Testimony - 03-31-2016 - VT House, on the legalization of the smoking of marijuana bill.

1st part - for those that VOTE YES, and support the VT law, as proposed, passed by the VT Senate, that allows the SMOKING of Marijuana (even allowing maybe your fellow legislators to LIGHT UP in this very chamber (and, expose others to the 2nd hand smoke cancer risk)... Please consider voting NO instead.

However, IF you VOTE YES, for legalizing the smoking of marijuana in VT, then please sign this pledge:

I, _____ swear, that by voting for this law allowing the smoking of marijuana, that I know of evidence of the fact that California Prop 65 list marijuana smoke as a cause of cancer (both for the user and due to 2nd hand smoke exposure), that I will:

- will pay 100% out of my own private pocket the full health care related costs (co-pay, and/or out of pocket costs potentially around $12,000 per year for a family, **for multiple years**), and will not pass costs on to tax payers (medicaid, or single payer) or via premium charges (any exchange health care system).
- will visit ALL of those with the resulting cancer in the cancer wards, or hospice, at least once per week.
- will pay all sickness or death lost income related costs of any citizen or business, due to the cancer.
- will pay any and all sickness or death cause potential bankruptcy costs (personal or small business)
- will go to all resulting funerals, and will pay for all funeral costs.
- will visit the grieving families yearly, incl all children, explaining how sorry you are for their loss.

X SIGNED: _____ DATE: _____

2nd Part, Dear VT Legislators: In your consideration of supporting the legalization of the SMOKING of marijuana in Vermont, DID YOU KNOW, that in 2009, that California (via a annual investigation and response to a Prop 65 citizen mandate) listed marijuana smoke as known carcinogen (for both user, and 2nd hand smoke), & where California stated that pot smoke shares 33 carcinogens with tobacco smoke? See:

http://oehha.ca.gov/prop65/docs_state/mjcrnr061909.html

Why create a law for pot smokers, where the SMOKING of marijuana, causes cancer, AND where other methods of use, that might not carry the same cancer risk that smoking does (are not considered as an more sensible, if proven safe, alternative)? Just the formaldehyde in pot smoke (Prop 65 long list of what is in pot smoke) is a world-wide known carcinogen.

Does the law even have a package labeling requirement for cancer & other health risk WARNINGS (where, the irony is, that all cancer causing tobacco products have this warning, and GMOs will have a place on labeling too, but not a warning for use of marijuana)?

Suggestion: Keep smoking of weed (plant burning of pectin) NOT legal, Where instead of traditional criminal penalty, then offenders of the law suffer a another penalty, where they be required to attend serious heath education that focuses on all the psycological and physiological risks, AND where they need to suffer MORE via a **MANDATORY long community service sentence, where the lengthy time served is performed at, both, a cancer ward and at a hospice** (so that users can see where they will end up at if they continue to smoke pot - and for kids if they get caught smoking tobacco, the same penalty).

The sad thing is, that all the taxes the state hopes to collect with current version of the law (with smoking pot as legal), will not pay for the many years of chemotherapy (at least $7,000 per treatment per Doug Baker who once served in this building) and other very expensive treatments... leaving non-user tax payers to pay via taxes that pay the the medicaid costs, or for non-user premium payers to pay for the pot smoker with cancer that has other insurance. That is not fair, make them pay for their own expensive treatments... same today that non-users of tobacco have to pay for the high cost of all the diseases that smoking tobacco causes (given that pot shares 33 of the same carcinogens, and the lack of research due to pot being not legal, we might find out that pot smoking causes more than just cancer).

-Here is the FULL Prop 65 (citizen mandated list) updated 12/2015

http://oehha.ca.gov/prop65/prop65_list/files/P65single061909.pdf

-- AND, Here is the 156 page, (pdf file link below) key document.
Titled - "EVIDENCE ON THE CARCINOGENICITY OF Marijuana Smoke"
Author - Reproductive and Cancer Hazard Assessment Branch Office of Environmental Health Hazard Assessment (California Environmental Protection Agency).

http://oehha.ca.gov/prop65/hazard_ident/pdf_zip/FinalMJsmokeHID.pdf

> Quote: "In summary, there is some evidence from studies in humans that marijuana smoke is associated with increased cancer risk. Studies in animals also provide some evidence that marijuana smoke induces tumors, with benign and malignant tumors observed in rats exposed via inhalation, malignant tumors in rats exposed via subcutaneous injection as newborns, and benign tumors in mice exposed dermally. Studies investigating the genotoxicity, immunotoxicity, and effects on endocrine function and cell signaling pathways provide additional evidence for the carcinogenicity of marijuana smoke. Finally, the similarities in chemical composition and in toxicological activity between marijuana smoke and tobacco smoke, and the presence of numerous carcinogens in marijuana (and tobacco) smoke, provide additional evidence of carcinogenicity".

There is no other way to say it, or write it, given the evidence, voting FOR allowing people to SMOKE (pot or tobacco), is voting for them to die (it is, at the least, a long term death by cancer sentence). It is morally wrong to think up ANY state legalized excuse to kill anyone. WRONG, to brush off their ultimate slow, painful, and disgusting death, in a way that their cancer death, becomes for them, and their loved ones, state supported, politically excusable collateral damage statistic (allowable by law, approved by legislators, signed by the governor), where those voting for the cancer causing law dismiss the importance of life, where instead, a citizen(s) cancer, or other pot smoking death(s), is OK due to a "greater good" excuse.

The Nazis used the same greater good logic to rationalize their "quick and easy" solution to a "perceived" problem (albeit their point of view, using their greater good logic, was far, far, more twisted, for sure).

This pot smoking bill is dollar driven eminent domain logic by the state. The state will profit due to taxes, others will profit growing/selling the pot, creating more jobs (death jobs). Where the state is, ultimately, given the evidence, validating, the taking away, of life itself. In all ways, history will see this as wrong.

Heck, why not carpet bomb them all, or increase speed limits to 100 MPH, because folks want drive fast?

Hopefully, we are a better society than that. Please do not pass pot smoking legalization bill, vote NO).

From: http://oehha.ca.gov/prop65/hazard_ident/pdf_zip/FinalMJsmokeHID.pdf

EVIDENCE ON THE CARCINOGENICITY OF
Marijuana Smoke

Reproductive and Cancer Hazard Assessment Branch
Office of Environmental Health Hazard Assessment
California Environmental Protection Agency

5.2 Conclusion

There is evidence from some epidemiological studies of marijuana smoke suggestive of increased cancer risk from both direct and parental marijuana smoking. However, this evidence is limited by validity issues and small numbers of studies for most types of cancer.

Direct marijuana smoking has been statistically significantly associated with cancer of the lung, head and neck, bladder, brain, and testis.

Parental marijuana smoking before or during gestation has been statistically significantly associated with childhood cancer.

- Childhood cancers that have been **associated with maternal marijuana smoking are acute myeloid leukemia, neuroblastoma, and rhabdomyosarcoma**.
- Childhood cancers that have been **associated with paternal marijuana smoking are leukemia (all types), infant leukemia (all types), acute lymphoblastic leukemia, acute myeloid leukemia, and rhabdomyosarcoma.**

In animal studies, increases in squamous cell papilloma of the skin were reported in mice exposed dermally to marijuana smoke condensate. **Malignant mesenchimatous tumors were reported following six subcutaneous injections of marijuana smoke condensate to newborn rats**.

In a marijuana smoke inhalation study in female rats, benign tumors of the ovary and **benign and malignant tumors of the uterus were observed**.

There is evidence that **marijuana smoke is genotoxic, immunosuppressive, and can alter endocrine function**. Studies of Δ 9 -THC and other cannabinoids provide evidence for alterations of multiple cell signaling pathways, in endocrine function, and suppression of the innate and adaptive immune response. **Prolonged exposures to marijuana smoke in animals and humans cause proliferative and inflammatory lesions in the lung.**

Marijuana smoke and tobacco smoke share many characteristics with regard to chemical composition and toxicological activity. Tobacco smoke is a Proposition 65 carcinogen, and **at least 33 individual constituents present in both marijuana smoke and tobacco smoke are Proposition 65 carcinogens**.

California Prop 65 - lists marijuana smoke as a carcinogen (shares 33 of the same carcinogens as tobacco smoke, but due to more tar, carries these more effectively into the lungs).

EVIDENCE ON THE CARCINOGENICITY OF

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August 2009

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Per this document Marijuana smoke contains both Methanol and Formaldehyde, both are carcinogenic.

Reproductive and Cancer Hazard Assessment Branch
Office of Environmental Health Hazard Assessment

California Environmental Protection Agency

1. EXECUTIVE SUMMARY

Marijuana smoke is formed when the dried flowers, leaves, stems, seeds and resins of plants in the genus *Cannabis* are burned. Marijuana smoke aerosol contains thousands of organic and inorganic chemicals, including psychoactive cannabinoids, which are unique to *Cannabis* plants. Inhaling marijuana smoke for its psychotropic properties became popular in western cultures in the 1960s, though marijuana has been used for medicinal and psychotropic purposes in other parts of the world for thousands of years. In California, use of marijuana for physician-recommended purposes has been legal under state law since 1996 when Proposition 215, the Compassionate Use Act, was passed by state voters. However, the vast majority of marijuana use continues to be for recreational purposes, which remains illegal.

Marijuana smoke and tobacco smoke share many characteristics with regard to chemical composition and toxicological properties. At least 33 individual constituents present in both marijuana smoke and tobacco smoke are already listed as carcinogens under Proposition 65.

In examining the potential carcinogenicity of marijuana smoke, a range of information was evaluated. Studies of cancer risk in humans and laboratory animals exposed to marijuana smoke were reviewed. Other relevant data, including studies investigating genotoxicity and effects on endocrine function, cell signaling pathways, and immune function caused by marijuana smoke, were all considered. Also of interest were the similarities in chemical composition and in toxicological properties between marijuana smoke and tobacco smoke, and the presence of numerous carcinogens in marijuana smoke. The findings of all these reviews are summarized below.

There is evidence from some epidemiological studies of people exposed to marijuana smoke suggestive of increased cancer risk from both direct and parental marijuana smoking. However, this evidence is limited by potential biases and small numbers of studies for most types of cancer. Studies reporting results for direct marijuana smoking have observed statistically significant associations with cancers of the lung, head and neck, bladder, brain, and testis. The strongest evidence of a causal association was for head and neck cancer, with two of four studies reporting statistically significant associations. The evidence was less strong but suggestive for lung cancer, with one of three studies conducted in populations that did not mix marijuana and tobacco reporting a significant association. Suggestive evidence also was seen for bladder cancer, with one of two studies reporting a significant association. For brain and testicular cancers, the single studies conducted of each of these endpoints reported significant associations. Among the epidemiological studies that reported results for parental marijuana smoking and childhood cancer, five of six found statistically significant associations. Maternal and paternal marijuana smoking were implicated, depending on the type of cancer. Childhood cancers that have been associated with maternal marijuana smoking are acute myeloid leukemia, neuroblastoma, and rhabdomyosarcoma. Childhood cancers that have been associated with paternal marijuana smoking are leukemia (all types), infant leukemia (all types), acute lymphoblastic leukemia, acute myeloid leukemia, and rhabdomyosarcoma.

A limitation common to the epidemiologic studies was potential bias from under-reporting of marijuana smoking due to its illegality, social stigma, lack of privacy during oral interviews, and

subject desire to please interviewers, and possibly different degrees of under-reporting between cancer patients and healthy controls. Another limitation of several studies was that they were conducted in geographic locations where marijuana and tobacco are commonly mixed before smoking (e.g., three of six lung cancer studies and one of two bladder cancer studies were conducted in northern Africa, and two of four oral cancer studies were conducted in England). Thus, the results of those studies may have been confounded by the effects of exposure to tobacco smoke.

In animal studies, increases in squamous cell papilloma of the skin were reported in mice exposed dermally to marijuana smoke condensate. Malignant mesenchymatous tumors were reported following six subcutaneous injections of marijuana smoke condensate to newborn rats. In a marijuana smoke inhalation study in female rats, benign tumors of the ovary (serous cytoma and follicular cysts) and benign and malignant tumors of the uterus (adenofibroma, adenosarcoma, and telengiectatic cyst and polyps) were observed. Marijuana smoke condensate also exhibited tumor promoting activity in a mouse skin tumor initiation-promotion assay.

Evidence indicating that marijuana smoke is genotoxic includes findings that marijuana smoke induces mutations in *Salmonella,* and several small cytogenetic studies in humans suggesting that exposure to marijuana smoke may be associated with increased mutations and chromosomal abnormalities. While the data on the genotoxicity of marijuana smoke *per se* are limited, many individual smoke constituents have been shown to form DNA adducts, induce gene mutations, and damage chromosomes.

Evidence indicating that marijuana smoke alters endocrine function includes findings for a number of different hormonal pathways. Marijuana smoke condensate has been shown to have estrogenic effects, including findings that it can activate the estrogen receptor (ER). Marijuana smoke also has been shown to have anti-estrogenic effects, through the induction of cytochrome P450 1A1 and the resultant increase in estrogen (E2) metabolism and through the inhibition of aromatase, an enzyme that converts testosterone to E2. Other studies indicate that marijuana smoke condensate has anti-androgenic effects, inhibiting binding of dihydrotestosterone (DHT) to the androgen receptor (AR). Studies of Δ^9-tetrahydrocannabinol (Δ^9-THC) and other cannabinoids provide evidence for disruption of the hypothalamic-pituitary-gonadal axis, including evidence that Δ^9-THC inhibits the release of follicle stimulating hormone, luteinizing hormone, prolactin, growth hormone, thyroid-stimulating hormone, and corticotrophin. These alterations in endocrine function can affect the growth of hormone responsive tissues, and might increase the risk of certain cancers (e.g., testes, ovary, uterus, and breast).

Evidence suggesting that marijuana smoke alters cell signaling pathways involved in cell cycle control comes from studies of the effects of Δ^9-THC and other cannabinoids on protein kinases. Depending upon the cell type and the dose administered, Δ^9-THC and other cannabinoids may either stimulate or inhibit cell proliferation.

There is evidence that marijuana smoke suppresses the innate and adaptive immune response. The bactericidal activity of rat alveolar macrophages was reduced by marijuana smoke *in vivo* and *in vitro.* Tumoricidal and bactericidal activities were reduced in alveolar macrophages from marijuana smokers, compared to non-smokers. In addition, in one study smoking marijuana was

associated with a more rapid progression of human immunodeficiency virus infection to acquired immunodeficiency syndrome. Δ^9-THC and other cannabinoids present in marijuana smoke have also been shown to suppress host resistance to microbial infection, macrophage function, natural killer and T cell cytolytic activity, cytokine production by macrophages and T cells, and to decrease antigen presentation by dendritic cells. These immunosuppressive effects could lead to an increased risk of cancer by reducing immunosurveillance capacity against neoplastic cells.

Prolonged exposures to marijuana smoke in animals and humans cause proliferative and inflammatory lesions in the lung, such as cellular disorganization, squamous metaplasia, and hyperplasia of basal and goblet cells (observed in the bronchial epithelial tissues of marijuana smokers).

In summary, there is some evidence from studies in humans that marijuana smoke is associated with increased cancer risk. Studies in animals also provide some evidence that marijuana smoke induces tumors, with benign and malignant tumors observed in rats exposed via inhalation, malignant tumors in rats exposed via subcutaneous injection as newborns, and benign tumors in mice exposed dermally. Studies investigating the genotoxicity, immunotoxicity, and effects on endocrine function and cell signaling pathways provide additional evidence for the carcinogenicity of marijuana smoke. Finally, the similarities in chemical composition and in toxicological activity between marijuana smoke and tobacco smoke, and the presence of numerous carcinogens in marijuana (and tobacco) smoke, provide additional evidence of carcinogenicity.

2. INTRODUCTION

2.1 Identity of Marijuana Smoke

Marijuana smoke is formed when the dried flowers, leaves, stems, seeds and resins of plants in the genus *Cannabis* are burned. *Cannabis sativa* and *Cannabis indica* are the species most commonly smoked. The following is a list of common marijuana plant products that are smoked:

- Bud. The flower tops of unpollinated female marijuana plants. Buds have the highest Δ^9-tetrahydrocannabinol (Δ^9-THC) content of all parts of the plant. Bud is probably the most common form of marijuana smoked currently in the U.S.
- Ganja (India); kif, kief, kef, keef (Morocco and Algeria); tekrouri, takrouri (Tunisia); and dagga (southern Africa). A mixture of flowering tops and leaves from female plants, dried and diced or powdered.
- Hashish (Middle East) and charas (Far East). Crude resin from flowering tops of unfertilized female marijuana plants. Processed differently in different parts of the world. Often collected by rubbing onto hands, cloth, or leather jackets, or by sifting. Compressed into blocks.
- Leaf. Less potent than buds or flower tops with regard to Δ^9-THC content, leaves were commonly smoked in the U.S. when marijuana first became popular in the 1960s and 1970s.

- Bhang (India and Bangladesh). Generally prepared from the leaves of male plants. Most often used for making beverages but sometimes smoked.

Marijuana smoke contains several thousand different compounds (Sparacino *et al.*, 1990). Some are released unchanged from the plant material as it burns, and the rest are products of either pyrolysis or incomplete combustion. Marijuana smoke consists of some chemicals present in the gas phase, some present in particulate matter, and some semi-volatile compounds that transition between the gas and particulate phase. Marijuana smoke includes a large variety of organic and inorganic chemicals, including amines, aromatic amines, aza-arenes, polycyclic aromatic hydrocarbons (PAHs), carbonyls, phenolics, pyrazines, pyrimidines, pyrroles, pyridines, isoxazoles, metals (arsenic, cadmium, chromium, lead, nickel, and selenium), hydrogen cyanide, carbon monoxide (CO), nitric oxide (NO), other nitrogen oxides (NO_x), ammonia, and over 60 cannabinoid compounds (Hoffmann *et al.,* 1975; Lee *et al.*, 1976; Sparacino *et al.*, 1990; Moir *et al.*, 2008).

Phytocannabinoid compounds are present in plants in the genus *Cannabis.* They are terpenophenolic compounds, commonly containing 21 carbons. The major cannabinoids present in marijuana smoke are Δ^9-THC, which is the most potent psychoactive compound present in marijuana (ELSohly, 2002), Δ^8 - THC, cannabinol (CBN), cannabidiol (CBD), cannabichromine and 11-OH-Δ^9 -THC. In the past, the levels of Δ^9-THC in marijuana smoked in the U.S. typically ranged from 1-3%. However, over the last 20 years levels of Δ^9-THC have been increasing as a result of the selective cultivation of plants. Typical levels of Δ^9-THC are now greater than 6%. Addition of hashish oil (a cannabinoid-rich extract from *Cannabis* plant material) to the dried material can boost Δ^9-THC levels even higher (e.g., 20%).

Approximately 350 of the thousands of chemicals present in marijuana smoke have been identified by various investigators (Moir *et al.*, 2008, Gieringer *et al.*, 2004, Sparacino *et al.*, 1990; Hoffmann *et al.*, 1975; Lee *et al.*, 1976). These are shown in Table 1 below. Five main studies of the major constituents present in marijuana smoke were designed as follows:

- Moir *et al.* (2008) used standardized marijuana, which was harvested in May 2004 and produced by Prairie Plant Systems Inc., of Saskatoon, Canada, for Health Canada. The material tested consisted of flowering heads only (reference: H55-MS17/338-FH). Smoke was generated using a smoking machine, operating under two different smoking conditions. The first smoking condition involved a puff volume of 35 milliliters (ml), a puff duration of two seconds, and a puff interval of sixty seconds, while the second smoking condition, referred to as 'extreme,' involved a puff volume of 70 ml, a puff duration of two seconds, and a puff interval of 30 seconds.
- Gieringer *et al.* (2004) used standard National Institute on Drug Abuse (NIDA) marijuana obtained from an independent laboratory. The mean Δ^9-THC content was 4.15%. Smoke was generated by combusting the marijuana in a glass pipe bowl, and collected in a volatile gas trap.
- Sparacino *et al.* (1990) generated marijuana smoke from two samples of Mexican marijuana, one with a "low" Δ^9-THC content (1.3%) and another with a "high" Δ^9-THC content (4.4%). Smoking machines employed either a constant draft apparatus, or an intermittent puff smoking system.

- Hoffmann *et al.* (1975) analyzed marijuana leaves obtained from the Division of Cancer Cause and Prevention of the National Cancer Institute (NCI). The NCI material was prepared from confiscated Mexican marijuana. The low concentration of Δ^9-THC (0.61%) in the marijuana suggested to Hoffmann *et al.* (1975) that the material had been diluted with domestic marijuana. Smoke was generated using a smoking machine.
- Lee *et al.* (1976) obtained Mexican marijuana containing 2.8% Δ^9-THC from the National Institute of Mental Health, in Rockville, Maryland, and generated smoke using a smoking machine under conditions simulating that of an average tobacco cigarette smoker.

Differences in the analytical methods (e.g., sample preparation and fractionation, instrumentation, limit of detection) employed in these studies preclude reaching any conclusions regarding the comparability of marijuana smoke constituents from different samples of marijuana.

Many of the chemical constituents that have been identified in marijuana smoke are carcinogens. The following 33 marijuana smoke constituents included in Table 1 are listed under Proposition 65 as causing cancer: acetaldehyde, acetamide, acrylonitrile, 4-aminobiphenyl, arsenic, benz[*a*]anthracene, benzene, benzo[*a*]pyrene, benzo[*b*]fluoranthene, benzo[*j*]fluoranthene, benzo[*k*]fluoranthene, benzofuran, 1,3-butadiene, cadmium, carbazole, catechol, chromium (hexavalent compounds), chrysene, dibenz[*a,h*]anthracene, dibenz[*a,i*]pyrene, dibenzo[*a,e*]pyrene, diethylnitrosamine, dimethylnitrosamine, formaldehyde, indeno[*1,2,3,-c,d*]pyrene, isoprene, lead, mercury, 5-methylchrysene, naphthalene, nickel, pyridine, and quinoline.

Table 1. Chemicals detected in marijuana smoke.

acenaphthene
acenaphthylene
acetaldehyde
acetamide
acetone
8-acetoxy-pyrazolobenzo-as-triazine
3-acetylpyridine
acrolein
acrylonitrile
alkyl nitrile
aminobenzamide
3-aminobiphenyl
4-aminobiphenyl
aminodimethylpyrimidine
aminodiphenylene oxide
aminomethylquinoline
1-aminonaphthalene
2-aminonaphthalene
m-aminophenol
aminoquinoline
β-amiryn
ammonia
anthanthrene
anthracene
arsenic
1-azidonaphthalene
1,2,3,3a,4,5,6,7, 5-azulenemethanol
benz[*a*]anthracene
benzacenaphthylene
benzene
benzeneacetonitrile
1,2-benzenedicarboxylic acid, bis (2)
1,2-benzenediol
1,3-benzenediol, 2-(3,7-dimethyl-2
benzimidazole
benzo[*a*]fluorene
benzo[*a*]pyrene
benzo[*b*]fluoranthene
benzo[*b*]fluorene
benzo[*c*]fluorene
benzo[*e*]pyrene
benzo[*g,h,i*]perylene
benzo[*j*]fluoranthene
benzo[*k*]fluoranthene
benzofluoranthene
benzofuran
2H-1-benzopyran-5-ol, 2-methyl-2-(4
1,4-benzoquinone
benzyl acetate
benzyl acetophenone
N-benzyl-4-aminobutyronitrile
binaphthyl
α-bisabolol
1,3-butadiene
1-butoxy-2-propanol
tert-butyl-parahydroxybenzoate
butyraldehyde
butyroamide
cadmium
caffeine
DL-cannabichromene
cannabinol (CBN)
carbazole
β-carboline
carbon monoxide (CO)
caryophyllene
caryophyllene oxide
catechol
1-chloro-octadecane
cholesta-3,5-dien-7-one
cholesterol
cholesteryl acetate
chromium
chrysene
m,o,p-cresol
crotonaldehyde
p-cumyl phenol
cyclododecane
cyclohexadecane
4*H*-cyclopenta[*d,e,f*]phenanthrene
cyclopentadiene
1a,2,3,1H-cyclopropa[a]naphthalene
cyclopropanenanoic acid, 2-[(2-bu
4,7,10-cycloundecatriene
decahydro-4a-methyl-1-naphthalene
1-decanol
1-decene
dibenz[*a,h*]anthracene
dibenz[*a,i*]anthracene
dibenz[*a,i*]pyrene
dibenzo[*a,e*]pyrene
dibenzofuran
d-dibenzopyrene
dibutyl phthalate
diethyl biphenyl
2,2'-diethyl-1,1'-biphenyl
diethylnitrosamine
diethylphenylene diamine
1,2-dihydro-3-isobutyl-1-methylpyrazine-2-one
2,3-dihydrobenzofuran
dihydroxymethyl phenyl quinazoline
2,3-dihyroxyprohexadeacanoic acid
dimethoxybenzene isomer
dimethyl naphthyridine
dimethyl tetrazine
7,11-dimethyl-1,6,10-dodetatriene
dimethylbenzimidazole
3,4-dimethylbenzoic acid
3,3-dimethylcyclobutane-carbonitrile
10,10-dimethylenebicyc
dimethylethanamide imidazole
dimethylethylpyrrole
1-(1,5-dimethylhexyl) cyclohexane
1,2-dimethylimidazole
N,N-dimethyl-N-(p-methoxyphenyl) formamide
N,N'-dimethyl-N,N'-diethyl-p-phenylene diamine
dimethylnaphtho(2,3,6-)thiophene
dimethylnaphthyridine
dimethylnitrosamine
3,3-dimethyloxetase
2,4-dimethylphenol
2,5-dimethylphenol
dimethylpiperazine
dimethylpyrimidone

2,4-dimethylquinazoline
dimethyltrisulfide
dimethyl-β-carboline isomer
dioctyl phthalate
diphenylamine
diphenylethyne
diphenylpyridine isomer
2,6-diterbutylnaphthalene
ditolyl ethane
docosane
2-dodecen-1-yl (-)succinic anhydride
5-dodecyldihydro-2 (3H)-furanone
dronabinol (THC)
eicosane
(E)-3-eicosene
3-eicosene
ethoxy benzaldehyde
ethoxyquinazoline
ethyl hydroxyl acetophenone
ethyl-4*H*-cyclopenta[*d,e,f*]phenanthrene
ethylbinaphthyl
ethylindole
ethylmethylbiphenyl
ethylphenol, 4-
fluoranthene
fluorine
formaldehyde
glaucyl alcohol
heneicosane
henricosyl formate, 1-
heptacosane
heptadecane
2-heptadecanol
2-heptadecanone
hexacosane
hexadecanal
hexadecanamide
hexadecane
(Z)-3-hexadecane
hexadecanoic acid
hexadecanoic acid, hexadecyl ester
1-hexadecanol
2-hexadecanol
n-hexadecanol
cis-11-hexadecen-1-yl acetate
9-hexadecenoic acid eicosyl
9-hexadecenoic acid eicosyl ester
hexanedioic acid dioctyl ester
hexanenitrile 3(pyrrolidnylmethylene)
2-hexyl-1-decanol
hydrogen cyanide
hydroquinone
5-hydroxyindole
hydroxymethylquinoline
4,5,6,7-1H-indazole
indeno[*1,2,3,-c,d*]pyrene
indole
isoprene
lead
2-p-mentha-1,8-dien-3-y resocinol
mercury
1H-3a,7-methanoazulene, octahydro-1
methanol
methoxy propyl pyrazine
2-methoxy-3-methylpyrazine
methoxybenzaldehyde
methyl acetyl pyrrole
methyl benzimidazole
3-methyl benzoic acid
4-methyl carbostyril
methyl ethyl ketone
methyl ethyl pyrazine
methyl ethyl pyrrole
1-methyl imidazole
methyl palmitate
methyl phenyl cinnoline
methyl pyridine carboxylic acid
methyl pyrimidine
methyl stearate
16-methyl-, met heptadecanoic acid
2-methyl-1,4-benzenedoil
3-methyl-1,8-naphthyridine
2-methyl-1-hexadecanol
1-methyl-1H-indene
3-methyl-1H-indole
4-methyl-1H-indole
N-methyl-2-pyridinamine
1-methyl-4-(5-methyl-1-cylohexene
3-methyl-4-ethylpyrrole
3-methyl-5-triazolo(4,3-a)pyrazine
methylacenaphthylene
methylaminonaphthyridine
1-methylanthracene
2-methylanthracene
10-methylbenz[*a*]anthracene
2-methylbenz[*a*]anthracene
3-methylbenz[*a*]anthracene
4-methylbenz[*a*]anthracene
5-methylbenz[*a*]anthracene
6-methylbenz[*a*]anthracene
8-methylbenz[*a*]anthracene
9-methylbenz[*a*]anthracene
methylbenzoxazole
methylbinaphthyl
methylcarbazole
1-methylchrysene
2-methylchrysene
3-methylchrysene
5-methylchrysene
6-methylchrysene
N-methyldiphenylamine
methylethylnitrosamine
1-methylfluoranthene
2-methylfluoranthene
3-methylfluoranthene
7-methylfluoranthene
8-methylfluoranthene
1-methylfluorene
2-methylfluorene
2-methylfuran
3-methylheneicosane
methylindole
methyl-n-(pyrid-2-yl) dihydropyrrole
N-methyl-N-[4-[4-4-methoxy acetamide
N-methyl-N-[4[4-methoxy-acetamide
1-methylnaphthalene
2-methylnaphthalene
1-methylphenanthrene
2-methylphenanthrene
3-methylphenanthrene
9-methylphenanthrene

1-methylphenazine
methylphenyl quinoxaline
methylpropionyl furan
methyl-pteridinone isomer
methylpyrazine
1-methylpyrene
2-methylpyrene
4-methylpyrene
methylpyriloindole
methylquinoline
methylthiazolopyrimidine
methylthiopyridine
1-methyl-β-carboline
naphthalene
naptho-sydinone
nickel
nitric oxide (NO)
nitroacetanilide
nitrogen oxides (NO_x)
nitropicoline
nonacosane
nonadecane
nonadecene
1-nonadecene
octacosane
octadecane
1-octadecanethiol
2,3-octadecanoic acid, dihydroxypro
1-octadecene
5-octadecene
1,2,3,5,6,7,8,8a-octanaphthalene
1-octdecanethiol
6-octen-1-ol, 3,7-dimethyl acetate
1,1'-oxybis-octane
pentacosane
pentadecane
pentadecanoic acid
1-pentadecene
pentyl cannabinol, 3-n-
3-n-pentyl-delta-9-tetrahydrocannabinol
perylene
phenanthrene
1,2,1-phenanthrenecarboxylic acid
1-phenantthrenecarboxylic acid, 7-et
phenoxy ethanol
N-phenyl acrylamide
phenyl alcohol
phenyl benzothiazole
1-phenyl decane
phenyl methyl quanidine
phenyl methyl urea
phenyl pyrazoline
phenyl pyridine
phenyl urea
phenylbenzimidazole
(α-picolidene)-n-propylamine, N-
α-picoline
2-pmemtha-1,8-dien-3-y-resorcinol
propionaldehyde
propionamide
2-(propylamino)benzothiazole propylbenzimidazole
pyrene
pyridine
quaterphenyl
quaterphenyl diphenylacenaphtylene
quinoline
resorcinol
selenium
squalene
styrene
tetracosane
tetradecanoic acid
2- (tetradecyloxy)-ethanol
Δ,8-tetrahydrocannabinol
Δ,9-tetrahydrocannabinol
tetramethylcyclopentanedione
2,6,10,14-tetramethylhexadecane
3,5,6,7-tetra-s-indacen-1(2H)-one
2,3,5,6-tetra-s-indacene-1,7-dione
2-thiocyanatodiphenylamine
toluene
tolyl azide
tricosane
(Z)-9-tricosene
1,7,11-trimethyl cyclotetradecane
trimethyl-2-oxo-1,2,3,4-tetrahydropyrimidine
trimethylnaphthyridine
2,2,4-trimethylpenta-1,3-diol-di-isobutyrate
1,3,5-trimethylpyrazole
2,6,10-trimethyl-tetradecane
tropolone
1-undecanol
valeramide
2-vinyl pyridine
vitamin E