Arbeidstilsynet

Grunnlag for fastsettelse av grenseverdi

Grunnlagsdokument for akrylsyre ($C_3H_4O_2$)

Kommisjonsdirektiv 2017/164/EU

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Denne rapporten omhandler det toksikologiske grunnlaget og vurderinger, samt tekniske og økonomiske hensyn for fastsettelse av grenseverdi for akrylsyre ($C_3H_4O_2$).

Innhold

Forord

Grunnlagsdokumenter for fastsettelse av grenseverdier utarbeides av Arbeidstilsynet i samarbeid med Statens arbeidsmiljøinstitutt (STAMI) og partene i arbeidslivet (Næringslivets hovedorganisasjon/Norsk Industri og Landsorganisasjonen i Norge) i henhold til *Strategi for utarbeidelse og fastsettelse av grenseverdier for forurensninger i arbeidsatmosfæren*. Dette dokumentet er utarbeidet ved implementering av kommisjonsdirektiv 2017/164/EU fastsatt 31. januar 2017.

EU-rådets direktiv 98/24/EC (Vern av helse og sikkerhet til arbeidstakere mot risiko i forbindelse med kjemiske agenser på arbeidsplassen) av 7. april 1998 stiller krav om at EU- kommisjonen skal legge frem forslag til indikative grenseverdier for eksponering av visse kjemikalier som medlemslandene må innføre på nasjonalt nivå. De nasjonale grenseverdiene kan være høyere enn de som står oppført i direktivet, dersom et medlemsland mener at det er nødvendig av tekniske og/eller økonomiske hensyn, men landene bør nærme seg den indikative grenseverdien. Direktivet stiller krav om at indikative grenseverdier vedtas gjennom kommisjonsdirektiv.

I Norge ble de indikative grenseverdiene innført som veiledende administrative normer. Da nye Arbeidsmiljøforskrifter trådte i kraft 1.1.2013 ble de veiledende administrative normene forskriftsfestet i forskrift om tiltaks- og grenseverdier og fikk betegnelsen tiltaksverdier. I 2015 ble begrepet «grenseverdi» for kjemikalier presisert og begrepet «tiltaksverdi» for kjemikalier ble opphevet i forskrift om tiltaks- og grenseverdier. I vedlegg 1 til forskriften ble det innført en tydeliggjøring av anmerkningene.

Arbeidstilsynet har ansvaret for revisjonsprosessen og utarbeidelse av grunnlagsdokumenter for stoffene som blir vurdert. Det toksikologiske grunnlaget for stoffene i denne revisjonen baserer seg i hovedsak på kriteriedokumenter fra EUs vitenskapskomité for fastsettelse av grenseverdier, Scientific Committee for Occupational Exposure Limits (SCOEL). SCOEL utarbeider de vitenskapelige vurderingene som danner grunnlaget for anbefalinger til helsebaserte grenseverdier, og disse legges fram for kommisjonen.

Statens arbeidsmiljøinstitutt (STAMI) ved Toksikologisk ekspertgruppe for administrative normer (TEAN) bidrar med faglige vurderinger i dette arbeidet. TEAN vurderer og evaluerer de aktuelle SCOEL dokumentene, presiserer kritiske effekter og vurderer behov for korttidsverdier ut i fra den foreliggende dokumentasjonen. Videre søker og evaluerer TEAN nyere litteratur etter utgivelsen av dokumentet. TEAN bruker kriteriene gitt i SCOEL's metodedokument "Methodology for the derivation of occupational exposure limits: Key documentation (version 7, June 2013)". Dette er inkludert i TEANs Metodedokument del B (Prosedyre for utarbeidelse av toksikologiske vurderinger for stoffer som skal implementeres i det norske regelverket for grenseverdier etter direktiv fra EU-kommisjonen) utarbeidet for denne revisjonen.

Informasjon om bruk og eksponering i Norge innhentes fra Produktregisteret, EXPO databasen ved STAMI og eventuelle tilgjengelige måledata fra virksomheter/næringer. Beslutningsprosessen skjer gjennom drøftingsmøter der Arbeidstilsynet, Næringslivets hovedorganisasjon/Norsk Industri og Landsorganisasjonen i Norge deltar, samt orienteringsmøter og offentlig høring. Konklusjonene fra høringen med forskriftsendringer og nye grenseverdier forelegges Arbeids- og sosialdepartementet som tar den endelige beslutningen.

Innledning

Dette grunnlagsdokumentet omhandler vurderingsgrunnlaget for fastsettelse av grenseverdi for akrylsyre. Innholdet bygger spesielt på anbefalinger fra Scientific Committee on Occupational Exposure Limits (SCOEL) i EU for akrylsyre, og vurderinger og kommentarer fra Toksikologisk Ekspertgruppe for Administrative Normer (TEAN).

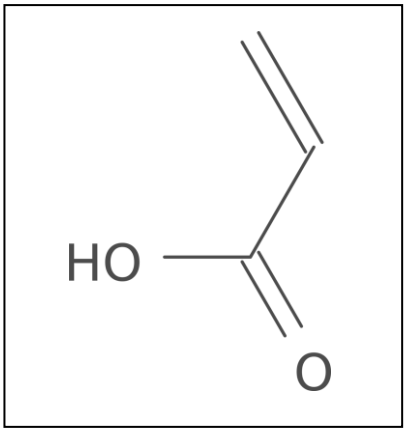
1. Stoffets identitet

Stoffet akrylsyre og dets molekylformel, synonym av stoffets navn, stoffets identifikasjonsnummer i Chemical Abstract Service Registry number (CAS-nr.) og European Inventory of Existing Commercial Chemical Substances (EINECS-nr. og/eller EC-nr.) og indeks-nr. der disse er kjent er gitt i tabell 1. Strukturformel av akrylsyre er vist i figur 1.

Tabell 1. Stoffets navn og identitet.

Kjemisk navn **Molekylformel**	**akrylsyre** **$C_3H_4O_2$**
Synonymer	2-propensyre,
CAS-nr.	79-10-7
EINECS-nr.	201-177-9
INDEX-nr.	-

Index-nr. er ikke oppgitt for akrylsyre.



Figur 1. Strukturformel av akrylsyre (https://snl.no/akrylsyre).

2. Fysikalske og kjemiske data

Stoffet akrylsyre er en klar, fargeløs væske med karakteristisk sur stikkende plastlukt, og den er lettløselig i vann. Stoffet kan fremstilles ved oksidasjon av akrolein, men de største mengdene blir laget fra etylen via etylenoksid. Det vises til tabell 2 for fysikalske og kjemiske data for akrylsyre.

Tabell 2. Fysikalske og kjemiske data for akrylsyre.

Kjemisk formel	**akrylsyre**
Molekylvekt (g/mol)	72,06
Fysisk tilstand	Fargeløs væske
Smeltepunkt (°C)	14
Kokepunkt (°C)	141
Flamme punkt (°C)	54
Selvantennelsestemperatur (°C)	395
Tetthet (20 °C)	1,05
Damptetthet (air = 1) (g/cm^3)	2,5
Damptrykk ved 20 °C (Pa)	413
Fordelingskoeffisient n-oktanol/vann (log K_{ow})	0,35
Løselighet i vann (25 °C, g/l)	1000
Løselighet i andre løsemidler	Løselig i metanol, etanol, 2-propanol, etylenglykol
Eksplosjonsgrenser (%)	Nedre (UEL): 2,9 Øvre (LEL): 8
Luktterskel (ppm)	0,067-1,047
Omregningsfaktor (20 °C, 101 kPa)	1 ppm = 2,947 mg/m^3; 1 mg/m^3 = 0,339 ppm

Data gitt av TEAN.

2.1 Forekomst og bruk

Det årlige produksjonsvolumet av akrylsyre i EU er anslått til å være ca. 810 000 tonn. Om lag 830 000 tonn akrylsyre blir forbrukt årlig i EU. Det importeres ca. 20 000 tonn og eksporteres minst 15 000 tonn utenfor EU. Markedet gjennomgikk en rask vekst på 1990-tallet.

Akrylsyre blir brukt til å produsere polyakrylat. Ved oppvarming polymeriserer akrylsyre og denne egenskapen utnyttes i visse derivater, f.eks. estere og til fremstilling av diverse plastprodukter. Akrylsyre (opp til 10%) blir brukt som ingrediens i produkter som lim, maling, bindemidler og trykksverte. Arbeidstakere kan bli eksponert for akrylsyre under produksjon og behandling av stoffet i kjemisk industri og i produksjon av produkter som inneholder akrylsyre. Eksponering for akrylsyre kan også oppstå ved bruk av klebemidler og andre produkter som inneholder dette stoffet, under nedbryting av fotoresistent materialer med ultrafiolett lys (f.eks under produksjon av integrerte kretser) og under fjerning av maling ved hjelp av flamme.

3. Grenseverdier

3.1 Nåværende grenseverdi

Nåværende grenseverdi (8 timer) i Norge for akrylsyre er:

10 ppm (30 mg/m^3).

3.2. Grenseverdier fra EU

Den europeiske vitenskapskomiteen, SCOEL foreslår for akrylsyre i sitt kriteriedokument av juni 2012[1]:

IOELV (Indicative Occupational Exposure Limit Value) (8 timer): 10 ppm (29 mg/m^3)

STEL (Short Term Exposure Limit) (1 min): 20 ppm (59 mg/m^3)

Ingen anmerkning er foreslått for akrylsyre.

3.3. Grenseverdier fra andre land og organisasjoner

Grenseverdier for akrylsyre fra andre land og organisasjoner er gitt i tabell 3 nedenfor.

Tabell 3. Grenseverdier for akrylsyre fra andre land og organisasjoner. Land og organisasjoner som ikke har grenseverdier for akrylsyre er markert med -.

Land Organisasjon	Grenseverdi (8 timer)	Korttidsverdi (15 min)	Anmerkning Kommentar
Sverige[1]	10 ppm; 30 mg/m^3	15 ppm; 45 mg/m^3	V, veiledende korttidsverdi
Danmark[2]	2 ppm; 5,9 mg/m^3	-	H, hudopptak
Finland[3]	2 ppm; 6 mg/m^3	15 ppm; 45 mg/m^3	-
Storbritannia[4]	-	-	-
Nederland[5]	5,9 mg/m^3	-	-
ACGIH, USA[6]	2 ppm; 5,9 mg/m^3	-	hud
NIOSH, USA[6]	2 ppm; 6 mg/m^3	-	hud
Tyskland, MAK[6]	10 ppm; 30 mg/m^3	I (1)	Gjelder korttidsverdi: Overskridelsesfaktor C, takverdi
Tyskland, Myndighetene[7]	10 ppm; 30 mg/m^3	-	1(I) Overskridelsesfaktor **Y**, ikke fare for skade på foster dersom grenseverdi overholdes.

[1] Arbetsmiljöverkets Hygieniska gränsvärden AFS 2015:7, https://www.av.se/globalassets/filer/publikationer/foreskrifter/hygieniska-gransvarden-afs-2015-7.pdf .
[2] At-vejledning, stoffer og materialer - C.0.1, 2007, https://arbejdstilsynet.dk/da/regler/at-vejledninger/g/c-0-1-graensevaerdi-for-stoffer-og-mat.
[3] Social og hälsovårdsministeriet, HTP-värden, Koncentrationer som befunnits skadliga, Helsingfors, 2016, http://julkaisut.valtioneuvosto.fi/bitstream/handle/10024/79110/STM_9_2016_HTP-varden_2016_Ruotsi_22122016_NETTI.pdf .
[4] EH40 andre utgave, 2013, http://www.hse.gov.uk/pubns/priced/eh40.pdf .
[5] http://www.ser.nl/en/oel_database.aspx; http://www.ser.nl/en/grenswaarden/2%20butyne%201%204%20diol.aspx
[6] Guide to occupational exposure values compiled by ACGIH, 2017.
[8] Baua, TRGS 900, oppdatert 2016, https://www.baua.de/DE/Angebote/Rechtstexte-und-Technische-Regeln/Regelwerk/TRGS/pdf/TRGS-900.pdf;jsessionid=439FFF321DF2323E60F868CD08E9CD3A.s1t2?__blob=publicationFile&v=2

3.4. Stoffets klassifisering

Stoffet akrylsyre er klassifisert og merket i henhold til CLP Annex VI (Forordning EC No 1272/2008), og klassifisering og merking med koder i henhold til fareklasse, kategori og faresetninger er gitt i tabell 4.

Tabell 4. Fareklasser, farekategori med forkortelse, merkekoder og faresetninger for akrylsyre.

Fareklasse **Farekategori** **Forkortelse**	**Merkekode**	**Faresetning**
Brannfarlige væsker Kategori 3 Flam. Liq. 3	H 226	Brannfarlig væske og damp
Akutt giftighet Kategori 4 Acute Tox. 4	H 302	Farlig ved svelging
Akutt giftighet Kategori 4 Acute Tox. 4	H 312	Farlig ved hudkontakt
Etsende/irriterende for huden Kategori 1 Underkategori 1A, Skin Corr. 1A	H 314	Gir alvorlige etseskader på hud og øyne
Akutt giftighet Kategori 4 Acute Tox. 4	H 332	Farlig ved innånding
Spesifikt målorgantoksisitet – enkelteksponering C ≥ 1 % Kategori 3 STOT SE 3	H335	Kan forårsake irritasjon av luftveiene
Farlig for vannmiljøet Akutt kategori 1 Aquatic Acute 1	H 400	Meget giftig for liv i vann

CLP ((Forordning (EC) Nr. 1272/2008), http://www.miljodirektoratet.no/Documents/publikasjoner/M259/M259.pdf, https://echa.europa.eu/information-on-chemicals/cl-inventory-database

3.5. Biologisk overvåking

For å vurdere grad av eksponering for forurensning i luften på arbeidsplassen kan man anvende konsentrasjonen av forurensningen i arbeidstakerens urin, blod eller utåndingsluft, eller annen respons på eksponeringen i kroppen. EU har satt verdier for dette kalt biologisk grenseverdi (BLV).

SCOEL har ikke fremmet et forslag til biologisk grenseverdi for akrylsyre.

4. Toksikologiske data og helseeffekter

4.1. Anbefaling fra SCOEL

EUs vitenskapskomite (SCOEL) har utarbeidet kriteriedokumentasjon for akrylsyre datert juni 2012[1] hvor de anbefaler en grenseverdi (8 timer) for stoffet lik 10 ppm (29 mg/m^3) og en korttidsverdi (1 min) lik 20 ppm (59 mg/m^3), se Vedlegg 1. SCOEL har ikke anbefalt noen anmerkninger for stoffet.

SCOEL rapporterer en NOAEL på 75 mg/m^3 (25 ppm) fra rottestudier og en NOAEL fra hunn musestudier på 15 mg/m^3 (5 ppm) og LOAEL lik 75 mg/m^3 (25 ppm).

SCOEL viser til at det ikke finnes resultater som tyder på at akrylsyre kan forårsake luftveissykdommer eller hudallergi, så dermed er ikke en anmerkning for sensibiliserende egenskaper nødvendig, men SCOEL rapporterer at det er ingen garanti for slik egenskap ikke kan oppstå.

4.2. Kommentarer fra TEAN

SCOEL-dokumentet er fra 2012[1]. TEAN har i tillegg gjennomgått kriteriedokumenter fra MAK fra 2006 (også gjennomgått av SCOEL), ACGIH fra 2001[2], AEGL fra 2004[3] og en NIOSH-publikasjon fra 2017[4]. Det ble gjort litteratursøk i PubMed. Det er ikke funnet relevante studier publisert i etterkant av SCOEL-dokumentet.

Den kritiske effekten til akrylsyre er etsende effekt på hud og øyne og irritasjon i luftveier hos menneske. Stoffet har ingen sensibiliserende effekt. Det er ikke dokumentert at det har karsinogene egenskaper og in vivo tester av mutagenisitet har i hovedsak gitt negative resultater.
Selv om stoffet produseres i store mengder, og er i bruk i en rekke industrielle prosesser, så er det svært lite litteratur på helseeffekter hos menneske og eksponeringsdata er mangelfulle.
Det er beskrevet noen kasuistikker (i perioden 1967-95) av arbeidere som har vært utsatt for ulykker med akrylsyre der etsende effekt på hud og øyne var hovedeffekten i MAK dokumentet fra 2006.
En god human epidemiologisk studie foreligger (Tucek M et al., 2002). I denne studien ble 60 arbeidere, eksponert for ulike kjemikalier i forbindelse med produksjon av akrylsyre, fulgt i åtte år (1992-1999). Arbeiderne ble sammenliknet med like mange kontroller. Eksponeringen for kjemikaliene var lav. Selv om de eksponerte arbeiderne kunne ha flyktige symptomer på irritasjon i øyne, hud og hals, ble det ikke funnet helseeffekter som kunne relateres til akrylsyre, spesielt ingen endring av lungefunksjonen.

Den toksiske egenskapen til stoffet ser ut til å dreie seg om lokal irritasjon av slimhinner og hud, som er av forbigående karakter. Rotte- og humandata danner basis når det gjelder å utlede grenseverdier (Miller et al., 1981 og van Thriel et. al., 2006). Det er brukt data fra rottestudier til å ekstrapolere til menneske siden det ser ut til at deponeringen av stoffet i nesen hos menneske minner om den hos rotte (Miller et al., 1981). På bakgrunn av disse betraktningene er en 8-timers TWA satt til 10 ppm. Van Thriel et al., (2006) har sett på lukt- og irritasjonsgrensen hos en stor gruppe av forsøkspersoner og finner at irritasjonsgrensen kan settes til 30 ppm. SCOEL foreslår på bakgrunn av disse data en STEL, begrenset til ett minutt, på 20 ppm. En hudnotasjon er ikke nødvendig ifølge SCOEL.

For å beskytte mot irritasjon mener TEAN det er behov for en korttidsverdi. TEAN har ingen bemerkninger til SCOELs vurderinger.

5. Bruk og eksponering

I Norge brukes akrylsyre i en rekke produkter, blant annet i bygningsartikler, trevarer og varer av kork, strå og flettematerialer, papir og papp, kjemikalier og kjemiske produkter, maling og lakk, trykkfarger og tetningsmidler, såpe og vaskemidler, rense- og polermidler, gummi- og plastprodukter og metaller.

5.1. Opplysning fra Produktregistret

Data fra Produktregisteret er innhentet oktober 2016, og inneholder opplysninger om mengde og bruk av akrylsyre i deklareringspliktige produkter. Produktregisterdata for akrylsyre viser at stoffet blir brukt i totalt 238 produkter som utgjør en total netto mengde av produkter lik 8,08 tonn (avrundet).

Det henvises til tabell 5 for detaljert oversikt over bransjebeskrivelser med tilhørende bransjekode for de produkter det kan rapporteres på (minimum 4 produkter) og total mengde i tonn (over 0,4 tonn) for akrylsyre.

Tabell 5. Bransjekoder og beskrivelser av bransjer hvor akrylsyre benyttes og total mengde forbruk i tonn.

Bransjekode	**Bransjebeskrivelse**	**Netto mengde (tonn)**
31.0	PRODUKSJON AV MØBLER	1,07
43	SPESIALISERT BYGGE- OG ANLEGGSVIRKSOMHET	0,41
45.2	VEDLIKEHOLD OG REPARASJON AV MOTORVOGNER, UNNTATT MOTORSYKLER	0,92

Akrylsyre inngår i produksjon av møbler og til vedlikehold og reparasjon av motorvogner (unntatt motorsykler) samt brukt i mindre grad i bygge- og anleggsvirksomheter.

Opplysninger om produkttypekode, produkttype og netto mengde er gitt i tabell 6 for akrylsyre.

Tabell 6. Oversikt over produkttyper med beskrivelser som inneholder akrylsyre og totale mengder av produktene.

Produkttype	Beskrivelse av produkttype	Netto mengde (tonn)
M05243	MALING OG LAKK FLYKTIGE ORGANISK LØSEMIDDEL DEKORATIV/BESKYTTELSE INDUSTRIELT BRUK	0,50
M05443	MALING OG LAKK LØSEMIDDELFRITT DEKORATIV/BESKYTTELSE INDUSTRIELT BRUK	1,06

På grunn av sikkerhetsbestemmelsene i Produktregisteret kan vi ikke gi eksakte opplysninger ut over denne informasjon.

5.2. Eksponering og måledokumentasjon

Det er ikke p.t. registrert eksponeringsmålinger for stoffet akrylsyre i STAMIs eksponeringsdatabase.

5.2.1 Prøvetakings- og analysemetode av akrylsyre.

I tabell 7 er anbefalte metoder for prøvetaking og analyser av akrylsyre presentert.

Tabell 7. Anbefalte metoder for prøvetaking og analyse av akrylsyre.

Prøvetakingsmetode	Analysemetode	Referanse
Rør m/Anasorb 708 (2 i serie)	Desorpsjon m/MeOH/H_2O, HPLC-UV[1]	OSHA-metode 28 [2], OSHA-metode PV 2005 [2]

[1] Væskekromatografi HPLC med standard ultrafiolett (UV) detektor for høyoppløselighet.
[2] OSHA metoder: OSHA Sampling and Analytical Methods finnes på www.osha.gov/dts/sltc/methods .

6. Vurdering

Toksikologiske data for akrylsyre er beskrevet i SCOEL-dokumentet i vedlegg 1, og kommentert av STAMI (TEAN) i kapittel 4.

Utgangspunktet for beregning av grenseverdi er en subkronisk NOAEL på 25 ppm i rotter. Akrylsyre trenger ikke å omsettes for å forårsake irritasjon, så individuelle forskjeller i sensorisk irritasjon er små.

Derfor, en 8-timers grenseverdi lik 10 ppm anses hensiktsmessig for å beskytte arbeidstakere fra histologiske endringer og irritasjon.

Tilgjengelige data støtter at den kritiske effekten av eksponering for akrylsyre er etsende effekt på hud og øyne samt irritasjon i luftveier hos mennesker. Derfor anbefaler SCOEL en korttidsverdi for akrylsyre. På bakgrunn av forelagte studier foreslår komiteen en korttidsverdi for akrylsyre lik 20 ppm, men begrenset til 1 minutt. Derfor foreslås en korttidsverdi for akrylsyre av 20 ppm som skal være begrenset til 1 min. Det rapporteres om at det ikke er måletekniske problemer ved den anbefalte grenseverdien og korttidsverdi på 1 minutt.

Akrylsyre er klassifisert og er farlig ved hudkontakt, farlig ved innånding, kan gi alvorlige etseskader på hud og øyne og kan forårsake irritasjon av luftveiene. SCOEL viser til at det ikke finnes resultater som tyder på at akrylsyre kan forårsake luftveissykdommer eller hudallergi, så dermed er ikke en anmerkning for sensibiliserende egenskaper nødvendig, men SCOEL rapporterer at det er ingen garanti for at en slik egenskap ikke kan oppstå. En hudnotasjon er heller ikke nødvendig ifølge SCOEL.

På bakgrunn av kunnskap om at akrylsyre kan gi irritasjon i øyne eller luftveier jf. klassifiseringen, og som forebyggende virkning, anbefales en anmerkning A (definert som kjemikalier som skal betraktes som at de fremkaller allergi eller annen overfølsomhet i øynene eller luftveier, eller som skal betraktes som at de fremkaller allergi ved hudkontakt) som i dette tilfellet gjelder overfølsomhet i øyne og luftveier.

Siden det ikke finnes resultater som tyder på at akrylsyre kan forårsake hudallergi, eller hudopptak anbefales ingen hudanmerkning for akrylsyre.

7. Konklusjon med forslag til ny grenseverdi

På bakgrunn av den foreliggende dokumentasjon og en avveiing mellom de toksikologiske dataene og tekniske og økonomiske hensyn, foreslås at grenseverdien (8 timer) på 10 ppm, 29 mg/m^3 for akrylsyre opprettholdes (men korrigert i henhold til omregningsfaktoren), og at det innføres en korttidsverdi (1 minutt) lik 20 ppm, 59 mg/m^3 for akrylsyre.

I tillegg innføres en anmerkning A (Kjemikalier som skal betraktes som at de fremkaller allergi eller annen overfølsomhet i øynene eller luftveier, eller som skal betraktes som at de fremkaller allergi ved hudkontakt) og E (EU har fastsatt en grenseverdi) for akrylsyre.

Anbefalt grenseverdi og korttidsverdi for akrylsyre:

Grenseverdi (8 timer): 10 ppm, 29 mg/m^3

Korttidsverdi (1 minutt): 20 ppm, 59 mg/m^3

Anmerkning: S (korttidsverdi), E (EU har fastsatt en grenseverdi for stoffet) og A (Kjemikalier som skal betraktes som at de fremkaller allergi eller annen overfølsomhet i øynene eller luftveier, eller som skal betraktes som at de fremkaller allergi ved hudkontakt)

8. Nye grenseverdier

På grunnlag av drøftinger med partene og høringsuttalelser ble ny grenseverdi og korttidsverdi for akrylsyre fastsatt til:

Grenseverdi (8-timer): 10 ppm, 29 mg/m^3

Korttidsverdi (1 min): 20 ppm, 59 mg/m^3

Anmerkninger: S (korttidsverdi), A (allergifremkallende)og E (EU har fastsatt grenseverdi for stoffet)

Stoffet har fått anmerkningen A med bakgrunn i dagens definisjon for anmerkningen: Kjemikalier som skal betraktes som at de fremkaller allergi eller annen overfølsomhet i øynene eller luftveier, eller som skal betraktes som at de fremkaller allergi ved hudkontakt.

9. Referanser

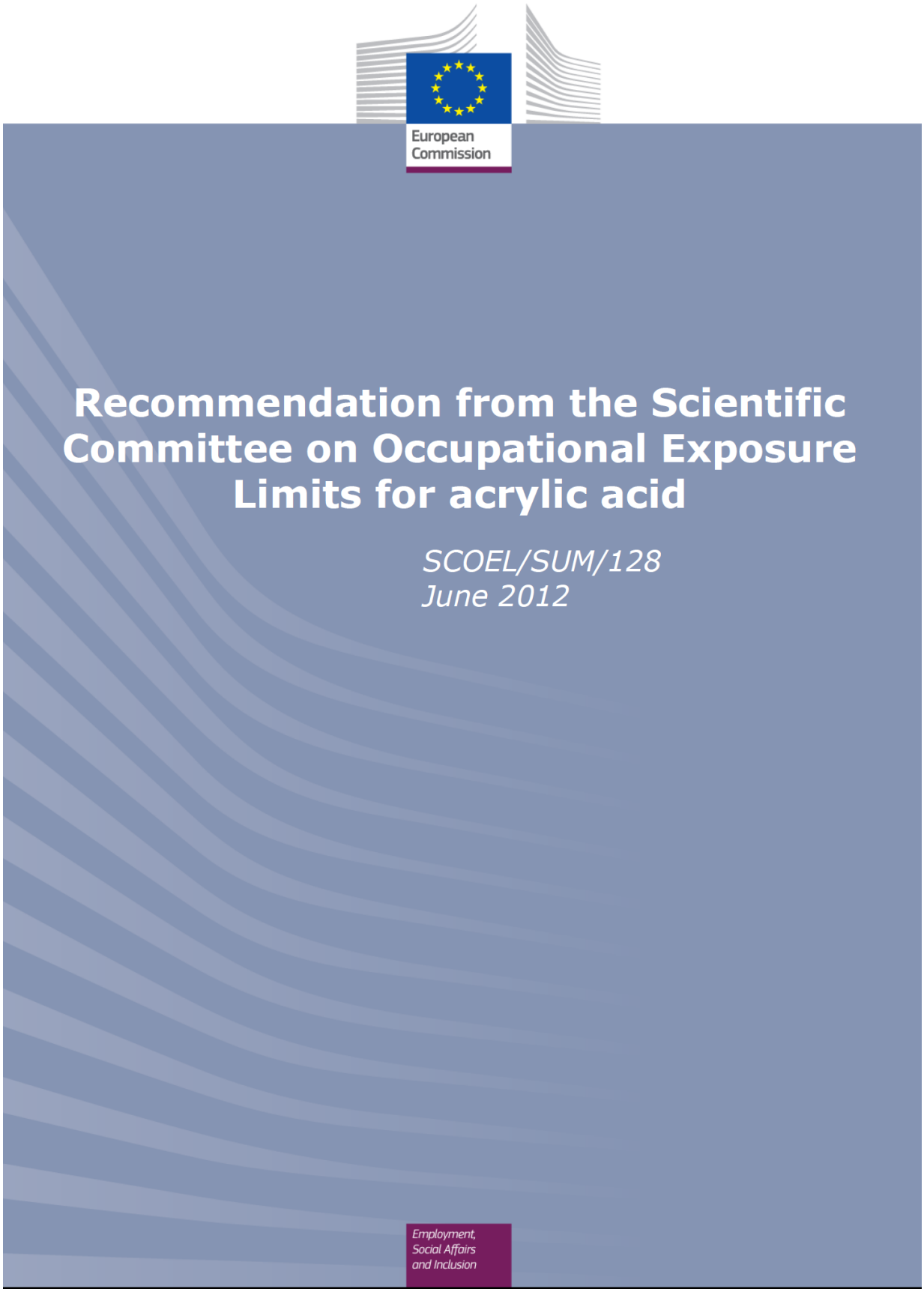
1. Recommendation from the Scientific Committee on Occupational Exposure Limits for acrylic acid, SCOEL/SUM/128, juni 2012.

2. American Conference of Governmental Industrial Hygienists (ACGIH®),
Threshold Limit Values (TLVs®) and Biological Exposure Indices (BEIs®)
1330 Kemper Meadow Drive | Cincinnati, Ohio; Acrylic acid, 2001.

3. NIOSH [2017]. NIOSH skin notation profile: Acrylic acid. By Hudson NL, Dotson GS. Cincinnati, OH: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication No. 2017-133.

4. AEGL Acute exposure guideline Levels (AEGLS), Acrylic acid Interim 2: 1/2004
United States Environmental Protection Agencies, EPA
https://www.epa.gov/sites/production/files/2014-08/documents/tsd304.pdf .

Vedlegg SCOEL



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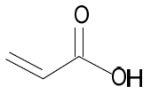
Recommendation from the Scientific Committee on Occupational Exposure Limits for acrylic acid

8-hour TWA:	10 ppm (29 mg/m^3)
STEL (1-min):	20 ppm (59 mg/m^3)
Notation:	None

Substance identification

Chemical name:	Acrylic acid
Synonyms:	2-propenoic acid, vinylformic acid
CAS No.:	79-10-7
EINECS No.:	201-177-9
Molecular formula:	$C_3H_4O_2$
Structural formula:	
Molecular weight:	72.06 g/mol
Conversion factor (25 °C):	1 ppm = 2.947 mg/m^3; 1 mg/m^3 = 0.339 ppm

EU Classification:

Flam. Liq. 3	H226	Flammable liquid and vapour
Acute Tox. 4 *	H332	Harmful if inhaled
Acute Tox. 4 *	H312	Harmful in contact with skin
Acute Tox. 4 *	H302	Harmful if swallowed
Skin Corr. 1A	H314	Causes severe skin burns and eye damage
Aquatic Acute 1	H400	Very toxic to aquatic life
STOT SE 3, C ≥ 1 %	H335	May cause respiratory irritation

This document is largely based on the EU-RAR (2002) and the references therein, along with some additional more recent published studies identified using the on-line database PubMed.

Physico-chemical properties

At 20 °C and 1 013 hPa, pure acrylic acid is a clear colourless liquid with an irritating acrid odour. The average odour threshold of acrylic acid in air is between 0.20–3.14 mg/m^3 (0.067 and 1.047 ppm). It is miscible with water and most organic solvents (IPCS 1997). Acrylic acid is flammable and combustible. The melting temperature is 14 °C and the boiling temperature is given as 141 °C. Acrylic acid has a flash point of 48–55 °C and the vapour pressure is 3.8 hPa at 20 °C. The pKa is given as 4.25 (EU RAR).

1. Occurrence/use and occupational exposure

The annual production volume of acrylic acid in the EU is estimated to be about 810 000 tonnes. About 20 000 tonnes are imported and at least 15 000 tonnes are exported outside the EU (EU RAR). The annual consumption of acrylic acid within the EU is about 830 000 tonnes. The market was reported to be undergoing rapid growth during the 1990s.

Acrylic acid is an industrial intermediate used to produce polyacrylate directly or polymerised via the intermediate stage of an acrylate ester. It is also used as an ingredient in products such as adhesives, which may contain up to 10 % acrylic acid, paints, binding agents and printing inks. Occupational exposure may arise during the production and processing of acrylic acid in the chemical industry and in the manufacture of products containing acrylic acid. Exposure may also occur during the use of adhesives and other products containing acrylic acid, during the decomposition of photoresistant materials with UV light (e.g. during the production of integrated circuits) and during the flame removal of paints.

1.1. Methods of exposure monitoring and analysis

1.1.1. Concentrations of acrylic acid in air

OSHA have published a partially validated method (OSHA Method PV2005, July 1996), which at a flowrate of 0.1 l/min during 4 hours onto Anasorb 708 tubes detects 0.2 µg.

The method proposed by Zanella *et al* (1999) also using diffusion denuder tubes with a subsequent HPLC technique reports detection of 2.9 µg/m^3 for a sample of 15 l (30 min at 0.5 l/min), which corresponds to an absolute detectable amount of 0.0435 µg.

Since a 1-min sampling time detects 5.9 µg (at 0.1 l/min - OSHA) or 29.4 µg (at 0.5 l/min - Zanella) a STEL of 20 ppm, which is restricted to 1 min, can be controlled. For practical reasons (very short sampling time), the use of a direct reading instrument (such as a photo ionisation detector) to assess the peak exposures to acrylic acid is recommended.

At the recommended OEL and STEL, no difficulties in measurement are expected.

1.1.2. Biological monitoring

There were no published methods of biological monitoring.

2. Health significance

2.1. Toxicokinetics

2.1.1. Absorption

Acrylic acid is almost completely absorbed via the inhalation and oral route. Dermal uptake was shown to be up to 26 %. The calculated nasal tissue dose for mice after inhalation of acrylic acid was 88 % higher than for rats (Barrow 1984). In rats, 97 % of the acrylic acid was deposited in the upper airways, indicating almost complete absorption after inhalation (Morris and Frederick 1995). Under similar exposure conditions, the uptake of acrylic acid by the human olfactory epithelium was predicted by a computational fluid dynamics and physiologically based pharmacokinetic dosimetry model to be 2- to 3-fold lower than that of rats (Frederick *et al* 1998, cited

in EU RAR 2002). Further refinements of the model predicted similar local deposition of acrylic acid in the olfactory epithelium of rats and humans at 4 and 25 ppm. The model used 43 parameters concerning respiration, tissue and mucus diffusivity, nasal surface and lumen, epithelia and mucus thickness, partition coefficients and gas phase mass transfer, amongst others, for rat and man (Andersen *et al* 2000, Frederick *et al* 2001). Some doubts were raised concerning the validity of the model, because important parameters (gas phase diffusivity, diffusivity in mucus, diffusivity in squamous epithelium and tissue diffusivity) were not measured but modelled (DFG 2006).

A simplified approach based on an even distribution of acrylic acid over the surface of the nasal epithelium and with the parameters listed in Table 1 led to the conclusion that the dose of the olfactory epithelium is about 1.6-fold higher in humans than in rats, also if the increased respiratory rate at the workplace is considered. However, as acrylic acid is highly water soluble it is mostly deposited anteriorly. In contrast to rats where a great part of the olfactory epithelium is located near the port of entry, the human olfactory epithelium is relatively small (Table 1) and located posteriorly, which implies that the dose of the olfactory epithelium in humans probably is not higher than that in rats (DFG 2006) and about 50 % lower than in mice (Barrow 1984).

Table 1. Dose and model parameters for the olfactory epithelium of rat and man at an exposure to 25 ppm acrylic acid (75 µg/l) (DFG 2006).

Parameter	Rat	Human
Minute volume	0.175 l/min, 250 g rat	20.8 l/min (10 m^3/8 h)
Acrylic acid inhaled/min	13.1 µg	1 563 µg
Surface of nasal epithelium	13.79 cm^2	245.9 cm^2
Surface of olfactory epithelium	6.72 cm^2	13.2 cm^2
Deposition in nose	97 %	ca. 50 %
Mean dose rate/cm^2 nasal epithelium (assuming constant nasal deposition)	0.92 µg/cm^2/min (13.1 µg/min/13.79 cm^2 × 0.97)	3.18 µg/cm^2/min (1 563 µg/min/245.9 cm^2 × 0.50)
Airstream over olfactory epithelium	ca. 15 %	ca. 7 %
Relative dose rate of acrylic acid for olfactory epithelium	1	1.6 (3.18/0.92 x 7 %/15 %)

The dermal absorption of acrylic acid is strongly dependent on the vehicle and pH of the solution. After dermal (occlusive) application of 5 mg acrylic acid/kg body weight to rats, cumulative absorption after 24 hours was dependent on the vehicle, with 22 % for acetone, 19 % for phosphate buffer pH 6 and 9 % for phosphate buffer pH 7.4. Under the condition of open application, 73 % of the acrylic acid (4 % solution) evaporated within 3 days, 21 % was absorbed and 6 % remained in the skin (EU RAR 2002).

Acrylic acid is rapidly and efficiently absorbed following ingestion (EU RAR).

Distribution

In a radiolabel study, Kutzman *et al* (1982) exposed rats (nose-only) to acrylic acid vapour for 1 minute. Ninety seconds after exposure, 18.3 % of the delivered dose remained in the rats. Approximately 28.0 % of this radioactivity was associated with the snout and 42.9 % of the radioactivity in the head. After 65 min, the activity in the snout was reduced to 8.1 %, and the radioactivity retained in liver and fat had increased markedly. Kutzman *et al* also administered an aqueous solution of radio-labelled acrylic acid by oral gavage to rats. The acrylic acid was rapidly absorbed and the radiolabel mainly expired as carbon dioxide within an hour of administration. The relative retention after 65 minutes was greatest in the liver. Approximately 6 % of the radiolabel was eliminated in the urine within 65 minutes. The authors used the short-lived ^{11}C as radiolabel, casting some doubt on the validity of the data.

Metabolism and excretion

Acrylic acid is rapidly metabolised to carbon dioxide which is formed via acrylyl-CoA by the non-vitamin-B12-dependent pathway of mammalian propionate catabolism (EU RAR). High doses of acrylic acid leading to tissue damage cause the formation of small amounts of mercapturic derivatives. About 80 % of an ingested dose of acrylic acid is exhaled as carbon dioxide within 24 hours. The kidneys and liver may be major sites of acrylic acid metabolism (Black *et al* 1993). A small proportion of absorbed acrylic acid is eliminated as urinary metabolites. The major urinary metabolite is 3-hydroxypropionic acid (EU RAR). Epoxidised metabolites of acrylic acid were not detected (EU RAR).

2.2. Acute toxicity

Pure acrylic acid is a very reactive chemical substance that exhibits severe corrosive properties when it comes into direct contact with biological material. The toxicity of acrylic acid is strongly dependent on its concentration, both in air and in the aqueous solution.

2.2.1. Human data

There were no reports of acute acrylic acid poisoning in humans (EU RAR, IPCS 1997).

2.2.2. Animal data

The EU RAR (2002) cites an LC_{50} of 3 600 mg/m^3 (4-hour exposure) in a poorly reported study in rats. Exposure to high concentrations of acrylic acid is reported to produce irritation of the nasal mucosa, the upper and lower airways and the eyes, corneal opacities and dermal toxicity (IPCS 1997). Silver *et al* (1981) reported a dose-dependent decrease in respiratory frequency and minute volume in rats exposed for 1 hour to 300, 900 and 1 500 mg/m^3 (100, 300, 500 ppm). The reduction was approximately 10–15 % at 100 ppm.

In rats, reported oral LD_{50} values range from 140 to 1 400 mg/kg body weight.

Acute dermal toxicity is dominated by severe local corrosion. The influence on uptake and toxicity due to the corrosivity of acrylic acid has to be considered. Dermal LD_{50} values of 300 and 640 mg/kg body weight in rabbits have been reported for undiluted acrylic acid (EU RAR). An LD_{50} of 1 350 mg/kg body weight was reported for male rats in a study with a 10 % aqueous solution of acrylic acid (pH of 2.5) in which it was believed that effects could be specifically attributed to acrylic acid *per se*, rather than the corrosive effects of acidity (EU RAR).

2.3. Irritation

2.3.1. Human data

The EU RAR cites three cases of accidental occupational exposure to acrylic acid that resulted in two admissions to hospital for skin corrosion and one admission for respiratory irritation.

The lateralisation threshold, indicative of a sensory irritation via trigeminal stimulation, was determined in 72 male and female persons. The median was 31 ppm (5-percentile 13 ppm) (van Thriel *et al* 2006).The results cannot be extrapolated to an 8-hour exposure, but are useful for setting a STEL. However, the irritation effect observed during a few minutes does not necessarily mean that longer exposure times result in increasing sensitivities to this reaction. This is supported by the literature review by Shusterman *et al* (2006) about time effects in human sensory irritation, which describes non-linearities in the time effects. They either showed a plateau or there was a reversal of the effects over time.

2.3.2. Animal data

Animal tests have demonstrated that acrylic acid is severely irritating to the respiratory tract. Majka *et al* (1974) reported severe irritation of the bronchial mucosa, exudate into the bronchial lumen, macrophages in the vesicle lumen and focal intraparenchymal irritation of the lungs in rabbits exposed to 2 970 mg/m^3.

Acrylic acid is severely corrosive to the skin, and exposure of rabbit skin to a 10 % solution of acrylic acid caused skin irritation after 5 minutes of exposure (unpublished report cited by EU RAR).

Acrylic acid causes severe damage to the eye in animals with irreversible corneal opacity and scarring of the eyelid. The serious damage to eyes caused by acrylic acid is not due to the acidic properties of this chemical, because in another study, neutralising the acid still led to irreversible corneal opacity (EU RAR).

Signs of local irritation (nasal discharge) were seen after repeated exposure to 300 and 1 500 ppm acrylic acid (Gage et al 1970; see Section 2.5.5).

2.4. Sensitisation

2.4.1. Human data

Workers exposed to acrylic acid can develop contact dermatitis but there is no strong evidence of skin sensitisation. There are two case reports of individuals displaying a positive response to acrylic acid in patch tests (Fowler 1990, Daecke *et al* 1993). Negative results were found in 6 other patch tested workers (Conde-Salazar *et al* 1988) and regular testing of more than 450 production workers during the 1990s failed to find evidence of skin sensitisation (EU RAR). It is possible that the reported cases of sensitisation to acrylic acid were actually due to sensitisation to an impurity of acrylic acid (EU RAR).

Respiratory sensitisation has not been reported.

2.4.2. Animal data

Pure acrylic acid has not shown skin sensitising properties in animal sensitisation tests (EU RAR). Positive results in older studies may have been due to 2,3-di(acryloxy)

propionic acid, a strongly sensitising impurity (DFG 2006) nowadays not contained anymore in commercial samples (EU RAR). There were no data for inhalation.

2.5. Repeated dose toxicity

2.5.1. Human data

No human data were available concerning chronic health effects of acrylic acid exposure, despite the widespread industrial use. A study of occupational exposure to chemicals during the production of acrylic acid by Tucek *et al* (2002) did not specifically measure exposure to acrylic acid or investigate the effects of acrylic acid exposure. Workers were exposed to a wide range of other chemicals but measured concentrations of chemicals in the working atmosphere were generally low and no health effects were found that could be attributed solely to acrylic acid.

2.5.2. Animal data

Inhalation

In a 90-day OECD-guideline compliant study, rats and mice were exposed for 6 hours each day to concentrations of 0, 15, 75 or 225 mg/m^3 (0, 5, 25 or 75 ppm) acrylic acid (Miller *et al* 1981). There was a reduction in the mean body weight gain of female mice in the 75- and 225-mg/m^3 (25 and 75 ppm) exposure groups. There were no significant differences in organ weights, clinical chemistry parameters, urine analysis parameters or gross pathology that could clearly be related to exposure. Slight focal degeneration of the nasal olfactory epithelium was observed in rats at 225 mg/m^3 (75 ppm), but no effects were seen at 15 or 75 mg/m^3 (5 or 25 ppm). In mice, there was a clear exposure-related increase in focal degeneration of the olfactory nasal epithelium with lesions being found in all animals in the 225-mg/m^3 (75 ppm) exposure group. The lesions were described as very slight at 15 mg/m^3 (5 ppm; 1/10 males and 4/10 females).

A brief summary is given below of the other studies described in the EU RAR (2002) that were not OECD-guideline compliant.

In a 2-week inhalation study, Miller *et al* (1981) exposed F-344 rats and B6C3F1 mice (five of each sex per group) to concentrations of 0, 75, 225 or 675 mg/m^3 (0, 25, 75 or 225 ppm) acrylic acid vapour for 6 hours each day, 5 days per week. Significant decreases in body weight gain were seen in animals exposed to 675 mg/m^3 (225 ppm) together with a reduction of adipose tissue in females exposed to this concentration. Lesions of the nasal mucosa and focal squamous metaplasia of nasal tissue were observed in rats at 675 mg/m^3 (225 ppm). In mice, lesions of the nasal mucosa were observed in 2 out of 5 males and 4 of the 5 females at 75 mg/m^3 (25 ppm) and in all mice exposed to 675 mg/m^3 (225 ppm).

Female B6C3F1 mice (15 per group) were exposed to acrylic acid vapour concentrations of 0, 5 or 25 ppm (6 or 22 hours/day) or to 25 ppm (4.4 hours/day) for 15 days. At the end of the exposure, 10 animals were sacrificed. The other 5 animals were sacrificed after a 6-week recovery period. Clinical parameters were recorded. Histopathologically, only the nasal cavity was examined. Exposure to 5 ppm for 6 hours/day did not result in effects. Concentrations of 5 ppm for 22 hours/day as well as 25 ppm for 4.4 hours/day resulted in concentration- and time-dependent changes in the olfactory epithelium with atrophy, basal cell hypertrophy, necrosis and degeneration of the Bowman gland. The findings after the 22-hour/day exposure to 5 ppm as well as after the 4.4- and 6-hour exposures to 25 ppm were fully reversible after 6 weeks. However, exposure to 25 ppm for 22 hours/day resulted in limited

regions of olfactory epithelium being replaced with respiratory-like epithelium (respiratory metaplasia) (Lomax *et al* 1994, Rohm and Haas Company 1994).

Gage (1970) found no effects in rats exposed to 240 mg/m^3 (80 ppm) acrylic acid vapour, 6 hours each day, 5 days per week for 4 weeks, whereas rats exposed at 900 mg/m^3 (300 ppm) showed signs of nasal irritation, lethargy and reduced body weight gain. Four exposures to 4 500 mg/m^3 (1 500 ppm) for 6 hours, resulted in nasal discharge, lethargy, retarded weight gain and kidney congestion.

Barrow (1986) found a reduction in respiratory function in rats and mice after exposure to 225 mg/m^3 (75 ppm) acrylic acid vapour for 6 hours per day for 5 days. The cell proliferation in the olfactory epithelium of these animals was increased 17-fold in mice as compared to only 4-fold in rats (Swenberg *et al* 1987).

Dermal exposure

One study cited by the EU RAR observed no irritant effects in mice following long term application of 1% acrylic acid in acetone. Another study reported that the incidence and severity of skin irritation was greater following exposure to 4 % than to 1 % in acetone, implying that some effects were observed in the lower dose group.

Oral administration

The EU RAR (2002) summarises data from several studies. The NOAEL in two 90-day studies were 40 mg/kg/day in male rats and 83 mg/kg/day female rats. An oral dose of 150 mg/kg/day has been reported to cause severe damage to the mucosa of the stomach and higher doses were associated with premature deaths and tubular degeneration/necrosis in the kidneys.

Summary on repeated dose administration

The toxic effects of acrylic acid are dominated by its local irritation. Prolonged inhalation of acrylic acid adversely affects the olfactory epithelium with a LOAEL of 15 mg/m^3 (5 ppm) in mice. For rats, a local NOAEL of 25 ppm was obtained.

2.6. Genotoxicity

Bacterial mutation studies have given negative results, whereas tests with mammalian cells yielded mixed results. The EU RAR (2002) concluded that the mutagenic potential of acrylic acid was limited to clastogenicity. Most *in vivo* assays gave negative results, and taking account of data available for structurally related acrylic compounds, the EU RAR (2002) considered it unlikely that acrylic acid is mutagenic *in vivo*.

2.7. Carcinogenicity

There were no human data, and no inhalation experiments have been undertaken in animals. Acrylic acid esters are rapidly metabolised in the nasal tissues to acrylic acid and the corresponding alcohol, therefore inhalation studies with acrylic esters can be used to assess the local carcinogenic potential of the acid. *n*-Butyl, ethyl and methyl acrylate were not carcinogenic in inhalation studies with rats and mice (DFG 2006). In rats exposed to doses equivalent to a mean dose of 9, 31 or 88 mg/kg/day acrylic acid in drinking water for 26 months (males) or 28 months (females), no treatment-related clinical, haematological or histopathological changes were detected in comparison with the controls other than a slightly reduced water consumption in high-dose males (Hellwig 1993). No skin tumours or skin irritation were observed in two lifetime studies in mice receiving repeated dermal applications of acrylic acid (EU RAR).

Overall, acrylic acid did not cause cancer in animals following oral or dermal administration and it can be expected from the results with esters that the acid is also not carcinogenic after inhalation.

2.8. Reproductive toxicity

Inhalation

Rats exposed to 0, 120, 360 and 1 080 mg/m^3 (0, 40, 120 and 360 ppm) during days 6–15 of gestation (6 hours/day) showed a concentration-related reduction in food and water intake leading to a reduction in body weight gain from the lowest concentration. Irritation of the respiratory tract and eyes was observed in the highest concentration group. No effects on reproductive performance including any evidence of developmental toxicity were observed. The NOAEL for developmental toxicity was 360 ppm with minimal maternal toxicity at 40 ppm (Klimisch and Hellwig 1991).

Rabbits exposed to concentrations of 0, 75, 225 and 675 mg/m^3 (0, 25, 75 and 225 ppm) during days 6–18 of gestation (6 hours/day whole body) showed no treatment related effects on gestational parameters. Concentration-related clinical signs (perinasal/perioral wetness, nasal congestion, reduced body weight gain and food consumption) were seen in the 225- and 675-mg/m^3 (75 and 225 ppm) groups. The NOAEL for developmental toxicity was 225 ppm (Bushy Run Research Center 1993, Neeper-Bradley *et al* 1997).

Offspring of rats exposed for 6 hours each day, during days 6–20 of gestation, to 150, 300, 600 or 900 mg/m^3 (50, 100, 200 or 300 ppm) acrylic acid showed signs of developmental toxicity (reduced foetal body weight) at 300 ppm acrylic acid, in the presence of overt signs of maternal toxicity (reduced body weight gain). The NOAEL for developmental toxicity was 200 ppm (Saillenfait *et al* 1999).

Oral

No effects on fertility were observed in oral reproductive toxicity studies.

DePass *et al* (1983) exposed male and female rats to 0, 83, 250 or 750 mg/kg/day for 13 weeks (before mating and throughout gestation and lactation). Each male was mated with 2 females. Dose-related reductions in food and water consumption and in body weight gain were observed. A non-significant reduction in the fertility of males and females, number of live pups and number of pups weaned was seen in the 750-mg/kg/day group.

In an OECD-guideline compliant 2-generation study (Hellwig *et al* 1997), a NOAEL of 460 mg/kg/day was derived for effects on fertility in rats exposed to 0, 500, 2 500 and 5 000 ppm in drinking water (53, 240 and 460 mg/kg body weight, resp.). Toxicity in the parent animals was expressed as a reduction in food and drinking water consumption accompanied by reduced body weights and reduced body weight gain. A parental NOAEL for general toxicity of 240 mg/kg/day was reported in the F_0 generation, but for the F_1 generation, the NOAEL was 53 mg/kg/day (EU RAR).

Conclusions

Acrylic acid is not considered to be a reproductive toxicant. The NOAEL for developmental toxicity was 200 ppm in rats and 225 ppm in rabbits. The LOAEL for rats was 300 ppm and was associated with maternal toxicity. For toxicity to fertility, the NOAEL was 460 mg/kg body weight in rats.

3. Recommendation

Acrylic acid is rapidly absorbed following inhalation, skin contact or ingestion, and is mainly metabolised by oxidative pathways to carbon dioxide, which is eliminated in exhaled air. Acrylic acid is severely irritating to the respiratory tract, severely corrosive to the skin and causes severe damage to the eyes. Despite the widespread industrial use of acrylic acid, there have been no studies of the effects of workplace exposure. There is no evidence that acrylic acid is likely to cause cancer. The mutagenic potential of acrylic acid in *in vitro* assays appears to be limited to clastogenicity and it is unlikely that acrylic acid is mutagenic *in vivo* (EU RAR).

The toxic effects of acrylic acid are dominated by local irritation. The NOAEL for effects on the olfactory epithelium was 75 mg/m^3 (25 ppm) in rats (Miller *et al* 1981), but not established for mice (Lomax *et al* 1994). The LOAEL in mice was 15 mg/m^3 (5 ppm). In female mice, the NOAEL for systemic toxicity was 15 mg/m^3 (5 ppm) and the LOAEL 75 mg/m^3 (25 ppm) (Miller *et al* 1981).

The NOAEL for developmental toxicity was 200 ppm in rats and 225 ppm in rabbits (Saillenfait *et al* 1999).

As has been argued in the EU RAR, from comparison of the 2- and 13-week studies with acrylic acid and studies with methyl acrylate and methyl methacrylate, the "nasal irritation threshold for acrylic acid will not substantially change when extrapolation is made from experimentally-tested subchronic exposure to chronic exposure". There are clear species differences in the deposition rate between rats and mice. Calculations suggest that humans have approximately the same deposition rate for acrylic acid as rats. Therefore, the rat, and not the mouse, is the most appropriate model for extrapolation to humans. The starting point for the calculation of the OEL is therefore the subchronic NOAEL of 25 ppm in rats. Acrylic acid does not have to be metabolised to cause irritation, so interindividual differences in sensory irritation thresholds should be small. Therefore, an 8-hour TWA of 10 ppm is considered appropriate to protect workers from histological changes and irritation.

The recommended OEL should not be exceeded significantly as irritation is to be expected in a significant number of workers, which is supported by the study of van Thriel *et al* (2006) who reported a lateralisation threshold (beginning of irritation in volunteers) of 30 ppm. Therefore, a STEL for acrylic acid of 20 ppm is proposed, which should be limited to 1 min.

No measurement difficulties are foreseen at the recommended OEL and the STEL of 1 min.

Due to the corrosive properties, routine dermal exposure to undiluted acrylic acid is unlikely. From the systemic oral NOAEL of 40 mg/kg body weight and the reported dermal uptake of 26 % from 1 % solutions (non-irritant) in rats, the corresponding amount of a 1 % solution can be calculated as 1 120 g for a 70-kg human. This amount is very large so that prolonged exposure to non-irritant solutions should not lead to systemic intoxications.

A "skin" notation is therefore not warranted.

There is no evidence that pure acrylic acid can cause respiratory or skin sensitisation, thus a "sensitiser" notation is not warranted.

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