City of Fort Bragg

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Receive Informational Report on Glyphosate Containing Pesticides and Discuss Applicability for the City of Fort Bragg

CITY OF FORT BRAGG
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COUNCIL COMMITTEE ITEM SUMMARY REPORT

MEETING DATE:	**MAY 8, 2019**
TO:	**PUBLIC WORKS AND FACILITIES COMMITTEE**
FROM:	**CHANTELL O'NEAL; ENGINEERING TECHNICIAN**
AGENDA ITEM TITLE:	**INFORMATIONAL REPORT FOR A DISCUSSION ABOUT GLYPHOSATE CONTANING PESTICIDE USE IN THE CITY OF FORT BRAGG**

BACKGROUND AND OVERVIEW:

At the regular meeting of the Public Works and Facilities Committee of April 10, 2019, Committee Member Morsell-Haye requested a discussion regarding the use of glyphosate containing pesticides in the city be discussed at an upcoming meeting. Below is a list of general information about glyphosate.

- Glyphosate is the active in ingredient in several readily available herbicide brands including Roundup®, Vision®, Accord®, and Rodeo®.
- On July 7, 2017, California became the first state in the nation to issue a warning on glyphosate by adding the chemical to the state's Proposition 65 list of chemicals and substances known to cause cancer. Proposition 65 does not provide guidance regarding how to determine whether a warning is required or a discharge is prohibited. The Office of Environmental Health Hazard Assessment (OEHHA) is the implementing agency for Proposition 65 and has the resources and expertise to examine the scientific literature and calculate a level of exposure, in this case a No Significant Risk Level (NSRL), that does not require a warning or for which a discharge is not prohibited.
- The City of Fort Bragg discontinued the use of any herbicides containing glyphosate over a year ago.

- The California Environmental Protection Agency's Notice of Amendment of Title 27 (April 10, 2018) establishes a NSRL of 1100 micrograms per day for glyphosate. This is considerably more restrictive than the United States Environmental Protection Agency (USEPA) established limits. The image below shows the comparison of the two standards.

Figure 1 Environmental Working Group Article California's proposed limit vs. the amount allowed by USEPA

- Since 1988, the USEPA has listed Glyphosate as a group D for cancer risk, which means there is not enough evidence and not enough data to demonstrate that it is a cancer risk. As of April 30, 2019 the USEPA issued a press release indicating the "EPA continues to find that there are no risks to public health when glyphosate is used in accordance with its current label and that glyphosate is not a carcinogen." (Office of Chemical Safety and Pollution Prevention (OCSPP)).
- World Health Organization's International Agency for Research on Cancer (IARC) listed the chemical glyphosate–the active ingredient in Roundup–as a 'probable carcinogen' Group 2A (International Agency for Research on Cancer, 2015).
- The following Cities in California have initiated an herbicide/pesticide reduction plan, pest management plan, or passed a municipal ordinance to reduce or ban the use of glyphosate containing herbicides.

California Cities	Description of Efforts
Arcarta	Arcata, California – Initiated a pesticide reduction plan that urges pesticides to only be used as a last resort.
Belvedere	Belvedere, California – Passed municipal ordinance initiating Integrated Pest Management program that restricts toxic pesticide use and urges pesticide use as last resort.
Benicia	Benicia, California – City decided to go glyphosate-free following the verdict in Johnson v. Monsanto Co.
Berkley	Berkeley, California – Implemented pest management program to minimize or eliminate the use of pesticides.
Burbank	Burbank, California – City Council members voted to discontinue the use of Roundup in city parks for one year, and Burbank Unified School District will no longer use the herbicide due to cancer concerns.
Carlsbad	Carlsbad, California – The City Council voted unanimously to adopt a policy that makes organic pesticides the preferred method for killing weeds. "Asked to choose between aesthetics and public health…I'm going to choose public health every time," said Councilwoman Cori Schumacher.

Corte Madera	Corte Madera, California – Passed ordinance calling for Integrated Pest Management (IPM) program restricting highly toxic pesticides, while also urging for pesticide use to be a last resort.
Davis	Davis, California – Passed ordinance implementing Integrated Pest Management (IPM) program designed to reduce the use of pesticides. Some city parks do not allow the use of glyphosate.
Encinitas	Encinitas, California – Banned the use of Roundup and other glyphosate-based weed killers in city parks.
Fairfax	Fairfax, California – Passed municipal ordinance restricting use of toxic pesticides on public property in favor of alternative methods.
Fresno	Fresno, California – After hearing from concerned parents and employees, Fresno Unified School District is investigating the use of alternative herbicides that do not contain glyphosate, citing health risks.
Irvine	Irvine, California – City Council passed resolution to cease spraying Roundup and other chemicals on public parks, streets and playgrounds.
Laguna Hills	Laguna Hills, California – Passed a resolution to test an organics-only pesticide program on two parks.
Lodi	Lodi, California –The city decided to ban the use of Roundup within 25 feet of playgrounds.
Los Angeles County	Long Beach, California – Citing the landmark $289 million verdict in Johnson v. Monsanto Co., Long Beach Parks & Recreation Director Gerardo Mouet announced an immediate halt on the spraying of Roundup in Long Beach Parks.
Los Angeles County	Los Angeles County, California – The Los Angeles County Board of Supervisors issued a moratorium on glyphosate-based herbicides, including Roundup weed killer.
Malibu	Malibu, California – The city may implement an Earth Friendly Management Policy (EFMP) to avoid the use of pesticides and other chemicals.
Marin County	Marin County, California – The county stopped using glyphosate, the active ingredient in Monsanto's Roundup weed killer, on all county-maintained parks, landscaping, playgrounds, walkways and parking areas.
Mill Valley	Mill Valley, California – Passed ordinance initiating Integrated Pest Management program that restricts toxic pesticide use and urges pesticide use as last resort.
Napa	Napa, California – A policy announced in March of 2019 banned glyphosate use on city property, completing a phase-out campaign that started three years ago.
Novato	Novato, California – Following the $289 million Monsanto verdict, Novato Mayor Josh Fryday said the city will no longer use Roundup weed killer.

Oakland	Oakland, California – Passed ordinance initiating Integrated Pest Management program that restricts toxic pesticide use and promotes pesticide use as last resort. On Sept. 1, 2018, the city formally halted the use of Roundup. Alameda County is reviewing its chemical spraying practices.
Palo Alto	Palo Alto, California – Pest management program calls for Integrated Pest Management that restricts pesticide use in favor of less harmful methods.
Petaluma	Petaluma, California – City officials are considering a ban on glyphosate for use in public parks.
Richmond	Richmond, California – Issued an ordinance to ban the use of glyphosate for all weed abatement activities conducted by the city.
San Anselmo	San Anselmo, California – Passed city resolution promoting an Integrated Pest Management program restricting the use of toxic pesticides. The program only allows pesticide use as a last resort.
San Francisco	San Francisco, California – Restricts the use of toxic pesticides on public property in favor of alternative, organic methods.
San Juan Capistrano	San Juan Capistrano, California – Implemented an organics-first policy to control weeds in city parks and open spaces.
San Lorenzo Valley	San Lorenzo Valley, California – The San Lorenzo Valley Water District voted 4-1 for a permanent ban of glyphosate pesticide use by the district.
Santa Rosa	Santa Rosa, California – Banned the use of Roundup at city parks.
Sonoma	Sonoma, California – The Sonoma City Council voted to ban the use of glyphosate in all city parks.
Thousand Oaks	Thousand Oaks, California – City instituted a ban on glyphosate use on public golf courses.
Woodland	Woodland, California – Woodland Joint Unified School District suspended the use of Roundup on school campuses.

RECOMMENDATION:

Discuss and consider options appropriate for the City of Fort Bragg.

ATTACHMENTS:

1. State of California Environmental Protection Agency Prop 65 List of Chemicals Known to the State to Cause Cancer or Reproductive Toxicity
2. Glyphosate Final Statement of Reason; Title 27, California Code of Regulations, issued by CalEPA Office of Environmental Health Hazard Assessment (OEHHA)
3. Notice of Amendment of Title 27 (April 10, 2018)
4. EPA Press Release of April 30, 2019

STATE OF CALIFORNIA
ENVIRONMENTAL PROTECTION AGENCY
OFFICE OF ENVIRONMENTAL HEALTH HAZARD ASSESSMENT
SAFE DRINKING WATER AND TOXIC ENFORCEMENT ACT OF 1986

CHEMICALS KNOWN TO THE STATE TO CAUSE CANCER OR REPRODUCTIVE TOXICITY
March 8, 2019

The Safe Drinking Water and Toxic Enforcement Act of 1986 requires that the Governor revise and republish at least once per year the list of chemicals known to the State to cause cancer or reproductive toxicity. The identification number indicated in the following list is the Chemical Abstracts Service (CAS) Registry Number. No CAS number is given when several substances are presented as a single listing. The date refers to the initial appearance of the chemical on the list. For easy reference, chemicals which are shown underlined are newly added. Chemicals or endpoints shown in strikeout were placed on the Proposition 65 list on the date noted, and have subsequently been removed.

Chemical	Type of Toxicity	CAS No.	Date Listed
A-alpha-C (2-Amino-9H-pyrido [2,3-b]indole)	Cancer	26148-68-5	January 1, 1990
Abiraterone acetate	developmental, female, male	154229-18-2	April 8, 2016
Acetaldehyde	Cancer	75-07-0	April 1, 1988
Acetamide	cancer	60-35-5	January 1, 1990
Acetazolamide	Developmental	59-66-5	August 20, 1999
Acetochlor	Cancer	34256-82-1	January 1, 1989
Acetohydroxamic acid	Developmental	546-88-3	April 1, 1990
2-Acetylaminofluorene	Cancer	53-96-3	July 1, 1987
Acifluorfen sodium	Cancer	62476-59-9	January 1, 1990
Acrylamide	Cancer	79-06-1	January 1, 1990
Acrylamide	developmental, male	79-06-1	February 25, 2011
Acrylonitrile	Cancer	107-13-1	July 1, 1987
Actinomycin D	Cancer Developmental	50-76-0	October 1, 1989 October 1, 1992
AF-2;[2-(2-furyl)-3-(5-nitro-2-furyl)] acrylamide	Cancer	3688-53-7	July 1, 1987
Aflatoxins	Cancer	---	January 1, 1988
Alachlor	Cancer	15972-60-8	January 1, 1989
Alcoholic beverages	Cancer	---	April 29, 2011
Alcoholic beverages, when associated with alcohol abuse	Cancer	---	July 1, 1988
Aldrin	cancer	309-00-2	July 1, 1988
All-trans retinoic acid	developmental	302-79-4	January 1, 1989
~~Allyl chloride~~ Delisted October 29, 1999	~~cancer~~	~~107-05-1~~	~~January 1, 1990~~
Aloe Vera, non-decolorized whole leaf extract	cancer	---	December 4, 2015
Alprazolam	developmental	28981-97-7	July 1, 1990
Altretamine	developmental, male	645-05-6	August 20, 1999
Amantadine hydrochloride	developmental	665-66-7	February 27, 2001
Amikacin sulfate	developmental	39831-55-5	July 1, 1990
2-Aminoanthraquinone	cancer	117-79-3	October 1, 1989
p-Aminoazobenzene	cancer	60-09-3	January 1, 1990
o-Aminoazotoluene	cancer	97-56-3	July 1, 1987

4-Aminobiphenyl (4-amino-diphenyl)	cancer	92-67-1	February 27, 1987
1-Amino-2,4-dibromo-anthraquinone	cancer	81-49-2	August 26, 1997
3-Amino-9-ethylcarbazole hydrochloride	cancer	6109-97-3	July 1, 1989
2-Aminofluorene	cancer	153-78-6	January 29, 1999
Aminoglutethimide	developmental	125-84-8	July 1, 1990
Aminoglycosides	developmental	---	October 1, 1992
1-Amino-2-methylanthraquinone	cancer	82-28-0	October 1, 1989
2-Amino-5-(5-nitro-2-furyl)-1,3,4-thiadiazole	cancer	712-68-5	July 1, 1987
4-Amino-2-nitrophenol	cancer	119-34-6	January 29, 1999
Aminopterin	developmental, female	54-62-6	July 1, 1987
Amiodarone hydrochloride	developmental, female, male	19774-82-4	August 26, 1997
Amitraz	developmental	33089-61-1	March 30, 1999
Amitrole	cancer	61-82-5	July 1, 1987
Amoxapine	developmental	14028-44-5	May 15, 1998
Amsacrine	cancer	51264-14-3	August 7, 2009
~~tert-Amyl methyl ether~~ Delisted December 13, 2013	~~developmental~~	~~994-05-8~~	~~December 18, 2009~~
Anabolic steroids	female, male	---	April 1, 1990
Analgesic mixtures containing phenacetin	cancer	---	February 27, 1987
Androstenedione	cancer	63-05-8	May 3, 2011
Angiotensin converting enzyme (ACE) inhibitors	developmental	---	October 1, 1992
Aniline	cancer	62-53-3	January 1, 1990
Aniline hydrochloride	cancer	142-04-1	May 15, 1998
o-Anisidine	cancer	90-04-0	July 1, 1987
o-Anisidine hydrochloride	cancer	134-29-2	July 1, 1987
Anisindione	developmental	117-37-3	October 1, 1992
Anthraquinone	cancer	84-65-1	September 28, 2007
Antimony oxide (Antimony trioxide)	cancer	1309-64-4	October 1, 1990
Aramite	cancer	140-57-8	July 1, 1987
Areca nut	cancer	---	February 3, 2006
Aristolochic acids	cancer	---	July 9, 2004
Arsenic (inorganic arsenic compounds)	cancer	--	February 27, 1987
Arsenic (inorganic oxides)	developmental	---	May 1, 1997
Asbestos	cancer	1332-21-4	February 27, 1987
Aspirin (NOTE: It is especially important not to use aspirin during the last three months of pregnancy, unless specifically directed to do so by a physician because it may cause problems in the unborn child or complications during delivery.)	developmental, female	50-78-2	July 1, 1990
Atenolol	developmental	29122-68-7	August 26, 1997
Atrazine	developmental, female	1912-24-9	July 15, 2016
Auramine	cancer	492-80-8	July 1, 1987
Auranofin	developmental	34031-32-8	January 29, 1999
Avermectin B1 (Abamectin)	developmental	71751-41-2	December 3, 2010
Azacitidine	cancer	320-67-2	January 1, 1992
Azaserine	cancer	115-02-6	July 1, 1987

Azathioprine	cancer	446-86-6	February 27, 1987
Azathioprine	developmental	446-86-6	September 1, 1996
Azobenzene	cancer	103-33-3	January 1, 1990
Barbiturates	developmental	---	October 1, 1992
Beclomethasone dipropionate	developmental	5534-09-8	May 15, 1998
Benomyl	developmental, male	17804-35-2	July 1, 1991
Benthiavalicarb-isopropyl	cancer	177406-68-7	July 1, 2008
Benz[a]anthracene	cancer	56-55-3	July 1, 1987
Benzene	cancer	71-43-2	February 27, 1987
Benzene	developmental, male	71-43-2	December 26, 1997
Benzidine [and its salts]	cancer	92-87-5	February 27, 1987
Benzidine-based dyes	cancer	---	October 1, 1992
Benzodiazepines	developmental	---	October 1, 1992
Benzo[b]fluoranthene	cancer	205-99-2	July 1, 1987
Benzo[j]fluoranthene	cancer	205-82-3	July 1, 1987
Benzo[k]fluoranthene	cancer	207-08-9	July 1, 1987
Benzofuran	cancer	271-89-6	October 1, 1990
Benzophenone	cancer	119-61-9	June 22, 2012
Benzo[a]pyrene	cancer	50-32-8	July 1, 1987
Benzotrichloride	cancer	98-07-7	July 1, 1987
Benzphetamine hydrochloride	developmental	5411-22-3	April 1, 1990
Benzyl chloride	cancer	100-44-7	January 1, 1990
Benzyl violet 4B	cancer	1694-09-3	July 1, 1987
Beryllium and beryllium compounds	cancer	---	October 1, 1987
Betel quid with tobacco	cancer	---	January 1, 1990
Betel quid without tobacco	cancer	---	February 3, 2006
Bevacizumab	developmental, female	216974-75-3	March 8, 2019
2,2-Bis(bromomethyl)-1,3-propanediol	cancer	3296-90-0	May 1, 1996
Bis(2-chloroethyl)ether	cancer	111-44-4	April 1, 1988
N,N-Bis(2-chloroethyl)-2-naphthylamine (Chlornapazine)	cancer	494-03-1	February 27, 1987
Bischloroethyl nitrosourea (BCNU) (Carmustine)	cancer	154-93-8	July 1, 1987
Bischloroethyl nitrosourea (BCNU) (Carmustine)	developmental	154-93-8	July 1, 1990
Bis(chloromethyl)ether	cancer	542-88-1	February 27, 1987
Bis(2-chloro-1-methylethyl)ether, technical grade	cancer	---	October 29, 1999
Bisphenol A (BPA)	female	80-05-7	May 11, 2015
~~Bisphenol A (BPA)~~ Delisted April 19, 2013	~~developmental~~	~~80-05-7~~	~~April 11, 2013~~
Bitumens, extracts of steam-refined and air refined	cancer	---	January 1, 1990
Bracken fern	cancer	---	January 1, 1990
Bromacil lithium salt	developmental	53404-19-6	May 18, 1999
Bromacil lithium salt	male	53404-19-6	January 17, 2003
Bromate	cancer	15541-45-4	May 31, 2002
Bromochloroacetic acid	cancer	5589-96-8	April 6, 2010
Bromodichloroacetic acid	cancer	71133-14-7	July 29, 2016
Bromodichloromethane	cancer	75-27-4	January 1, 1990
Bromoethane	cancer	74-96-4	December 22, 2000
Bromoform	cancer	75-25-2	April 1, 1991
1-Bromopropane (1-BP)	cancer	106-94-5	August 5, 2016
1-Bromopropane (1-BP)	developmental, female,	106-94-5	December 7, 2004

	male		
2-Bromopropane (2-BP)	female, male	75-26-3	May 31, 2005
Bromoxynil	developmental	1689-84-5	October 1, 1990
Bromoxynil octanoate	developmental	1689-99-2	May 18, 1999
Butabarbital sodium	developmental	143-81-7	October 1, 1992
1,3-Butadiene	cancer	106-99-0	April 1, 1988
1,3-Butadiene	developmental, female, male	106-99-0	April 16, 2004
1,4-Butanediol dimethanesulfonate (Busulfan)	cancer	55-98-1	February 27, 1987
1,4-Butanediol dimethanesulfonate (Busulfan)	developmental	55-98-1	January 1, 1989
Butylated hydroxyanisole	cancer	25013-16-5	January 1, 1990
Butyl benzyl phthalate (BBP)	developmental	85-68-7	December 2, 2005
~~n-Butyl glycidyl ether~~ Delisted April 4, 2014	~~male~~	~~2426-08-6~~	~~August 7, 2009~~
beta-Butyrolactone	cancer	3068-88-0	July 1, 1987
Cacodylic acid	cancer	75-60-5	May 1, 1996
Cadmium	developmental, male	---	May 1, 1997
Cadmium and cadmium compounds	cancer	---	October 1, 1987
Caffeic acid	cancer	331-39-5	October 1, 1994
Captafol	cancer	2425-06-1	October 1, 1988
Captan	cancer	133-06-2	January 1, 1990
Carbamazepine	developmental	298-46-4	January 29, 1999
Carbaryl	cancer	63-25-2	February 5, 2010
Carbaryl	developmental, female, male	63-25-2	August 7, 2009
Carbazole	cancer	86-74-8	May 1, 1996
Carbon black (airborne, unbound particles of respirable size)	cancer	1333-86-4	February 21, 2003
Carbon-black extracts	cancer	---	January 1, 1990
Carbon disulfide	developmental, female, male	75-15-0	July 1, 1989
Carbon monoxide	developmental	630-08-0	July 1, 1989
Carbon tetrachloride	cancer	56-23-5	October 1, 1987
Carboplatin	developmental	41575-94-4	July 1, 1990
N-Carboxymethyl-N-nitrosourea	cancer	60391-92-6	January 25, 2002
Catechol	cancer	120-80-9	July 15, 2003
Ceramic fibers (airborne particles of respirable size)	cancer	---	July 1, 1990
Certain combined chemotherapy for lymphomas	cancer	---	February 27, 1987
Chenodiol	developmental	474-25-9	April 1, 1990
Chloral	cancer	75-87-6	September 13, 2013
Chloral hydrate	cancer	302-17-0	September 13, 2013
Chlorambucil	cancer	305-03-3	February 27, 1987
Chlorambucil	developmental	305-03-3	January 1, 1989
~~Chloramphenicol~~ Delisted January 4, 2013	~~cancer~~	~~56-75-7~~	~~October 1, 1989~~
Chloramphenicol sodium succinate	cancer	982-57-0	September 27, 2013
Chlorcyclizine hydrochloride	developmental	1620-21-9	July 1, 1987
Chlordane	cancer	57-74-9	July 1, 1988
Chlordecone (Kepone)	cancer	143-50-0	January 1, 1988
Chlordecone (Kepone)	developmental	143-50-0	January 1, 1989

Chlordiazepoxide	developmental	58-25-3	January 1, 1992
Chlordiazepoxide hydrochloride	developmental	438-41-5	January 1, 1992
Chlordimeform	cancer	6164-98-3	January 1, 1989
Chlorendic acid	cancer	115-28-6	July 1, 1989
Chlorinated paraffins (Average chain length, C12; approximately 60 percent chlorine by weight)	cancer	108171-26-2	July 1, 1989
p-Chloroaniline	cancer	106-47-8	October 1, 1994
p-Chloroaniline hydrochloride	cancer	20265-96-7	May 15, 1998
~~Chlorodibromomethane~~ Delisted October 29, 1999	~~cancer~~	~~124-48-1~~	~~January 1, 1990~~
Chloroethane (Ethyl chloride)	cancer	75-00-3	July 1, 1990
1-(2-Chloroethyl)-3-cyclohexyl-1-nitrosourea (CCNU) (Lomustine)	cancer	13010-47-4	January 1, 1988
1-(2-Chloroethyl)-3-cyclohexyl-1-nitrosourea (CCNU) Lomustine)	developmental	13010-47-4	July 1, 1990
1-(2-Chloroethyl)-3-(4-methyl-cyclohexyl) -1-nitrosourea (Methyl-CCNU)	cancer	13909-09-6	October 1, 1988
Chloroform	cancer	67-66-3	October 1, 1987
Chloroform	developmental	67-66-3	August 7, 2009
Chloromethyl methyl ether (technical grade)	cancer	107-30-2	February 27, 1987
3-Chloro-2-methylpropene	cancer	563-47-3	July 1, 1989
1-Chloro-4-nitrobenzene	cancer	100-00-5	October 29, 1999
4-Chloro-*o*-phenylenediamine	cancer	95-83-0	January 1, 1988
Chloroprene	cancer	126-99-8	June 2, 2000
2-Chloropropionic acid	male	598-78-7	August 7, 2009
Chlorothalonil	cancer	1897-45-6	January 1, 1989
p-Chloro-*o*-toluidine	cancer	95-69-2	January 1, 1990
p-Chloro-*o*-toluidine, strong acid salts of	cancer	---	May 15, 1998
5-Chloro-*o*-toluidine and its strong acid salts	cancer	---	October 24, 1997
Chlorotrianisene	cancer	569-57-3	September 1, 1996
Chlorozotocin	cancer	54749-90-5	January 1, 1992
Chlorpyrifos	developmental	2921-88-2	December 15, 2017
~~Chlorsulfuron~~ Delisted June 6, 2014	~~developmental, female, male~~	~~64902-72-3~~	~~May 14, 1999~~
Chromium (hexavalent compounds)	cancer	---	February 27, 1987
Chromium (hexavalent compounds)	developmental, female, male	---	December 19, 2008
Chrysene	cancer	218-01-9	January 1, 1990
C.I. Acid Red 114	cancer	6459-94-5	July 1, 1992
C.I. Basic Red 9 monohydrochloride	cancer	569-61-9	July 1, 1989
C.I. Direct Blue 15	cancer	2429-74-5	August 26, 1997
C.I. Direct Blue 218	cancer	28407-37-6	August 26, 1997
C.I. Disperse Yellow 3	cancer	2832-40-8	February 8, 2013
C.I. Solvent Yellow 14	cancer	842-07-9	May 15, 1998
Ciclosporin (Cyclosporin A; Cyclosporine)	cancer	59865-13-3 79217-60-0	January 1, 1992
Cidofovir	cancer, developmental, female, male	113852-37-2	January 29, 1999
Cinnamyl anthranilate	cancer	87-29-6	July 1, 1989
Cisplatin	cancer	15663-27-1	October 1, 1988
Citrus Red No. 2	cancer	6358-53-8	October 1, 1989

Cladribine	developmental	4291-63-8	September 1, 1996
Clarithromycin	developmental	81103-11-9	May 1, 1997
Clobetasol propionate	developmental, female	25122-46-7	May 15, 1998
Clofibrate	cancer	637-07-0	September 1, 1996
Clomiphene citrate	cancer	50-41-9	May 24, 2013
Clomiphene citrate	developmental	50-41-9	April 1, 1990
Clorazepate dipotassium	developmental	57109-90-7	October 1, 1992
CMNP (pyrazachlor)	cancer	6814-58-0	August 25, 2015
Cobalt metal powder	cancer	7440-48-4	July 1, 1992
Cobalt [II] oxide	cancer	1307-96-6	July 1, 1992
Cobalt sulfate	cancer	10124-43-3	May 20, 2005
Cobalt sulfate heptahydrate	cancer	10026-24-1	June 2, 2000
Cocaine	developmental, female	50-36-2	July 1, 1989
Coconut oil diethanolamine condensate (cocamide diethanolamine)	cancer	---	June 22, 2012
Codeine phosphate	developmental	52-28-8	May 15, 1998
Coke oven emissions	cancer	---	February 27, 1987
Colchicine	developmental, male	64-86-8	October 1, 1992
Conjugated estrogens	cancer	---	February 27, 1987
Conjugated estrogens	developmental	---	April 1, 1990
Creosotes	cancer	---	October 1, 1988
p-Cresidine	cancer	120-71-8	January 1, 1988
Cumene	cancer	98-82-8	April 6, 2010
Cupferron	cancer	135-20-6	January 1, 1988
Cyanazine	developmental	21725-46-2	April 1, 1990
Cycasin	cancer	14901-08-7	January 1, 1988
Cycloate	developmental	1134-23-2	March 19, 1999
~~Cyclohexanol~~ Delisted January 25, 2002	~~male~~	~~108-93-0~~	~~November 6, 1998~~
Cycloheximide	developmental	66-81-9	January 1, 1989
Cyclopenta[cd]pyrene	cancer	27208-37-3	April 29, 2011
Cyclophosphamide (anhydrous)	cancer	50-18-0	February 27, 1987
Cyclophosphamide (anhydrous)	developmental, female, male	50-18-0	January 1, 1989
Cyclophosphamide (hydrated)	cancer	6055-19-2	February 27, 1987
Cyclophosphamide (hydrated)	developmental, female, male	6055-19-2	January 1, 1989
Cyhexatin	developmental	13121-70-5	January 1, 1989
Cytarabine	developmental	147-94-4	January 1, 1989
Cytembena	cancer	21739-91-3	May 15, 1998
D&C Orange No. 17	cancer	3468-63-1	July 1, 1990
D&C Red No. 8	cancer	2092-56-0	October 1, 1990
D&C Red No. 9	cancer	5160-02-1	July 1, 1990
D&C Red No. 19	cancer	81-88-9	July 1, 1990
Dacarbazine	cancer	4342-03-4	January 1, 1988
Dacarbazine	developmental	4342-03-4	January 29, 1999
Daminozide	cancer	1596-84-5	January 1, 1990
Danazol	developmental	17230-88-5	April 1, 1990
Dantron (Chrysazin; 1,8-Dihydroxyanthraquinone)	cancer	117-10-2	January 1, 1992
Daunomycin	cancer	20830-81-3	January 1, 1988
Daunorubicin hydrochloride	developmental	23541-50-6	July 1, 1990
2,4-D butyric acid	~~developmental~~, male	94-82-6	June 18, 1999
DDD (Dichlorodiphenyl-	cancer	72-54-8	January 1, 1989

dichloroethane)			
DDE (Dichlorodi-phenyldichloroethylene)	cancer	72-55-9	January 1, 1989
DDT (Dichlorodi-phenyltrichloroethane)	cancer	50-29-3	October 1 , 1987
o,p'-DDT	developmental, female, male	789-02-6	May 15, 1998
p,p'-DDT	developmental, female, male	50-29-3	May 15, 1998
DDVP (Dichlorvos)	cancer	62-73-7	January 1, 1989
Demeclocycline hydrochloride (internal use)	developmental	64-73-3	January 1, 1992
Des-ethyl atrazine (DEA)	developmental, female	6190-65-4	July 15, 2016
Des-isopropyl atrazine (DIA)	developmental, female	1007-28-9	July 15, 2016
N,N'-Diacetylbenzidine	cancer	613-35-4	October 1, 1989
2,4-Diaminoanisole	cancer	615-05-4	October 1, 1990
2,4-Diaminoanisole sulfate	cancer	39156-41-7	January 1, 1988
2,4-Diamino-6-chloro-*s*-triazine (DACT)	developmental, female	3397-62-4	July 15, 2016
4,4'-Diaminodiphenyl ether (4,4'-Oxydianiline)	cancer	101-80-4	January 1, 1988
2,4-Diaminotoluene	cancer	95-80-7	January 1, 1988
~~Diaminotoluene (mixed)~~ Delisted November 20, 2015	~~cancer~~	---	~~January 1, 1990~~
Diazepam	developmental	439-14-5	January 1, 1992
Diazoaminobenzene	cancer	136-35-6	May 20, 2005
Diazoxide	developmental	364-98-7	February 27, 2001
Dibenz[a,h]acridine	cancer	226-36-8	January 1, 1988
Dibenz[a,j]acridine	cancer	224-42-0	January 1, 1988
Dibenzanthracenes	cancer	---	December 26, 2014
Dibenz[a,c]anthracene	cancer	215-58-7	December 26, 2014
Dibenz[a,h]anthracene	cancer	53-70-3	January 1, 1988
Dibenz[a,j]anthracene	cancer	224-41-9	December 26, 2014
7H-Dibenzo[c,g]carbazole	cancer	194-59-2	January 1, 1988
Dibenzo[a,e]pyrene	cancer	192-65-4	January 1, 1988
Dibenzo[a,h]pyrene	cancer	189-64-0	January 1, 1988
Dibenzo[a,i]pyrene	cancer	189-55-9	January 1, 1988
Dibenzo[a,l]pyrene	cancer	191-30-0	January 1, 1988
Dibromoacetic acid	cancer	631-64-1	June 17, 2008
Dibromoacetonitrile	cancer	3252-43-5	May 3, 2011
1,2-Dibromo-3-chloropropane (DBCP)	cancer	96-12-8	July 1, 1987
1,2-Dibromo-3-chloropropane (DBCP)	male	96-12-8	February 27, 1987
2,3-Dibromo-1-propanol	cancer	96-13-9	October 1, 1994
Dichloroacetic acid	cancer	79-43-6	May 1, 1996
Dichloroacetic acid	developmental, male	79-43-6	August 7, 2009
p-Dichlorobenzene	cancer	106-46-7	January 1, 1989
3,3'-Dichlorobenzidine	cancer	91-94-1	October 1, 1987
3,3'-Dichlorobenzidine dihydrochloride	cancer	612-83-9	May 15, 1998
1,1-Dichloro-2,2-bis(*p*-chlorophenyl)ethylene (DDE)	developmental, male	72-55-9	March 30, 2010
1,4-Dichloro-2-butene	cancer	764-41-0	January 1, 1990
3,3'-Dichloro-4,4'-diaminodiphenyl ether	cancer	28434-86-8	January 1, 1988
1,1-Dichloroethane	cancer	75-34-3	January 1, 1990

Dichloromethane (Methylene chloride)	cancer	75-09-2	April 1, 1988
Dichlorophene	developmental	97-23-4	April 27, 1999
1,2-Dichloropropane	cancer	78-87-5	January 1, 1990
1,3-Dichloro-2-propanol (1,3-DCP)	cancer	96-23-1	October 8, 2010
1,3-Dichloropropene	cancer	542-75-6	January 1, 1989
Dichlorphenamide	developmental	120-97-8	February 27, 2001
Diclofop-methyl	cancer	51338-27-3	April 6, 2010
Diclofop methyl	developmental	51338-27-3	March 5, 1999
Dicumarol	developmental	66-76-2	October 1, 1992
Dieldrin	cancer	60-57-1	July 1, 1988
~~Dienestrol~~ Delisted January 4, 2013	~~cancer~~	~~84-17-3~~	~~January 1, 1990~~
Diepoxybutane	cancer	1464-53-5	January 1, 1988
Diesel engine exhaust	cancer	---	October 1, 1990
Diethanolamine	cancer	111-42-2	June 22, 2012
Di(2-ethylhexyl)phthalate (DEHP)	cancer	117-81-7	January 1, 1988
Di(2-ethylhexyl)phthalate (DEHP)	developmental, male	117-81-7	October 24, 2003
1,2-Diethylhydrazine	cancer	1615-80-1	January 1, 1988
Diethylstilbestrol (DES)	cancer	56-53-1	February 27, 1987
Diethylstilbestrol (DES)	developmental	56-53-1	July 1, 1987
Diethyl sulfate	cancer	64-67-5	January 1, 1988
Diflunisal	developmental, female	22494-42-4	January 29, 1999
~~Diglycidyl ether~~ Delisted April 4, 2014	~~male~~	~~2238-07-5~~	~~August 7, 2009~~
Diglycidyl resorcinol ether (DGRE)	cancer	101-90-6	July 1, 1989
Di-*n*-hexyl phthalate (DnHP)	female, male	84-75-3	December 2, 2005
Di-*n*-butyl phthalate (DBP)	developmental, female, male	84-74-2	December 2, 2005
Dihydroergotamine mesylate	developmental	6190-39-2	May 1, 1997
Dihydrosafrole	cancer	94-58-6	January 1, 1988
Di-isodecyl phthalate (DIDP)	developmental	68515-49-1/ 26761-40-0	April 20, 2007
Diisononyl phthalate (DINP)	cancer	---	December 20, 2013
Diisopropyl sulfate	cancer	2973-10-6	April 1, 1993
Diltiazem hydrochloride	developmental	33286-22-5	February 27, 2001
3,3'-Dimethoxybenzidine (*o*-Dianisidine)	cancer	119-90-4	January 1, 1988
3,3'-Dimethoxybenzidine dihydrochloride	cancer	20325-40-0	October 1, 1990
3,3'-Dimethoxybenzidine-based dyes metabolized to 3,3'-dimethoxybenzidine	cancer	---	June 11, 2004
N,N-Dimethylacetamide	developmental, male	127-19-5	May 21, 2010
4-Dimethylaminoazobenzene	cancer	60-11-7	January 1, 1988
trans-2-[(Dimethylamino)methyl-imino]-5-[2-(5-nitro-2-furyl)vinyl]-1,3,4-oxadiazole	cancer	55738-54-0	January 1, 1988
7,12-Dimethylbenz(a)anthracene	cancer	57-97-6	January 1, 1990
3,3'-Dimethylbenzidine (ortho-Tolidine)	cancer	119-93-7	January 1, 1988
3,3'-Dimethylbenzidine-based dyes metabolized to 3,3'-dimethylbenzidine	cancer	---	June 11, 2004
3,3'-Dimethylbenzidine dihydrochloride	cancer	612-82-8	April 1, 1992
Dimethylcarbamoyl chloride	cancer	79-44-7	January 1, 1988
N,N-Dimethylformamide	cancer	68-12-2	October 27, 2017

1,1-Dimethylhydrazine (UDMH)	cancer	57-14-7	October 1, 1989
1,2-Dimethylhydrazine	cancer	540-73-8	January 1, 1988
2,6-Dimethyl-N-nitrosomorpholine (DMNM)	cancer	1456-28-6	February 8, 2013
Dimethyl sulfate	cancer	77-78-1	January 1, 1988
N,N-Dimethyl-*p*-toluidine	cancer	99-97-8	May 2, 2014
Dimethylvinylchloride	cancer	513-37-1	July 1, 1989
m-Dinitrobenzene	male	99-65-0	July 1, 1990
o-Dinitrobenzene	male	528-29-0	July 1, 1990
p-Dinitrobenzene	male	100-25-4	July 1, 1990
3,7-Dinitrofluoranthene	cancer	105735-71-5	August 26, 1997
3,9-Dinitrofluoranthene	cancer	22506-53-2	August 26, 1997
1,3-Dinitropyrene	cancer	75321-20-9	November 2, 2012
1,6-Dinitropyrene	cancer	42397-64-8	October 1, 1990
1,8-Dinitropyrene	cancer	42397-65-9	October 1, 1990
Dinitrotoluene (technical grade)	female, male	---	August 20, 1999
2,4-Dinitrotoluene	cancer	121-14-2	July 1, 1988
2,4-Dinitrotoluene	male	121-14-2	August 20, 1999
2,6-Dinitrotoluene	cancer	606-20-2	July 1, 1995
2,6-Dinitrotoluene	male	606-20-2	August 20, 1999
Dinitrotoluene mixture, 2,4-/2,6-	cancer	---	May 1, 1996
Dinocap	developmental	39300-45-3	April 1, 1990
Dinoseb	developmental, male	88-85-7	January 1, 1989
Di-*n*-propyl isocinchomeronate (MGK Repellent 326)	cancer	136-45-8	May 1, 1996
1,4-Dioxane	cancer	123-91-1	January 1, 1988
Diphenylhydantoin (Phenytoin)	cancer	57-41-0	January 1, 1988
Diphenylhydantoin (Phenytoin)	developmental	57-41-0	July 1, 1987
Diphenylhydantoin (Phenytoin), sodium salt	cancer	630-93-3	January 1, 1988
Direct Black 38 (technical grade)	cancer	1937-37-7	January 1, 1988
Direct Blue 6 (technical grade)	cancer	2602-46-2	January 1, 1988
Direct Brown 95 (technical grade)	cancer	16071-86-6	October 1, 1988
Disodium cyanodithioimido-carbonate	developmental	138-93-2	March 30, 1999
Disperse Blue 1	cancer	2475-45-8	October 1, 1990
Diuron	cancer	330-54-1	May 31, 2002
Doxorubicin hydrochloride (Adriamycin)	cancer	25316-40-9	July 1, 1987
Doxorubicin hydrochloride (Adriamycin)	developmental, male	25316-40-9	January 29, 1999
Doxycycline (internal use)	developmental	564-25-0	July 1, 1990
Doxycycline calcium (internal use)	developmental	94088-85-4	January 1, 1992
Doxycycline hyclate (internal use)	developmental	24390-14-5	October 1, 1991
Doxycycline monohydrate (internal use)	developmental	17086-28-1	October 1, 1991
~~2,4-DP (dichloroprop)~~ Delisted January 25, 2002	~~developmental~~	~~120-36-5~~	~~April 27, 1999~~
Emissions from combustion of coal	cancer	---	August 7, 2013
Emissions from high-temperature unrefined rapeseed oil	cancer	---	January 3, 2014
Endrin	developmental	72-20-8	May 15, 1998
Environmental tobacco smoke (ETS)	developmental	---	June 9, 2006
Epichlorohydrin	cancer	106-89-8	October 1, 1987

Epichlorohydrin	male	106-89-8	September 1, 1996
Epoxiconazole	cancer	135319-73-2	April 15, 2011
Ergotamine tartrate	developmental	379-79-3	April 1, 1990
Erionite	cancer	12510-42-8/ 66733-21-9	October 1, 1988
Estradiol 17B	cancer	50-28-2	January 1, 1988
Estragole	cancer	140-67-0	October 29, 1999
Estrogens, steroidal	cancer	---	August 19, 2005
Estrogen-progestogen (combined) used as menopausal therapy	cancer	---	November 4, 2011
Estrone	cancer	53-16-7	January 1, 1988
Estropipate	cancer, developmental	7280-37-7	August 26, 1997
Ethinylestradiol	cancer	57-63-6	January 1, 1988
Ethionamide	developmental	536-33-4	August 26, 1997
Ethoprop	cancer	13194-48-4	February 27, 2001
Ethyl acrylate	cancer	140-88-5	July 1, 1989
Ethyl alcohol in alcoholic beverages	developmental	---	October 1, 1987
Ethylbenzene	cancer	100-41-4	June 11, 2004
~~Ethyl-tert-butyl ether~~ Delisted December 13, 2013	~~male~~	~~637-92-3~~	~~December 18, 2009~~
Ethyl dipropylthiocarbamate	developmental	759-94-4	April 27, 1999
Ethyl-4,4'-dichlorobenzilate	cancer	510-15-6	January 1, 1990
Ethylene dibromide	cancer	106-93-4	July 1, 1987
Ethylene dibromide	developmental, male	106-93-4	May 15, 1998
Ethylene dichloride (1,2-Dichloroethane)	cancer	107-06-2	October 1, 1987
Ethylene glycol (ingested)	developmental	107-21-1	June 19, 2015
Ethylene glycol monoethyl ether	developmental, male	110-80-5	January 1, 1989
Ethylene glycol monoethyl ether acetate	developmental, male	111-15-9	January 1, 1993
Ethylene glycol monomethyl ether	developmental, male	109-86-4	January 1, 1989
Ethylene glycol monomethyl ether acetate	developmental, male	110-49-6	January 1, 1993
Ethyleneimine (Aziridine)	cancer	151-56-4	January 1, 1988
Ethylene oxide	cancer	75-21-8	July 1, 1987
Ethylene oxide	female	75-21-8	February 27, 1987
Ethylene oxide	developmental, male	75-21-8	August 7, 2009
Ethylene thiourea	cancer	96-45-7	January 1, 1988
Ethylene thiourea	developmental	96-45-7	January 1, 1993
~~2-Ethylhexanoic acid~~ Delisted December 13, 2013	~~developmental~~	~~149-57-5~~	~~August 7, 2009~~
Ethyl methanesulfonate	cancer	62-50-0	January 1, 1988
Etodolac	developmental, female	41340-25-4	August 20, 1999
Etoposide	cancer	33419-42-0	November 4, 2011
Etoposide	developmental	33419-42-0	July 1, 1990
Etoposide in combination with cisplatin and bleomycin	cancer	---	November 4, 2011
Etretinate	developmental	54350-48-0	July 1, 1987
Fenoxaprop ethyl	developmental	66441-23-4	March 26, 1999
Fenoxycarb	cancer	72490-01-8	June 2, 2000
Filgrastim	developmental	121181-53-1	February 27, 2001
Fluazifop butyl	developmental	69806-50-4	November 6, 1998
Flunisolide	developmental, female	3385-03-3	May 15, 1998
Fluorouracil	developmental	51-21-8	January 1, 1989
Fluoxymesterone	developmental	76-43-7	April 1, 1990

Flurazepam hydrochloride	developmental	1172-18-5	October 1, 1992
Flurbiprofen	developmental, female	5104-49-4	August 20, 1999
Flutamide	developmental	13311-84-7	July 1, 1990
Fluticasone propionate	developmental	80474-14-2	May 15, 1998
Fluvalinate	developmental	69409-94-5	November 6, 1998
Folpet	cancer	133-07-3	January 1, 1989
Formaldehyde (gas)	cancer	50-00-0	January 1, 1988
2-(2-Formylhydrazino)-4-(5-nitro-2-furyl)thiazole	cancer	3570-75-0	January 1, 1988
Fumonisin B_1	cancer	116355-83-0	November 14, 2003
Furan	cancer	110-00-9	October 1, 1993
Furazolidone	cancer	67-45-8	January 1, 1990
Furfuryl alcohol	cancer	98-00-0	September 30, 2016
Furmecyclox	cancer	60568-05-0	January 1, 1990
Fusarin C	cancer	79748-81-5	July 1, 1995
Gallium arsenide	cancer	1303-00-0	August 1, 2008
Ganciclovir	cancer, developmental, male	82410-32-0	August 26, 1997
Ganciclovir sodium	developmental, male	107910-75-8	August 26, 1997
Gasoline engine exhaust (condensates/extracts)	cancer	---	October 1, 1990
Gemfibrozil	cancer	25812-30-0	December 22, 2000
Gemfibrozil	female, male	25812-30-0	August 20, 1999
Gentian violet (Crystal violet)	cancer	548-62-9	November 23, 2018
Glass wool fibers (inhalable and biopersistent)	cancer	---	July 1, 1990
Glu-P-1 (2-Amino-6-methyldipyrido [1,2- a:3',2'-d]imidazole)	cancer	67730-11-4	January 1, 1990
Glu-P-2 (2-Aminodipyrido [1,2-a:3',2'-d]imidazole)	cancer	67730-10-3	January 1, 1990
Glycidaldehyde	cancer	765-34-4	January 1, 1988
Glycidol	cancer	556-52-5	July 1, 1990
Glyphosate	cancer	1071-83-6	July 7, 2017
Goldenseal root powder	cancer	---	December 4, 2015
Goserelin acetate	developmental, female, male	65807-02-5	August 26, 1997
Griseofulvin	cancer	126-07-8	January 1, 1990
Gyromitrin (Acetaldehyde methylformylhydrazone)	cancer	16568-02-8	January 1, 1988
Halazepam	developmental	23092-17-3	July 1, 1990
Halobetasol propionate	developmental	66852-54-8	August 20, 1999
Haloperidol	developmental, female	52-86-8	January 29, 1999
Halothane	developmental	151-67-7	September 1, 1996
HC Blue 1	cancer	2784-94-3	July 1, 1989
Heptachlor	cancer	76-44-8	July 1, 1988
Heptachlor	developmental	76-44-8	August 20, 1999
Heptachlor epoxide	cancer	1024-57-3	July 1, 1988
Herbal remedies containing plant species of the genus *Aristolochia*	cancer	---	July 9, 2004
Hexachlorobenzene	cancer	118-74-1	October 1, 1987
Hexachlorobenzene	developmental	118-74-1	January 1, 1989
Hexachlorobutadiene	cancer	87-68-3	May 3, 2011

Hexachlorocyclohexane (technical grade)	cancer	---	October 1, 1987
Hexachlorodibenzodioxin	cancer	34465-46-8	April 1, 1988
Hexachloroethane	cancer	67-72-1	July 1, 1990
2,4-Hexadienal (89% trans, trans isomer; 11% cis, trans isomer)	cancer	---	March 4, 2005
Hexafluoroacetone	developmental, male	684-16-2	August 1, 2008
Hexamethylphosphoramide	cancer	680-31-9	January 1, 1988
Hexamethylphosphoramide	male	680-31-9	October 1, 1994
n-Hexane	male	110-54-3	December 15, 2017
2,5-Hexanedione	male	110-13-4	December 4, 2015
Histrelin acetate	developmental	---	May 15, 1998
Hydramethylnon	developmental, male	67485-29-4	March 5, 1999
Hydrazine	cancer	302-01-2	January 1, 1988
Hydrazine sulfate	cancer	10034-93-2	January 1, 1988
Hydrazobenzene (1,2-Diphenylhydrazine)	cancer	122-66-7	January 1, 1988
Hydrogen cyanide (HCN) and cyanide salts (CN salts)	male	---	July 5, 2013
1-Hydroxyanthraquinone	cancer	129-43-1	May 27, 2005
Hydroxyurea	developmental	127-07-1	May 1, 1997
Idarubicin hydrochloride	developmental, male	57852-57-0	August 20, 1999
Ifosfamide	developmental	3778-73-2	July 1, 1990
Iodine-131	developmental	10043-66-0	January 1, 1989
Imazalil	cancer	35554-44-0	May 20, 2011
Indeno[1,2,3-cd]pyrene	cancer	193-39-5	January 1, 1988
Indium phosphide	cancer	22398-80-7	February 27, 2001
IQ (2-Amino-3-methylimidazo [4,5-f] quinoline)	cancer	76180-96-6	April 1, 1990
Iprodione	cancer	36734-19-7	May 1, 1996
Iprovalicarb	cancer	140923-17-7 140923-25-7	June 1, 2007
Iron dextran complex	cancer	9004-66-4	January 1, 1988
Isobutyl nitrite	cancer	542-56-3	May 1, 1996
Isoprene	cancer	78-79-5	May 1, 1996
Isopyrazam	cancer	881685-58-1	July 24, 2012
~~Isosafrole~~ Delisted December 8, 2006	~~cancer~~	~~120-58-1~~	~~October 1, 1989~~
Isotretinoin	developmental	4759-48-2	July 1, 1987
Isoxaflutole	cancer	141112-29-0	December 22, 2000
Kresoxim-methyl	cancer	143390-89-0	February 3, 2012
Lactofen	cancer	77501-63-4	January 1, 1989
Lasiocarpine	cancer	303-34-4	April 1, 1988
Lead	developmental, female, male	---	February 27, 1987
Lead and lead compounds	cancer	---	October 1, 1992
Lead acetate	cancer	301-04-2	January 1, 1988
Lead phosphate	cancer	7446-27-7	April 1, 1988
Lead subacetate	cancer	1335-32-6	October 1, 1989
Leather dust	cancer	---	April 29, 2011

Leuprolide acetate	developmental, female, male	74381-53-6	August 26, 1997
Levodopa	developmental	59-92-7	January 29, 1999
Levonorgestrel implants	female	797-63-7	May 15, 1998
Lindane and other hexachloro-cyclohexane isomers	cancer	---	October 1, 1989
Linuron	developmental	330-55-2	March 19, 1999
Lithium carbonate	developmental	554-13-2	January 1, 1991
Lithium citrate	developmental	919-16-4	January 1, 1991
Lorazepam	developmental	846-49-1	July 1, 1990
Lovastatin	developmental	75330-75-5	October 1, 1992
Lynestrenol	cancer	52-76-6	February 27, 2001
Malathion	cancer	121-75-5	May 20, 2016
Malonaldehyde, sodium salt	cancer	24382-04-5	May 3, 2011
Mancozeb	cancer	8018-01-7	January 1, 1990
Maneb	cancer	12427-38-2	January 1, 1990
Marijuana smoke	cancer	---	June 19, 2009
Me-A-alpha-C (2-Amino-3-methyl-9H-pyrido[2,3-b]indole)	cancer	68006-83-7	January 1, 1990
Mebendazole	developmental	31431-39-7	August 20, 1999
Medroxyprogesterone acetate	cancer	71-58-9	January 1, 1990
Medroxyprogesterone acetate	developmental	71-58-9	April 1, 1990
Megestrol acetate	cancer	595-33-5	March 28, 2014
Megestrol acetate	developmental	595-33-5	January 1, 1991
MeIQ (2-Amino-3,4-dimethyl-imidazo[4,5-f]quinoline)	cancer	77094-11-2	October 1, 1994
MeIQx (2-Amino-3,8-dimethyl-imidazo[4,5-f]quinoxaline)	cancer	77500-04-0	October 1, 1994
Melphalan	cancer	148-82-3	February 27, 1987
Melphalan	developmental	148-82-3	July 1, 1990
Menotropins	developmental	9002-68-0	April 1, 1990
Mepanipyrim	cancer	110235-47-7	July 1, 2008
Meprobamate	developmental	57-53-4	January 1, 1992
2-Mercaptobenzothiazole	cancer	149-30-4	October 27, 2017
Mercaptopurine	developmental	6112-76-1	July 1, 1990
Mercury and mercury compounds	developmental	---	July 1, 1990
Merphalan	cancer	531-76-0	April 1, 1988
Mestranol	cancer	72-33-3	April 1, 1988
Metam potassium	cancer	137-41-7	December 31, 2010
Methacycline hydrochloride	developmental	3963-95-9	January 1, 1991
Metham sodium	cancer	137-42-8	November 6, 1998
Metham sodium	developmental	137-42-8	May 15, 1998
Methanol	developmental	67-56-1	March 16, 2012
Methazole	developmental	20354-26-1	December 1, 1999
Methimazole	developmental	60-56-0	July 1, 1990
Methotrexate	developmental	59-05-2	January 1, 1989
Methotrexate sodium	developmental	15475-56-6	April 1, 1990
5-Methoxypsoralen with ultraviolet A therapy	cancer	484-20-8	October 1, 1988
8-Methoxypsoralen with ultraviolet A therapy	cancer	298-81-7	February 27, 1987
2-Methylaziridine (Propyleneimine)	cancer	75-55-8	January 1, 1988
Methylazoxymethanol	cancer	590-96-5	April 1, 1988
Methylazoxymethanol acetate	cancer	592-62-1	April 1, 1988
Methyl bromide, as a structural	developmental	74-83-9	January 1, 1993

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Methyl carbamate	cancer	598-55-0	May 15, 1998
Methyl chloride	developmental	74-87-3	March 10, 2000
Methyl chloride	male	74-87-3	August 7, 2009
3-Methylcholanthrene	cancer	56-49-5	January 1, 1990
5-Methylchrysene	cancer	3697-24-3	April 1, 1988
4,4'-Methylene bis(2-chloroaniline)	cancer	101-14-4	July 1, 1987
4,4'-Methylene bis(N,N-dimethyl) benzenamine	cancer	101-61-1	October 1, 1989
4,4'-Methylene bis(2-methylaniline)	cancer	838-88-0	April 1, 1988
4,4'-Methylenedianiline	cancer	101-77-9	January 1, 1988
4,4'-Methylenedianiline dihydrochloride	cancer	13552-44-8	January 1, 1988
Methyleugenol	cancer	93-15-2	November 16, 2001
Methylhydrazine and its salts	cancer	---	July 1, 1992
2-Methylimidazole	cancer	693-98-1	June 22, 2012
4-Methylimidazole	cancer	822-36-6	January 7, 2011
Methyl iodide	cancer	74-88-4	April 1, 1988
Methyl isobutyl ketone	cancer	108-10-1	November 4, 2011
Methyl isobutyl ketone (MIBK)	developmental	108-10-1	March 28, 2014
Methyl isocyanate (MIC)	developmental, female	624-83-9	November 12, 2010
~~Methyl isopropyl ketone~~ Delisted April 4, 2014	~~developmental~~	~~563-80-4~~	~~February 17, 2012~~
Methyl mercury	developmental	---	July 1, 1987
Methylmercury compounds	cancer	---	May 1, 1996
Methyl methanesulfonate	cancer	66-27-3	April 1, 1988
Methyl-n-butyl ketone	male developmental	591-78-6	August 7, 2009 December 4, 2015
2-Methyl-1-nitroanthraquinone (of uncertain purity)	cancer	129-15-7	April 1, 1988
N-Methyl-N'-nitro-N-nitrosoguanidine	cancer	70-25-7	April 1, 1988
N-Methylolacrylamide	cancer	924-42-5	July 1, 1990
N-Methylpyrrolidone	developmental	872-50-4	June 15, 2001
α-Methyl styrene (alpha-Methylstyrene)	cancer	98-83-9	November 2, 2012
~~α-Methyl styrene~~ Delisted April 4, 2014	~~female~~	~~98-83-9~~	~~July 29, 2011~~
Methyltestosterone	developmental	58-18-4	April 1, 1990
Methylthiouracil	cancer	56-04-2	October 1, 1989
Metiram	cancer	9006-42-2	January 1, 1990
Metiram	developmental	9006-42-2	March 30, 1999
Metronidazole	cancer	443-48-1	January 1, 1988
Michler's ketone	cancer	90-94-8	January 1, 1988
Midazolam hydrochloride	developmental	59467-96-8	July 1, 1990
Minocycline hydrochloride (internal use)	developmental	13614-98-7	January 1, 1992
Mirex	cancer	2385-85-5	January 1, 1988
Misoprostol	developmental	59122-46-2	April 1, 1990
Mitomycin C	cancer	50-07-7	April 1, 1988
Mitoxantrone hydrochloride	cancer	70476-82-3	January 23, 2015
Mitoxantrone hydrochloride	developmental	70476-82-3	July 1, 1990
Molinate	developmental, female, male	2212-67-1	December 11, 2009
MON 4660 (dichloroacetyl-1-oxa-4-azaspiro(4,5)-decane)	cancer	71526-07-3	March 22, 2011
MON 13900 (furilazole)	cancer	121776-33-8	March 22, 2011

3-Monochloropropane-1,2-diol (3-MCPD)	cancer	96-24-2	October 8, 2010
Monocrotaline	cancer	315-22-0	April 1, 1988
MOPP (vincristine-prednisone-nitrogen mustard-procarbazine mixture)	cancer	113803-47-7	November 4, 2011
5-(Morpholinomethyl)-3-[(5-nitrofurfuryl-idene)-amino]-2-oxazolidinone	cancer	139-91-3	April 1, 1988
Mustard Gas	cancer	505-60-2	February 27, 1987
MX (3-chloro-4-(dichloromethyl) 5-hydroxy-2(5H)-furanone)	cancer	77439-76-0	December 22, 2000
Myclobutanil	developmental, male	88671-89-0	April 16, 1999
beta-Myrcene	cancer	123-35-3	March 27, 2015
Nabam	developmental	142-59-6	March 30, 1999
Nafarelin acetate	developmental	86220-42-0	April 1, 1990
Nafenopin	cancer	3771-19-5	April 1, 1988
Nalidixic acid	cancer	389-08-2	May 15, 1998
Naphthalene	cancer	91-20-3	April 19, 2002
1-Naphthylamine	cancer	134-32-7	October 1, 1989
2-Naphthylamine	cancer	91-59-8	February 27, 1987
Neomycin sulfate (internal use)	developmental	1405-10-3	October 1, 1992
Netilmicin sulfate	developmental	56391-57-2	July 1, 1990
Nickel (Metallic)	cancer	7440-02-0	October 1, 1989
Nickel acetate	cancer	373-02-4	October 1, 1989
Nickel carbonate	cancer	3333-67-3	October 1, 1989
Nickel carbonyl	cancer	13463-39-3	October 1, 1987
Nickel carbonyl	developmental	13463-39-3	September 1, 1996
Nickel compounds	cancer	---	May 7, 2004
Nickel (soluble compounds)	developmental, male	---	October 26, 2018
Nickel hydroxide	cancer	12054-48-7; 12125-56-3	October 1, 1989
Nickelocene	cancer	1271-28-9	October 1, 1989
Nickel oxide	cancer	1313-99-1	October 1, 1989
Nickel refinery dust from the pyrometallurgical process	cancer	---	October 1, 1987
Nickel subsulfide	cancer	12035-72-2	October 1, 1987
Nicotine	developmental	54-11-5	April 1, 1990
Nifedipine	developmental, female, male	21829-25-4	January 29, 1999
Nimodipine	developmental	66085-59-4	April 24, 2001
Niridazole	cancer	61-57-4	April 1, 1988
Nitrapyrin	cancer	1929-82-4	October 5, 2005
Nitrapyrin	developmental	1929-82-4	March 30, 1999
Nitrilotriacetic acid	cancer	139-13-9	January 1, 1988
Nitrilotriacetic acid, trisodium salt monohydrate	cancer	18662-53-8	April 1, 1989
5-Nitroacenaphthene	cancer	602-87-9	April 1, 1988
~~5-Nitro-*o*-anisidine~~ Delisted December 8, 2006	~~cancer~~	~~99-59-2~~	~~October 1, 1989~~
o-Nitroanisole	cancer	91-23-6	October 1, 1992
Nitrobenzene	cancer	98-95-3	August 26, 1997
Nitrobenzene	male	98-95-3	March 30, 2010
4-Nitrobiphenyl	cancer	92-93-3	April 1, 1988
6-Nitrochrysene	cancer	7496-02-8	October 1, 1990

Nitrofen (technical grade)	cancer	1836-75-5	January 1, 1988
2-Nitrofluorene	cancer	607-57-8	October 1, 1990
Nitrofurantoin	male	67-20-9	April 1, 1991
Nitrofurazone	cancer	59-87-0	January 1, 1990
1-[(5-Nitrofurfurylidene)-amino]-2-imidazolidinone	cancer	555-84-0	April 1, 1988
N-[4-(5-Nitro-2-furyl)-2-thiazolyl] acetamide	cancer	531-82-8	April 1, 1988
Nitrogen mustard (Mechlorethamine)	cancer	51-75-2	January 1, 1988
Nitrogen mustard (Mechlorethamine)	developmental	51-75-2	January 1, 1989
Nitrogen mustard hydrochloride (Mechlorethamine hydrochloride)	cancer	55-86-7	April 1, 1988
Nitrogen mustard hydrochloride (Mechlorethamine hydrochloride)	developmental	55-86-7	July 1, 1990
Nitrogen mustard N-oxide	cancer	126-85-2	April 1, 1988
Nitrogen mustard N-oxide hydrochloride	cancer	302-70-5	April 1, 1988
Nitromethane	cancer	75-52-5	May 1, 1997
2-Nitropropane	cancer	79-46-9	January 1, 1988
1-Nitropyrene	cancer	5522-43-0	October 1, 1990
4-Nitropyrene	cancer	57835-92-4	October 1, 1990
N-Nitrosodi-*n*-butylamine	cancer	924-16-3	October 1, 1987
N-Nitrosodiethanolamine	cancer	1116-54-7	January 1, 1988
N-Nitrosodiethylamine	cancer	55-18-5	October 1, 1987
N-Nitrosodimethylamine	cancer	62-75-9	October 1, 1987
p-Nitrosodiphenylamine	cancer	156-10-5	January 1, 1988
N-Nitrosodiphenylamine	cancer	86-30-6	April 1, 1988
N-Nitrosodi-*n*-propylamine	cancer	621-64-7	January 1, 1988
N-Nitroso-N-ethylurea	cancer	759-73-9	October 1, 1987
N-Nitrosohexamethyleneimine	cancer	932-83-2	November 23, 2018
3-(N-Nitrosomethylamino)-propionitrile	cancer	60153-49-3	April 1, 1990
4-(N-Nitrosomethylamino)-1-(3-pyridyl)1-butanone	cancer	64091-91-4	April 1, 1990
N-Nitrosomethyl-*n*-butylamine	cancer	7068-83-9	December 26, 2014
N-Nitrosomethyl-*n*-decylamine	cancer	75881-22-0	December 26, 2014
N-Nitrosomethyl-*n*-dodecylamine	cancer	55090-44-3	December 26, 2014
N-Nitrosomethylethylamine	cancer	10595-95-6	October 1, 1989
N-Nitrosomethyl-*n*-heptylamine	cancer	16338-99-1	December 26, 2014
N-Nitrosomethyl-*n*-hexylamine	cancer	28538-70-7	December 26, 2014
N-Nitrosomethyl-*n*-nonylamine	cancer	75881-19-5	December 26, 2014
N-Nitrosomethyl-*n*-octylamine	cancer	34423-54-6	December 26, 2014
N-Nitrosomethyl-*n*-pentylamine	cancer	13256-07-0	December 26, 2014
N-Nitrosomethyl-*n*-propylamine	cancer	924-46-9	December 26, 2014
N-Nitrosomethyl-*n*-tetradecylamine	cancer	75881-20-8	December 26, 2014
N-Nitrosomethyl-*n*-undecylamine	cancer	68107-26-6	December 26, 2014
N-Nitroso-N-methylurea	cancer	684-93-5	October 1, 1987
N-Nitroso-N-methylurethane	cancer	615-53-2	April 1, 1988
N-Nitrosomethylvinylamine	cancer	4549-40-0	January 1, 1988
N-Nitrosomorpholine	cancer	59-89-2	January 1, 1988
N-Nitrosonornicotine	cancer	16543-55-8	January 1, 1988
N-Nitrosopiperidine	cancer	100-75-4	January 1, 1988
N-Nitrosopyrrolidine	cancer	930-55-2	October 1, 1987
N-Nitrososarcosine	cancer	13256-22-9	January 1, 1988
o-Nitrotoluene	cancer	88-72-2	May 15, 1998

Nitrous oxide	developmental, female	10024-97-2	August 1, 2008
Norethisterone (Norethindrone)	cancer	68-22-4	October 1, 1989
Norethisterone (Norethindrone)	developmental	68-22-4	April 1, 1990
Norethisterone acetate (Norethindrone acetate)	developmental	51-98-9	October 1, 1991
Norethisterone (Norethindrone) /Ethinyl estradiol	developmental	68-22-4/ 57-63-6	April 1, 1990
Norethisterone (Norethindrone)/Mestranol	developmental	68-22-4/ 72-33-3	April 1, 1990
Norethynodrel	cancer	68-23-5	February 27, 2001
Norgestrel	developmental	6533-00-2	April 1, 1990
Ochratoxin A	cancer	303-47-9	July 1, 1990
Oil Orange SS	cancer	2646-17-5	April 1, 1988
Oral contraceptives, combined	cancer	---	October 1, 1989
Oral contraceptives, sequential	cancer	---	October 1, 1989
Oryzalin	cancer	19044-88-3	September 12, 2008
Oxadiazon	cancer	19666-30-9	July 1, 1991
Oxadiazon	developmental	19666-30-9	May 15, 1998
Oxazepam	cancer	604-75-1	October 1, 1994
Oxazepam	developmental	604-75-1	October 1, 1992
~~p,p'-Oxybis(benzenesulfonyl hydrazide)~~ Delisted December 13, 2013	~~developmental~~	~~80-51-3~~	~~August 7, 2009~~
Oxydemeton methyl	female, male	301-12-2	November 6, 1998
Oxymetholone	cancer	434-07-1	January 1, 1988
Oxymetholone	developmental	434-07-1	May 1, 1997
Oxytetracycline (internal use)	developmental	79-57-2	January 1, 1991
Oxytetracycline hydrochloride (internal use)	developmental	2058-46-0	October 1, 1991
Oxythioquinox (Chinomethionat)	cancer	2439-01-2	August 20, 1999
Oxythioquinox (Chinomethionat)	developmental	2439-01-2	November 6, 1998
Paclitaxel	developmental, female, male	33069-62-4	August 26, 1997
Palygorskite fibers (> 5μm in length)	cancer	12174-11-7	December 28, 1999
Panfuran S	cancer	794-93-4	January 1, 1988
Paramethadione	developmental	115-67-3	July 1, 1990
Parathion	cancer	56-38-2	May 20, 2016
Penicillamine	developmental	52-67-5	January 1, 1991
Pentabromodiphenyl ether mixture [DE-71 (technical grade)]	cancer	---	July 7, 2017
Pentachlorophenol	cancer	87-86-5	January 1, 1990
Pentachlorophenol and by-products of its synthesis (complex mixture)	cancer	---	October 21, 2016
Pentobarbital sodium	developmental	57-33-0	July 1, 1990
Pentosan polysulfate sodium	cancer	---	April 18, 2014
Pentostatin	developmental	53910-25-1	September 1, 1996
Perfluorooctane sulfonate (PFOS)	developmental	1763-23-1	November 10, 2017
Perfluorooctanoic acid (PFOA)	developmental	335-67-1	November 10, 2017
Pertuzumab	developmental	380610-27-5	January 27, 2017
Phenacemide	developmental	63-98-9	July 1, 1990
Phenacetin	cancer	62-44-2	October 1, 1989
Phenazopyridine	cancer	94-78-0	January 1, 1988
Phenazopyridine hydrochloride	cancer	136-40-3	January 1, 1988

Phenesterin	cancer	3546-10-9	July 1, 1989
Phenobarbital	cancer	50-06-6	January 1, 1990
Phenolphthalein	cancer	77-09-8	May 15, 1998
Phenoxybenzamine	cancer	59-96-1	April 1, 1988
Phenoxybenzamine hydrochloride	cancer	63-92-3	April 1, 1988
Phenprocoumon	developmental	435-97-2	October 1, 1992
o-Phenylenediamine and its salts	cancer	95-54-5	May 15, 1998
Phenyl glycidyl ether	cancer	122-60-1	October 1, 1990
~~Phenyl glycidyl ether~~ Delisted April 4, 2014	~~male~~	~~122-60-1~~	~~August 7, 2009~~
Phenylhydrazine and its salts	cancer	---	July 1, 1992
o-Phenylphenate, sodium	cancer	132-27-4	January 1, 1990
o-Phenylphenol	cancer	90-43-7	August 4, 2000
Phenylphosphine	~~developmental~~ male	638-21-1	August 7, 2009
PhiP(2-Amino-1-methyl-6-phenylimidazol[4,5-b]pyridine)	cancer	105650-23-5	October 1, 1994
Pimozide	developmental, female	2062-78-4	August 20, 1999
Pioglitazone	cancer	111025-46-8	April 18, 2014
Pipobroman	developmental	54-91-1	July 1, 1990
Pirimicarb	cancer	23103-98-2	July 1, 2008
Plicamycin	developmental	18378-89-7	April 1, 1990
Polybrominated biphenyls	cancer	---	January 1, 1988
Polybrominated biphenyls	developmental	---	October 1, 1994
Polychlorinated biphenyls	cancer	---	October 1, 1989
Polychlorinated biphenyls	developmental	---	January 1, 1991
Polychlorinated biphenyls (containing 60 or more percent chlorine by molecular weight)	cancer	---	January 1, 1988
Polychlorinated dibenzo-*p*-dioxins	cancer	---	October 1, 1992
Polychlorinated dibenzofurans	cancer	---	October 1, 1992
Polygeenan	cancer	53973-98-1	January 1, 1988
Ponceau MX	cancer	3761-53-3	April 1, 1988
Ponceau 3R	cancer	3564-09-8	April 1, 1988
Potassium bromate	cancer	7758-01-2	January 1, 1990
Potassium dimethyldithiocarbamate	developmental	128-03-0	March 30 1999
Pravastatin sodium	developmental	81131-70-6	March 3, 2000
Prednisolone sodium phosphate	developmental	125-02-0	August 20, 1999
Primidone	cancer	125-33-7	August 20, 1999
Procarbazine	cancer	671-16-9	January 1, 1988
Procarbazine hydrochloride	cancer	366-70-1	January 1, 1988
	developmental		July 1, 1990
Procymidone	cancer	32809-16-8	October 1, 1994
Progesterone	cancer	57-83-0	January 1, 1988
Pronamide	cancer	23950-58-5	May 1, 1996
Propachlor	cancer	1918-16-7	February 27, 2001
1,3-Propane sultone	cancer	1120-71-4	January 1, 1988
Propargite	cancer	2312-35-8	October 1, 1994
Propargite	developmental	2312-35-8	June 15, 1999
Propazine	developmental, female	139-40-2	July 15, 2016
beta-Propiolactone	cancer	57-57-8	January 1, 1988
Propoxur	cancer	114-26-1	August 11, 2006
Propylene glycol mono-*t*-butyl ether	cancer	57018-52-7	June 11, 2004
Propylene oxide	cancer	75-56-9	October 1, 1988
Propylthiouracil	cancer	51-52-5	January 1, 1988
Propylthiouracil	developmental	51-52-5	July 1, 1990
Pulegone	cancer	89-82-7	April 18, 2014
Pymetrozine	cancer	123312-89-0	March 22, 2011

Pyridine	cancer	110-86-1	May 17, 2002
Pyrimethamine	developmental	58-14-0	January 29, 1999
Quazepam	developmental	36735-22-5	August 26, 1997
Quinoline and its strong acid salts	cancer	---	October 24, 1997
Quizalofop-ethyl	male	76578-14-8	December 24, 1999
Radionuclides	cancer	---	July 1, 1989
Reserpine	cancer	50-55-5	October 1, 1989
Residual (heavy) fuel oils	cancer	---	October 1, 1990
Resmethrin	cancer	10453-86-8	July 1, 2008
Resmethrin	developmental	10453-86-8	November 6, 1998
Retinol/retinyl esters, when in daily dosages in excess of 10,000 IU, or 3,000 retinol equivalents. (NOTE: Retinol/retinyl esters are required and essential for maintenance of normal reproductive function. The recommended daily level during pregnancy is 8,000 IU.)	developmental	---	July 1, 1989
Ribavirin	developmental	36791-04-5	April 1, 1990
Ribavirin	male	36791-04-5	February 27, 2001
Riddelliine	cancer	23246-96-0	December 3, 2004
Rifampin	developmental, female	13292-46-1	February 27, 2001
~~Saccharin~~ Delisted April 6, 2001	~~cancer~~	~~81-07-2~~	~~October 1, 1989~~
~~Saccharin, sodium~~ Delisted January 17, 2003	~~cancer~~	~~128-44-9~~	~~January 1, 1988~~
Safrole	cancer	94-59-7	January 1, 1988
Salted fish, Chinese-style	cancer	---	April 29, 2011
Secobarbital sodium	developmental	309-43-3	October 1, 1992
Sedaxane	cancer	874967-67-6	July 1, 2016
Selenium sulfide	cancer	7446-34-6	October 1, 1989
Sermorelin acetate	developmental	---	August 20, 1999
Shale-oils	cancer	68308-34-9	April 1, 1990
Silica, crystalline (airborne particles of respirable size)	cancer	---	October 1, 1988
Simazine	developmental, female	122-34-9	July 15, 2016
Sodium dimethyldithiocarbamate	developmental	128-04-1	March 30 1999
Sodium fluoroacetate	male	62-74-8	November 6, 1998
Soots, tars, and mineral oils (untreated and mildly treated oils and used engine oils)	cancer	---	February 27, 1987
Spirodiclofen	cancer	148477-71-8	October 8, 2010
Spironolactone	cancer	52-01-7	May 1, 1997
Stanozolol	cancer	10418-03-8	May 1, 1997
Sterigmatocystin	cancer	10048-13-2	April 1, 1988
Streptomycin sulfate	developmental	3810-74-0	January 1, 1991
Streptozocin (streptozotocin)	developmental, female, male	18883-66-4	August 20, 1999
Streptozotocin (streptozocin)	cancer	18883-66-4	January 1, 1988
Strong inorganic acid mists containing sulfuric acid	cancer	---	March 14, 2003

Styrene	cancer	100-42-5	April 22, 2016
Styrene oxide	cancer	96-09-3	October 1, 1988
Sulfallate	cancer	95-06-7	January 1, 1988
Sulfasalazine (Salicylazosulfapyridine)	cancer	599-79-1	May 15, 1998
Sulfasalazine (Salicylazosulfapyridine)	male	599-79-1	January 29, 1999
Sulfur dioxide	developmental	7446-09-5	July 29, 2011
Sulindac	developmental, female	38194-50-2	January 29, 1999
Talc containing asbestiform fibers	cancer	---	April 1, 1990
Tamoxifen and its salts	cancer	10540-29-1	September 1, 1996
Tamoxifen citrate	developmental	54965-24-1	July 1, 1990
Temazepam	developmental	846-50-4	April 1, 1990
Teniposide	developmental	29767-20-2	September 1, 1996
Terbacil	developmental	5902-51-2	May 18, 1999
Teriparatide	cancer	52232-67-4	August 14, 2015
Terrazole	cancer	2593-15-9	October 1, 1994
Testosterone and its esters	cancer	58-22-0	April 1, 1988
Testosterone cypionate	developmental	58-20-8	October 1, 1991
Testosterone enanthate	developmental	315-37-7	April 1, 1990
Tetrabromobisphenol A	cancer	79-94-7	October 27, 2017
3,3′,4,4′-Tetrachloroazobenzene	cancer	14047-09-7	July 24, 2012
2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD)	cancer	1746-01-6	January 1, 1988
2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD)	developmental	1746-01-6	April 1, 1991
1,1,1,2-Tetrachloroethane	cancer	630-20-6	September 13, 2013
1,1,2,2-Tetrachloroethane	cancer	79-34-5	July 1, 1990
Tetrachloroethylene (Perchloroethylene)	cancer	127-18-4	April 1, 1988
p-a,a,a-Tetrachlorotoluene	cancer	5216-25-1	January 1, 1990
Tetrachlorvinphos	cancer	22248-79-9	May 20, 2016
Tetracycline (internal use)	developmental	60-54-8	October 1, 1991
Tetracyclines (internal use)	developmental	---	October 1, 1992
Tetracycline hydrochloride (internal use)	developmental	64-75-5	January 1, 1991
Tetrafluoroethylene	cancer	116-14-3	May 1, 1997
Tetranitromethane	cancer	509-14-8	July 1, 1990
Thalidomide	developmental	50-35-1	July 1, 1987
Thioacetamide	cancer	62-55-5	January 1, 1988
4,4'-Thiodianiline	cancer	139-65-1	April 1, 1988
Thiodicarb	cancer	59669-26-0	August 20, 1999
Thioguanine	developmental	154-42-7	July 1, 1990
Thiophanate methyl	female, male	23564-05-8	May 18, 1999
Thiouracil	cancer	141-90-2	June 11, 2004
Thiourea	cancer	62-56-6	January 1, 1988
Thorium dioxide	cancer	1314-20-1	February 27, 1987
Titanium dioxide (airborne, unbound particles of respirable size)	cancer	---	September 2, 2011
Tobacco, oral use of smokeless products	cancer	---	April 1, 1988
Tobacco smoke	cancer	---	April 1, 1988
Tobacco smoke (primary)	developmental, female, male	---	April 1, 1988

Tobramycin sulfate	developmental	49842-07-1	July 1, 1990
Toluene	developmental	108-88-3	January 1, 1991
	~~female~~	~~108-88-3~~	~~August 7, 2009~~
Toluene diisocyanate	cancer	26471-62-5	October 1, 1989
o-Toluidine	cancer	95-53-4	January 1, 1988
o-Toluidine hydrochloride	cancer	636-21-5	January 1, 1988
~~para-Toluidine~~ Delisted October 29, 1999	~~cancer~~	~~106-49-0~~	~~January 1, 1990~~
Topiramate	developmental	97240-79-4	November 27, 2015
Toxaphene (Polychlorinated camphenes)	cancer	8001-35-2	January 1, 1988
Toxins derived from *Fusarium* Moniliforme (Fusarium verticillioides)	cancer	---	August 7, 2009
Treosulfan	cancer	299-75-2	February 27, 1987
Triadimefon	developmental, female, male	43121-43-3	March 30, 1999
Triamterene	cancer	396-01-0	April 18, 2014
Triazolam	developmental	28911-01-5	April 1, 1990
S,S,S-Tributyl phosphorotrithioate (Tribufos, DEF)	cancer	78-48-8	February 25, 2011
Tributyltin methacrylate	developmental	2155-70-6	December 1, 1999
Trichlormethine (Trimustine hydrochloride)	cancer	817-09-4	January 1, 1992
Trichloroacetic acid	cancer	76-03-9	September 13, 2013
Trichloroethylene	cancer	79-01-6	April 1, 1988
Trichloroethylene	developmental, male	79-01-6	January 31, 2014
2,4,6-Trichlorophenol	cancer	88-06-2	January 1, 1988
1,2,3-Trichloropropane	cancer	96-18-4	October 1,1992
Trientine hydrochloride	developmental	38260-01-4	February 27, 2001
Triforine	developmental	26644-46-2	June 18, 1999
~~1,3,5-Triglycidyl-s-triazinetrione~~ Delisted December 13, 2013	~~male~~	~~2451-62-9~~	~~August 7, 2009~~
Trilostane	developmental	13647-35-3	April 1, 1990
Trimethadione	developmental	127-48-0	January 1, 1991
2,4,5-Trimethylaniline and its strong acid salts	cancer	---	October 24, 1997
Trimethyl phosphate	cancer	512-56-1	May 1, 1996
Trimetrexate glucuronate	developmental	82952-64-5	August 26, 1997
TRIM® VX	cancer	---	May 25, 2018
2,4,6-Trinitrotoluene (TNT)	cancer	118-96-7	December 19, 2008
Triphenyltin hydroxide	cancer	76-87-9	July 1, 1992
Triphenyltin hydroxide	developmental	76-87-9	March 18, 2002
~~Tris(aziridinyl)-*p*-benzoquinone (Triaziquone)~~ Delisted December 8, 2006	~~cancer~~	~~68-76-8~~	~~October 1, 1989~~
Tris(1-aziridinyl)phosphine sulfide (Thiotepa)	cancer	52-24-4	January 1, 1988
Tris(2-chloroethyl) phosphate	cancer	115-96-8	April 1, 1992
Tris(2,3-dibromopropyl)phosphate	cancer	126-72-7	January 1, 1988
Tris(1,3-dichloro-2-propyl) phosphate (TDCPP)	cancer	13674-87-8	October 28, 2011
Trp-P-1 (Tryptophan-P-1)	cancer	62450-06-0	April 1, 1988
Trp-P-2 (Tryptophan-P-2)	cancer	62450-07-1	April 1, 1988
Trypan blue (commercial grade)	cancer	72-57-1	October 1, 1989

Unleaded gasoline (wholly vaporized)	cancer	---	April 1, 1988
Uracil mustard	cancer	66-75-1	April 1, 1988
	developmental, female, male		January 1, 1992
Urethane (Ethyl carbamate)	cancer	51-79-6	January 1, 1988
	developmental		October 1, 1994
Urofollitropin	developmental	97048-13-0	April 1, 1990
Valproate (Valproic acid)	developmental	99-66-1	July 1, 1987
Vanadium pentoxide (orthorhombic crystalline form)	cancer	1314-62-1	February 11, 2005
Vinblastine sulfate	developmental	143-67-9	July 1, 1990
Vinclozolin	cancer	50471-44-8	August 20, 1999
	developmental		May 15, 1998
Vincristine sulfate	developmental	2068-78-2	July 1, 1990
Vinyl bromide	cancer	593-60-2	October 1, 1988
Vinyl chloride	cancer	75-01-4	February 27, 1987
4-Vinylcyclohexene	cancer	100-40-3	May 1, 1996
4-Vinyl-cyclohexene	female, ~~male~~	100-40-3	August 7, 2009
4-Vinyl-1-cyclohexene diepoxide (Vinyl cyclohexene dioxide)	cancer	106-87-6	July 1, 1990
Vinyl cyclohexene dioxide (4-Vinyl-1-cyclohexene diepoxide)	female, ~~male~~	106-87-6	August 1, 2008
Vinyl fluoride	cancer	75-02-5	May 1, 1997
Vinylidene chloride (1,1-Dichloroethylene)	cancer	75-35-4	December 29, 2017
Vinyl trichloride (1,1,2-Trichloroethane)	cancer	79-00-5	October 1, 1990
Vismodegib	developmental, female, male	879085-55-9	January 27, 2017
Warfarin	developmental	81-81-2	July 1, 1987
Wood dust	cancer	---	December 18, 2009
2,6-Xylidine (2,6-Dimethylaniline)	cancer	87-62-7	January 1, 1991
Zalcitabine	cancer	7481-89-2	August 7, 2009
Zidovudine (AZT)	cancer	30516-87-1	December 18, 2009
Zileuton	cancer, developmental, female	111406-87-2	December 22, 2000

~~Zineb~~ Delisted October 29, 1999

Date: March 8, 2019

FINAL STATEMENT OF REASONS
TITLE 27, CALIFORNIA CODE OF REGULATIONS

SECTION 25705(b) SPECIFIC REGULATORY LEVELS POSING NO SIGNIFICANT RISK

NO SIGNIFICANT RISK LEVEL: GLYPHOSATE

This is the Final Statement of Reasons for the adoption of a No Significant Risk Level (NSRL)[1] for glyphosate. On June 26, 2017, the Office of Environmental Health Hazard Assessment (OEHHA) announced the listing of glyphosate, effective July 7, 2017, as a chemical known to the state to cause cancer for purposes of Proposition 65[2]. OEHHA issued a Notice of Proposed Rulemaking to adopt a proposed amendment to Section 25705, Specific Regulatory Levels Posing No Significant Risk, identifying an NSRL of 1100 micrograms per day (µg/day) for glyphosate under Title 27, California Code of Regulations, section 25705(b)[3]. The Initial Statement of Reasons sets forth the grounds for the amendment to the regulation.

Briefly, in developing the NSRL for glyphosate, OEHHA relied on Volume 112 in the series of International Agency for Research on Cancer (IARC) Monographs on the Evaluation of Carcinogenic Risks to Humans, entitled "Some Organophosphate Insecticides and Herbicides: Diazinon, Glyphosate, Malathion, Parathion, and Tetrachlorvinphos"[4], which summarizes the available data from rodent carcinogenicity studies of glyphosate, as well as other information relevant to the carcinogenic activity of this chemical. The NSRL is based upon the results of the most sensitive scientific study deemed to be of sufficient quality[5]. OEHHA agrees with IARC's determination that the increased incidence of hemangiosarcomas observed in a study of male CD-1 mice is treatment-related and is using that study as the basis for the NSRL.

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[1] No Significant Risk Levels (NSRLs) for cancer-causing chemicals have been established for many of the chemicals listed under Proposition 65. A business would not be required to provide a Proposition 65 warning for an exposure to a listed carcinogen that is at or below the NSRL.
[2] The Safe Drinking Water and Toxic Enforcement Act of 1986, codified at Health and Safety Code section 25249.5 *et. seq*., hereafter referred to as "Proposition 65" or "The Act".
[3] All further regulatory references are to sections of Title 27 of the Cal. Code of Regs., unless otherwise indicated.
[4] International Agency for Research on Cancer (IARC, 2015). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Volume 112, Some Organophosphate Insecticides and Herbicides: Diazinon, Glyphosate, Malathion, Parathion, and Tetrachlorvinphos. IARC, World Health Organization, Lyon, France. Available from: http://monographs.iarc.fr/ENG/Monographs/vol112/index.php
[5] Section 25703(a)(4)

The Notice of Proposed Rulemaking was published in the California Regulatory Notice Register on April 7, 2017 (Register 2017, No. 14-Z) and initiated a 45-day public comment period that was scheduled to close on May 22, 2017. OEHHA received several requests to extend the public comment period and it was extended until June 21, 2017. OEHHA received over 1,300 oral and written public comments on the proposed rulemaking from several organizations and numerous individuals.

PEER REVIEW

As required by Section 25302(e) of the regulations, on May 17, 2017, OEHHA provided the notice of proposed rulemaking and the initial statement of reasons for the proposed NSRL for glyphosate to the members of the Carcinogen Identification Committee for their individual review and comment. OEHHA received peer-review comments from committee members Thomas McDonald, M.P.H., Ph.D., Luoping Zhang, PhD, Shanaz Dairkee, PhD, and Jason Bush, Ph.D.

UPDATED INFORMATION

There are no updates to the information contained in the ISOR, and no new documents were relied upon or added to the rulemaking file. Non-substantive revisions were made to the final regulation text to align the text with the text currently printed in the California Code of Regulations.

SUMMARY AND RESPONSE TO RELEVANT COMMENTS RECEIVED

OEHHA's responses to the oral and written comments received throughout this rulemaking process are incorporated in this Final Statement of Reasons (FSOR). Some commenters analyzed IARC's scientific conclusions, supporting or disagreeing with IARC's classification of glyphosate as a Group 2A carcinogen and providing their own scientific analyses and conclusions, cited the conclusions of other international regulatory or scientific bodies that were contrary to IARC's, or expressed or reiterated general disagreement with the addition of glyphosate to the Proposition 65 list; such comments are not directed to the subject of this rulemaking, which is the establishment of an NSRL for glyphosate. OEHHA responded to these types of comments in the listing documents for glyphosate and does not respond to them again here.

Other commenters discussed the US Environmental Protection Agency's (US EPA's) report entitled 'Glyphosate Issue Paper: Evaluation of Carcinogenic Potential'[6],

[6] US EPA (2016). Glyphosate Issue Paper: Evaluation of Carcinogenic Potential. Office of Pesticide Programs, US Environmental Protection Agency. September 12, 2016. Available from: https://www.epa.gov/sites/production/files/2016-09/documents/glyphosate_issue_paper_evaluation_of_carcincogenic_potential.pdf

critiquing the analysis and conclusions therein, including comments that US EPA did not follow good laboratory practices in its weight of the evidence evaluation by omitting relevant studies[7], as well as concerns that the cancer-related data provided by the US EPA has been brought into question based on allegations of collusion with Monsanto. These comments are not directed to the subject of this rulemaking and are not responded to here.

OEHHA additionally received many comments during the regulatory process that included observations or opinions regarding the use of glyphosate; suggestions that OEHHA conduct further studies into the health effects of glyphosate; statements that the NSRL does not consider impacts other than carcinogenicity; concerns of increased chronic illness among children and the lack of studies of the effects of pesticides on children[8]; opinions that glyphosate is safe, regulated, and effective; statements of support for other actions that are not the subject of this rulemaking (such as banning or restricting use of the chemical); and recommendations to use methods of clinical testing of 0.5 parts per billion or lower, and much lower for urine and water testing[9]. Some commenters expressed concern over the negative effects of genetically modified organisms (GMOs), that all GMOs should be banned, or that the US Food and Drug Administration should adopt mandatory regulations concerning genetically engineered plants and animals[10]. Some commenters also stated that Monsanto is greedy, corrupt, or withholding scientific evidence of glyphosate's toxicity to humans and animals[11]. Such remarks do not constitute an objection or recommendation specifically directed at the proposed action, or the procedures followed in this rulemaking action. Accordingly, OEHHA is not required under the Administrative Procedure Act to respond to such comments in this FSOR. Because OEHHA is constrained by limitations upon its time and resources, and is not obligated by law to respond to irrelevant comments[12], OEHHA does not provide responses to all of these remarks in this FSOR. However, the absence of responses to such remarks should not be construed to mean that OEHHA agrees with them.

Many commenters made the same or similar comments, and this document does not provide an exhaustive accounting of all commenters addressing the same point. A summary of the comments relevant to this rulemaking is provided below, along with OEHHA's responses to those comments. As explained in detail in the responses to comments, OEHHA declines to change the proposed NSRL based on the comments.

[7] Comment from Kurt Wallace.
[8] Comment from Michelle Perro
[9] Comment from Diane Rude
[10] Comment from Stephanie Easton
[11] Comment from Kathleen Furey
[12] California Government Code section 11346.9(a)(3)

Comment 1 (Baum, Hedlund, Aristei & Goldman, P.C., A Voice for Choice, Donna R. Farmer, Ph.D., on behalf of Monsanto and others): The potency estimate for the NSRL should be based on cancer findings from human epidemiological studies, rather than on findings from animal carcinogenicity studies. Many commenters assert that in failing to consider epidemiologic studies, the proposed safe harbor level does not conform to "quantitative risk assessment" and that OEHHA did not follow Section 25703 of the regulations.

Some of these commenters went on to state that prioritizing animal bioassays over epidemiological data overlooks the risk to individuals exposed to glyphosate during its application as a pesticide. They further argue that use of epidemiological data would provide a more robust and comprehensive evaluation of a chemical which most users absorb via cutaneous and respirational contact.

Paul Eusey, Tricia Brooks, and several other commenters stated that OEHHA should review the lowest levels of glyphosate in the epidemiological studies, but should always err on the side of caution and public health (see also Response #29 and discussion of precautionary principle).

Response 1: As stated in Section 25703 of the regulations, the assessment used to derive the NSRL "shall be based on evidence and standards of comparable scientific validity to the evidence and standards which form the scientific basis for the listing of the chemical as known to the state to cause cancer"[13]. Glyphosate was listed pursuant to the Labor Code listing mechanism[14] as a result of IARC's classification of glyphosate in Group 2A ("probably carcinogenic to humans"), with a finding of sufficient evidence of carcinogenicity in experimental animals[15,16]. IARC also found "there is *limited evidence* in humans for the carcinogenicity of glyphosate", noting "[a] positive association has been observed for non-Hodgkin lymphoma." Given that the listing of glyphosate is based on findings of limited evidence in humans and sufficient evidence in animals, basing the potency estimate for the NSRL on animal studies is both appropriate and consistent with Section 25703.

Animal bioassays are more frequently used than epidemiological data in quantitatively assessing the health risks of chemicals, including carcinogens. The epidemiological

[13] Section 25703(a)(4)
[14] Section 25249.8(a) of the Act
[15] OEHHA (2015). Notice of Intent to List - Tetrachlorvinphos, Parathion, Malathion, Glyphosate. https://oehha.ca.gov/media/downloads/crnr/090415noillcset27.pdf
[16] IARC (2015). Full citation provided in footnote 3.

studies evaluated by IARC, like many human studies, do not provide the type of information on levels of exposure that is needed for dose-response analysis. Specifically, these studies broadly characterized glyphosate exposure to individuals as either 'never' or 'ever' exposed, or as 'duration' of exposure, and were unable to quantify the individuals' specific levels of exposure to the chemical. Since the epidemiology studies did not measure or estimate the dose level to which participants were exposed, a cancer potency cannot be calculated using these studies.

OEHHA disagrees with the commenters' assertions that the use of animal cancer bioassay data to estimate cancer potency results in a less robust or comprehensive risk assessment than would the use of epidemiologic data, or that the use of animal data in some way overlooks risks to workers or other individuals exposed to glyphosate. As noted above, the epidemiologic studies available to date on glyphosate only provide limited evidence of a causal relationship between exposure and cancer risk, and they do not provide the type of information on levels of exposure needed in order to estimate cancer potency. Thus, OEHHA's use of animal cancer bioassay data from the most sensitive study of sufficient quality to estimate human cancer potency for this chemical is appropriate and consistent with the Proposition 65 regulations[17], other cancer risk assessment guidance from OEHHA[18], and guidance from US EPA[19]. The estimate of human cancer potency is equally valid for estimating risks to occupationally exposed workers and to other individuals exposed to glyphosate, and the NSRL for glyphosate is not limited to a specific route of exposure[20,21]. No change to the regulatory proposal was made based on these comments.

Comment 2 (Moms Across America, Marty Eustis, Majorie Golden, Gloria Anderson and other commenters): Glyphosate induces breast cancer in humans. Marty Eustis commented that the NSRL should be "substantially lower" than the proposed 1100 micrograms/day in order to actually be safe to Californians. Majorie Golden, Gloria Anderson, and Marty Eustis commented that until a comprehensive independent study is done, the NSRL should be at or "well below 0.0001 mg/day" (Thongprakisang et al.), the concentration where it stimulated breast cancer cells in vitro.

[17] Section 25703
[18] OEHHA (2009). Technical Support Document for Cancer Potency Factors. https://oehha.ca.gov/media/downloads/crnr/tsdcancerpotency.pdf
[19] US EPA (2005). Guidelines for Carcinogen Risk Assessment. March, 2005. Risk Assessment Forum, US Environmental Protection Agency, Washington, DC.
[20] OEHHA (2017). Initial Statement of Reasons, Title 27, California Code of Regulations, Proposed Amendment to: Section 25705(b) Specific Regulatory Levels Posing No Significant Risk. Glyphosate. Available at https://oehha.ca.gov/media/downloads/crnr/glyphosate032917isor.pdf
[21] Section 25703(a)(4)

Response 2: These comments all appear to be based on an *in vitro* study by Thongprakaisang et al. (2013)[22], in which glyphosate was shown to induce proliferation in a hormone-dependent human breast cancer cell line (T47D cells derived from ductal carcinoma cells), but not in a hormone-independent human breast cancer cell line (MDA-MB231 breast adenocarcinoma cells). This study is not a human epidemiology study and thus it does not provide evidence that glyphosate induces breast cancer in humans. Rather, it is a study of the effect of glyphosate on the proliferation of cultured cells, and it does not provide information that can be used to derive the NSRL for glyphosate. No changes were made to the regulatory proposal based on this comment.

Comment 3 (Monsanto, Ramboll Environ on behalf of The Scotts Company LLC, and others): Reviews by others have concluded that there are no treatment-related tumors in animal cancer bioassays of glyphosate, nor are there other datasets that provide evidence of a strong dose-response relationship of carcinogenicity that could be relied upon to estimate the potential for health effects in humans following exposure to expected concentrations and that the lack of an adequate dataset is consistent with conclusions reached by JMPR (2006) and US EPA (2016) that any tumor findings are not treatment-related. OEHHA has no basis to quantify an NSRL using experimental animal studies.

Response 3: Glyphosate was listed under Proposition 65 via the "Labor Code" listing mechanism, based on IARC's classification[23] of glyphosate as *probably carcinogenic to humans* (Group 2A), and its conclusion that there is *sufficient evidence* of carcinogenicity in experimental animals for glyphosate. IARC's conclusion of sufficient evidence in experimental animals is based on findings from two studies in male mice. Specifically, IARC cited "a significant positive trend in the incidence of haemangiosarcoma [a malignant neoplasm] in male CD-1 mice" in a two-year diet study[24], and "a positive trend in the incidence of renal tubule carcinoma [a malignant neoplasm] and of renal tubule adenoma and carcinoma (combined) [an appropriate combination of benign and malignant neoplasms]" in male CD-1 mice in a different

[22] Thongprakaisang S, Thiantanawat A, Rangkadilok N, Suriyo T, Satayavivad J., 2013. Glyphosate induces human breast cancer cells growth via estrogen receptors. *Food Chem Toxicol* **59**:129-36.
[23] IARC (2015). Full citation provided in footnote 3.
[24] As noted in the Initial Statement of Reasons, this study of glyphosate (purity 98.6%) met the criterion in Section 25703 as the most sensitive study of sufficient quality, and was used to derive the NSRL. This study was performed by Inveresk Research International and summarized in the 2006 Joint FAO/WHO Meeting on Pesticide Residues report (JMPR, 2006. Glyphosate. In: Joint FAO/WHO Meeting on Pesticide Residues. Pesticide residues in food – 2004: toxicological evaluations. Report No. WHO/PCS/06.1. Geneva: World Health Organization; pp. 95 – 169.) and by IARC (IARC, 2015, IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Volume 112. Some Organophosphate Insecticides and Herbicides: Diazinon, Glyphosate, Malathion, Parathion, and Tetrachlorvinphos. IARC, World Health Organization, Lyon, France).

two-year diet study[25], with IARC noting that these malignant kidney tumors are rare in this strain of mice. OEHHA agrees with IARC's determination that these tumor findings are treatment-related and demonstrate statistically significant dose-response relationships.

In developing the NSRL for glyphosate, OEHHA followed the guidance set forth in Section 25703 that the assessment "be based on evidence and standards of comparable scientific validity to the evidence and standards which form the scientific basis for the listing of the chemical as known to the state to cause cancer", and based the NSRL on the results of the most sensitive scientific study deemed to be of sufficient quality. OEHHA determined that the two-year study conducted in male CD-1 mice fed glyphosate (purity 98.6%) in the diet, in which a significant positive trend in the incidence of hemangiosarcomas was observed, met the criteria in 25703 as the most sensitive study of sufficient quality. OEHHA used this data to derive the NSRL for glyphosate. No changes were made to the regulatory proposal based on this comment.

Comment 4 (Monsanto): The commenter cited the decision in *Baxter Healthcare Corp. v. Denton,* 120 Cal. App. 4th 333, 15 Cal. Rptr. 3d 430 (2004) to support its assertion that OEHHA is required to determine that a glyphosate exposure at any level does not pose a "significant risk", and as such requires OEHHA to establish an "infinite" NSRL. Baum, Hedlund, Aristei & Goldman, P.C. and others stated that Monsanto's reliance on *Baxter v. Denton* is inappropriate.

Response 4: OEHHA disagrees that the *Baxter* decision mandates the establishment of an infinite NSRL. The decision in *Baxter* is factually distinguishable from the proposed NSRL for glyphosate[26]. The commenter provides no evidence that the mechanism of action for glyphosate does not operate in humans, which was the pivotal issue in that case. In *Baxter*, the Appellate Court focused on evidence that the mechanism by which DEHP increased the incidence of liver tumors in animals was not relevant to humans[27]. This notably included evidence regarding the classification of DEHP by IARC[28]. At the time of the *Baxter* decision, IARC had downgraded its earlier classification of DEHP as Group 2B ("possibly carcinogenic to humans") to Group 3 ("not classifiable as to its carcinogenicity to humans"). Glyphosate, on the other hand,

[25] In summarizing this study of glyphosate (purity 99.7%), IARC cited four US EPA documents (US EPA 1985a, b, 1986, 1991a) (IARC, 2015, IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Volume 112. Some Organophosphate Insecticides and Herbicides: Diazinon, Glyphosate, Malathion, Parathion, and Tetrachlorvinphos. IARC, World Health Organization, Lyon, France).
[26] See comment letters from Baum, Hedlund, Aristei & Goldman, P.C., (Comment #9945) and Center for Biological Diversity, et al. (Comment #9974)]
[27] Baxter Healthcare Corp. v. Denton, 120 Cal. App. 4th 333, 15 Cal. Rptr. 3d 430 (2004), at 438.
[28] *Id.*

has received a higher Group 2A classification from IARC[29]. IARC's Group 2A classification of glyphosate is based on "sufficient evidence" in animal studies and "limited evidence" in human (epidemiological) studies. IARC found that mechanistic and other relevant data support the Group 2A classification of glyphosate (e.g., "strong" evidence for genotoxicity, both for "pure" glyphosate and for glyphosate formulations) and concluded, "[t]here is evidence that these effects can operate in humans". IARC has not reclassified glyphosate, or modified its findings that animal studies provided sufficient evidence of carcinogenicity and human studies provided limited evidence of carcinogenicity. No changes to the regulatory proposal were made based on this comment.

Comment 5 (Monsanto, Chris Portier, SafeAgSafeSchools, Anthony Samsel, Baum, Hedlund, Aristei & Goldman, P.C., and others): Monsanto commented that according to Section 25703, OEHHA's assessment is not limited to the specific studies used as the basis for listing the chemical, but instead OEHHA's "assessment shall be based on evidence and standards of comparable scientific validity to the evidence and standards which form the scientific basis for listing the chemical as known to the state to cause cancer." Monsanto went on to say that OEHHA's basis for listing is IARC's classification of glyphosate as a category 2A chemical on the basis of sufficient evidence in animals and that OEHHA should consider all available rodent studies and not just the select few that IARC chose to evaluate. The other studies contradict the conclusions reached by IARC's working group with respect to the four referenced animal studies.

Additionally, Chris Portier, Safe Ag Safe Schools, Anthony Samsel, Baum, Hedlund, Aristei & Goldman, P.C., Sonoma County Conservation Action[30] and others requested that OEHHA analyze and incorporate additional bioassay data in the derivation of an NSRL for glyphosate, not just studies reviewed by IARC. This includes the studies discussed in the review article by Greim et al. (2015). Some of these studies, including Wood et al. (2009), Lankas (1981), and Stout and Ruecker (1990), as cited by Baum, Hedlund, Aristei & Goldman, P.C. and Safe Ag Safe Schools, observed tumors or lymphomas at much lower doses than the study used to derive the NSRL. Baum, Hedlund, Aristei & Goldman, P.C, stated that if the data from these studies were used, a significantly lower NSRL would have been reached. Safe Ag Safe Schools stated that the NSRL is not based on the most sensitive study of acceptable quality and should be based on a dose of 31.49 mg/kg/day. Chris Portier and the Center for Biological Diversity commented that the Atkinson study is not the most sensitive study of sufficient

[29] IARC (2015). Full citation provided in footnote 3.
[30] The commenter suggested a revised NSRL based on a dose of 31.39/mg/kg/day, which is related to the Lankas study discussed in Greim et al.

quality to guide the suggested NSRL, and that other studies provide a more scientifically sound and health- protective basis for calculating the NSRL (i.e., Wood et al. [2009], Lankas [1981], and Stout and Ruecker [1990]), and that OEHHA must do an independent analysis of these studies and not rely on US EPA's conclusions.

During the public hearing for this rulemaking, Dr. Donna Farmer, senior toxicologist at Monsanto's Regulatory Product Safety Center, commented that OEHHA's reliance on male mouse hemangiosarcomas is not justified for the derivation of a NSRL.

Seosamh Devine commented that OEHHA relied too much on Monsanto's scientific opinions.

Response 5: As noted by the commenters, Section 25703 of the regulations states that the assessment used to derive the cancer potency "shall be based on evidence and standards of comparable scientific validity to the evidence and standards which form the scientific basis for the listing of the chemical as known to the state to cause cancer"[31]. Glyphosate was listed under Proposition 65 via the "Labor Code" listing mechanism, based on IARC's classification[32] of glyphosate as *probably carcinogenic to humans* (Group 2A), and its conclusion that there is *sufficient evidence* of carcinogenicity in experimental animals for glyphosate. As discussed in response to comment 3, IARC's conclusion of sufficient evidence in experimental animals is based on findings from two studies in male mice. Specifically, IARC cited "a significant positive trend in the incidence of haemangiosarcoma in male CD-1 mice" in a two-year diet study[33], and "a positive trend in the incidence of renal tubule carcinoma and of renal tubule adenoma and carcinoma (combined)" in male CD-1 mice in a different two-year diet study[34], with IARC noting that these malignant kidney tumors are rare in this strain of mice.

In contrast to the commenters implication that IARC only evaluated a select few studies in its monograph on glyphosate, IARC[35] discussed each of the 14 sets of animal cancer

[31] Section 25703(a)(4)
[32] IARC (2015). Full citation provided in footnote 3.
[33] As noted in the Initial Statement of Reasons, this study of glyphosate (purity 98.6%) met the criterion in Section 25703 as the most sensitive study of sufficient quality, and was used to derive the NSRL. This study was performed by Inveresk Research International and summarized in the 2006 Joint FAO/WHO Meeting on Pesticide Residues report (JMPR, 2006. Glyphosate. In: Joint FAO/WHO Meeting on Pesticide Residues. Pesticide residues in food – 2004: toxicological evaluations. Report No. WHO/PCS/06.1. Geneva: World Health Organization; pp. 95 – 169.) and by IARC (IARC, 2015, IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Volume 112. Some Organophosphate Insecticides and Herbicides: Diazinon, Glyphosate, Malathion, Parathion, and Tetrachlorvinphos. IARC, World Health Organization, Lyon, France).
[34] In summarizing this study of glyphosate (purity 99.7%), IARC cited four US EPA documents (US EPA 1985a, b, 1986, 1991a) (IARC, 2015, IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Volume 112. Some Organophosphate Insecticides and Herbicides: Diazinon, Glyphosate, Malathion, Parathion, and Tetrachlorvinphos. IARC, World Health Organization, Lyon, France).
[35] IARC (2015). Full citation provided in footnote 3

studies (five in mice and nine in rats)[36] included in the review by Greim *et al*. (2015)[37], as well as two additional sets of studies in rats, for a total of 16 sets of animal cancer studies. IARC noted in particular that the information reported in the article by Greim *et al*. and provided in the supplemental materials lacked sufficient detail regarding "statistical methods, choice of doses, body-weight gain, survival data, details of histopathological examination, and/or stability of dosed feed mixture" to be evaluated[38]. IARC evaluations "rely only on data that are in the public domain and available for independent scientific review"[39]. Utilizing additional sources in the public domain, IARC was able to conduct independent scientific review of two of the five sets of mouse studies included in Greim *et al*., five of the nine sets of rat studies included in Greim *et al*., and two additional sets of rat studies not included in Greim *et al*.

OEHHA is not aware of any additional animal cancer studies of glyphosate, other than the 16 sets of studies discussed by IARC. Of those 16 sets, IARC found that two sets of studies in mice and six sets of studies in rats were *adequate* for the evaluation of glyphosate carcinogenicity (emphasis added).

Of those eight sets of rodent studies, treatment-related increases in the incidence of malignant tumors were observed in one study in male mice, and treatment-related increases in the incidence of combined malignant and benign tumors were observed in a second male mouse study. Treatment-related increases in benign tumors were observed in two male rat studies and one female rat study; in each case, IARC noted there was no apparent progression of the benign tumors to malignancy.

Thus, OEHHA reviewed the available data from the rodent carcinogenicity studies of glyphosate in light of the requirement of Section 25703 that the assessment "be based on evidence and standards of comparable scientific validity to the evidence and standards which form the scientific basis for the listing of the chemical as known to the state to cause cancer", and determined that the two-year study conducted in male CD-1 mice fed glyphosate (purity, 98.6%) in the diet met the criterion in Section 25703 as the most sensitive study of sufficient quality. OEHHA agrees with IARC's determination that the increased incidence of hemangiosarcomas observed in this study of male CD-1 mice is treatment-related.

[36] Each set of studies consists of two experiments, one in males and one in females.

[37] Greim H, Saltmiras D, Mostert V, Strupp C (2015). Evaluation of carcinogenic potential of the herbicide glyphosate, drawing on tumor incidence data from fourteen chronic/carcinogenicity rodent studies. *Crit Rev Toxicol* 45(3):185-208.

[38] IARC (2015). Full citation provided in footnote 3.

[39] IARC (2015). IARC monograph Volume 112, General Remarks. p. 35.

OEHHA used this data to derive the NSRL for glyphosate. OEHHA did not rely on US EPA's conclusions to derive the NSRL for glyphosate; nor did OEHHA rely on Monsanto's scientific opinions to derive the NSRL (see also Response #3).

No changes were made to the regulatory proposal based on this comment.

Comment 6 (Valerie Noble and several commenters): The proposed NSRL does not account for bioaccumulation of glyphosate. Food Democracy Now further stated that a 2004 joint report from the United Nations Food and Agriculture [Organization] Program [*sic*] and the World Health Organization determined that glyphosate accumulates in the bones of lab animals.

Response 6: Valerie Noble did not provide a citation for the finding she attributed to Kruger et al. regarding bioaccumulation of glyphosate. OEHHA performed a literature search and identified one publication authored by Monika Kruger[40] . Contrary to the commenter's assertion, this publication provides no data indicating that glyphosate bioaccumulates. OEHHA is not aware of any evidence from studies in humans that demonstrate that glyphosate bioaccumulates. Similarly, there is no evidence that glyphosate bioaccumulates in non-human primates, or other mammals. For example, in rhesus monkeys, nearly all of an intravenous dose of glyphosate was eliminated within 24 hours[41], and in Fischer 344 rats greater than 90% of an oral dose of glyphosate was eliminated within 72 hours[42]. In another rat study, the total body burden of radiolabeled glyphosate residues measured 7 days after a single oral dose was approximately 1% of the administered dose. Further, no evidence of glyphosate bioaccumulation was observed in two repeated dosing studies conducted in rats[43].

The report referred to by the commenters appears to be the 2006 Joint FAO/WHO Meeting on Pesticide Residues (JMPR) report. However, the report does not conclude that glyphosate accumulates in the bones of lab animals. The report states that, after reviewing studies in mammals, there is no evidence of accumulation of glyphosate in

[40] Krüger M, Shehata AA, Schrödl W, and Rodloff A (2013). Glyphosate suppresses the antagonistic effect of Enterococcus spp. on Clostridium botulinum. Anaerobe 20: 74–78. Available from: https://www.ncbi.nlm.nih.gov/pubmed/23396248
[41] IARC (2015) p. 45, full citation provided in footnote 3.
[42] IARC (2015) p. 44, full citation provided in footnote 3.
[43] IARC (2015) p. 43, full citation provided in footnote 3.

mammals[44][45]. No changes were made to the regulatory proposal based on this comment.

Comment 7 (Meghan Lawler, Pesticide Free Zone, and Laura Hayes, Linda Causey, Zen Honeycutt and other commenters): OEHHA should consider effects other than carcinogenicity in setting the NSRL, such as evidence of induction of liver disease at 4 nanograms/kg, teratogenicity, breakdown of the blood-brain barrier, and evidence of destruction of gut bacteria at 0.1 ppm. Meghan Lawler and Laura Hayes stated that glyphosate is a neurotoxin, endocrine disruptor, mineral chelator, and antibiotic, and that it causes liver disease.

Some commenters stated that the NSRL fails to account for the potential transgenerational effects of endocrine disruptors, and asserted that an appropriate study to determine the NSRL should involve mice studies for three generations. Pesticide Free Zone commented that by excluding low dose studies from consideration, OEHHA may not be accounting for harmful endocrine-disrupting chemical actions. Laura Hayes commented that the most serious negative health consequences result when glyphosate substitutes for glycine during protein synthesis.

Response 7: Proposition 65 requires the maintenance and updating of a list of chemicals that cause cancer or reproductive toxicity, and requires businesses that knowingly cause exposures to listed chemicals to provide warnings. Other health effects – including liver disease, breakdown of the blood-brain barrier and destruction of gut bacteria – are outside the scope of the law. Following the guidance set forth in Section 25703, OEHHA bases NSRLs on cancer dose-response assessments, which are conducted using data from the most sensitive scientific studies deemed to be of sufficient quality. Observations of liver disease, teratogenicity, breakdown of the blood-brain barrier, destruction of gut bacteria, and endocrine disruption are not observations of cancer, and thus studies relating to such health effects do not provide data that can be used in a cancer dose-response assessment. The NSRL for glyphosate is based on animal carcinogenicity studies, and dose-response analysis of tumor incidence data from these studies.

[44] JMPR (2006). Glyphosate. In: Pesticide residues in food – 2004. Evaluations 2004 Part II – Toxicological evaluations, Joint Meeting of the FAO Panel of Experts on Pesticides Residues in Food and the Environment and the WHO Core Assessment Group, Rome, Italy, 20-29 September 2004, p. 95–116, 172. Available from: whqlibdoc.who.int/publications/2006/9241665203_eng.pdf

[45] JMPR (2016). Glyphosate. In: Joint FAO/WHO Meeting on Pesticide Residues. Pesticide residues in food 2016. Special Session of the Joint FAO/WHO Meeting on Pesticide Residues, Geneva, 9 to 13 May 2016. Rome: Food and Agriculture Organization of the United Nations/Geneva, World Health Organization (WHO) (FAO Plant Production and Protection Paper No. 227), p. 19–28, 45, 72–82. Available from: http://www.fao.org/3/a-i5693e.pdf

In reviewing the mechanistic data available for glyphosate, IARC did not conclude that glyphosate is carcinogenic via endocrine disruption. Rather, IARC concluded that there was strong evidence for genotoxicity and oxidative stress, and weak evidence for receptor-mediated effects. There are no data to suggest that glyphosate acts as a carcinogen via a transgenerational mechanism. OEHHA is not aware of any multi-generational cancer studies of glyphosate.

No changes were made to the regulatory proposal based on these comments.

Comment 8 (K. Paul Stoller, MD, Nancy O'Mara, MPH, Mei-Ling Stefan, Anthony Samsel and others): Urge consideration of the possible human health effects of other chemicals present in commercial formulations of glyphosate, e.g. adjuvants, surfactants, and inert ingredients, as well as consideration of possible synergism of glyphosate with other xenobiotic chemicals. There are no safe levels of the N-nitrosamines of glyphosate that are found in every glyphosate product.

Response 8: The Proposition 65 warning requirement applies only to chemicals listed for causing cancer or reproductive toxicity. In this case, the substance listed as causing cancer is glyphosate[46], not commercial formulations of glyphosate. Analysis of possible effects (*e.g.,* additive, synergistic, or antagonistic) of other exposures that may co-occur with glyphosate is outside the scope of Proposition 65 and is not relevant to the derivation of the NSRL for glyphosate. Thus, the NSRL is based on the results of the most sensitive scientific study of *glyphosate* deemed to be of sufficient quality. No changes were made to the regulatory proposal based on this comment.

Comment 9 (Dr. Stephen C. Frantz, Nancy O'Mara, MPH, and others): Urge consideration of a non-linear dose-response relationship, stating that endocrine disrupting chemicals, such as glyphosate, do not demonstrate the common default monotonic dose-response relationship.

Response 9: No data were provided to support the assertions that a non-monotonic cancer dose-response relationship exists for glyphosate.

[46] As noted in the Notice of Intent to List Glyphosate (https://oehha.ca.gov/proposition-65/crnr/notice-intent-list-tetrachlorvinphos-parathion-malathion-glyphosate) and the Notice of Listing (https://oehha.ca.gov/proposition-65/crnr/glyphosate-listed-effective-july-7-2017-known-state-california-cause-cancer), the 2015 IARC monograph on glyphosate indicates the following chemicals are "also relevant: 38641-94-0 (glyphosate-isopropylamine salt) 40465-66-5 (monoammonium salt) 69254-40-6 (diammonium salt) 34494-03-6 (glyphosate-sodium) 81591-81-3 (glyphosate-trimesium)" (IARC, 2015b), because these salts dissociate to free glyphosate.

As discussed in the Initial Statement of Reasons (ISOR)[47] for this action, OEHHA reviewed the available data from the rodent carcinogenicity studies of glyphosate discussed by IARC and determined that the most sensitive scientific study of sufficient quality for the cancer dose-response assessment was a study in male mice in which a statistically significant increasing trend in hemangiosarcoma was observed. The data from this study exhibited a monotonic dose-response relationship. Based upon consideration of the available mechanistic and other relevant data, OEHHA fit a multistage polynomial cancer model to the dose-response data to estimate cancer potency and derive the NSRL for glyphosate. This is consistent with the guidance set forth in Section 25703. No changes were made to the regulatory proposal based on this comment.

Comment 10 (Anthony Samsel): Glyphosate is a synthetic amino acid and an analogue of glycine. Glyphosate ligates with lysozyme, which may impact fibrocystic cytokines and human and animal immune systems. Glyphosate inhibits several enzymes, including protease, lipase, and pepsins, which can have effects on human health.

The commenter submitted three publications that were not included in IARC's review (Table 1).

Response 10: This comment is essentially a summary of Samsel and Seneff's 2016 article, entitled "Glyphosate pathways to modern disease V: Amino acid analogue of glycine in diverse proteins"[48]. This paper proposes a number of hypotheses regarding possible mechanisms by which glyphosate may effect human health. However, these hypotheses are not supported by experimental data and the relevance of the hypothesized health effects to cancer induction is unclear.

OEHHA reviewed each of the three publications in the context of the guidance set forth in Section 25703, which provides that "the assessment shall be based on evidence and standards of comparable scientific validity to the evidence and standards which form the scientific basis for the listing of the chemical as known to the state to cause cancer"[49] and determined that none of the studies provide data that would affect the cancer dose-response analysis (See Table 1). No changes were made to the regulation based on this comment.

[47] OEHHA (2017). Initial Statement of Reasons, Title 27, California Code of Regulations, Proposed Amendment to: Section 25705(b) Specific Regulatory Levels Posing No Significant Risk. Glyphosate. Available at https://oehha.ca.gov/media/downloads/crnr/glyphosate032917isor.pdf

[48] Samsel A and Seneff S (2016). Glyphosate pathways to modern disease V: Amino acid analogue of glycine in diverse proteins. *J Biol Phys Chem* **16**:9-46.

[49] Section 25703(a)(4)

Table 1. Publications submitted by Anthony Samsel

Reference	Comments
Samsel A and Seneff S (2015). Glyphosate pathways to modern disease IV: Cancer and related pathologies. *Journal of Biological Physics and Chemistry* **15**:121-159.	This article reviews epidemiological evidence of cancers in humans exposed to glyphosate and mechanistic information on glyphosate, and discusses possible carcinogenic mechanisms. "Glyphosate has a large number of tumorigenic effects on biological systems, including direct damage to DNA in sensitive cells, disruption of glycine homeostasis, succinate dehydrogenase inhibition, chelation of manganese, modification to more carcinogenic molecules such as N-nitrosoglyphosate and glyoxylate, disruption of fructose metabolism, etc." This article does not provide data that would affect the cancer dose-response analysis that forms the basis for the NSRL.
Samsel A and Seneff S (2016). Glyphosate pathways to modern disease V: Amino acid analogue of glycine in diverse proteins. *Journal of Biological Physics and Chemistry* **16**:9-46.	This article proposes that glyphosate is a synthetic amino acid and analogue of glycine, which can be incorporated into peptides, affect various enzymes, and lead to numerous diseases. "Glyphosate, acting as a glycine analogue, may be mistakenly incorporated into peptides during protein synthesis." "…the combination of activation of kinases and suppression of phosphatases that can plausibly be induced through glyphosate's displacement of conserved glycines in the enzymes can be predicted to lead to an overabundance of phosphorylated molecules, systemically." "Phosphorylation is a widespread modification with profound effects on affected molecules, which can increase risk to both Alzheimer's disease and cancer."

	"VLA-4 [very late antigen-4] is required for normal development of both T- and B-cells in the bone marrow, in part by regulating the balance between proliferation and differentiation of haematopoietic progenitors [291]. It can therefore be expected that impaired function would lead to pathologies such as immune dysfunction and cancer. Two conserved glycine residues at positions 130 and 190 are essential for its adhesive activity [292]. Glyphosate's link to NHL may therefore be explained through substitution of glyphosate for glycine at one or both of these conserved residues." This paper proposes a number of theories regarding disease mechanisms. However, these theories are not supported by experimental data. This article does not provide data that would affect the cancer dose-response analysis that forms the basis for the NSRL.
Samsel A and Seneff S (2017). Glyphosate pathways to modern disease VI: Prions, amyloidoses and autoimmune neurological diseases. *Journal of Biological Physics and Chemistry* **17**:8-32.	This article is a review of glyphosate and autism, multiple sclerosis, and other autoimmune disorders. The only reference to cancer is the reporting of a correlation between the incidence of thyroid cancer in the US and an increase in glyphosate usage on corn and soy crops. However, statistical correlations of cancer incidence with usage/exposure are not enough to presume causation. This article does not provide data that would affect the cancer dose-response analysis that forms the basis for the NSRL.

Comment 11 (Dr. Stephen C. Frantz): "Developing an NSRL that relies on 'acceptable calculated reference doses' supplied by the USEPA and its international counterparts is generally troublesome. That is, the EU 'standard' for daily chronic exposure to [glyphosate] is 0.5 mg/kg body weight, a level that is 3.5 fold *lower* than the U.S. 'standard' of 1.75 mg/kg body weight. Obviously, both levels cannot be acceptable and safe; and the EU version is already less than half of the proposed 1.1 mg by OEHHA."

Response 11: The NSRL for glyphosate does not rely on "acceptable calculated reference doses" or other values calculated by other agencies. Following the guidance

set forth in Section 25703, NSRLs are based on cancer dose-response assessments, which are conducted using data from the most sensitive scientific studies deemed to be of sufficient quality. As discussed in the ISOR for this rulemaking[50], OEHHA determined that the two-year study conducted in male CD-1 mice fed glyphosate (purity, 98.6%) in the diet met this criterion. OEHHA used this data to derive the NSRL for glyphosate.

Furthermore, as stated in Section 25703, an NSRL is defined as "[the level] which is calculated to result in one excess case of cancer in an exposed population of 100,000, assuming lifetime exposure at the level in question." NSRLs are intended to aid businesses in determining if they must comply with the warning and discharge provisions of Proposition 65; NRSLs are not intended to establish exposure or risk levels for other regulatory purposes (Section 25701(d)).

While reference doses set by other agencies are not relevant to this rulemaking, OEHHA notes that the European Union has set the *acceptable daily intake* (ADI) for glyphosate at 0.5 mg/kg[51], and US EPA has set the *chronic reference dose* (cRfD) for glyphosate at 1.00 mg/kg-day[52]; each of these values was developed by applying an uncertainty factor to a No Observed Adverse Effect Level (NOAEL) derived from developmental toxicity studies in rabbits. Neither value was based on cancer dose-response assessment and neither was developed specifically to protect against cancer. And finally, the ADI set by the European Union is not less than half of the proposed NSRL for glyphosate. The NSRL is expressed as an intake of µg/day, while the ADI (and cRfD) are expressed as mg/kg-day. Normalized to body weight, the NSRL would be less than the ADI or cRfD, not greater. No changes were made to the regulatory proposal based on this comment.

Comment 12 (The California League of Food Processors): Establishing an NSRL conflicts with tolerances set by US EPA for residues in food.

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[50] OEHHA (2017). Initial Statement of Reasons, Title 27, California Code of Regulations, Proposed Amendment to: Section 25705(b) Specific Regulatory Levels Posing No Significant Risk. Glyphosate. Available at https://oehha.ca.gov/media/downloads/crnr/glyphosate032917isor.pdf

[51] European Food Safety Authority (EFSA, 2015). Conclusion on the peer review of the pesticide risk assessment of the active substance glyphosate. EFSA Journal 2015;13 (11):4302. doi:10.2903/j.efsa.2015.4302. Available from: http://onlinelibrary.wiley.com/doi/10.2903/j.efsa.2015.4302/epdf, page 13

[52] The commenter refers to the former cRfD set by US EPA. The value has been updated since the comment was submitted, as shown in US EPA (2017). Glyphosate. Dietary Exposure Analysis in Support of Registration Review. Office of Chemical Safety and Pollution Prevention. Available from: https://www.epa.gov/sites/production/files/2017-12/documents/glyphosate_dietary_exposure_analysis_in_support_of_registration_review.pdf

Response 12: There is no direct correlation between a tolerance level set by US EPA and an NSRL adopted for purposes of Proposition 65. The two standards are developed under different laws and have different purposes. Whereas tolerances are mandatory maximum allowable pesticide residues on foods, NSRLs identify levels of exposure to listed carcinogens associated with a 1 in 100,000 cancer risk. If a food exposure to a pesticide listed as a carcinogen results in a cancer risk greater than 1 in 100,000, Proposition 65 requires a warning even if the food complies with US EPA's tolerances and can be legally sold in California. In such an instance, Proposition 65 gives Californians the right to be informed of the exposure and to make their own decision as to whether they wish to purchase or consume the food. No changes were made to the regulatory proposal based on this comment.

Comment 13 (K. Paul Stoller, MD): Regulators should not rely on just one study to determine acceptable daily intake.

Response 13: No Significant Risk Levels (NSRLs) are distinct from Acceptable Daily Intakes (ADIs). The NSRL is defined in the Proposition 65 regulations as "[the level] which is calculated to result in one excess case of cancer in an exposed population of 100,000, assuming lifetime exposure at the level in question." ADI values, on the other hand, are based on non-cancer health effects, and are neither defined nor used under Proposition 65.

In developing the NSRL for glyphosate, OEHHA followed the guidance set forth in Section 25703 and based the NSRL on the results of the most sensitive scientific study deemed to be of sufficient quality. No changes were made to the regulatory proposal based on this comment.

Comment 14 (Anthony Samsel): A thorough consideration cannot be had without a deep investigation and understanding of the nitrosamines of glyphosate which are carcinogens.

Response 14: Nitrosamines of glyphosate are not listed under Proposition 65 as causing cancer, nor are they the subject of this rulemaking. As discussed in response to comment 8, an NSRL applies specifically to the particular substance or chemical that has been listed as known to the state to cause cancer[53]. Therefore, studies of other chemicals, such as nitrosamines of glyphosate, do not provide information relevant to the derivation of the NSRL for glyphosate. No changes were made to the regulatory proposal based on this comment.

[53] Health and Safety Code section 25249.10(c) and Title 27, Cal. Code of Regs. section 25701.

Comment 15 (Ramboll Environ, on behalf of The Scotts Company, LLC): OEHHA and IARC failed to consider additional conclusions from the 2006 JMPR report on the study used to derive the NSRL, namely the lack of a dose-response relationship, the lack of statistically significant comparisons between treated animals and control animals, and the fact that the incidences were within the historical ranges for controls, and thus improperly reached conclusions regarding use of this data. Dr. Thomas McDonald, a peer reviewer and member of the Carcinogen Identification Committee, also stated that the dataset selected as the basis for the NSRL does not appear to be well supported as a treatment-related effect.

Response 15: As discussed in response to comment 5, IARC conducted an independent scientific review of the two-year study conducted in male CD-1 mice fed glyphosate (purity, 98.6%) in the diet, which OEHHA used to derive the NSRL. IARC concluded that a treatment-related increase in hemangiosarcomas was observed in this study, with a statistically significant positive trend. The tumor incidence data and positive trend test results, shown in Table 1 of the ISOR[54], demonstrate the dose-response relationship observed for hemangiosarcoma in this study.

While the pairwise comparison between the tumor incidence in animals in the high dose group and those in the control group did not rise to the $p < 0.05$ level of statistical significance, data from Charles River Laboratories indicate that hemangiosarcomas are infrequently observed in untreated male CD-1 mice, with a mean incidence of 1.13% (range 0% – 12.00%) reported in 2000[55], and 0.56% (range 0% - 4.55%) in 2010[56]. More specifically, no hemangiosarcomas were observed in untreated controls in 38 of the 46 studies summarized in 2000[57], or in 13 of the 14 studies summarized in 2010[58].

[54] OEHHA (2017). Initial Statement of Reasons, Title 27, California Code of Regulations, Proposed Amendment to: Section 25705(b) Specific Regulatory Levels Posing No Significant Risk. Glyphosate. Available at https://oehha.ca.gov/media/downloads/crnr/glyphosate032917isor.pdf

[55] Giknis MLA and Clifford CB (2000). Spontaneous Neoplastic Lesions in the Crl:CD-1®(ICR)BR Mouse. Charles River Laboratories, Wilmington, MA.

[56] Giknis MLA and Clifford CB (2010). Spontaneous Neoplastic Lesions in the Crl:CD1 (ICR) Mouse in Control Groups from 18 Month to 2 Year Studies. Charles River Laboratories, Wilmington, MA. Available from: http://animalab.eu/sites/all/pliki/produkty-dopobrania/spontaneous-neoplastic-lesions-in-the-crlcd1icr-mouse-in-control-groups-from-18-month-to-2-year-studies-march-2010.pdf

[57] Giknis MLA and Clifford CB (2000). Spontaneous Neoplastic Lesions in the Crl:CD-1®(ICR)BR Mouse. Charles River Laboratories, Wilmington, MA.

[58] Giknis MLA and Clifford CB (2010). Spontaneous Neoplastic Lesions in the Crl:CD1 (ICR) Mouse in Control Groups from 18 Month to 2 Year Studies. Charles River Laboratories, Wilmington, MA. Available from: http://animalab.eu/sites/all/pliki/produkty-dopobrania/spontaneous-neoplastic-lesions-in-the-crlcd1icr-mouse-in-control-groups-from-18-month-to-2-year-studies-march-2010.pdf

While JMPR[59] stated that the tumor "incidences recorded in this study fell within the historical ranges for controls", OEHHA notes, "concurrent controls are considered the most relevant comparison group for evaluating potential exposure-related tumor effects"[60]. In discussing the use of historical control data, IARC states "less weight is given to historical controls when they show a high degree of variability, and greater weight when they show little variability. It is generally not appropriate to discount a tumour response that is significantly increased compared with concurrent controls by arguing that it falls within the range of historical controls, particularly when historical controls show high between-study variability and are, thus, of little relevance to the current experiment"[61].

OEHHA agrees with IARC's determination that the increased incidence of hemangiosarcomas observed in this study of male CD-1 mice is treatment-related.

No changes were made based on this comment.

Comment 16: (Ramboll Environ, on behalf of The Scotts Company, LLC): The data used to derive the NSRL does not establish consistency across studies that is needed to provide a causal connection between exposure to glyphosate and cancer: there was no dose-related incidence of hemangiosarcoma reported in the female mouse study and no statistically significant increases in any tumors in another study with comparable concentrations.

Response 16: Section 25703(1) specifies that animal cancer bioassays must meet generally accepted scientific principles (e.g., the thoroughness of experimental protocol, the degree to which dosing resembles the expected manner of human exposure, the temporal exposure pattern, the duration of study, the purity of test material, the number and size of exposed groups, the route of exposure, and the extent of tumor occurrence) in order to be used in the development of NSRLs. In carcinogenicity testing there is no requirement or expectation that the same tumors will be seen in male and female animals of the same species and strain. It is also recognized that differences in study design (*e.g.*, doses tested; length of exposure; length of study) and implementation (*e.g.*, test substance purity/composition/lot; animal strain/substrain/colony/supplier of

[59] JMPR (2006). Glyphosate. In: Joint FAO/WHO Meeting on Pesticide Residues. Pesticide residues in food – 2004: toxicological evaluations. Report No. WHO/PCS/06.1. Geneva: World Health Organization; pp. 95 – 169

[60] National Toxicology Program (NTP, 2015). Handbook for Preparing Report on Carcinogens Monographs. Office of the Report on Carcinogens, Division of the NTP, National Institute of Environmental Health Sciences, US Department of Health and Human Services. Available online at https://ntp.niehs.nih.gov/pubhealth/roc/handbook/index.html

[61] IARC (2006). Preamble. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. IARC, World Health Organization, Lyon, France, p. 14. Available online at: http://monographs.iarc.fr/ENG/Preamble/CurrentPreamble.pdf

origin; diet composition; laboratory site, other animal husbandry conditions) may result in differences in response across animal carcinogenicity studies. Thus, consistency across animal studies is not required to establish a causal connection.

IARC concluded "[t]here is *sufficient evidence* in experimental animals for the carcinogenicity of glyphosate" based on findings from two studies in male mice. Specifically, IARC found "a significant positive trend in the incidence of haemangiosarcoma in male CD-1 mice" in a two-year diet study[62], and "a positive trend in the incidence of renal tubule carcinoma and of renal tubule adenoma and carcinoma (combined)" in male CD-1 mice in a different two-year diet study[63], with IARC noting that these malignant kidney tumors are rare in this strain of mice. Thus, IARC found dose-related increases in tumor incidence in these studies and OEHHA agrees with this determination.

In developing the NSRL for glyphosate, OEHHA followed the guidance set forth in Section 25703 and based the NSRL on the results of the most sensitive scientific study deemed to be of sufficient quality. No changes were made to the regulation based on this comment.

Comment 17 (Ramboll Environ, on behalf of The Scotts Company, LLC):
"Conducting dose-response modeling with a limited dataset – such as the dataset used in the derivation of the NSRL for glyphosate, which provides the observation of incidence above zero only at the highest concentration – creates significant model uncertainty." They also state that "this type of dataset lacks the necessary information to inform the shape of the dose-response curve in the low concentration region, which is needed for extrapolation to concentrations relevant to the human population and thus to estimate the NSRL."

Response 17: The proposed NSRL for glyphosate is based on the results of the most sensitive scientific study deemed to be of sufficient quality from which an NSRL can be derived, pursuant to Section 25703. Use of the multistage cancer model is generally

[62] As noted in the Initial Statement of Reasons, this study of glyphosate (purity 98.6%) met the criterion in Section 25703 as the most sensitive study of sufficient quality, and was used to derive the NSRL. This study was performed by Inveresk Research International and summarized in the 2006 Joint FAO/WHO Meeting on Pesticide Residues report (JMPR, 2006. Glyphosate. In: Joint FAO/WHO Meeting on Pesticide Residues. Pesticide residues in food – 2004: toxicological evaluations. Report No. WHO/PCS/06.1. Geneva: World Health Organization; pp. 95 – 169.) and by IARC (IARC, 2015, IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Volume 112. Some Organophosphate Insecticides and Herbicides: Diazinon, Glyphosate, Malathion, Parathion, and Tetrachlorvinphos. IARC, World Health Organization, Lyon, France).

[63] In summarizing this study of glyphosate (purity 99.7%), IARC cited four US EPA documents (US EPA 1985a, b, 1986, 1991a) (IARC, 2015, IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Volume 112. Some Organophosphate Insecticides and Herbicides: Diazinon, Glyphosate, Malathion, Parathion, and Tetrachlorvinphos. IARC, World Health Organization, Lyon, France).

accepted as the default approach to modeling lifetime cancer data as it is considered sufficiently flexible to fit most cancer bioassay data[64]. As stated in the ISOR for glyphosate[65], OEHHA determined that the study it used to derive the NSRL demonstrated a treatment-related increase in hemangiosarcomas, with a statistically significant positive trend. OEHHA disagrees with the commenter that modeling this data using the multistage cancer model "creates significant model uncertainty"; in fact, examination of the goodness-of-fit criteria[66,67] subsequent to fitting the model supports the appropriateness of the default approach. In particular, the global goodness-of-fit p-value is 0.9365, which is well above the cutoff of 0.05, the scaled residuals are all less than two in absolute value, and the plot shows that the multistage cancer model fits the data very well. The relatively low incidence of hemangiosarcoma in the high dose group (8%) effectively limits the possibilities the shape of the curve fit to the data can take. In fitting the multistage cancer model to this data, OEHHA followed the guidance in Section 25703, which is consistent with scientific practices in other OEHHA programs[68] and other scientific guidance, including US EPA's 2005 cancer risk assessment guidelines[69]. No changes were made to the proposed regulation based on this comment.

Comment 18 (Baum, Hedlund, Aristei & Goldman, P.C.): Section 25703(a)(1) requires that OEHHA consider the "degree to which dosing resembles the expected manner of human exposure" and "the route of exposure." The dietary ingestion of glyphosate as evaluated in the animal cancer bioassay considered by OEHHA does not resemble the expected manner of human exposure through application.

Response 18: The commenter has quoted only a portion of Section 25703(a)(1); OEHHA provides the full statement from the regulations for context and clarity:

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[64] US EPA (2014). Module 5: Benchmark Dose Modeling - Cancer Models [Webinar]. In *Benchmark Dose Software (BMDS) Training Webinars.* Available from: https://clu-in.adobeconnect.com/_a1089459318/p3a32k3l8of/?launcher=false&fcsContent=true&pbMode=normal&archiveOffset=488800

[65] OEHHA (2017). Initial Statement of Reasons, Title 27, California Code of Regulations, Proposed Amendment to: Section 25705(b) Specific Regulatory Levels Posing No Significant Risk. Glyphosate. Available at https://oehha.ca.gov/media/downloads/crnr/glyphosate032917isor.pdf

[66] US EPA (2014). Module 5: Benchmark Dose Modeling - Cancer Models [Webinar]. In *Benchmark Dose Software (BMDS) Training Webinars.* Available from: https://clu-in.adobeconnect.com/_a1089459318/p3a32k3l8of/?launcher=false&fcsContent=true&pbMode=normal&archiveOffset=488800

[67] US EPA (2012). Benchmark Dose Technical Guidance. Washington, DC: US EPA. Available from: https://www.epa.gov/sites/production/files/2015-01/documents/benchmark_dose_guidance.pdf

[68] OEHHA (2009). Technical Support Document for Cancer Potency Factors. https://oehha.ca.gov/media/downloads/crnr/tsdcancerpotency.pdf

[69] US EPA (2005). US Environmental Protection Agency. Guidelines for Carcinogen Risk Assessment. Risk Assessment Forum. Washington, DC. EPA/630/P-03/001B. March 2005.

> "Animal bioassay studies for quantitative risk assessment shall meet generally accepted scientific principles, including the thoroughness of experimental protocol, the degree to which dosing resembles the expected manner of human exposure, the temporal exposure pattern, the duration of study, the purity of test material, the number and size of exposed groups, the route of exposure, and the extent of tumor occurrence."

As can be seen in the full quotation of Section 25703(a)(1) above, "the degree to which dosing resembles the expected manner of human exposure" is one of *several* key considerations in determining whether or not an animal cancer bioassay is suitable for use in the development of an NSRL. OEHHA found the data used to derive the NSRL for glyphosate to be sufficient with respect to each of these considerations. With regard to the manner in which animals were dosed, diet is one of the expected routes of glyphosate exposure in humans and thus deriving the NSRL from study data in which test animals were administered glyphosate through the diet is consistent with the regulations. Animal bioassays employing dietary exposure are commonly used and routinely accepted for toxicity testing of pesticides.

Comment 19 (Dr. Thomas McDonald): OEHHA should make its own determination on the genotoxicity of glyphosate and not rely on IARC. He states that other authoritative bodies have concluded that glyphosate poses no genotoxicity risk in mammals, and that a Margin of Exposure (MOE) approach [to dose-response assessment] appears more appropriate.

Response 19: To the extent that the comment is directed toward the listing of glyphosate, it is not relevant to the determination of an NSRL for this chemical. OEHHA has reviewed the discussion of the mechanistic data for glyphosate provided in the IARC monograph and agrees with IARC's conclusion that "Overall, the mechanistic data provide strong evidence for genotoxicity and oxidative stress. There is evidence that these effects can operate in humans."[70]

OEHHA notes that IARC[71] further elaborated on this evidence, stating:

- "There is strong evidence that exposure to glyphosate or glyphosate-based formulations is genotoxic based on studies in humans in vitro and studies in experimental animals. One study in several communities in individuals exposed to glyphosate-based formulations also found chromosomal damage in blood cells; in this study, markers of chromosomal damage (micronucleus formation)

[70] IARC (2015) p. 78, full citation provided in footnote 3.
[71] IARC (2015) pp. 78-79, full citation provided in footnote 3.

were significantly greater after exposure than before exposure in the same individuals."

- "There is strong evidence that glyphosate, glyphosate-based formulations, and aminomethylphosphonic acid can act to induce oxidative stress based on studies in experimental animals, and in studies in humans in vitro. This mechanism has been challenged experimentally by administering antioxidants, which abrogated the effects of glyphosate on oxidative stress. Studies in aquatic species provide additional evidence for glyphosate-induced oxidative stress."

OEHHA disagrees that a Margin of Exposure approach is more scientifically appropriate for derivation of the NSRL for glyphosate than the procedure used by OEHHA. Section 25703 sets forth a default approach, using a multistage model for deriving a cancer potency estimate, which is used "in the absence of principles or assumptions scientifically more appropriate"[72]. No information has been provided in support of another mechanism of action that would suggest a different approach to dose-response analysis.

In deriving the NSRL, OEHHA used the Benchmark Dose (BMD) method, as described both in OEHHA's guidance[73] and in the US EPA guidelines[74], applying a multistage mathematical model to describe the relationship between the risk of cancer and the dose. As part of the procedure OEHHA used for determining the cancer potency using the BMD method, a determination is made as to the proper type of extrapolation from the point of departure (typically the 95% lower confidence limit of the ED_{05} or ED_{10} for tumor induction) to low doses. OEHHA considered whether there was a more scientifically appropriate method for the NSRL derivation than linear extrapolation, but did not identify one, stating in the Initial Statement of Reasons:

"Based on consideration of the available mechanistic information on glyphosate and the above conclusions reached by IARC[75], a multistage model is applied to derive a cancer potency estimate, following the guidance in Section 25703. There are no principles or assumptions scientifically more appropriate, based on the available data, than this approach."[76]

[72] Section 25703(a)
[73] OEHHA (2009). Technical Support Document for Cancer Potency Factors. Available from: https://oehha.ca.gov/media/downloads/crnr/tsdcancerpotency.pdf
[74] US EPA (2005). Guidelines for Carcinogen Risk Assessment, March 2005. Risk Assessment Forum, US Environmental Protection Agency, Washington, DC.
[75] IARC (2015). Full citation provided in footnote 3.
[76] OEHHA (2017). Initial Statement of Reasons, Title 27, California Code of Regulations, Proposed Amendment to: Section 25705(b) Specific Regulatory Levels Posing No Significant Risk. Glyphosate. Available at https://oehha.ca.gov/media/downloads/crnr/glyphosate032917isor.pdf

No changes were made to the regulatory proposal based on this comment.

Comment 20 (Food Democracy Now, The Agricultural Council of California, The California Farm Bureau Federation, Monsanto, Ramboll Environ on behalf of The Scotts Company LLC, Anthony Samsel, Jessica Denning, and PT Rothschild): Suggest alternative values for the NSRL for glyphosate:

Anthony Samsel, Frank Menhams and others commented that a value of 0 µg/day should be used because there is no safe level of glyphosate.

PT Rothschild recommended setting an NSRL based on a concentration of 0.01 parts per trillion.

Jessica Denning recommended setting an NSRL based on a concentration of a concentration of 0.01 parts per trillion because at a part per trillion, breast cell proliferation occurs.

Food Democracy Now suggested 0.1 µg/day.

The Agricultural Council of California and the California Farm Bureau Federation request that the proposed NSRL [1,100 µg/day] be considered a minimum value and that no consideration be given to anything lower.

Monsanto states that glyphosate does not cause cancer, therefore, exposure at any level poses no significant risk of cancer to humans, therefore the NSRL should be infinite.

Ramboll Environ on behalf of The Scotts Company LLC, states that if OEHHA insists on setting an NSRL for glyphosate, it should be infinite.

Response 20: Section 25703 of the regulations states that the assessment used to derive the NSRL "shall be based on evidence and standards of comparable scientific validity to the evidence and standards which form the scientific basis for the listing of the chemical as known to the state to cause cancer"[77]. Section 25703 further states that "risk analysis shall be based on the most sensitive study deemed to be of sufficient quality." No data that met these criteria were provided to support setting the NSRL at 0 or 0.1 µg/day, or setting an NSLR based on a concentration of 0.01 parts per trillion or 10 parts per quadrillion, nor were such data provided to support setting an infinite NSRL.

[77] Section 25703(a)(4)

In developing the NSRL for glyphosate, OEHHA followed the guidance set forth in Section 25703 and based the NSRL on the results of the most sensitive scientific study deemed to be of sufficient quality. OEHHA determined that the two-year study conducted in male CD-1 mice fed glyphosate (purity 98.6%) in the diet met the criteria in 25703 precisely because this study led to the highest cancer potency and subsequently the lowest NSRL among studies deemed to be of comparable scientific validity as those which formed the scientific basis for the listing of glyphosate. As already noted, OEHHA agrees with IARC's determination that the increased incidence of hemangiosarcomas observed in this study of male CD-1 mice is treatment-related.

No changes were made to the regulatory proposal based on this comment.

Comment 21 (The Environmental Working Group): OEHHA should set the limit at 10 µg/day which factors in a tenfold safety factor to account for the increased vulnerability of children, a one-in-a-million cancer risk standard used for carcinogens in drinking water, and rounding.

The commenter states that including a tenfold safety factor in the development of the NSRL for glyphosate is supported by OEHHA's 2009 report "*In Utero* and Early Life Susceptibility to Carcinogens", NRC's 1993 report "Pesticides in the Diets of Infants and Children", NRC's 2009 report "Science and Decisions" which advises public health agencies to include a factor of up to 25 to account for individual variation in susceptibility, and the 1996 Food Quality Protection Act which specifically required pesticide risk assessors to consider children's susceptibility to pesticides using a tenfold safety factor.

The commenter also states that OEHHA should use the one-in-a-million standard applied for carcinogens in drinking water for setting the NSRL for all exposures.

Response 21: The Food Quality Protection Act (FQPA), a federal law, is separate and distinct from Proposition 65, a California state law. Moreover, these two laws were established for different purposes and have different regulations and requirements. In particular, the FQPA relates to the setting of safety standards for pesticide residues in food, while Proposition 65 requires businesses to provide a warning when they cause an exposure to a listed chemical unless they can show the exposure does not exceed the safe harbor level, and prohibits the discharge of listed chemicals to sources of drinking water. Proposition 65 warnings are not required and the discharge prohibition does not apply when exposures are at or below the safe harbor level.

The NSRL is defined as "[the level] which is calculated to result in one excess case of cancer in an exposed population of 100,000, assuming lifetime exposure at the level in

question"[78]. Thus, OEHHA cannot use a one-in-a-million level of risk in setting the NSRL. Similarly, OEHHA cannot apply a tenfold safety factor to the NSRL. The NSRL for glyphosate was derived according to the requirements set forth in Section 25703.

NSRLs do not conflict with permissible levels set by the federal government or with the one-in-a-million cancer risk standard for carcinogens in drinking water. These other laws have no bearing on Proposition 65, and it has no bearing on them. No changes to the regulatory proposal were made based on this comment.

Comment 22 (Food Democracy Now!, Joanie Blaxter): OEHHA should wait to consider a high NSRL for glyphosate until the studies showing carcinogenic effects in human populations can be replicated and extended. Joanie Blaxter commented that the testing model should be replaced with a more real life model of the effects of sub-acute low-level exposure over long periods of time in combination with exposure to other potentially activating chemicals and heavy metals.

Response 22: As stated in the response to Comment 1, glyphosate was listed pursuant to the Labor Code listing mechanism[79] as a result of IARC's classification of glyphosate in Group 2A ("probably carcinogenic to humans"), with a finding of "sufficient evidence of carcinogenicity in experimental animals" and "limited evidence" in humans[80,81]. Section 25703 of the regulations states that the assessment used to derive the NSRL "shall be based on evidence and standards of comparable scientific validity to the evidence and standards which form the scientific basis for the listing of the chemical as known to the state to cause cancer"[82]. Given that the listing of glyphosate is based on sufficient evidence in animals, basing the potency estimate for the NSRL on animal studies is both appropriate and consistent with Section 25703. It is not appropriate for OEHHA to wait for additional studies to be conducted in humans, or otherwise delay the adoption of the NSRL for glyphosate, which is intended to aid businesses in complying with Proposition 65. Should additional scientific studies become available in the future that meet the criteria set out in Section 25703, OEHHA can consider revising the NSRL for glyphosate at that time. No changes were made to the regulatory proposal based on this comment.

Comment 23 (Comments from Food Democracy Now): "A two year study on rats published in 2015 found that just 0.05 ppb changed the function of more than 4000 genes. It would behoove the commission to pay attention to any and all studies which

[78] Section 24703.
[79] Section 25249.8(a) of the Act
[80] OEHHA (2015). Notice of Intent to List - Tetrachlorvinphos, Parathion, Malathion, Glyphosate. https://oehha.ca.gov/media/downloads/crnr/090415noillcset27.pdf
[81] IARC (2015). Full citation provided in footnote 3.
[82] Section 25703(a)(4)

suggest adverse human health effects at such miniscule levels. The study found steatohepatosis which predisposes to liver cancer at a glyphosate equivalent dose of only 4 nanograms per kg per day. The amount of glyphosate ingested by these rats is approximately 4000 times lower than what is typically ingested based on levels found in urine. This is the only study of its type providing a direct causative link between an environmentally relevant dose of Roundup and a serious disease."

Response 23: The commenter appears to be referring to a 2015 publication by Mesnage *et al*.[83], that analyzed differences in gene expression, not gene function, in the liver and kidney of female rats administered a glyphosate-based herbicide in drinking water for two years, as compared with controls receiving "plain water". Changes in gene expression levels were observed for more than 4000 genes in the liver and kidney of treated animals, as compared with controls. Treatment-related tumors were not reported in this study. This study does not provide data that would affect the cancer dose-response analysis that forms the basis for the NSRL. No changes were made to the regulatory proposal based on this comment.

Comment 24 (A number of commenters, including Meghan Lawler): Raised concerns about exposure to glyphosate, whether through food, consumer products, the environment, or during application as a pesticide. Some state that the proposed NSRL does not reflect real-world exposure scenarios or expected exposure concentrations. Some state that it is unclear how the increased exposure of agricultural workers will be factored in, when setting an NSRL. Some have reported various levels that a typical adult is exposed to on a daily basis. Some state that there is no way to establish or enforce a safe level because it is impossible to quantify cumulative exposure. Meghan Lawler commented that no comprehensive, long term, independent study has been done that shows real life exposure levels for glyphosate.

Response 24: Following the guidance set forth in Section 25703, NSRLs are based on cancer dose-response assessments, which are conducted using tumor incidence data from the most sensitive scientific studies deemed to be of sufficient quality. Cancer dose-response assessments are performed to estimate a carcinogen's cancer potency, and the NSRL is derived based on the cancer potency estimate. Specifically, the NSRL is defined as "[the level] which is calculated to result in one excess case of cancer in an exposed population of 100,000, assuming lifetime exposure at the level in question"[84]. Thus, the NSRL is a level of exposure or intake, expressed in units of µg/day that is associated with a risk of cancer of one-in-100,000.

[83] Mesnage R, Arno M, Costanzo M, Malatesta M, Seralini G-E, Antoniou MN (2015). Transcriptome profile analysis reflects rat liver and kidney damage following chronic ultra-low dose Roundup exposure. *Environmental Health* 14:70 DOI 10.1186/s12940-015-0056-1.
[84] Section 24703.

Exposure information (*e.g.*, exposure routes, exposure levels) is not used in dose-response assessment. Rather, estimates of exposure may be used *together* with estimates of cancer potency to predict cancer risk within a population.

As noted in response to comment 1, the estimate of cancer potency for glyphosate is equally valid for estimating risks to agricultural workers and to other exposed individuals, and the NSRL for glyphosate is not limited to a specific route of exposure[85,86].

Many conventional regulatory standards are developed using the kind of real-world exposure information cited by the commenters. Those standards identify legally mandated, health-protective levels of exposures to chemicals that can be feasibly achieved by manufacturers and employers. The NSRL is not a conventional regulatory standard, as it is based strictly on the scientific criteria cited above. It is intended to guide businesses in determining whether a warning is necessary or whether discharges of a chemical into drinking water sources are prohibited. A Proposition 65 warning enables Californians to make informed choices about their exposures to listed chemicals.

Comment 25 (Several commenters): The proposed level is too high, and one commenter stated that, in comparison, the NSRL for TCDD is much lower.

Response 25: The comment compares the proposed NSRL for glyphosate, 1100 µg/day, to the NSRL for 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD), which is 0.000005 µg/day. It has long been recognized that carcinogens vary in strength, or potency, with some being extremely potent, and others much less potent[87]. Indeed, the cancer potencies of carcinogens vary by several orders of magnitude[88]. NSRLs, which are derived from cancer potency estimates, can similarly vary by orders of magnitude, as can be seen when comparing the NSRL for glyphosate to that for TCDD. Thus, the fact that the NSRL for glyphosate is much higher than the NSRL for TCDD is not an indication that the glyphosate NSRL is too high, or otherwise inappropriate.

In developing the NSRL for glyphosate, OEHHA followed the guidance set forth in Section 25703 and based the NSRL on the results of the most sensitive scientific study deemed to be of sufficient quality. OEHHA determined that the two-year study

[85] OEHHA (2017). Initial Statement of Reasons, Title 27, California Code of Regulations, Proposed Amendment to: Section 25705(b) Specific Regulatory Levels Posing No Significant Risk. Glyphosate. Available at https://oehha.ca.gov/media/downloads/crnr/glyphosate032917isor.pdf
[86] Section 25703(a)(4)
[87] Gold, L. S., et al. (1984) A carcinogenic potency database of the standardized results of animal bioassays. *Environ Health Perspect* **58**: 9-319.
[88] *Ibid*.

conducted in male CD-1 mice fed glyphosate (purity 98.6%) in the diet met the criteria in 25703 precisely because this study led to the highest cancer potency and subsequently the lowest NSRL among studies deemed to be of comparable scientific validity as those which formed the scientific basis for the listing of glyphosate. As already noted, OEHHA agrees with IARC's determination that the increased incidence of hemangiosarcomas observed in this study of male CD-1 mice is treatment-related. No changes were made based on this comment.

Comment 26 (Laura Hayes, Pesticide Free Zone and Tamsin Lisa Kelly, JD, MD): The proposed level is a random rate that cannot be accurately monitored or enforced. Pesticide Free Zone asked how OEHHA would determine the amount that humans are exposed to on a daily basis. Tamsin Lisa Kelly, JD, MD, stated that if use is allowed, testing of food and water supplies must be required regularly to assure exposure is limited.

Response 26: OEHHA disagrees with the statement that the proposed NSRL for glyphosate is a random rate. As described in more detail in Response 19 OEHHA followed standard cancer dose-response assessment practice in deriving an NSRL of 1100 µg/day for glyphosate, which is based on the most sensitive study of sufficient quality. OEHHA's approach is consistent with Section 25703, scientific practices in other OEHHA programs[89] and other scientific guidance, including US EPA's 2005 cancer risk assessment guidelines[90].

OEHHA has no authority under Proposition 65 to monitor exposures to listed chemicals. Businesses are responsible for determining if they are causing exposures to listed chemicals at levels that require warnings. The purpose of the NSRL is to assist businesses in making these determinations. Similarly, OEHHA has no authority under Proposition 65 to require testing of food and water supplies. No changes were made to the regulatory proposal based on this comment.

Comment 27 (A Voice for Choice, Organic Sacramento, and several others): The NSRL does not account for differences in vulnerability due to size, age, stage of development, health status, or socioeconomic status.

Response 27: As specified in Section 25703, the "risk analysis shall be based on the most sensitive study deemed to be of sufficient quality", and "the risk level which represents no significant risk shall be one which is calculated to result in one excess

[89] OEHHA (2009). Technical Support Document for Cancer Potency Factors. https://oehha.ca.gov/media/downloads/crnr/tsdcancerpotency.pdf

[90] US EPA (2005). US Environmental Protection Agency. Guidelines for Carcinogen Risk Assessment. Risk Assessment Forum. Washington, DC. EPA/630/P-03/001B. March 2005.

case of cancer in an exposed population of 100,000, **assuming lifetime exposure at the level in question**". (Emphasis added)

In developing the NSRL for glyphosate, OEHHA followed the guidance set forth in Section 25703 and based the NSRL on the results of the most sensitive scientific study deemed to be of sufficient quality. The calculation assumes lifetime exposure at the level in question to an average person in the general population. No changes were made to the regulation based on this comment.

Comment 28 (A number of commenters): Urge OEHHA to ban glyphosate, that it be declared a "restricted use" chemical, that it not be available to the public, or that OEHHA should ensure labeling of all products, businesses, and public spaces containing any amount of glyphosate. Bob Sanders commented that instead of considering an NSRL, OEHHA should be discussing glyphosate as "not safe for human consumption" (NSFHC) and including 10 mile safety zones to protect children and families.

Response 28: Proposition 65 does not give OEHHA authority to remove products or chemicals from the market or to restrict their use. While OEHHA has regulatory authority to broadly identify acceptable methods and content for Proposition 65 warnings, OEHHA does not have the authority to directly regulate product labeling as suggested by the commenters. Similarly, Proposition 65 does not give OEHHA the authority to categorize glyphosate as not safe for human consumption or to impose safety zones as suggested by the commenter. These comments are outside the scope of the current rulemaking and no changes were made based on this comment.

Comment 29 (Larry Wartels, Susan[91] and others): OEHHA should use the precautionary principle in developing the NSRL for glyphosate. OEHHA should only allow use of the lowest effective levels of glyphosate so that plants do not become glyphosate resistant.

Response 29: In developing the NSRL for glyphosate, OEHHA followed the guidance set forth in Section 25703 of the regulations, which states that the assessment used to derive the NSRL "shall be based on evidence and standards of comparable scientific validity to the evidence and standards which form the scientific basis for the listing of the chemical as known to the state to cause cancer"[92]. Glyphosate was listed pursuant to the Labor Code listing mechanism[93] as a result of IARC's classification of glyphosate in Group 2A ("probably carcinogenic to humans"), with a finding of sufficient evidence of

[91] The commenter did not provide a last name.
[92] Section 25703(a)(4)
[93] Section 25249.8(a) of the Act

carcinogenicity in experimental animals[94,95]. OEHHA reviewed the available data from the rodent carcinogenicity studies of glyphosate discussed by IARC[96], and determined that the two-year study conducted in male CD-1 mice fed glyphosate (purity, 98.6%) in the diet met the criterion in Section 25703 as the most sensitive study of sufficient quality. OEHHA agrees with IARC's determination that the increased incidence of hemangiosarcomas observed in this study of male CD-1 mice is treatment-related. OEHHA then performed a standard dose-response assessment using the data from this study to derive the NSRL for glyphosate. The resistance of plants to glyphosate is not relevant for purposes of deriving an NSRL. No changes were made based on this comment.

Comment 30 (One commenter (anonymous)): Extrapolating cancer risk to humans from hemangiosarcomas, which are very rare in humans, seems misleading and to use this to determine the NSRL seems unscientific.

Response 30: The premise underlying this comment is incorrect. It is a generally accepted principle that the ability of a chemical to cause cancer in animals is predictive that the chemical also poses a cancer hazard in humans[97]. However, it is not assumed that the same tumor type observed in animals will be observed in humans[98]. Similarly, the fact that cancer potency is estimated based on animal tumor data for a particular tumor type does not imply that the cancer potency applies specifically to that same tumor type in humans. The human cancer potency estimate is a measure of the carcinogenic hazard posed by a particular carcinogen, and can be used to estimate the risk of cancer (at all sites that may be affected by this carcinogen) associated with a specific level of exposure in humans. No changes were made in response to this comment.

Comment 31 (Baum, Hedlund, Aristei & Goldman, P.C., Meredith Newton, Timothy Litzenburg and others): Raised concerns over OEHHA meeting with representatives from Monsanto in October 2015. The commenters state that OEHHA should be presented with an impartial and comprehensive scope of data in determining the NSRL and that industry meetings with regulators should be open to public scrutiny. Timothy

[94] OEHHA (2015). Notice of Intent to List - Tetrachlorvinphos, Parathion, Malathion, Glyphosate. Available from: https://oehha.ca.gov/media/downloads/crnr/090415noillcset27.pdf
[95] IARC (2015). Full citation provided in footnote 3.
[96] IARC (2015). Full citation provided in footnote 3.
[97] IARC (2006). Preamble. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. World Health Organization, International Agency for Research on Cancer, Lyon, France, 2006. Available from: http://monographs.iarc.fr/ENG/Preamble/CurrentPreamble.pdf)
[98] US EPA (2005). Guidelines for Carcinogen Risk Assessment, March 2005. Risk Assessment Forum, US Environmental Protection Agency, Washington, DC.

Litzenburg] requested that OEHHA schedule a meeting with stakeholders before making a decision on the safe harbor threshold.

Response 31: This comment is not directed towards the rulemaking. In compliance with the Administrative Procedure Act (APA), OEHHA published a Notice of Proposed Rulemaking, thereby opening a 45-day public comment period, and held a public hearing where all interested parties were allowed to provide their input regarding the proposed rulemaking. OEHHA provided the public with the opportunity to provide written comments during the comment period. OEHHA is publicly responding to all the oral and written comments received during the rulemaking in this Final Statement of Reasons. Nothing in the APA prohibits OEHHA from meeting with stakeholders to hear all viewpoints on an issue. The October 2015 meeting occurred before glyphosate was added to the Proposition 65 list of chemicals and before the current rulemaking proposal. OEHHA also met with many of the commenters, including representatives of Baum, Hedlund, Aristei & Goldman, P.C. and Timothy Litzenburg and others in August 2017 to understand their position concerning the NSRL. No changes were made to the proposed regulation based of this comment.

Comment 32 (Zen Honeycutt): Section 25703 requires OEHHA to consider all available studies showing harm. Provided many references for OEHHA's consideration, many of which were not included in IARC's review (Table 2.)

Response 32: Section 25703 does not mandate a review of all available studies showing harm. Rather Section 25703 requires that the assessment "be based on evidence and standards of comparable scientific validity to the evidence and standards which form the scientific basis for the listing of the chemical as known to the state to cause cancer", and the NSRL must be based on the results of the most sensitive scientific study deemed to be of sufficient quality.

Of the 72 published scientific articles listed in the comments from Zen Honeycutt, 54 were not cited in the IARC Monograph[99] that OEHHA relied on in developing the NSRL for glyphosate. These 54 publications are listed in Table 2. OEHHA reviewed each of these publications in the context of the guidance set forth in Section 25703, i.e., "the assessment shall be based on evidence and standards of comparable scientific validity to the evidence and standards which form the scientific basis for the listing of the chemical as known to the state to cause cancer"[100] and determined that none of the studies provide data that would affect the cancer dose-response analysis (See Table 2). No changes were made to the regulatory proposal based on this comment.

[99] IARC (2015). Full citation provided in footnote 3.
[100] Section 25703(a)(4)

Table 2. Studies related to glyphosate that were identified by Zen Honeycutt and not considered by IARC

Reference	Comments
Arbuckle TE, Lin Z, Mery LS (2001). An exploratory analysis of the effect of pesticide exposure on the risk of spontaneous abortion in an Ontario farm population. *Environ Health Perspec*t **109**(8):851-7.	This human epidemiological study investigated the effects of glyphosate exposure on spontaneous abortion. This reproductive toxicity study is not relevant to cancer dose-response analysis.
Astiz M, Alaniz MJT de, Marra CA (2009). The impact of simultaneous intoxication with agrochemicals on the antioxidant defense system in rat. *Pesticide Biochemistry and Physiology* **94**:93-99.	This study in rats examined the effects of glyphosate on oxidative stress, and hormone levels. This mechanistic study does not provide data that would affect the cancer dose-response analysis that forms the basis for the NSRL.
Barbosa ER, Leiros da Costa MD, Bacheschi LA, Scaff M, Leite CC (2001). Parkinsonism after glycine-derivate exposure. *Mov Disord* **16**(3):565-8.	This is a case report of an incidence of Parkinson's disease following exposure to glyphosate, and is not relevant to cancer dose-response analysis.
Bellé R, Le Bouffant R, Morales J, Cosson B, Cormier P, Mulner-Lorillon O (2007). Sea urchin embryo, DNA-damaged cell cycle checkpoint and the mechanisms initiating cancer development. *J Soc Biol* **201**(3):317-27. [Article in French]	This study examined the effects of glyphosate on sea urchin development. This toxicity study may provide data on possible mechanisms of action, but it does not provide data that can be used in the cancer dose-response analysis.
Benedetti AL, Vituri Cde L, Trentin AG, Domingues MA, Alvarez-Silva M (2004). The effects of sub-chronic exposure of Wistar rats to the herbicide Glyphosate-Biocarb. *Toxicol Lett* **153**(2):227-32.	This study examined the effects of Glyphosate-Biocarb® on the livers of Wistar rats following 75 days of exposure. This subchronic toxicity study does not provide data that can be used in the cancer dose-response analysis.
Benedetti D, Nunes E, Sarmento M, Porto C, Dos Santos CE, Dias JF, da Silva J (2013). Genetic damage in soybean workers exposed to pesticides: evaluation with the comet	This study in farm workers assessed the effects of exposure to complex mixtures of pesticides, including glyphosate, on DNA. The authors reported that DNA damage and genomic hypermethylation of DNA were significantly increased in individuals exposed

and buccal micronucleus cytome assays. *Mutat Res* **752**(1-2):28-33.	to pesticide mixtures, but it does not provide data that can be used in the cancer dose-response analysis.
Beuret CJ, Zirulnik F, Giménez MS (2005). Effect of the herbicide glyphosate on liver lipoperoxidation in pregnant rats and their fetuses. *Reprod Toxicol* **19**(4):501-4.	This study investigated the effects of glyphosate on pregnant female Wistar rats and their fetuses. This reproductive toxicity study provides no data that can be used in the cancer dose-response analysis.
Cox C (2004). Herbicide factsheet: glyphosate. *Journal of Pesticide Reform* **24**(4):10-15.	This factsheet is a short review and does not provide data that would affect the cancer dose-response analysis that forms the basis for the NSRL.
da Costa Mdo D, Gonçalves LR, Barbosa ER, Bacheschi LA (2003). Neuroimaging abnormalities in parkinsonism: study of five cases. *Arq Neuropsiquiatr* **61**(2B):381-6. [Article in Portuguese]	This study reports neuroimaging results in five patients with Parkinson's disease, one of whom was exposed to glyphosate. This study is not relevant to cancer dose-response analysis.
Dallegrave E, Mantese FD, Coelho RS, Pereira JD, Dalsenter PR, Langeloh A (2003). The teratogenic potential of the herbicide glyphosate-Roundup in Wistar rats. *Toxicol Lett* **142**(1-2):45-52.	This study examined the teratogenicity of glyphosate-Roundup® to Wistar rats. This developmental toxicity study provides no data relevant to cancer dose-response analysis.
Dallegrave E, Mantese FD, Oliveira RT, Andrade AJ, Dalsenter PR, Langeloh A (2007). Pre- and postnatal toxicity of the commercial glyphosate formulation in Wistar rats. *Arch Toxicol* **81**(9):665-73.	This study investigated the reproductive effects of glyphosate-Roundup® on male and female offspring of Wistar rats exposed during pregnancy and lactation. This reproductive toxicity study provides no data relevant to cancer dose-response analysis.
Daruich J, Zirulnik F, Gimenez MS (2001). Effect of the herbicide glyphosate on enzymatic activity in pregnant rats and their fetuses. *Environ Res* **85**(3):226-31.	This study investigated the effects of glyphosate exposure to pregnant Wistar rats on enzymes in the dams and their fetuses. This reproductive toxicity study provides no data relevant to cancer dose-response analysis.
de Liz Oliveira Cavalli VL, Cattani D, Heinz Rieg CE, Pierozan P, Zanatta L, Benedetti Parisotto E, Wilhelm	This study investigated the effects of glyphosate on male rat Sertoli cells and testis

Filho D, Mena Barreto Silva FR, Pessoa-Pureur R, Zamoner A (2013). Roundup disrupts male reproductive functions by triggering calcium-mediated cell death in rat testis and Sertoli cells. *Free Radic Biol Med* **65**:335-46.	*in vitro*. This *in vitro* toxicity study provides no data to cancer dose-response analysis.
de Souza JS, Kizys MM, da Conceição RR, Glebocki G, Romano RM, Ortiga-Carvalho TM, Giannocco G, da Silva ID, Dias da Silva MR, Romano MA, Chiamolera MI (2017). Perinatal exposure to glyphosate-based herbicide alters the thyrotrophic axis and causes thyroid hormone homeostasis imbalance in male rats. *Toxicology* **377**:25-37.	This study investigated the effects of a glyphosate-based herbicide on the hypothalamic-pituitary-thyroid axis of male rats following *in utero* exposure. The authors reported that exposure affected thyroid hormone homeostasis. While this study contributes to the data on possible mechanisms of action, it does not provide data that can be used in the cancer dose-response analysis.
Geng D et al. (2000). Study of Herbicide Roundup impact on yellow eel mutagenic. *Journal of Xuzhou Normal University* (Natural Science Edition) **2**. [Article in Chinese] Available from http://www.cnki.com.cn/ Article/ CJFDTotal-XZSX200002018.htm	This study investigated the genotoxicity of glyphosate in the erythrocytes of *Monopterus albus* (Asian swamp eel) *in vivo*. It suggests that glyphosate induces chromosomal aberrations. While this study contributes to the data on possible mechanisms of action, it does not provide data that can be used in the cancer dose-response analysis.
Hokanson R, Fudge R, Chowdhary R, Busbee D (2007). Alteration of estrogen-regulated gene expression in human cells induced by the agricultural and horticultural herbicide glyphosate. *Hum Exp Toxicol* **26**:747-52.	This *in vitro* study investigated the effects of glyphosate on human MCF-7 cells and found altered gene expression. This mechanistic study does not provide data that can be used in the cancer dose-response analysis.
Huang C, Li B, Xu K, Liu D, Hu J, Yang Y, Nie H, Fan L, Zhu W (2017). Decline in semen quality among 30,636 young Chinese men from 2001 to 2015. Fertil Steril **107**(1):83-88.	This study provides no information or data that is specific to glyphosate.
Jayawardena UA, Rajakaruna RS, Navaratne AN, Amerasinghe PH	This toxicity study of glyphosate and other pesticides observed malformations in exposed

(2010). Toxicity of agrochemicals to common hourglass tree frog (*Polypedates cruciger*) in acute and chronic exposure. *International Journal of Agriculture and Biology*, 12, 641-648.	tree frogs. This study provides no data relevant to cancer dose-response analysis.
Kamel F, Tanner C, Umbach D, Hoppin J, Alavanja M, Blair A, Comyns K, Goldman S, Korell M, Langston J, Ross G, Sandler D (2007). Pesticide exposure and self-reported Parkinson's disease in the agricultural health study. *Am J Epidemiol* **165**(4):364-74.	This study on Parkinson's disease is not relevant to cancer dose response-analysis.
Kang J et al. (2008). Study of glyphosate effect causing mutagenic on rats. Carcinogenesis, Teratogenesis & Mutagenesis **3**. [Article in Chinese] Available from http://www.cnki.com.cn/ Article/ CJFDTotal-ABJB200803018.htm	The hyperlink provided by the commenter leads to an article by Kang et al. (2008), named "Study on mutagenesis induced by glyphosate in mice". The full text also indicates that this study was in mice, but not rats. Other than the title, the rest of the citation is correct. This study reports that glyphosate induced micronucleus formation in bone marrow polychromatic erythrocytes of Kunming mice, increased sperm aberrations, and decreased sperm count. While this study contributes to the data on possible mechanisms of action, it does not provide data that would affect the cancer dose-response analysis that forms the basis for the NSRL.
Kruger M, Schledorn P, Schrodl W, Hoppe HW, Lutz W, Shehata AA (2014). Detection of Glyphosate Residues in Animals and Humans. *J Environ Anal Toxicol* **4**(2):210.	This study measured glyphosate residues in animals and humans using ELISA and gas chromatography-mass spectroscopy. Glyphosate residues were detected in the kidney, liver, lung, spleen, muscles, and intestine in dairy cows (minimum = 1.36 µg/g; maximum of 108 µg/mg). Glyphosate residues were detected in the urine of dairy cows (minimum = 0 µg/ml; maximum = 164

	µg/ml), rabbits (minimum = 2.37 µg/ml; maximum = 70 µg/ml) and humans (minimum = 0.1 µg/ml; maximum = 71.3 µg/ml). Significantly higher urinary glyphosate residues were reported in chronically ill humans than in healthy individuals. This study provides no data relevant to cancer dose response analysis.
Lajmanovich RC, Sandoval MT, Peltzer PM (2003). Induction of mortality and malformation in Scinax nasicus tadpoles exposed to glyphosate formulations. *Bull Env Contam Toxicol* **70**:612–618.	This study investigated the effects of glyphosate on tadpoles exposed for 96 hours. This acute toxicity study in amphibians provides no data relevant to cancer dose-response analysis.
Li Q, et al. (2010). Acute toxicity of eight types of pesticides to sea urchin embryos during different phases of development. *Asian Journal of Ecotoxicology.* [Article in Chinese] Available from http://d.wanfangdata.com.cn/Periodical_cyyhj 201002014.aspx	This study investigated the acute toxicity of glyphosate on the development of sea urchin embryos. This study provides no data relevant to cancer dose-response analysis.
Lioi MB, Scarfi MR, Santoro A, Barbieri R, Zeni O, Salvemini F, Di Berardino D, Ursini MV (1998). Cytogenetic damage and induction of pro-oxidant state in human lymphocytes exposed in vitro to glyphosate, vinclozolin, atrazine, and DPXE9636. *Environ Mol Mutagen* **32**(1):39-46.	This *in vitro* study in human peripheral lymphocytes reported that glyphosate exposure increased chromosomal aberrations, sister chromatid exchanges, and a change in the redox state of the cell. This study contributes to the data on possible mechanisms of action, but it does not provide data that would affect the cancer dose-response analysis that forms the basis for the NSRL.
Marc J, Mulner-Lorillon O, Boulben S, Hureau D, Durand G, Bellé R (2002). Pesticide Roundup provokes cell division dysfunction at the level of CDK1/cyclin B activation. *Chem Res Toxicol* **15**(3):326-31.	This study investigated the effects of Roundup® and glyphosate on cell cycle regulation in sea urchin embryos. This mechanistic study does not provide data that can be used in the cancer dose-response analysis.

Marc J, Bellé R, Morales J, Cormier P, Mulner-Lorillon O (2004a). Formulated glyphosate activates the DNA-response checkpoint of the cell cycle leading to the prevention of G2/M transition. *Toxicol Sci* **82**(2):436-42.	This *in vitro* study investigated the effects of glyphosate on the cell cycle of sea urchins. This mechanistic study does not provide data that can be used in the cancer dose-response analysis.
Marc J, Mulner-Lorillon O, Bellé R (2004b). Glyphosate-based pesticides affect cell cycle regulation. *Biol Cell* **96**(3):245-9.	This paper investigated the effects of several glyphosate-based pesticides on cell cycle regulation in sea urchins. This mechanistic study does not provide data that can be used in the cancer dose-response analysis.
Marc J, Le Breton M, Cormier P, Morales J, Bellé R, Mulner-Lorillon O (2005). A glyphosate-based pesticide impinges on transcription. Toxicol Appl Pharmacol **203**(1):1-8.	This study investigated the effects of glyphosate on sea urchin development and found effects on transcription in early development. This study does not provide data that can be used in the cancer dose-response analysis.
McComb BC, Curtis L, Chambers CL, Newton M, Bentson K (2008). Acute toxic hazard evaluations of glyphosate herbicide on terrestrial vertebrates of the Oregon Coast Range. *Environ Sci Pollut Res Int* **15**(3):266-72.	This study evaluated the effects of acute exposure to glyphosate on white lab mice and 9 wild vertebrate species from the Oregon coast (deer mouse, chipmunk, shrew, vole, newt, frog, and three types of salamanders). This acute toxicity study does not provide data that can be used in the cancer dose-response analysis.
Mesnage R, Clair E, Spiroux de Vendômois J, Séralini GE (2010). Two cases of birth defects overlapping Stratton-Parker syndrome after multiple pesticide exposure. *Occup Environ Med* **67**(5):359.	This is a report of two instances of congenital malformations in children whose parents had been exposed to multiple pesticides, including glyphosate. These case reports are not relevant to cancer dose-response analysis.
Mesnage R, Renney G, Seralini GE, Ward M (2017) Multiomics reveal non-alcoholic fatty liver disease in rats following chronic exposure to an ultra-low dose of Roundup herbicide. *Sci Rep* **7**:39328.	This study used metabolome and proteome analyses of rat liver tissue to investigate the effects of low-dose exposure of rats to a glyphosate-based herbicide. The authors concluded that the metabolome and proteome changes observed were indicative of non-alcoholic fatty liver disease. This study does

	not provide data that can be used in the cancer dose-response analysis.
Nan X (2001). Impact of glyphosate herbicide on carp peripheral blood erythrocyte micronucleus and nuclear anomalies, *Journal of Anhui Normal University* (Natural Science Edition) **24**(4): 329-331. [Article in Chinese] Available from http://www.cqvip.com/ qk/97138X/200006/ 4887295.html	The hyperlink provided by the commenter leads to a study by Nan et al. (2000), titled "Effects of Herbicide (Glyphosate) on Micronuclei and Nuclear Anomalies in Erythrocyte of Bufo bufo Gargarizans". It was conducted in Asiatic toads, not carp as the title provided by the commenter states. This study found that glyphosate increased the frequency of micronuclei and nuclear abnormalities in the erythrocytes of Asiatic toads after oral treatment. While this study contributes to the data on possible mechanisms of action, it does not provide data that can be used in the cancer dose-response analysis.
Nan X (2002). Study of impact of glyphosate herbicide on carp blood cells and hemoglobin. *Gansu Science* **2**. [Article in Chinese] Available from http://www.cnki.com.cn/ Article/CJFDTotal-GSKX200204015.htm	This study investigated the toxicity of glyphosate on carp (*Carassius auratus*) by measuring hemoglobin levels and erythrocyte and leucocyte counts. This study provides no data relevant to cancer dose-response analysis.
Nan X et al. (2003). Impact of glyphosate herbicide on loach white blood cells. *Journal of Wenzhou Normal University* (Natural Science Edition) **24**(2): 72-74. [Article in Chinese] Available from http://www.cnki.com.cn/ Article/CJFDTotal-WZSF200302019.htm	The hyperlink provided by the commenter leads to an article by Nan et al. (2003), titled "Effect of Mi[s]gurnus Anguillicaudatus induced by glyphosate". Other than the title, the rest of the citation is correct. This study investigated the effect of glyphosate on lymphocyte and granulocyte counts in the peripheral blood of *Misgurnus Anguillicaudatus* (pond loach, a fresh water fish). This study provides no data relevant to cancer dose-response analysis.

Negga R, Stuart JA, Machen ML, Salva J, Lizek AJ, Richardson SJ, Osborne AS, Mirallas O, McVey KA, Fitsanakis VA (2012). Exposure to glyphosate- and/or Mn/Zn-ethylene-bis-dithiocarbamate-containing pesticides leads to degeneration of γ-aminobutyric acid and dopamine neurons in *Caenorhabditis elegans*. *Neurotox Res* **21**(3):281-90.	This study on the effect of glyphosate on neurons in the roundworm *C. elegans* provides no data relevant to cancer dose response-analysis.
Oliveira AG, Telles LF, Hess RA, Mahecha GA, Oliveira CA (2007). Effects of the herbicide Roundup on the epididymal region of drakes Anas platyrhynchos. *Reprod Toxicol* **23**(2):182-91.	This study investigated the effects of Roundup® on the epididymis and testes of adult male ducks exposed for 15 days. This male reproductive toxicity study provides no data relevant to cancer dose-response analysis.
Perkins PJ, Boermans HJ, Stephenson GR (2000). Toxicity of glyphosate and triclopyr using the frog embryo teratogenesis assay—*Xenopus*. *Environmental Toxicology and Chemistry* **19**: 940–945.	The effects of glyphosate were studied on the embryonic development of *Xenopus laevis.* This developmental toxicity study is not relevant to cancer dose-response analysis.
Relyea RA (2012). New effects of Roundup on amphibians: predators reduce herbicide mortality; herbicides induce antipredator morphology. *Ecol Appl* **22**(2):634-47.	This study examined the effects of Roundup on the response of amphibians to predators. This behavioral study is not relevant to cancer dose-response analysis.
Romano RM, Romano MA, Bernardi MM, Furtado PV, Oliveira CA (2010). Prepubertal exposure to commercial formulation of the herbicide glyphosate alters testosterone levels and testicular morphology. *Arch Toxicol* **84**(4):309-17.	This paper reports the effects of glyphosate on testicular development in male rats exposed on postnatal days 23 to 53. This study does not provide data that can be used in the cancer dose-response analysis.
Roy NM, Ochs J, Zambrzycka E, Anderson A (2016). Glyphosate induces cardiovascular toxicity in *Danio rerio*. *Environmental Toxicology and Pharmacology*	This study investigated the effects of glyphosate on heart development in zerbrafish. This developmental toxicity study is not relevant to cancer dose-response analysis.

46:292–300.	
Savitz DA, Arbuckle T, Kaczor D, Curis KM (1997). Male pesticide exposure and pregnancy outcome. *Am J Epidemiol* **146**(12):1025-35.	This human epidemiology study assessed pesticide exposure, including exposure to glyphosate, on male reproductive outcomes. This male reproductive toxicity study is not relevant to cancer dose-response analysis.
Soso AB, Barcellos LJ, Ranzani-Paiva MJ, Kreutz LC, Quevedo RM, Anziliero D, Lima M, Silva LB, Ritter F, Bedin AC, Finco JA (2007). Chronic exposure to sub-lethal concentration of a glyphosate-based herbicide alters hormone profiles and affects reproduction of female Jundiá (Rhamdia quelen). *Environ Toxicol Pharmacol* **23**:308–313.	This study examined the effects of glyphosate on the Jundia fish and found effects on reproductive status. This fish reproductive toxicity study does not provide data that can be used in the cancer dose-response analysis.
Soto AM, Sonnenschein C (2010). Environmental causes of cancer: endocrine disruptors as carcinogens. *Nat Rev Endocrinol* **6**(7):363-70.	This study provides no data specific to glyphosate.
Sparling DW, Matson C, Bickham J, Doelling-Brown P (2006). Toxicity of glyphosate as Glypro and LI700 to red-eared slider (trachemys scripta elegans) embryos and early hatchlings. *Environ Toxicol Chem* **25**(10):2768-74.	This study examined the effects of glyphosate on the development of turtle eggs. This developmental toxicity study in turtles is not relevant to cancer dose-response analysis.
Swanson NL, Leu A, Abrahamson J, Wallet B (2014). Genetically engineered crops, glyphosate and the deterioration of health in the United States of America. *Journal of Organic Systems* **9**(2).	This descriptive study conducted correlation analyses based on time trends in genetically engineered crop data, glyphosate application data, and disease rates in the US. A significant correlation was reported between glyphosate application rates and incidence of thyroid, liver, bladder, pancreas, and kidney cancer, and myeloid leukemia. Incidences of these cancers were also correlated with percentages of genetically engineered corn and soy planted in the US.

	This descriptive study provides correlations between glyphosate usage and disease rates. However, a descriptive study does not provide evidence of causation. Additionally, there is a latency period between exposure to a carcinogen and development of cancer. In this study, however, there was often a temporal overlap between increases in glyphosate use and increases in cancer incidence (e.g., no evidence of latency between exposure and cancer). In some cases, cancer incidences increased before glyphosate use did. Descriptive studies such as this do not provide data that can be used in cancer dose-response analysis.
van der Mark M, Brouwer M, Kromhout H, Nijssen P, Huss A, Vermeulen R (2012). Is pesticide use related to Parkinson disease? Some clues to heterogeneity in study results. *Environ Health Perspect* **120**(3):340-7.	This paper conducted a systematic review and meta-analysis of pesticide use (including glyphosate) and Parkinson's disease. This study on Parkinson's disease is not relevant to cancer dose response-analysis.
Vandenberg LN, Colborn T, Hayes TB, Heindel JJ, Jacobs DR Jr, Lee DH, Shioda T, Soto AM, vom Saal FS, Welshons WV, Zoeller RT, Myers JP. Hormones and endocrine-disrupting chemicals: Low-dose effects and nonmonotonic dose responses. *Endocr Rev*. 2012;33(3):378-455.	This study provides no data specific to glyphosate.
Wang G, Fan XN, Tan YY, Cheng Q, Chen SD (2011). Parkinsonism after chronic occupational exposure to glyphosate. *Parkinsonism Relat Disord* **17**(6):486-7.	This study on Parkinson's disease is not relevant to cancer dose-response analysis.
Wu H (1996). Glyphosate impact on rat cytochrome P450 2 B1 and P450 2 c11 gene expression. *Health*	The hyperlink provided by the commenter leads to an article by Wu and Prough (1996), titled "CYP450 2B1 and 2C11 expression in

Toxicology Journal, **10**(4): 231-234 [Article in Chinese] Available from http://www.cnki.com.cn/Article/CJFDTotal-WSDL604.004.htm	rat by glyphosate". Other than the different title, the rest of the citation is correct. This study examined liver microsomal enzyme activity as well as expression levels of CYP450 2B1 and 2C11 mRNA in rats after glyphosate treatment by oral gavage. This study does not provide data that can be used in the cancer dose-response analysis.
Yousef MI, Salem MH, Ibrahim HZ, Helmi S, Seehy MA, Bertheussen K (1995). Toxic effects of carbofuran and glyphosate on semen characteristics in rabbits. *J Environ Sci Health B* **30**(4):513-34.	This study investigated the effects of glyphosate on body weight and semen in male New Zealand white rabbits exposed for six weeks. This male reproductive toxicity study is not relevant to cancer dose-response analysis.
Yu H et al. (2012). Progress in study of glyphosate toxicity **6**. [Article in Chinese] Available from http://www.cnki.com.cn/Article/CJFDTOTAL-BANG201206050.htm and http://www.doc88.com/p-666125982792.html	This is a review of literature on the toxicity of glyphosate. This review did not identify any studies that would affect the cancer dose-response analysis.
Zeng M, Huang T et al. (2014). Glyphosate toxicity to mice GC-1 sperm cells and the interference effect of N-acetyl cysteine, *Ecological Toxicology Bulletin* **1.** [Article in Chinese] Available from http://www.cnki.com.cn/Article/CJFDTotal-STDL201401031.htm	The hyperlink provided by the commenter leads to an article by Zeng et al. (2014), titled "Cytotoxicity of Glyphosate to GC-1 Mice Spermatogonium and Antagonistic Effects of N-acetylcysteine". Other than the title, the rest of the citation is correct. This study examined the cytotoxicity of glyphosate on GC-1 (mouse spermatogonia) cells. The study found that glyphosate induced DNA damage as shown by the Comet assay, and suggests that glyphosate may increase reactive oxygen species production in GC-1 cells. While this study contributes to the data on possible mechanisms of action, it does not provide data that would affect the cancer dose-response analysis that forms the basis for the NSRL.
Zhao W et al. (2011). Study of oxidative damage of the body	This study examined oxidative damage induced by glyphosate in Kunming mice.

caused by glyphosate. *Toxiology Journal* **25**(5):364-366 [Article in Chinese] Available from http://www.cnki.com.cn/Article/CJFDTotal-WSDL201105013.htm	Oxidative damage was measured as levels of total antioxidant capacity (TAC) and malondialdehyde (MDA) in serum and several tissues, and as serum levels of advanced oxidation products. While this study contributes to the data on possible mechanisms of action, it does not provide data that would affect the cancer dose-response analysis that forms the basis for the NSRL.
Zhao W, Yu H, Zhang J, Shu L (2013). Effects of glyphosate on apoptosis and expressions of androgen-binding protein and vimentin mRNA in mouse Sertoli cells. *Journal of Southern Medical University* **33**(11):1709-1713. [Article in Chinese]	This *in vitro* study investigated the effects of glyphosate on cultured mouse Sertoli cells. This male reproductive toxicity study does not provide data that can be used in the cancer dose-response analysis.

Comment 33: Teri Persico, Sandy DeSimone, William Brooks, Dr. Stephanie Seneff, and a number of other commenters provided lists of references for OEHHA's consideration.

Response 33: Of the published scientific articles listed in these comments, OEHHA carefully reviewed each of the cited documents in the context of the guidance set forth in Section 25703, in the same manner as was done in response to comment 32 above. Specifically the regulations states that "the assessment shall be based on evidence and standards of comparable scientific validity to the evidence and standards which form the scientific basis for the listing of the chemical as known to the state to cause cancer".[101] OEHHA determined that none of the cited studies provide data that would affect the cancer dose-response analysis[102]. No changes were made to the regulatory proposal based on these comments.

Comment 34 (**Baum, Hedlund, Aristei and Goldman, P.C.):** "Additional documents pertinent to the Safe Harbor NSRL and Roundup/glyphosate carcinogenicity are presently still under seal and it is strongly recommended that OEHHA obtain access to

[101] Section 25703(a)(4)

[102] In fact, most of the articles were unrelated to carcinogenicity and instead focused on topics such as ecotoxicity, environmental fate and transport, analytical methods, mechanisms unrelated to carcinogenicity, and more.

such documents before OEHHA takes the potentially precarious step of issuing an NSRL of 1100 micrograms."

Response 34: OEHHA used publicly available scientific studies to calculate the NSRL. There is no legal basis for OEHHA to ask a court in a third party matter to provide it with sealed documents. In the event these materials become publicly available in the future and they are relevant to the calculation of the NSRL, OEHHA can reconsider the NSRL at that time. No changes were made to the regulatory proposal based on this comment.

Comment 35: Carcinogen Identification Committee members Dr. Jason Bush, Dr. Luoping Zhang, and Dr. Shanaz Dairkee had no objections to the proposed NSRL. Colton Bond commented that the NSRL is a reasonably conservative benchmark. Chris Portier supported use of the multistage model and the extrapolation plan for the evaluation of glyphosate carcinogenicity. Anne Surdzial recommended that OEHHA adopt the NSRL as is, which is supported by science.

Response 35: No response is required. No changes were made to the regulatory proposal based on this comment.

Comment 36 (Linda Causey): Request determination on the economic cost to finding glyphosate in California wines.

Response 36: This comment is not related to the rulemaking. An NSRL does not require a business to test for the presence of glyphosate in California wines or any other products. In the Economic Impact Analysis for this rulemaking, OEHHA noted:

"One year after the date of listing, businesses that manufacture, distribute or sell products with glyphosate in the state must provide a warning if their product or activity exposes the public or employees to significant amounts of this chemical. *The regulatory proposal does not create additional compliance requirements, but instead provides a "safe harbor" value that aids businesses in determining whether a warning is required for a given exposure*. (Emphasis added.)

Benefits of this regulation include sparing businesses the expense of calculating their own NSRL and possibly enabling them to reduce or avoid litigation costs..."

No changes were made based on this comment.

Comment #37 (Timothy Litzenburg): The single mouse study that OEHHA relied on was done by a glyphosate producer.

Response #37: Studies conducted or contracted by the chemical manufacturer often form part of the scientific data supporting carcinogenicity. As noted in the response to

comment #5, IARC found that two sets of studies in mice and six sets of studies in rats were adequate for the evaluation of glyphosate carcinogenicity. OEHHA reviewed the available data from the rodent carcinogenicity studies of glyphosate in light of the requirement of Section 25703 that the assessment "be based on evidence and standards of comparable scientific validity to the evidence and standards which form the scientific basis for the listing of the chemical as known to the state to cause cancer", and determined that the two-year study conducted in male CD-1 mice fed glyphosate (purity, 98.6%) in the diet met the criterion in Section 25703 as the most sensitive study of sufficient quality. No changes were made based on this comment.

Local Mandate Determination

OEHHA has determined this regulatory action will not impose a mandate on local agencies or school districts nor does it require reimbursement by the State pursuant to Part 7 (commencing with Section 17500) of Division 4 of the Government Code. Local agencies and school districts are exempt from Proposition 65. OEHHA has also determined that no nondiscretionary costs or savings to local agencies or school districts will result from this regulatory action. The regulation does not create additional compliance requirements, but instead provides a "safe harbor" value that aids businesses in determining whether a warning is required for a given exposure.

Alternatives Determination

Pursuant to Government Code section 11346.5(a)(13), OEHHA initially determined that no reasonable alternative considered by OEHHA, or that has otherwise been identified and brought to the attention of OEHHA, would be more effective in carrying out the purpose for which the action is proposed, or would be as effective and less burdensome to affected private persons than the proposed action, or would be more cost-effective to affected private persons and equally effective in implementing the statutory policy or other provision of law.

In accordance with Government Code section 11346.9(a)(4), OEHHA has considered available alternatives to determine whether any alternative would be more effective in carrying out the purpose for which the regulations were proposed. OEHHA has also considered whether an alternative exists which would be as effective as and less burdensome to affected private persons than the proposed action. OEHHA has determined that no alternative considered would be more effective, or as effective as and less burdensome to affected private persons than the proposed regulation. No alternative that is less burdensome yet equally as effective in achieving the purposes of the regulation in a manner that achieves the purposes of the statute has been proposed. The NSRL provides a "safe harbor" value that aids businesses in determining if they are complying with the law. The regulation does not create additional compliance requirements, but instead provides a "safe harbor" value that aids

businesses in determining whether a warning is required for a given exposure. The alternative to the proposed amendment to Section 25705(b) would be to not adopt an NSRL for the chemical. Failure to adopt an NSRL would leave the business community without a “safe harbor” level to assist businesses in complying with Proposition 65. Some commenters proposed alternative NSRLs and approaches for deriving an NSRL. These comments were not reasonable alternatives and are fully discussed in responses to comments within this FSOR. There were no small-business specific alternatives submitted during the rulemaking process.

CALIFORNIA ENVIRONMENTAL PROTECTION AGENCY
OFFICE OF ENVIRONMENTAL HEALTH HAZARD ASSESSMENT

NOTICE OF AMENDMENT

TITLE 27, CALIFORNIA CODE OF REGULATIONS

AMENDMENT TO SECTION 25705
NO SIGNIFICANT RISK LEVEL - GLYPHOSATE
APRIL 10, 2018

On April 6, 2018, the Office of Administrative Law approved an amendment of Title 27, California Code of Regulations, section 25705, No Significant Risk Level (NSRL) for the chemical glyphosate. **The regulation will be effective on July 1, 2018.** This regulation establishes a NSRL of 1100 micrograms per day for glyphosate.

The regulatory text and the supporting rulemaking documents are available at the following links:

Notice of Proposed Rulemaking

Initial Statement of Reasons

Final Statement of Reasons

Final Adopted Regulatory Text

Questions regarding this regulatory action can be directed to Esther Barajas-Ochoa, at esther.barajas-ochoa@oehha.ca.gov or (916) 322-2068.

News Releases from Headquarters › Chemical Safety and Pollution Prevention (OCSPP)

EPA Takes Next Step in Review Process for Herbicide Glyphosate, Reaffirms No Risk to Public Health

04/30/2019

Contact Information:
EPA Press Office (press@epa.gov)

WASHINGTON – Today, the U.S. Environmental Protection Agency (EPA) is taking an important step in the agency's review of glyphosate. As part of this action, EPA continues to find that there are no risks to public health when glyphosate is used in accordance with its current label and that glyphosate is not a carcinogen. The agency's scientific findings on human health risk are consistent with the conclusions of science reviews by many other countries and other federal agencies. While the agency did not identify public health risks in the 2017 human health risk assessment, the 2017 ecological assessment did identify ecological risks. To address these risks, EPA is proposing management measures to help farmers target pesticide sprays on the intended pest, protect pollinators, and reduce the problem of weeds becoming resistant to glyphosate.

"EPA has found no risks to public health from the current registered uses of glyphosate," said **EPA Administrator Andrew Wheeler**. "Today's proposed action includes new management measures that will help farmers use glyphosate in the most effective and efficient way possible, including pollinator protections. We look forward to input from farmers and other stakeholders to ensure that the draft management measures are workable, realistic, and effective."

"If we are going to feed 10 billion people by 2050, we are going to need all the tools at our disposal, which includes the use the glyphosate," **U.S. Secretary of Agriculture Sonny Perdue** said. "USDA applauds EPA's proposed registration decision as it is science-based and consistent with the findings of other regulatory authorities that glyphosate does not pose a carcinogenic hazard to humans."

Glyphosate is the most widely used herbicide in U.S. agriculture and has been studied for decades. Glyphosate is used on more than 100 food crops, including glyphosate-resistant corn, soybean, cotton, canola and sugar beet. Non-agricultural uses include residential areas, aquatic areas, forests, rights of way, ornamentals and turf.

Once the Federal Register notice publishes, the public will be able to submit comments on EPA's proposed decision at www.regulations.gov in docket # EPA-HQ-OPP-2009-0361. Public comments will be due 60 days after the date of publication in Federal Register. EPA's responses to the comments received on the draft ecological and human health risk assessments and the benefits assessment will be in the docket.

For more information about glyphosate, including today's proposed interim decision and supporting documents, visit: https://www.epa.gov/ingredients-used-pesticide-products/glyphosate.

The glyphosate draft risk assessments and supporting documents can be found at: https://www.epa.gov/ingredients-used-pesticide-products/draft-human-health-and-ecological-risk-assessments-glyphosate.

LAST UPDATED ON APRIL 30, 2019

From: Keith F. Collins
To: Varga, Tom
Cc: Miller, Tabatha; O"Neal, Chantell
Subject: RE: Chemical ban question
Date: Thursday, May 02, 2019 12:50:15 PM

Tom,
You are correct that cities are preempted from regulating pesticides, except that they can regulate how pesticides are used by the city itself on its own property. The City of Malibu recently did this. As you can see from below, any City ordinance that attempts to regulate the use or sale of pesticides by the public would be invalidated if challenged. If necessary, we can do further research into what specific substances the City can regulate.
"…no ordinance or regulation of local government, including, but not limited to, an action by a local governmental agency or department, a county board of supervisors or a city council, or a local regulation adopted by the use of an initiative measure, may prohibit or in any way attempt to regulate any matter relating to the registration, sale, transportation, or use of pesticides, and any of these ordinances, laws, or regulations are void and of no force or effect."

Cal Food & Agr Code § 11501.1

Keith F. Collins
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Tel: (714) 446-1400
Fax: (714) 446-1448

From: Varga, Tom [mailto:TVarga@fortbragg.com]
Sent: Thursday, May 2, 2019 8:58 AM
To: Keith F. Collins
Cc: Miller, Tabatha; O'Neal, Chantell
Subject: Chemical ban question

Keith-
Thanks for making the trip up and saying hi.
I already have a question that I hope you can help with. One of our councilmembers, Jessica Morsell-Haye, has strong concerns about Round-Up and its primary active ingredient Glyphosate. She is asking if a full and total ban is possible for the possession, sale, or use city-wide including both private and public parties. We are making a brief presentation at next Wednesday's Public Works & Facilities Committee meeting for discussion and a possible recommendation to the full Council. Coincidentally, we just heard that there may be State pre-emption over local agency bans on the scale being considered. We did not get much detail beyond that. Can you give us more background on the matter? What can we or cannot regulate when it comes to glyphosate or similar substances. It appears that local agencies in California primarily limit their actions to public uses.
If you can find something by next Wednesday morning, that would be greatly appreciated. If it will take a little more time to chase down this information, that is understood as well.
Thanks for the help,

Tom Z. Varga
Director of Public Works

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