhand smoke had been conclusively proven to cause cancer. That report started a wave of actions to limit exposure to second-hand smoke. Koop wrote the foreword to "Merchants of Death" and stated "We need an informed public that understands the political and economic dimensions of this twentieth-century plague. Comprehensive reviews and analysis of the total problem are necessary for us to create this smoke-free society by the year 2000. This book, *(Merchants of Death)* because it tells the truth, is likely to be dangerous to the health of the tobacco industry."

It is now 20 long years from the year 2000. Cigarettes are still hugely profitable, and our state and all our states and our nation are now sharing in the profits of the tobacco industry. Vaping is now raising its ugly head, and many are proposing that we as states should share in the profits of that lethal and even MORE addicting addition to the tobacco industry instead of finally driving a stake in the heart of the tobacco industry.

We as legislators need to not listen to the myths of "bans don't work", and "remember prohibition", and "consider the right of the individual" and "how can we dishonor our returning soldiers who put their lives on the line by taking their cigarettes away." (that last ignores the fact that all services are tobacco free during basic training – all military trainees go "cold turkey" for 12 weeks). Those are the tropes and memes of the still very well heeled and deep pocketed tobacco industry and their pervasive and influential lobbyists. We need to buckle down and stop killing people, stop killing pregnant women, stop killing and disabling the children who are our future.

It only takes 26 votes in the House, 13 in the Senate and one governor to turn this around. That is our job. Let's do it.

Our sad history of government "profit sharing" with the tobacco industry

The Tobacco Master Settlement Agreement (MSA) was entered into in November 1998. In the MSA, the original participating manufacturers (OPM) agreed to pay a minimum of $206 billion over the first 25 years of the agreement. There were many other aspects to this settlement, but it basically was intended to

compensate the state for some of the medical costs of caring for persons with smoking-related illnesses. In the MSA, the original participating manufacturers (OPM) agreed to pay a minimum of $206 billion over the first 25 years of the agreement.

This has been viewed as a Faustian bargain (or a deal with the devil of the tobacco industry). The manufacture and sale of cigarettes would continue. Only a minimum part of the settlement funds was used to convince or aid smokers in quitting. The settlement appeared to be a cash cow for the settling states and the tobacco company were given a retrieve and allowed to pursue their course of manufacturing and selling cigarettes.

In addition, most states started to impose excise taxes on cigarettes with greatly different rates contributing to smuggling from low tax states to high tax states such as New York. Hawaii, per reports from various agencies, does not have a significant smuggling problem despite a high excise tax. The tax may be somewhat effective, but it disproportionately affects lower income segments, and does little to protect pregnant mothers and their innocent babies. Given the cost of the taxes it may limit mothers nutrition and actually damage the fetuses more.

In a study conducted on behalf of the New York State department of Health it revealed that low-income smokers (those in households making under $30,000), spent an average of 23.6% of their household income on cigarettes, compared to 2.2% for smokers in households making over $60,000.

While the price of cigarettes has continuously increased since 1965, the percentage of that price going towards taxes is now half of what it was then. As of 2011, Philip Morris list total government revenue, including federal, state and local and sales taxes as 55% of the estimated retail price of a pack of cigarettes in the US.

So, the tobacco companies were bringing in more money and the poor were disproportionately paying that money; and the health problems continued, albeit with a lower portion of the population still smoking. In my view it is time to lower the cost of living in Hawaii by getting rid of cigarettes (AND vaping) which will help the overall health and decrease the money wasted on cigarettes and vaping.

Developmental Cholinotoxicants: Nicotine and Chlorpyrifos

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Environmental Health Perspectives, Vol 107, Supplement 1. February 1999

The stimulation of cholinergic receptors in target cells during a critical developmental period provides signals that influence cell replication and differentiation. Accordingly, environmental agents that promote cholinergic activity evoke neurodevelopmental damage because of the inappropriate timing or intensity of stimulation. Nicotine evokes mitotic arrest in brain cells possessing high concentrations of nicotinic cholinergic receptors. In addition, the cholinergic overstimulation programs the expression of genes that evoke apoptosis and delayed cell loss.

Chlorpyrifos elicits damage by both non-cholinergic and cholinergic mechanisms extending from early stages of neural cell replication through late stages of axonogenesis and terminal differentiation. Accordingly, the window of developmental vulnerability to chlorpyrifos is likely to extend from the embryonic period into postnatal life. Environ Health Perpect 107 (Suppl 1);71-80 (1999).

Thus, unlike classical teratology, in which the first trimester of fetal development is the most sensitive target for adverse effects of drugs or chemicals, brain development is likely to be affected by exposures ranging from the early embryonic stage through adolescence (5). This review will focus on disruption of brain development elicited by agents targeting cholinergic transmission. Two of the most widespread chemical assaults on the fetus are cholinergic: nicotine, a direct cholinergic agonist delivered by maternal cigarette smoking, and insecticides,

Nicotine: Prototypic Cholinotoxicant

The largest toxic assault on fetal development is provided by maternal cigarette smoking, which involves approximately one-fourth of all pregnancies in the United States (13,14). Epidemiologic studies have established the tragic results: tens of thousands of spontaneous abortions and neonatal intensive care unit admissions annually, thousands of perinatal deaths and deaths from Sudden Infant Death Syndrome (crib death), and substantially increased incidence of learning disabilities, behavioral problems, and attention deficit/hyperactivity disorder (14-20).

We also identified a second mechanism for cell deficits caused by nicotine exposure (10): inhibition of DNA synthesis (Figure 3). Administration of even a single dose of nicotine to pregnant or neonatal rats elicits a precipitous and persistent (several hours long) decline in DNA synthesis, with specific targeting of brain regions with the highest concentrations of nicotinic cholinergic receptors.

Thus, fetal exposure to nicotine has lasting adverse effects on synaptic performance, effects that may not emerge fully until adolescence. We have also identified numerous adverse effects of prenatal nicotine exposure on postsynaptic signaling mechanisms, all of which are potential participants in neurobehavioral abnormalities.

Conclusions and Future Directions

Drugs or chemicals that target cholinergic neurotransmission probably represent the largest source of neurobehavioral teratogenesis. Nicotine exposure involves one-fourth of all pregnancies in the United States, and exposure to insecticides that target cholinesterase is ubiquitous.

We have shown that nicotine damages the developing brain at concentrations achieved in moderate smokers or with nicotine replacement therapies such as the transdermal patch. The sequelae of maternal smoking are already well established (14) and include high rated of miscarriage, fetal death, intrauterine growth retardation, deaths in the postnatal period, and behavioral and learning disturbances. The finding that a specific substance in tobacco (nicotine) is a major contributor to adverse outcomes provides the first definitive proof that tobacco is a direct cause of these problems.

Finally, it should not be overlooked that unlike standard teratogens, agents that target specific cell populations in the nervous system rather than general organogenesis, can be expected to have adverse effects that extent to the final stages of development: childhood and adolescence.

Developmental Cholinotoxicants: Nicotine and Chlorpyrifos

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The stimulation of cholinergic receptors in target cells during a critical developmental period provides signals that influence cell replication and differentiation. Accordingly, environmental agents that promote cholinergic activity evoke neurodevelopmental damage because of the inappropriate timing or intensity of stimulation. Nicotine evokes mitotic arrest in brain cells possessing high concentrations of nicotinic cholinergic receptors. In addition, the cholinergic overstimulation programs the expression of genes that evoke apoptosis and delayed cell loss. Effects of cholinesterase inhibitors exhibit many similarities to those of nicotine. Chlorpyrifos administered to developing rats in doses that do not evoke signs of overt toxicity decreased DNA synthesis and caused shortfalls in cell numbers in brain regions enriched in cholinergic innervation. In embryo cultures, chlorpyrifos also evoked apoptosis during neurulation. However, chlorpyrifos also evokes noncholinergic disruption of cell development by interfering with cell signaling via adenylyl cyclase, leading to widespread disruption that is not limited to cholinergic systems. We have tested this hypothesis *in vitro* with PC12 cells, which lack the enzymes necessary to produce chlorpyrifos oxon, the metabolite that inhibits cholinesterase. Chlorpyrifos inhibited DNA synthesis in undifferentiated PC12 cells, which have relatively few cholinergic receptors. Furthermore, chlorpyrifos was more effective than nicotine and its effects were not blocked by cholinergic antagonists. When cells were allowed to differentiate in the presence of chlorpyrifos, cell replication was inhibited even more profoundly and cell acquisition was arrested. At higher concentrations, chlorpyrifos also inhibited neuritic outgrowth. Thus, chlorpyrifos elicits damage by both noncholinergic and cholinergic mechanisms extending from early stages of neural cell replication through late stages of axonogenesis and terminal differentiation. Accordingly, the window of developmental vulnerability to chlorpyrifos is likely to extend from the embryonic period into postnatal life. — *Environ Health Perspect* 107(Suppl 1):71–80 (1999). *http://ehpnet1.niehs.nih.gov/docs/1999/Suppl-1/71-80slotkin/abstract.html*

Key words: adenylyl cyclase, apoptosis, chlorpyrifos, cholinergic neurotoxicants, DNA synthesis, nicotine

Neurotransmitters as Trophic Factors

Nearly four decades ago, Buznikov (*1,2*) demonstrated that neurotransmitters were present in high concentrations during specific phases of early development in sea urchin embryos, unrelated to their function in synaptic communication. Subsequently, transient expression of these substances and their specific receptors has been identified during ontogeny of the mammalian nervous system, and it is now certain that transmitters play essential roles in the cellular and architectural development of the brain (*3,4*). During this period, receptor stimulation uniquely communicates with the genes that control cell differentiation, changing the ultimate fate of the cell (Figure 1). As these changes are not typical for the mature nervous system, the ontogenetic state of the target cell is critical in determining whether the outcome of receptor stimulation is an effect on cell replication, differentiation, growth, death (apoptosis), or "learning," that is, determining the future set-point for responsiveness of the cell. At the same time, these multiple roles create a wide window of vulnerability in which exposure of the brain to neuroactive chemicals that elicit or block neurotransmitter responses can alter development. Thus, unlike classical teratology, in which the first trimester of fetal development is the most sensitive target for adverse effects of drugs or chemicals, brain development is likely to be affected by exposures ranging from the early embryonic stage through adolescence (*5*).

This review will focus on disruption of brain development elicited by agents targeting cholinergic transmission. Two of the most widespread chemical assaults on the fetus are cholinergic: nicotine, a direct cholinergic agonist delivered by maternal cigarette smoking, and insecticides, which enhance cholinergic effects through inhibition of cholinesterase, the enzyme that hydrolyzes acetylcholine. A focus on cholinergic mechanisms is also appropriate given the critical role played by acetylcholine in brain maturation. Cholinergic stimulation is essential for establishment of cerebrocortical cytoarchitecture, and even transient interference with cholinergic input during development produces permanent structural and behavioral damage (*6–8*). Similarly, cholinergic overstimulation at an inappropriate time leads to developmental anomalies. In the rat, the peak of cholinergic tone in the cortex ordinarily occurs during the second postnatal week (*9*). Administration of cholinergic agonists before that time or dietary alterations that evoke early onset of cholinergic activity result in premature cessation of neuronal mitosis, leading to shortfalls in cell numbers and deficient synaptic activity (*9–12*). Accordingly, it is important to explore the mechanisms underlying the actions of cholinotoxicants and their impact on the developing brain.

Nicotine: Prototypic Cholinotoxicant

The largest toxic assault on fetal development is provided by maternal cigarette smoking, which involves approximately one-fourth of all pregnancies in the United States (*13,14*). Epidemiologic studies have established the tragic results: tens of thousands of spontaneous abortions and neonatal intensive care unit admissions annually, thousands of perinatal deaths and deaths from Sudden Infant Death Syndrome (crib death), and substantially increased incidence of learning disabilities, behavioral problems, and attention deficit/hyperactivity disorder (*14–20*). These findings do not, however, obligate an underlying cholinotoxic mechanism. Cigarette smoke contains thousands of bioactive compounds, including hydrogen cyanide and carbon monoxide. In addition, the smoking "life style" is associated with multiple risk factors including poor prenatal care and low

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Abbreviations used: AChE, acetylcholinesterase; ANOVA, analysis of variance; CPF, chlorpyrifos; NGF, nerve growth factor; NIC, nicotine; ODC, ornithine decarboxylase; PN, postnatal; Rx, treatment.

socioeconomic status. Accordingly, animal models are needed to isolate the role of nicotine from these confounding variables.

Unfortunately the design of animal models of nicotine exposure has not proven simple. Injection of nicotine into pregnant rats does produce behavioral (*21–23*) and cellular (*24–26*) abnormalities, but many of these effects are caused by vasoconstrictor effects on uteroplacental circulation, evoking episodic hypoxia (*27–30*). Nicotine injections produce high peak plasma levels of drug, inducing obvious ischemic episodes (blanching, cyanosis) with each dose (*10,31*). Accordingly, in the mid-1980s, we developed the first animal model of fetal nicotine exposure to make use of continuous infusions delivered by implantable osmotic minipumps (*9,26, 31–34*), a delivery route that avoids hypoxia–ischemia, and that delivers a fixed dose of drug simulating the steady-state plasma levels seen in smokers or users of transdermal nicotine patches (*35,36*). Pharmacokinetic and pharmacodynamic differences dictate the use of higher overall doses in rats than in humans, so that the critical end point is matching the plasma concentrations and the corresponding pharmacologic effects (*36,37*). Thus, in rats, dose rates of 2 to 6 mg/kg/day are necessary to reproduce the nicotine plasma levels found in moderate (0.5 to 1 pack/day) to heavy (2 packs/day) smokers.

With the infusion model, we have been able to show definitive damage to developing rat brain by doses of nicotine that reproduce the plasma levels found in heavy smokers (*26,31–34*). Two indices of these adverse effects are illustrated in Figure 2. In animals exposed prenatally to nicotine, ornithine decarboxylase activity, a marker enzyme for cell damage, is elevated during the postnatal period in both early-developing (forebrain) and late-developing (cerebellum) brain regions even though nicotine exposure terminates at birth. During the same period, deficits in total cell number, as determined by DNA content, worsen. Subsequently, we found that genes associated with programmed cell death (apoptosis) are constitutively activated by prenatal nicotine exposure (*38,39*), with effects persisting into the period of maximal cell loss; direct morphological assessment of nicotine-exposed embryos confirmed the presence of numerous apoptotic cells (*40*). Nicotine-induced apoptosis in the developing brain is in direct contrast to the observation that nicotine exerts a neuroprotective effect in the adult brain (*41,42*), including protection from injury-induced apoptosis (*43,44*). Just as with c-*fos* itself (*45–47*), the developmental context in which nicotine exposure occurs is likely to be critical for determining whether apoptosis is evoked or suppressed. Indeed, cholinergic agonists and antagonists can both elicit apoptosis depending on whether the context involves active or desensitized receptors (*48*). In the context of extended exposure to nicotine during fetal development, persistent induction of c-*fos* clearly is associated with enhanced cell death (*31*), most likely from apoptosis (*40*).

We also identified a second mechanism for cell deficits caused by nicotine exposure (*10*): inhibition of DNA synthesis (Figure 3). Administration of even a single dose of nicotine to pregnant or neonatal

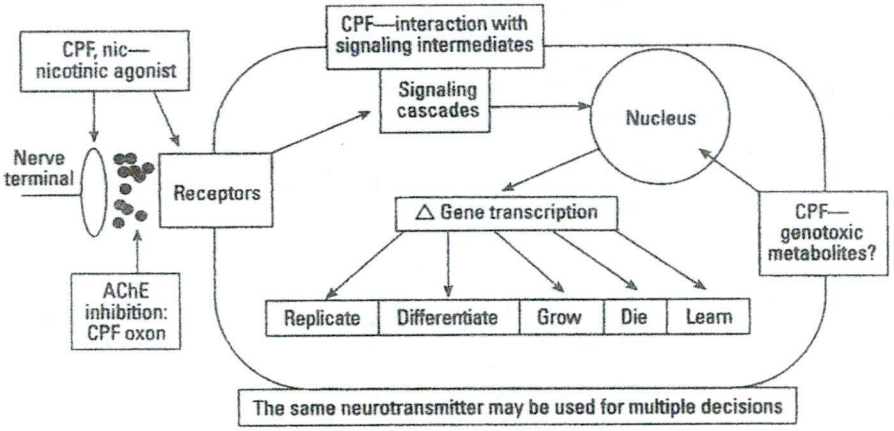


Figure 1. Cholinotoxicant targeting of cell development. Abbreviations: AChE, acetylcholinesterase; CPF, chlorpyrifos; Nic, nicotine. During development, neurotransmitters, through their receptors and associated signaling cascades, control the genes that influence differentiation. Depending on the context in which stimulation occurs, the same neurotransmitter can promote cell replication, can elicit a switch from replication to differentiation, can promote or arrest cell growth, can evoke apoptosis, or can program the genes that determine the future responsiveness of the cell to external stimulation. Nicotine targets nicotinic cholinergic receptors located on target cells, directly evoking changes in gene expression. Presynaptic nicotinic receptors that modulate release of other neurotransmitters produce secondary alterations of target cell development through the actions of these other transmitters on their respective receptors, signaling cascades and gene expression (*39*). Chlorpyrifos through its active oxon metabolite inhibits acetylcholinesterase, preventing the breakdown of acetylcholine and thus enhancing cholinergic activity. In addition, chlorpyrifos can exhibit agonistlike properties, opening and then desensitizing nicotinic cholinergic receptor/ion channels (*81*), can interact with signaling intermediates such as G-proteins and adenylyl cyclase (*80,82,83*), or can produce oxidative damage to DNA (*84,85*).

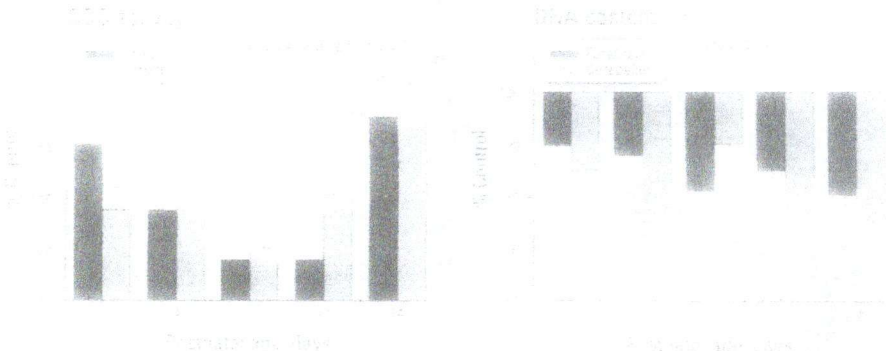


Figure 2. Effects of nicotine on biomarkers of cell damage (ornithine decarboxylase activity) and cell number (DNA content), evaluated in postnatal rat brain (*32*). Abbreviation: ODC, ornithine decarboxylase. Nicotine exposure elicits persistent damage (elevated ornithine decarboxylase activity) and cell loss (decreased DNA) despite discontinuing nicotine exposure at birth. Effects are discernible in both an early-developing region (forebrain) and late-developing region (cerebellum). Data represent means and standard errors obtained from 8 pups in each group at each age for each determination, with ANOVA main treatment effect indicated within the panels.

rats elicits a precipitous and persistent (several hours long) decline in DNA synthesis, with specific targeting of brain regions with the highest concentrations of nicotinic cholinergic receptors. The same effects are obtained when minute amounts of nicotine are introduced directly into the brain, bypassing any systemic drug effects (*10*).

Simply losing cells or preventing acquisition of the correct number of cells does not inherently account for neurobehavioral disruption by nicotine exposure; instead it is necessary to demonstrate that synaptic function is affected. Because nicotine works through cholinergic receptors, we first evaluated effects on cholinergic transmission (*9,49*). Using biochemical indices of neuronal impulse activity, we found that prenatal nicotine exposure blunted the ontogenetic rise of synaptic activity in the forebrain and produced persistent deficits in the hippocampus (Figure 4). However, adverse functional effects are not limited to cholinergic neurotransmission. Nicotinic receptors also play a prominent role in the activity of catecholaminergic systems, and we found that fetal nicotine treatment had adverse effects on these synapses as well, again with the effects appearing well after termination of nicotine exposure. Catecholaminergic function showed two phases of synaptic hypoactivity, one in the immediate postpartum period and another emerging with the onset of puberty (*33*), accompanied by behavioral anomalies (*36,50,51*). In the intervening stages, even though basal activity was within normal limits, the reactivity of noradrenergic systems to acute nicotine challenge was obtunded in the prenatal nicotine group (Figure 4): doses of nicotine that evoked norepinephrine release in brain regions of control animals were unable to do so in the group exposed to nicotine prenatally (*52*). Thus, fetal exposure to nicotine has lasting adverse effects on synaptic performance, effects that may not emerge fully until adolescence.

We also have identified numerous adverse effects of prenatal nicotine exposure on postsynaptic signaling mechanisms, all of which are potential participants in neurobehavioral abnormalities. These entail lasting changes in the expression of cell signaling intermediates (*53,54*), uncoupling of receptors and second messenger systems from downstream cellular events (*55,56*), and alterations in the expression of receptor proteins themselves (*26,53,55–57*). Developmental disruption by nicotine thus occurs at numerous loci and ranges from outright cell loss to specific alterations of neural activity to misprogramming of receptor signaling

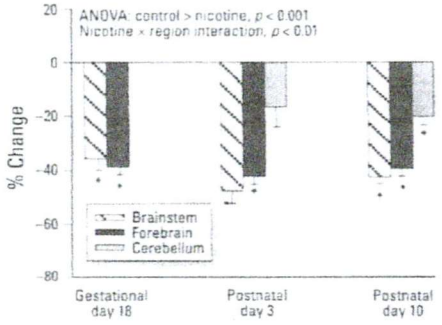


Figure 3. Inhibition of DNA synthesis in rat brain regions after a single dose of nicotine (*10,31,106*). Measurements of [^{3}H]thymidine incorporation into DNA were made in the first 30 min after nicotine administration. Susceptibility is directly related to the concentration of nicotinic cholinergic receptors in each region, namely brainstem ≥ forebrain > cerebellum. Data represent means and standard errors obtained from 18–30 rats at each age in each treatment group. ANOVA across all ages and regions is shown within the panel; asterisks (*) denote values that differ significantly from the corresponding control.

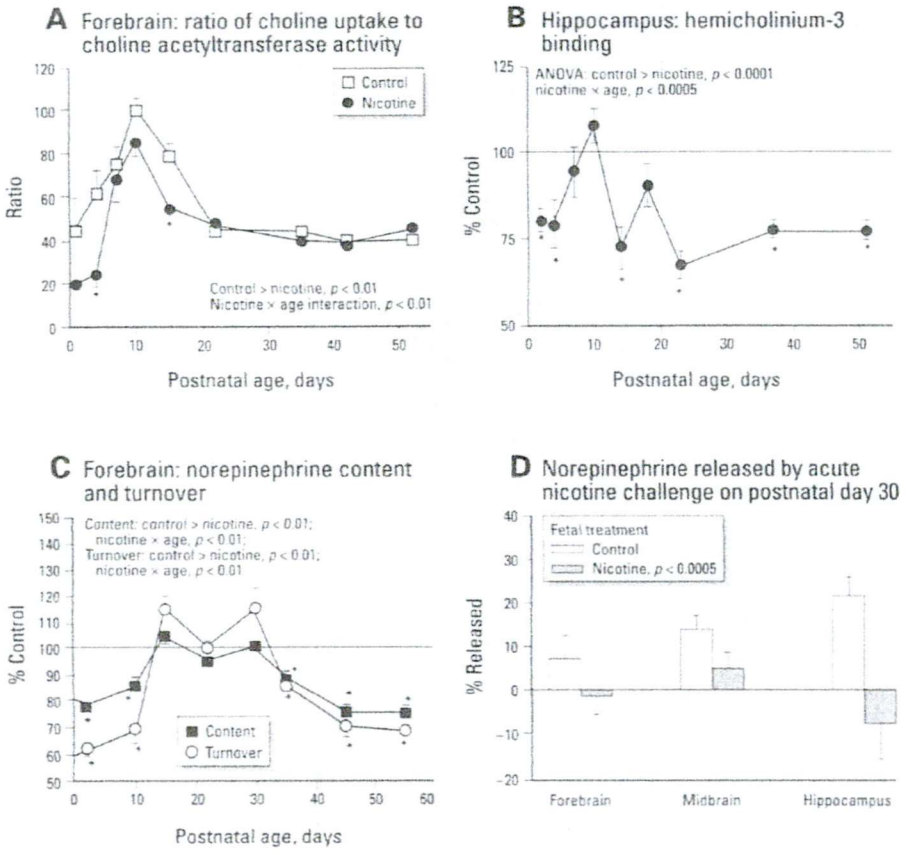


Figure 4. Synaptic hypoactivity elicited by prenatal nicotine exposure. (*A*) In the forebrain, the ratio of choline uptake to choline acetyltransferase activity (a biochemical marker of impulse activity in cholinergic projections) shows a naturally occurring peak at postnatal day 10; nicotine blunts activity before and during the developmental spike (*9*). (*B*) In the hippocampus, [^{3}H]hemicholinium-3 binding to the high-affinity choline transporter, which is regulated by nerve impulse activity, shows both initial postnatal deficits and a later-emerging, permanent deficit in the nicotine group (*49*). (*C*) Noradrenergic hypoactivity is also elicited by prenatal nicotine exposure. Norepinephrine content and turnover are suppressed in the forebrain during both the initial postnatal period, and more persistently with the onset of puberty (*33*). (*D*) Before the reemergence of deficits in the measures of basal activity, the nicotine group shows a subnormal responsiveness to acute challenges. A single injection of nicotine, which releases norepinephrine in the control group, fails to do so in the nicotine group (*52*). Data represent means and standard errors obtained from 7–10 animals in each group at each age, for each type of determination. ANOVA is shown within the panels and asterisks (*) denote individual ages at which the nicotine group differs from the corresponding control. Individual tests were not run for acute norepinephrine release because of the absence of a significant interaction of treatment × region.

mechanisms. In trying to determine whether these various outcomes all reflect a similar underlying basic mechanism, two interrelated questions emerge. First, are the effects present at doses corresponding to moderate smoking (one-half pack to one pack per day), where growth impairment, which can lead to nonspecific alterations, is absent? If so, this would imply a specific mechanism targeting the developing brain rather than effects secondary to a more general fetal insult. Selectivity for the developing brain would then raise the second question: Is stimulation of nicotinic cholinergic receptors the underlying target for the effects? The first question can be answered definitively. Lowering the dose of nicotine in rats to the point at which growth impairment vanishes and plasma levels match those of moderate smokers still produces all the signs of fetal brain damage that were seen at higher doses (*34,39*): elevated ornithine decarboxylase activity, progressive cell loss, and deficits of synaptic activity (Figure 5). These results are opposite from nonspecific insult, where brain development typically is spared relative to all other growth components (*58–60*). The most likely explanation for the exquisite sensitivity of the developing brain to nicotine-induced damage is the targeting of specific proteins, namely nicotinic cholinergic receptors, that have the ability to respond to nicotine at extremely low (nanomolar) concentrations (*26,61–63*). Nicotinic receptors originate in the fetal brain during neurulation and rise dramatically in late gestation and after birth (*26,62–65*). We have been able to show that these receptors are tonically stimulated by fetal nicotine exposure, as evidenced by receptor upregulation (*26*), even at doses that do not impair growth (*34*). Specific tests of each component of fetal brain cell loss evoked by nicotine have verified the involvement of nicotinic receptors, whether for inhibition of DNA synthesis (*10*), stimulation of damage markers (*66*), or promotion of apoptosis (*38,67*). Delayed functional sequelae such as late-appearing reductions in synaptic activity are more problematic because of the long temporal separation between initial injury and measurable consequences. However, just as for the more immediate markers of cell damage, the dose threshold for delayed neural effects also lies far below that of growth impairment, whether assessed biochemically (*34,52*) or behaviorally (*50,51,68–70*). By implication, the delayed effects are most likely linked to the initial receptor-mediated changes in cell development originating during and immediately after fetal nicotine exposure.

Targeting of nicotinic receptors can explain the widespread nature of the defects in cell number and synaptic activity seen after prenatal nicotine exposure. As shown in Figure 1, nicotinic receptors are located not only at postsynaptic sites but also are extremely prominent at the presynaptic terminals of a wide variety of neurotransmitter systems including acetylcholine, catecholamines (norepinephrine, dopamine), and excitatory amino acids, which are themselves potentially neurotoxic. Evoked release of other transmitters that alone exert neurotrophic control of their own targets thus is likely to produce disruption in all the sites "downstream" from nicotinic receptor activation. One issue for further consideration is whether a specific receptor subtype is involved in nicotinic cholinergic neurotrophic actions, and by implication, mediating the disruptive effects of prenatal nicotine exposure. Indeed, based on *in vitro* studies, specific roles have been postulated for control of synaptogenesis by nicotinic receptor subtypes containing the $\alpha 7$ subunit and for adverse effects of nicotine (*71*). It is also apparent that developing neurons show distinct ontogenetic profiles for expression of the genes encoding the individual subunits of nicotinic receptors (*72,73*). Nevertheless, some key elements are missing in the current understanding of the role of receptor subtypes in the developmental effects of nicotine, as nearly all studies of subtypes *in vivo* have been conducted at the level of mRNA but not receptor protein. Accordingly, it is unclear as to which subtypes are actually expressed at the cell surface and whether specific subtypes are linked differentially to neurotrophic stimuli. Nevertheless, this absence of knowledge concerning receptor subtypes does not obviate the clear-cut effects of nicotine on cell development and the linkage of these effects to nicotinic cholinergic receptors.

Our findings indicate conclusively that nicotine is a neuroteratogen, evoking cell damage and reducing cell numbers, impairing synaptic activity and behavioral performance, and eliciting these changes at doses commensurate with moderate smoking, below the level at which fetal growth is impaired. The underlying mechanisms are receptor mediated, accounting for selective effects on the brain at low-dose thresholds and for the involvement of brain regions and transmitter systems that have prominent cholinergic inputs. Receptor stimulation leads to two distinct errors in the program of cell development, a premature change from cell replication to differentiation, and after a delay, initiation of the program for cell damage and apoptosis. The next issue, then, is whether other potential cholinotoxicants, especially insecticides, share the same mechanisms and outcomes.

Figure 5. Nicotine damages the developing brain at doses that do not compromise growth (*31,34,52*). Abbreviation: ODC, ornithine decarboxylase. Administration of 2 mg/kg/day to pregnant rats, which simulates plasma levels of nicotine found in moderate smokers, results in normal body and brain region weights in the offspring. Nevertheless, cell damage (elevated ODC activity), cell loss (reduced DNA content) and synaptic hypoactivity (subnormal norepinephrine turnover) are still fully evident. Data represent means and standard errors obtained from 5–10 animals in each group at each age for each type of determination. Differences for weights are not significant (ANOVA), but effects on biomarkers are (main treatment effect, $p < 0.0001$ across all three biomarkers and for each biomarker taken individually).

Developmental Neurotoxicity of Chlorpyrifos *in Vivo*

Increasing use is being made of the long-lasting organophosphorus insecticide chlorpyrifos, largely because this agent does not elicit organophosphate pesticide-induced persistent neuropathies until the dose is raised above the threshold for lethality (*74*). Nevertheless, recent concern has arisen over domestic application, which can lead to infant exposures well above acceptable levels (*75,76*). Animal studies indicate that immature animals are far more susceptible to acute toxicity of chlorpyrifos (*77–79*) despite the fact that they recover from cholinesterase inhibition more quickly than adults (*78–80*). As with other organophosphate insecticides, chlorpyrifos, via its reactive metabolite, chlorpyrifos oxon, inhibits cholinesterase and prevents the breakdown of acetylcholine. An initial view of the potential impact of chlorpyrifos on signaling targets in brain development thus could resemble that of nicotine (Figure 1), with promotion of cholinergic signaling as the primary target. However, chlorpyrifos also exhibits direct cholinergic agonistlike properties, opening and then desensitizing nicotinic cholinergic receptor/ion channels (*81*); it interacts with signaling intermediates such as G-proteins and adenylyl cyclase (*80,82, 83*); and it may produce oxidative damage to DNA (*84,85*).

If the primary effect of chlorpyrifos on the developing brain is a reflection of its general mode of toxicity as seen in mature animals, namely cholinesterase inhibition, then the net effects during development should bear a strong resemblance to those of nicotine, which also elicits cholinergic hyperstimulation. When we administered chlorpyrifos to neonatal rats (Figure 6), we obtained acute inhibition of DNA synthesis (*77*). However, at 1 day of age, there was no regional selectivity to the effect: regions with low cholinergic innervation (cerebellum) were affected just as much as cholinergically enriched regions (brainstem, forebrain). Regional selectivity then emerged by the end of the first postnatal week, at which point cholinergic antagonists could block the effect. Thus, chlorpyrifos affects DNA synthesis by at least two different types of mechanisms, an initial, noncholinergic effect, and subsequently, actions mediated through cholinergic activity. In support of the unexpected finding of noncholinergic contributions to effects on DNA synthesis, we obtained the same inhibitory actions when minute amounts of chlorpyrifos were injected directly into the brain, bypassing hepatic activation to chlorpyrifos oxon, the metabolite that inhibits cholinesterase. The contributions of noncholinergic mechanisms to the net adverse effect on brain development are readily demonstrable. With repeated chlorpyrifos administration, we obtained persistent inhibition of DNA synthesis (*86*), leading to deficits in cell number (*87*) and suppression of macromolecular constituents (*88*). These effects were seen at chlorpyrifos exposure levels that were devoid of any overt toxicity and that reduced cholinesterase activity by only 20% (*80*), a degree of inhibition insufficient to produce signs of systemic toxicity.

Some of the postulated, noncholinergic effects of chlorpyrifos involve cell signaling intermediates common to multiple neuronal and hormonal inputs, especially the adenylyl cyclase transduction pathway (*82,83,89*). Cyclic AMP is universally involved in the control of cell replication and differentiation in virtually all prokaryotic and eukaryotic cells (*90–94*), so that perturbation of this pathway during development would be expected to have a significant impact on brain cell development. When we examined the effects of otherwise subtoxic doses of chlorpyrifos on adenylyl cyclase activity in the developing brain (*80*), we found profound effects on G-protein-mediated signaling, including that operating through neurotransmitter receptors known to play neurotrophic roles in cell replication/differentiation patterns (Figure 7). Importantly, low doses of chlorpyrifos administered early in development, with minimal cholinesterase inhibition, had a much greater effect on adenylyl cyclase activity than larger doses given later in development, even though the latter treatment produced much greater inhibition of cholinesterase. Again, this indicates that noncholinergic mechanisms play critical roles in the adverse effects of chlorpyrifos on brain development. Thus,

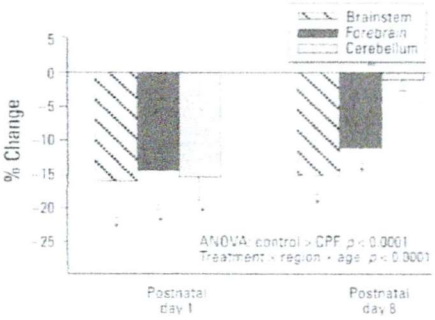


Figure 6. Inhibition of DNA synthesis after a single dose of chlorpyrifos (*77*). Abbreviation: CPF, chlorpyrifos. Inhibition does not display regional selectivity until the end of the first postnatal week, suggesting two separate mechanisms: an initial, noncholinergic effect, followed by a later-appearing, cholinergic effect. Data represent means and standard errors obtained from 29–63 animals in each treatment group at each age. ANOVA across both ages and all regions appears within the panel. Asterisks (*) denote individual values for which the chlorpyrifos group differs from the corresponding control.

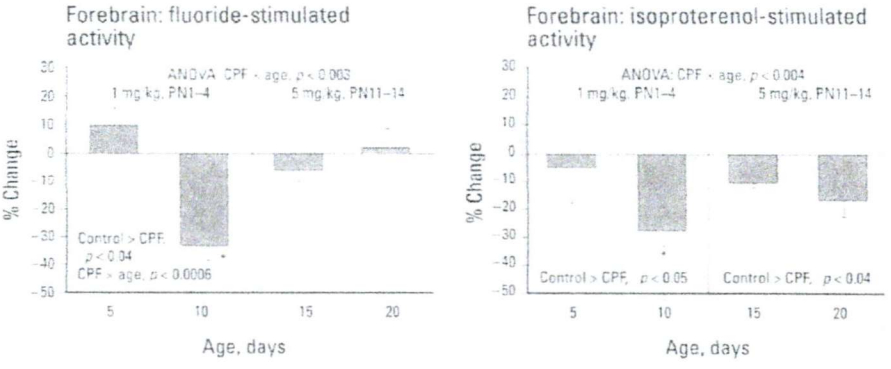


Figure 7. Effects of repeated chlorpyrifos administration on forebrain adenylyl cyclase activity (*80*). Abbreviations: CPF, chlorpyrifos; PN, postnatal. Treatment with a low dose on postnatal days 1–4 produces a larger deficit than a larger dose given later in development, despite the fact that the later treatment produces greater cholinesterase inhibition. The effect on adenylyl cyclase emerges after a delay of several days after cessation of treatment, at a time when cholinesterase activity has completely recovered to normal. Data represent means and standard errors obtained from 11–12 animals in each treatment group at each age. ANOVA across age is shown within the panels and asterisks (*) indicate individual values that differ significantly from the corresponding control.

conversion of chlorpyrifos to its oxon metabolite and the consequent inhibition of cholinesterase may not be the essential factors in determining neurobehavioral teratology by this compound or potentially for other insecticides as well.

Developmental Neurotoxicity of Chlorpyrifos Modeled *in Vitro*

A definitive demonstration that chlorpyrifos exerts direct effects on neurodevelopment requires control over the cellular environment, as provided by *in vitro* models. We have used PC12 rat pheochromocytoma cells, a cloned cell line that initially resembles sympathetic neuronal precursor cells but that differentiates to resemble sympathetic neurons morphologically, physiologically, and biochemically (*95,96*). The onset of differentiation is initiated by nerve growth factor after which the cells develop the appearance and function of cholinergic target neurons, including increased expression of cholinergic receptors, choline acetyltransferase, and acetylcholinesterase (*97,98*). Equally important, these cells lack cytochrome P450 (*99*), the enzyme that converts chlorpyrifos to its oxon, the metabolite that inhibits cholinesterase. Thus, if the actions of chlorpyrifos seen for brain development *in vivo* are paralleled by similar actions on PC12 cells *in vitro*, the effects cannot be secondary to cholinesterase inhibition, the standard biomarker of organophosphate-induced toxicity.

Using undifferentiated PC12 cells, we obtained immediate (1 hr) inhibition of DNA synthesis (Figure 8); effects on RNA or protein synthesis were much less notable, indicating a selectivity toward replicating cells (*100*). The effect on DNA synthesis in undifferentiated PC12 cells could not be blocked by cholinergic receptor antagonists, confirming that chlorpyrifos itself produces effect without a requirement for cholinesterase inhibition and its resultant cholinergic hyperstimulation. When PC12 cells were allowed to differentiate in the continuous presence of chlorpyrifos, the inhibition of DNA synthesis intensified and persisted throughout the period of cell development (Figure 9). As a consequence, acquisition of new cells (DNA level) was severely curtailed, or at the highest concentrations, completely arrested, replicating the effects found for chlorpyrifos *in vivo*. In contrast to the profound effects on DNA synthesis and cell acquisition, neurite extension, as measured by the increase in membrane surface area (protein/DNA ratio), was inhibited only at high chlorpyrifos concentrations. These results confirm a targeted, primary effect of chlorpyrifos on cell replication, with other developmental abnormalities requiring higher exposure levels. Just as was found for *in vivo* treatments, the progression of cell differentiation increases the sensitivity to chlorpyrifos, representing emergence of the cholinergic target phenotype; at that point, both direct and cholinergically mediated effects become additive (*77*), whereas only the direct effects can be expressed in the undifferentiated state.

We have also carried out *in vitro* studies in rat embryo cultures (*67*). Using chlorpyrifos concentrations that showed no evidence of growth reduction or dysmorphogenesis, we found clear-cut abnormalities of mitosis in the developing brain at the neural tube stage. Embryos were incubated with chlorpyrifos for 48 hr beginning on embryonic day 9.5 (Figure 10). Examination of the forebrain and hindbrain regions revealed reduced and altered mitotic figures with dispersion and disorientation of the mitotic layer. In

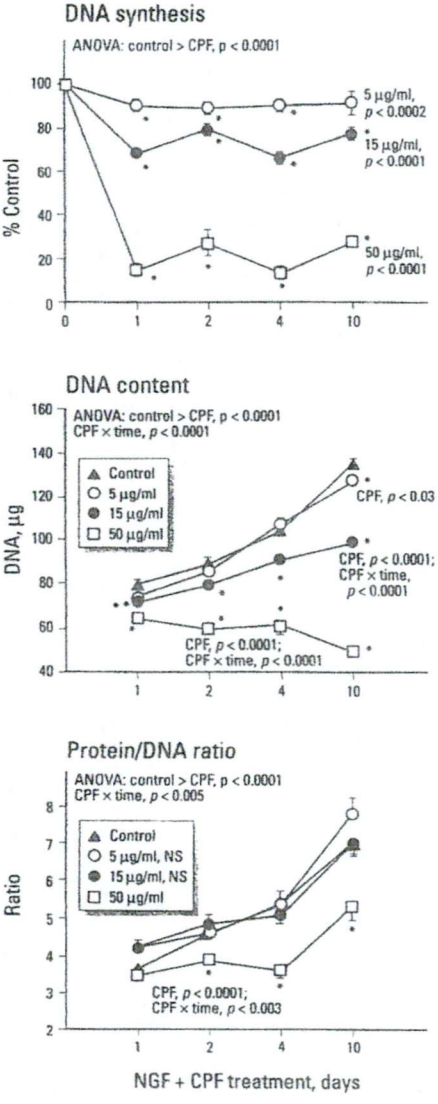
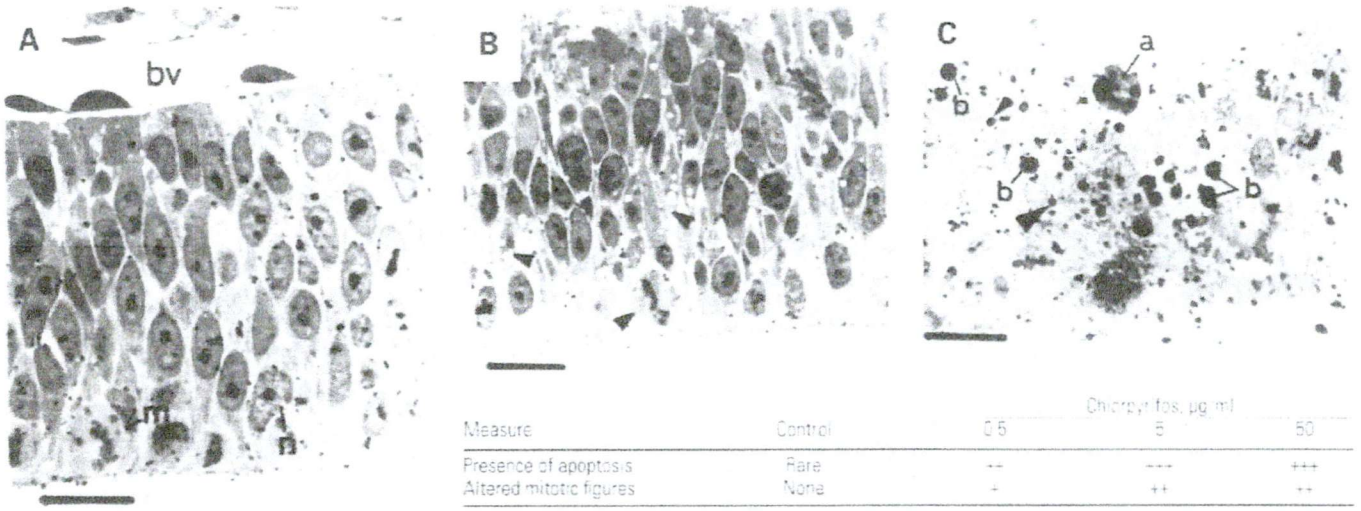


Figure 9. Effects of continuous exposure to chlorpyrifos on PC12 cells during differentiation (*100*). Abbreviation: CPF, chlorpyrifos. The inhibition of DNA synthesis is intensified and maintained throughout the developmental period, leading to reduced or arrested cell acquisition (DNA level). At the highest concentration, neurite extension (as measured by the protein/DNA ratio) is also inhibited. Data represent means and standard errors obtained from 20–36 determinations in each group at each time point. ANOVA across all time points and treatments appears within each panel and asterisks (*) denote individual values that differ from the corresponding control.

Figure 8. Inhibition of DNA synthesis by chlorpyrifos in undifferentiated PC12 cells (*100*). Abbreviations: ATR, atropine; CPF, chlorpyrifos; MEC, mecamylamine. Inhibition shows an immediate onset of action and is not mediated by cholinergic hyperstimulation, as receptor blocking agents for muscarinic (atropine) or nicotinic (mecamylamine) receptors do not prevent the effect. Data represent means and standard errors obtained from 6–17 determinations for each treatment and time point. ANOVA across all treatments and time points appear within the panels and asterisks (*) denote individual values for which the treated groups differ from the corresponding controls.



Measure	Control	Chlorpyrifos, µg/ml 0.5	5	50
Presence of apoptosis	Rare	++	+++	+++
Altered mitotic figures	None	+	++	++

Figure 10. Effects of chlorpyrifos on brain development in cultured rat embryos (*67*). Abbreviations: bv, blood vessel; CPF, chlorpyrifos; m, mitotic figure; n, inactive heterochromatin. (*A*) Forebrain neuroepithelium in control embryos at embryonic day 11.5, showing a bipolar pseudostratified epithelium: apical and basal processes contain a granular nucleus and inactive heterochromatin. Mitotic figure can be seen toward the internal limiting membrane. Mesenchyme around the germinal epithelium shows blood vessel. (*B*) Neuroepithelium from an embryo exposed to chlorpyrifos (50 µg/ml). Note the extensive vacuolation of the cytoplasm of the epithelial cells (arrowheads). (*C*) Forebrain neuroepithelium from a chlorpyrifos-exposed embryo showing extensive cell death (b) and extracellular bodies (arrowheads). A large cell (a) with multiple apoptotic condensations is also visible. Scale bar = 20 µm. For semi-quantitative measurements (table at bottom of figure), evaluations were made in numerous sections obtained from four otherwise morphologically normal embryos in each treatment group. Over a much larger cohort (>40 embryos per treatment), there was no evidence of gross dysmorphogenesis or of changes in developmental landmarks aside from the disruption of cell development in the neuroepithelium.

addition, cytotoxicity was evidenced by cytoplasmic vacuolation, enlargement of intercellular spaces, and the presence of a significant number of apoptotic figures. Significant effects were found even at concentrations as low as 0.5 µg/ml.

Our results with PC12 cells or rat embryo cultures support the idea that chlorpyrifos specifically targets brain development. However, a major problem is how to compare exposures *in vitro* with those likely to be experienced with environmental contamination. Certainly, the concentration and exposure period necessary to affect brain cell development *in vitro* lie well below those necessary for dysmorphogenesis, for chromosome damage (*101*) or for general cytotoxicity (*101,102*). Although scant information is available concerning the actual levels of chlorpyrifos achieved in fetal brain, we have already demonstrated that doses that cause only 20% cholinesterase inhibition nevertheless depress mitosis in neonatal rat brain *in vivo* (*77,80,86*), leading to deficiencies in cell numbers (*87*). A preliminary report on pregnant rats (*103*) found that a comparable degree of cholinesterase inhibition, which is well below the threshold for any observable signs of cholinergic hyperstimulation, produces peak fetal brain concentrations of the major metabolite of chlorpyrifos of approximately 0.25 µg/g, which on a molar basis, corresponds to the lowest concentration of chlorpyrifos used in our studies with embryos *in vitro* (*67*). On a body weight basis, the doses of chlorpyrifos needed for adult or developmental toxicity in rats range up to tens to hundreds of mg/kg (*78,79,104,105*) and certainly no lower than 2 mg/kg (*77*). Mitotic arrest *in vivo* occurs with brain concentrations of 2 µg/g (*77*), again well within the concentration range needed for *in vitro* effects. The likely acute exposure level for infants after home application of chlorpyrifos is also above this range: 350 µg/kg/day for a 2-week period, for a total of 5 mg/kg (*76*). Although there are clear limitations of extrapolation across species and between cultures and intact systems, *in vitro* evaluations nevertheless can point the way to likely mechanisms and adverse outcomes, and are likely to be within the range of relevant exposure levels *in vivo*.

Conclusions and Future Directions

Drugs or chemicals that target cholinergic neurotransmission probably represent the largest source of neurobehavioral teratogenesis. Nicotine exposure involves one-fourth of all pregnancies in the United States, and exposure to insecticides that target cholinesterase is ubiquitous. Establishing the underlying mechanisms, and hence safety thresholds, for these compounds must represent a major focus of future work. We have shown that nicotine damages the developing brain at concentrations achieved in moderate smokers or with nicotine replacement therapies such as the transdermal patch. The sequelae of maternal smoking are already well established (*14*) and include high rates of miscarriage, fetal death, intrauterine growth retardation, deaths in the postnatal period, and behavioral and learning disturbances. The finding that a specific substance in tobacco (nicotine) is a major contributor to adverse outcomes provides the first definitive proof that tobacco is a direct cause of these problems, not simply a covariable with other components of the smoking life style. In the case of chlorpyrifos, our findings indicate that inhibition of cholinesterase, the standard biomarker for organophosphate toxicity, is inadequate to explain the effects of this compound on brain development. The uncovering of alternative mechanisms indicates the need for research on screening methods that emphasize unique attributes of developing systems such as DNA synthesis, cell acquisition, apoptosis, and cytoarchitectural modeling of specific brain regions. *In vitro* systems such as neural cell lines or embryo cultures can play key roles in elaborating these mechanisms and in establishing new safety thresholds for insecticide exposure during

development. Finally, it should not be overlooked that unlike standard teratogens, agents that target specific cell populations in the nervous system rather than general organogenesis, can be expected to have adverse effects that extend to the final stages of development: childhood and adolescence. In the future, we will need to acquire new ways of evaluating potential postnatal effects of environmental contaminants.

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LATE

Testimony to House Committee on Health
Tuesday, February 4, 2020; 2:15 p.m.
State Capitol, Conference Room 329

Dear Chair Mizuno, Vice-Chair Kobayashi, and Committee Members,

My name is Ramic Santiago and I **support** HB 2457: RELATING TO THE YOUTH VAPING EPIDEMIC.

There are too many children under 18 that are choosing to vape in Hawaii. According to the Center for Disease Control and Prevention Hawai‘i ranks second in children ranging from 6th to 12th grade who choose to vape (1). According to Kaiser Health News numbers reported in 2018 translate to 3 million high school students nationally who vape. These staggering numbers continue to rise with advancements in vaping technology. Vaping devices can come in a variety of sizes and others like the JUUL product which is the size of a small USB port and can be used almost undetectable. Nicotine levels are manipulated and highly concentrated like the JUUL that can contain as much nicotine as 20 packs of cigarettes.

Nicotine is extremely unhealthy for youth because it can be extremely harmful to the developmental process in the youths brain and nicotine causes changes in the brains synapses which are built faster than the adults brain (1). Most e cigs contain nicotine and nicotine use in adolescence may further the risk for future addiction to drugs (1). There are many carcinogenic chemicals used in the production of e-cigs including cancer-causing compounds and heavy metals such as nickel, tin, and lead (1).

Many vaping products come with or are used with flavor additives such as candy flavorings. These flavorings are targeted at making nicotine products familiar to youth and make the vaping product more palatable to taste than traditional burning tobacco products which are restricted by the 2009 Family Smoking Prevention and Tobacco Control Act which banned fruit and candy flavorings in cigarettes but did not include smokeless tobacco products like chew and e-cigs.

As of January 2020 the Trump administration has enacted a ban on the flavoring of e-cigarettes which use pre filled cartridges but does not include tank based systems where users fill their own nicotine and flavor mixturing(2). Furthermore, mentol pre-filled vaping devices are excluded from the ban (2) . According to the National Institute on Drug Abuse 66% of teens vape only flavoring (2). Banning pre-filled e-cig products will have little effect when teens can still get ahold of products that contain flavored vaping products like fillable tank systems.

Hawaii needs to further the Trump administration's efforts to reduce the increasing number of underage children who use vaping products. Hawaii needs to ban all flavored vaping products in the state to fight the epidemic of underage teen vaping.

Sincerely,

Ramic P.H. Santiago, B.S.
1614 Emerson Street # 5
Honolulu, Hawaii 96813

Resources

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2. MarketWatch.(2020). Federal Government Bans Popular E-cigg Flavors to the Curb.
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Testimony to House Committee on Health
Tuesday, February 4, 2020; 2:15 p.m.
State Capitol, Conference Room 329

Dear Chair Mizuno, Vice-Chair Kobayashi, and Committee Members,

I support HB 2457: RELATING TO THE YOUTH VAPING EPIDEMIC.

Tobacco killed my husband last March from smoking. He ended up dying from COPD and had lung cancer. He suffered greatly before he passed away and died an early death.

We have 6 grandchildren that I don't want to have the same fate as their grandfather. Our keiki are a vulnerable group and must be protected. Don’t make it easy for electronic smoking devices to get into the hands of our kids from accessibility online and for being cheap to buy.

Please pass HB 2457 to save our children for the future.

Sincerely,

Jennifer Hausler
Pearl City, 96782

Date: February 4, 2020
To: The Honorable John M. Mizuno, Chair
Bertrand Kobayashi, Vice Chair
Members of the Committee on Health

The Honorable Justin H. Woodson, Chair
Mark J. Hashem, Vice Chair
Sean Quinlan, Vice
Members of the Committee on Lower and Higher Education

From: Ruthie Diaz, BSW
rddiaz@hawaii.edu

Re: Support for HB2457, RELATING TO THE YOUTH VAPING EPIDEMIC

Hearing: Tuesday, February 4, 2020 at 2:15 pm at Conference Room 309

Thank you for the opportunity to submit testimony in SUPPORT of HB2457, RELATING TO THE YOUTH VAPING EPIDEMIC

I am a non-traditional student at the University of Hawai'i, Mānoa, and completing the master's degree at the Myron B. Thompson School of Social Work. As a full-time student, social work practicum student, and a part-time employee, I have opportunities to engage with the communities at the University of Hawai'i as well as the Oahu community working with individuals and families. Most importantly, I am a mother of two children, ages five and an 11. I care deeply for my children as well as all the youth of Hawai'i, and prevention is key to many health risks.

Although there are various stories that I have encountered with electronic nicotine delivery systems ENDS and vape products, the one I can recall involves my four-year-old son. I live in a condominium with a wraparound lanai that welcomes cool trade wind breezes on most days. Because of this, my lanai doors are always wide open. My home is a smoke-free building, which should eliminate such behaviors and provide clean air for my family. However, addictive behaviors will often lead to the disregarding of such rules. Neighboring units smoking on their lanai lead to clouds of smoke to travel down to my family's unit. My four-year-old smelled the strawberry flavors and savored it. Luckily, he spoke out loud and mentioned that it smells like candy. I knew what it was, and I had no choice but to report it to our building management and close my lanai door. I realized that the smell of such flavors was very enticing to my four-year-old, and had I not stopped him, he would have continued to inhale the vape products contaminating our fresh air. This was a teaching moment for my older child about the health risk and the inconvenience it causes other people and families, but not necessarily to a four-year-old. This experience allowed me to see how dangerously enticing these ENDS flavors to even the youngest children. It made me aware of how normalized use or even continued exposure to ENDS and vape products may lead to experimentation and subsequent addiction.

Bill HB2457, if enacted, it will prevent many health problems that are related to electronic nicotine delivery systems (ENDS) and vape products. The increasing youth vaping epidemic, particularly in Hawai'i, is due to the lack of regulation pertaining to ENDS, enticement to youth using candy-flavored vape products as well as communities and social behaviors among youth normalizing the use of these items.

The opposition's argument that the use of ENDS and vape products is successful as a smoking cessation product has not been proven true, nor is it FDA approved. A study conducted in 2016 by Garcia-Arcos *et al.*, concluded

that mice who were exposed to aerosolized nicotine-free and nicotine-containing e-cigarette fluid "triggered effects normally associated with the development of COPD including cytokine expression, airway hyper-reactivity, and lung tissue destruction" (Garcia-Arcos et al., 2016). The medically proven and documented health risks and addictive behaviors of ENDS and vaping outweighs the unsubstantiated arguments of successful cessation for existing smokers.

The continuous use and exposures of the harmful products to minors may cause addiction, lead to dual use of combustible cigarettes and vape products, and damage to the developing adolescent brain. The banning of flavored tobacco products is an imperative next step in tobacco control if HB2457 becomes law.

Garcia-Arcos, I., Geraghty, P., Baumlin, N., Campos, M., Dabo, A. J., Jundi, B., … Foronjy, R. (2016). Chronic electronic cigarette exposure in mice induces features of COPD in a nicotine-dependent manner. *Thorax*, *71*(12), 1119–1129. doi: 10.1136/thoraxjnl-2015-208039

American Heart Association.

LATE

American Heart Association testimony for HB 2507, "Relating To Health"

For more information on the AHA's educational or research programs, visit www.heart.org or contact your nearest AHA office.

Office: (808) 377-6630
Fax: (808) 524-0556

Neighbor Islands:
Serviced by the Oahu office

The American Heart Association **supports the intent of** HB 2507, but **OPPOSES** the imposition of penalties for those underage who purchase of possess tobacco products.

Two new reports from the Centers for Disease Control and Prevention (CDC) found people who started smoking before age 21 are more likely to have a high nicotine dependence, and raising the age to buy tobacco to 21 impacts the sale of such products.

In one of the new reports, the CDC analyzed data from the 2014-'15 Tobacco Use Supplement from the Current Population Survey. Just over 25,000 adults answered questions about when they started smoking, their level of nicotine dependence and attempts to quit.

About half reported smoking regularly before age 18, 33% started at ages 18-20 and 17% started at 21 or older. About half reported trying to quit in the past year. Adjusted results showed those who started before age 18 and those who started at ages 18-20 were more likely to have high nicotine dependence compared to those who started at 21 or older. Those who started smoking when they were under 21 also were less likely to try to quit.

In another new study, the CDC looked at the impact of implementing Tobacco 21 (T21) laws in Hawaii in 2016 and compared it to California, which implemented a similar law the same year but with an exemption for military. It also compared Hawaii to the rest of the U.S.

Researchers used data on sales of cigarettes and cigars/cigarillos in large grocery stores from June 2012 to February 2017 and found average monthly cigarette sales in Hawaii dropped about 4.4% following the new law. California sales declined 11.7%, and mainland sales dropped 10.6%. However, Hawaii was the only one to see both a drop in sales and a drop in the share of menthol sales. Hawaii also had a 12.1% drop in cigar/cigarillo sales compared to 7.1% for California and 4.1% for the rest of the U.S. The share of menthol sales rose 7.1% in the U.S., but changes weren't statistically significant for Hawaii and California. Authors said this finding "suggests that T21 policies may have attenuated an otherwise upward trend."

"Taken together, these results indicate T21 policies may decrease sales of some tobacco products, especially cigars and flavoured/menthol products that are disproportionately consumed by youth, young adults and persons of color," authors wrote.

They also encouraged additional tobacco control measures such as increasing prices and restricting the sale of flavored products.

In addition to increasing the age of purchase, the American Heart Association notes the need to require permits/licenses for wholesalers and retailers of *all* tobacco products to allow for more effective education and enforcement against underage sales. It also recommends online sales retrictions to counteract the ability for those underage to purchase tobacco products from retailers outside the state.

The American Heart Association, however, strongly opposes youth possession and penalties (PUP) tobacco laws. The rise in PUP laws is linked to Big Tobacco's response to the Synar amendment which required states to enact and enforce laws prohibiting distribution and sale of tobacco products to minors. As states imposed restrictions on tobacco retail sales, the tobacco industry and retail merchants associations pressured lawmakers to penalize buyers and users as well as vendors.

Advocates for PUP laws hoped that the laws would play a central role in a multi-pronged approach to reducing youth initiation and smoking rates, but studies show little evidence of a deterrent effect over time.

Big Tobacco targeted youth for decades, seeking to create new generations of customers addicted to its products. Instead of holding industry and retailers accountable, PUP laws shift responsibility to their victims – young consumers who are purchasing and using a deadly and highly addictive product.

Psychologists have found that punishment is not an optimal strategy for behavior change – a finding that is even more relevant when the behavior in question is addictive. PUP laws are unlikely to reduce youth initiation and smoking prevalence at the population level. Some researchers suggest that they are counterproductive, actually increasing smoking rates among youth who seek to engage in behavior deemed deviant or behavior associated with adulthood.

PUP laws are inequitable because they disproportionately affect youth of color. Youth of color – as well as LGBT youth, youth with disabilities, and boys – are more likely to smoke because these populations have been targeted via advertising and retailer placement by the tobacco industry.

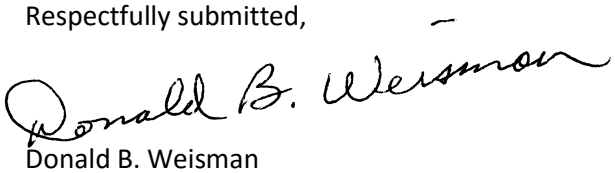
Enforcement of PUP laws also disproportionately affects youth from low-income communities. High smoking rates are correlated with low income, and there are more tobacco retailers and advertisements in less affluent areas. Consequently, low-income youth are more likely to smoke and to be affected by PUP laws. A child with a job, a single parent, or 2 parents who work outside the home may struggle to complete community service or pay fines. A child who is unable to complete community service or pay fines may be subject to escalating penalties that are increasingly difficult to resolve. Further, the resulting stress takes a toll on health and increases the likelihood of risky behaviors or involvement with juvenile justice, mental health, substance use, or other systems.

PUP laws stigmatize youth who smoke, yet smoking is an addictive behavior promoted by a billion-dollar industry that directly and deliberately targets them. Stigma is not an

effective public health intervention, and it may keep kids from seeking cessation treatment
or education. Problematic behaviors such as smoking may be more likely to continue in the face of punishment (as opposed to cessation interventions) because punishment provides an incentive to hide the
behavior and protect those engaged in it.

The American Heart Association instead recommends a focus on policies that have been proven effective in reducing youth tobacco use including increasing taxes, allocating state funding to meet the CDC's recommended level of tobacco prevention, control and cessation funding for the state, limiting sales to youth through effective enforcement of retailers for underage sales, restricting online sales, and comprehensive smoke-free air laws.

Respectfully submitted,



Donald B. Weisman
Hawaii Government Relations/Communications Director

LATE

HB-2507
Submitted on: 2/3/2020 4:22:01 PM
Testimony for HLT on 2/4/2020 8:30:00 AM

Submitted By	Organization	Testifier Position	Present at Hearing
Luke	Individual	Oppose	No

Comments:

LATE

RETAIL MERCHANTS OF HAWAII

TESTIMONY OF TINA YAMAKI
PRESIDENT
RETAIL MERCHANTS OF HAWAII
February 4, 2020

Re: HB 2507 Relating to Health

Good morning Chairperson Mizuno and members of the House Committee on Health. I am Tina Yamaki, President of the Retail Merchants of Hawaii and I appreciate this opportunity to testify.

The Retail Merchants of Hawaii (RMH) as founded in 1901 and is a statewide, not for profit trade organization committed to the growth and development of the retail industry in Hawaii.
The retail industry is one of the largest employers in the state, employing 25% of the labor force.

We OPPOSE HB 2507 Relating to Health. This measure prohibits the sale or furnishing of tobacco and tobacco products to persons under twenty-five years of age. Increases minimum age from twenty-one years of age to twenty-five years of age for purchase and possession of tobacco and tobacco products, including electronic smoking devices and increases fines.

Bans are not the simple solution and will not stop adults from smoking – noting that it is already illegal for minors. This type of ban would only push the sale of cigarettes and vaping devices into the black market and to online sales. Persons 21 years of age are deemed to be adults and should be allowed to make their own decisions when it comes to smoking or vaping.

The New England Journal of Medicine published an article last year that found that e-cigarettes were nearly twice as effective as conventional nicotine replacement products, like patches and gum, for quitting smoking. The study was conducted in Britain and funded by the National Institute for Health Research and Cancer Research UK.

We urge you to hold this measure.

Mahalo again for this opportunity to testify.

3610 Waialae Ave • Honolulu, HI 96816 (808) 592-4200 tyamaki@rmhawaii.org

HB-2507
Submitted on: 2/3/2020 5:27:58 PM
Testimony for HLT on 2/4/2020 8:30:00 AM

LATE

Submitted By	Organization	Testifier Position	Present at Hearing
Johnathon G. Myers	Individual	Oppose	No

Comments:

I am writing in STRONG OPPOSITION to this proposed Bill.

This Proposed Bill is Government overreach. The legal age to purchase tobacco products has already been raised to 21 years old, which is already impeding on adults making adult decisions for use of legal adult products. Increasing the age limit is not a viable couse of action for curbing behavior for products that are deemed legal at the State and Federal level. If you relate this to another legal adult product, alcohol, I do not see the House or Senate trying to curb the "youth epidemic" of drinking under age. Please focus on enforcement of the laws that are already on the books and propose a course of action and funding stream for enforcing the laws already in place.

Thank you for considering this testimony.

Aloha

Johnathon Myers

Concerned Constituent

DAVID Y. IGE
GOVERNOR OF HAWAII

BRUCE S. ANDERSON, PHD
DIRECTOR OF HEALTH

STATE OF HAWAII
DEPARTMENT OF HEALTH
P. O. Box 3378
Honolulu, HI 96801-3378
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LATE

Testimony in SUPPORT of H.B. 2507
RELATING TO HEALTH

REPRESENTATIVE JOHN M. MIZUNO, CHAIR
HOUSE COMMITTEE ON HEALTH
Hearing Date: February 4, 2020 Room Number: 329

Fiscal Implications: None

Department Testimony: The Department of Health (DOH) supports House Bill 2507 (H.B. 2507) that proposes to raise the minimum age for sale, purchase, and to possess tobacco and tobacco products (including electronic smoking devices) from age twenty-one years to age twenty-five years.

In 2015, the Institute of Medicine (IOM) released a study which examined the public health impact of raising the minimum age for purchase of tobacco products to either twenty-one or twenty-five years. The Committee on the Public Health Implications of Raising the Minimum Age for Purchasing Tobacco Products reviewed scientific literature across disciplines and tobacco policies, and developed mathematical modeling to quantify predictions. They found that raising the minimum age of legal access to tobacco products in the United States, particularly to ages twenty-one and twenty-five, would lead to a substantial decrease in smoking prevalence and smoking related mortality[1]. The findings from the IOM study provided DOH with the scientific evidence and justification for supporting the 2015 Hawaii legislation, Senate Bill 1030 that was enacted as Act 122, and raised the age for purchasing tobacco products to twenty-one.

The IOM report is relevant to the recommendations of H.B. 2507 to increase the minimum age of legal access to tobacco products to age twenty-five. The Food and Drug Administration (FDA) has encouraged states and localities to enact laws that are stricter than

[1] IOM (Institute of Medicine). 2015. Public health implications of raising the minimum age of legal access to tobacco products. Washington, DC: The National Academies Press.

existing federal legal age of sale of tobacco products, which was raised on December 20, 2019 to twenty-one years. The Department concurs with the description put forth in this measure's preamble describing the gravity and toll of tobacco use and addiction on this country and state. The risks of tobacco-related diseases and premature deaths would continue to be decreased. Immediate benefits from other tobacco-related health effects and consequences include inflammation and impaired immune functioning that result in hospitalizations and reduced capacity for wound healing.

The Department commends the legislature for setting bold goals and the inclusion of all tobacco products in in H.B. 2507 to protect Hawaii's youth and young adult population from initiating tobacco use of any kind. The Department does not support the increase in the possession, use, and purchase penalty for young people found in Section 2(6), page 9, lines 12 to line 14. Youth penalty is not nationally recommended because these laws punish and stigmatize young people who are aggressively marketed tobacco products while deflecting responsibility from the tobacco companies[2].

Offered Amendments: The Department suggests the following amendments to delete the increases to the possession, use, and penalty fines in Section 2(6), page 9, lines 12 to line 14.

. . . years of age who violates subsection (5) shall be fined $10 ~~$50~~ for the first offense. Any subsequent offense shall subject the violator to a fine of $50 ~~$100~~, no part of which shall be . . .

Thank you for the opportunity to testify.

[2] Bach, L. Campaign for Tobacco Free Kids. "Youth Purchase, Use, Or Possession Laws Are Not Effective Tobacco Prevention," September 20, 2018: https://www.tobaccofreekids.org/assets/factsheets/0074.pdf

LATE

Testimony of Kimo Haynes,
President of the Hawaii Petroleum Marketers Association

SUPPORTING THE INTENT OF HOUSE BILL 2507, RELATING TO HEALTH

House Committee on Health
The Honorable John Mizuno, Chair
The Honorable Bertrand Kobayashi, Vice Chair

Tuesday, February 4, 2020 at 8:30 a.m.
Hawaii State Capitol, Conference Room 329

Chair Mizuno, Vice Chair Kobayashi, and members of the Committee,

I am Kimo Haynes, president of the Hawaii Petroleum Marketers Association ("HPMA"). HPMA is a non-profit trade association comprised of members who directly market liquid motor fuel products across the Hawaiian Islands. Our membership includes individuals and companies who operate as independent marketers, jobbers or distributors of petroleum products and who buy liquid motor fuel products at the wholesale level and sell or distribute products to retail customers, other wholesalers, and other bulk consumers.

House Bill 2507, Relating to Health, prohibits the sale or furnishing of tobacco and tobacco products to persons under 25 years of age, and increases the minimum age from 21 to 25 for purchasing and possessing tobacco and tobacco products, including electronic smoking devices.

HPMA supports the intent of this bill, which seeks to further protect Hawaii's youths from the health dangers of using tobacco products by raising the minimum eligibility age to 25.

HPMA member companies have training programs and policies in place for their convenience store workforce to be disciplined in verifying the age of tobacco buyers.

Thank you for allowing HPMA the opportunity to submit written testimony on this bill.