#### NO ON 28 = YES ON 10

### Nicotine and Cocaine Use

#### An Editorial by Raymond Castellino, D.C.

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Proposition 10 was passed by the wise voters of California to add a tax of fifty cents per pack on cigarettes. These revenues are being distributed to counties for needed services for children ages from conception to five year old and their families. Proposition 10 is among the most exciting outcomes of the democratic process in recent times.

Proposition 28 was written to repeal the cigarette tax. Some of its proponents mistakenly contend that this money would better be spent on adults with smoking related illnesses. They imply there is no or little relationship between cigarette smoking and children.

Let us set the record straight!

The first five years of a child's life, especially the prenatal and infant periods, are the most crucial for brain and nervous system development. Smoking inhibits a prenate's growth and can cause congenital changes that affect a person for the rest of his or her life.

Approximately 25% of all pregnant mothers in this country smoke. Many more experience the effects of secondhand smoke. When a baby's mother smokes or is present to secondhand smoke, her baby becomes a captive passive smoker. Nicotine, carbon monoxide and other toxins enter the mother's blood and deny necessary oxygen to the baby's developing body. Tobacco smoke is known to stunt fetal growth, cause learning defects and brain damage. In addition, prenatal and infant death rates are higher in babies of smoking mothers. These babies are more susceptible to smoke related diseases including lung problems, asthma, heart disease and sudden infant death syndrome (SDS).

Any time a pregnant mom is exposed to tobacco smoke, either by smoking herself or by breathing secondhand smoke, she and her baby are exposed to these risks.

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Recent studies show that cocaine and nicotine have similar effects on the fetus.

Surprisingly, the difference is that effects from cocaine are shorter lived than those of nicotine.

The effects of nicotine actually appear to be worse. Nearly all crack cocaine users smoke tobacco and use alcohol. Evidence shows that the detrimental effects of these drugs on babies born to mothers who use crack cocaine are from the combined effects of tobacco, cocaine and alcohol use. While tobacco and alcohol are legal substances, all three pose serious health and social problems. A higher percentage of our population smokes cigarettes. Smoking cigarettes affects far more babies than cocaine use. (See the web site from Duke University at:

<a href="http://www.dukenews.duke.edu/nicotine/slotkin.htm">http://www.dukenews.duke.edu/nicotine/slotkin.htm</a> for scientific evidence. See the article below.)

Smoking affects everyone from the earliest prenate to the elderly. Prop 10 is designed to eliminate smoking and improve our health. Proposition 10 puts substantial sums of money into local hands to enrich the lives of pre-born babies, children and families across all walks of life. Prop 10 is for our children and grandchildren.

# VOTE NO ON PROP 28! NO ON 28 = YES ON 10! DO IT FOR THE CHILDREN!

Respectfully, Raymond Castellino, D.C.

Santa Barbara

Raymond Castellino is an internationally known teacher in the field of pre- and perinatal psychology and health. He is Executive Director of BEBA (Birthing Evolution – Birthing Awareness), a non profit family research clinic in Santa Barbara, California dedicated to helping babies and families heal from prenatal and birth-related stress and trauma.

## Fetal nicotine exposure

http://www.nietrokers.nl/e/g02109.html

The following article by Dr. Theodore Slotkin describes his research using nicotine injections into pregnant rats. They were

using only nicotine, so all the other poisons that are transplacental to the fetus were not used during the studies. Even at levels of nicotine equivalent to women smoking low enough not to affect birth weight (only a few cigarets a day), the brains of rat pups were changed. One of the concerns about smoking during pregnancy is lower birth weight and all of the adverse effects that result from that.

His conclusion if applied to humans is that nicotine by itself lowers IQ, increases AD&HD (Attention Deficit & Hyperactivity Disorder) and that could increase bad behavior plus the number of people with low IQ's, and lower the number of geniuses.

There have been other studies saying that IQ is lower in children of smokers, and one study recently says that tobacco smoking creates criminals in the womb. Dr. Joe DiFranzia in Boston did research in which women were tested for pregnancy every week. He figures that among US smoking women, there are an additional 190,000 miscarriages a year because of their smoking.

Fetal nicotine or cocaine exposure: which one is worse? Despite extensive adverse publicity, tobacco use continues in approximately 25% of all pregnancies in the United States, overshadowing illicit drugs of abuse, including cocaine. The societal cost of maternal smoking is seen most readily in underweight newborns, in high rates of perinatal morbidity, mortality and Sudden Infant Death Syndrome and in persistent deficits in learning and behavior.

We have designed animal models of nicotine exposure to prove that nicotine itself is a neuroteratogen, thus providing a causative link between tobacco exposure and adverse perinatal outcomes. In particular, nicotine infusion paradigms that, like the transdermal patch used in man, produce drug exposure without the confounds of other components of tobacco or of episodic hypoxic-ischemic insult, have enabled a mechanistic dissection of the role played by nicotine in fetal brain damage. Nicotine targets specific neurotransmitter receptors in the fetal brain, eliciting abnormalities of cell proliferation and differentiation, leading to shortfalls in the number of cells and eventually to altered synaptic activity. Because of the close regulatory association of cholinergic and catecholaminergic systems, adverse effects of nicotine involve multiple transmitter pathways and influence not only the immediate developmental events in fetal brain, but also the eventual programming of synaptic competence. Accordingly, defects may appear after a prolonged period of apparent normality, leading to cognitive and learning defects that appear in childhood or adolescence.

Comparable alterations occur in peripheral autonomic pathways, leading to increased susceptibility to hypoxia-induced brain damage, perinatal mortality and Sudden Infant Death. Identifying the receptor-driven mechanisms that underlie the neurobehavioral damage caused by fetal nicotine exposure provides a rational basis for decisions about nicotine substitution therapy for smoking cessation in pregnancy. In contrast to the effects of nicotine, animal models of crack cocaine use in pregnancy indicate a more restricted spectrum of effects, a reflection of differences both in pharmacokinetics and pharmacodynamics of the two drugs.

Notably, although cocaine, like nicotine, also targets cell replication, its effects are short-lived, permitting recovery to occur in between doses, so that the eventual consequences are much less severe. To some extent, the effects of cocaine on brain development resemble those of nicotine because the two share cardiovascular actions (vasoconstriction) that, under some circumstances, elicit fetal hypoxia-ischemia. In light of the fact that nearly all crack cocaine users smoke cigarettes, the identification of specific developmental effects of cocaine may prove difficult to detect.

Although scientists and the public continue to pay far more attention to fetal cocaine effects than to those of nicotine or tobacco use, a change of focus to concentrate on tobacco could have a disproportionately larger impact on human health.

Dr T.A. Slotkin (<u>t.slotkin@duke.edu</u>) Duke University Medical Center, Durham, North Carolina, USA J Pharmacol Exp Ther 1998 Jun;285(3):931-45