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REPUBLICA DE CHILE
COMISION NACIONAL DEL MEDIO AMBIENTE
ASR/PMC

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APRUEBA ANTEPROYECTO DE REVISIÓN DE LA NORMA DE CALIDAD PRIMARIA PARA MATERIAL PARTICULADO RESPIRABLE MP10.

SANTIAGO, 22 JUN **DE 1999**

EXENTA N° 867

VISTOS:

Los acuerdos del Consejo Directivo de CONAMA N°s 80/98 de 7 de agosto de 1998 que determina la revisión del Decreto Supremo N° 59 del Ministerio Secretaría General de la Presidencia, y 93 /99 de 28 de enero de 1999 que aprueba la creación del Comité Operativo para la revisión de la Norma y N° 99/99 de 26 de marzo de 1999, que aprueba cuarto programa priorizado de Normas; La Resolución Exenta N° 0060 de 2 de febrero de 1999 de la Dirección Ejecutiva de CONAMA; La Resolución Exenta N° 129 de 16 de febrero de 1999, que da inicio a la elaboración del anteproyecto, publicada en el Diario Oficial el 22 de Febrero de 1999; El Decreto Supremo N° 59 de 16 de marzo de 1998 del Ministerio Secretaría General de la Presidencia; El Decreto Supremo N° 93 de 15 de mayo de 1995 del Ministerio Secretaría General de la Presidencia; Las facultades que me confiere la ley 19.300 sobre Bases del Medio Ambiente; y la Resolución N° 520 de 1996 de la Contraloría General de la República.

CONSIDERANDO:

Que el Reglamento que fija el Procedimiento para la Dictación de Normas de Calidad Ambiental y de Emisión, Decreto Supremo N°93 de 1995 del Ministerio Secretaría General de la Presidencia, dispone en su artículo 17 que, elaborado el anteproyecto de norma, el Director Ejecutivo de la Comisión Nacional del Medio Ambiente (CONAMA) dictará la resolución que lo apruebe y lo someta a consulta.

RESUELVO:

1.- Apruébase el anteproyecto de Revisión de la Norma de Calidad Primaria para Material Particulado Respirable MP10, contenida en el Decreto Supremo N° 59 de 1998 del Ministerio Secretaría General de la Presidencia, cuyo texto es del siguiente tenor:

I. FUNDAMENTOS

- La influencia que tiene la contaminación atmosférica sobre el deterioro de la salud humana ha sido ampliamente reconocida por la literatura científica a nivel internacional, en particular respecto de los aspectos agudos durante episodios críticos de contaminación pero también se muestran evidencias respecto de los efectos crónicos y acumulativos.

- De manera general se ha podido establecer a través de diversos estudios, que los efectos de la contaminación atmosférica sobre la salud humana se pueden clasificar en efectos agudos, acumulativos y crónicos dependiendo de la concentración y del tipo de

contaminante, del tiempo y grado de exposición y de los factores personales que puedan influir en la acción deletérea sobre la salud, de estos contaminantes.

- Que, establecer las evidencias respecto de los efectos de la contaminación atmosférica sobre la salud de las personas es una tarea compleja por diversas razones, tales como:

- Distintos factores y/o agentes tóxicos que actúan simultáneamente sobre el mismo órgano cuya respuesta fisiopatológica es la misma. Por lo tanto, es difícil poder identificar la acción en forma aislada.
- Determinados factores y/o agentes que se potencian mutuamente, resultando su efecto conjunto mayor que la sumatoria de los efectos específicos de cada uno. Este sinergismo se ha descrito entre las partículas respirables (PM_{10}) y el anhídrido sulfuroso (SO_2) y también entre las partículas respirables y el frío.
- Se deben considerar también las variables que influyen en los efectos de los contaminantes y que tienden a confundir el real impacto de los últimos, como por ejemplo las variaciones de temperatura, humedad, el hábito de fumar, el fumador pasivo, la presencia de patologías preexistentes, la vivienda, la ocupación, la clase social, las características biológicas y culturales de las personas o grupos considerados.
- Las propiedades físicas y químicas del material particulado pueden variar sustancialmente debido a factores tales como los tipos de fuentes de emisión, las condiciones meteorológicas y las condiciones geográficas entre otros.
- Aún cuando se han podido establecer asociaciones entre la concentración de material particulado respirable (PM_{10}) y el incremento de enfermedades y muerte prematura tanto por efectos agudos como crónicos y acumulativos, no se conoce exactamente él o los mecanismos por el cual las partículas pueden causar dichos daños.

- Con todas las dificultades derivadas de la necesidad de abordar, en su complejidad, los problemas de salud de las personas en que concurren diversos factores etiológicos, condicionantes y/o determinantes en el proceso de la multicausalidad de las enfermedades, en lo que dice relación a los efectos crónicos y acumulativos de la contaminación atmosférica sobre la salud, la comunidad científica internacional ha venido acumulando evidencias durante el último decenio que permiten avanzar en dar respuesta a una serie de preocupaciones sobre los efectos del material particulado respirable PM_{10} y $PM_{2,5}$ como son:

  - Las muertes prematuras en adultos mayores, o en personas que tienen enfermedades cardíacas y pulmonares.
  - La agravación de enfermedades, lo que se traduce en aumento de las hospitalizaciones y de las atenciones de urgencia.
  - El deterioro de la función pulmonar y el aumento de síntomas respiratorios en niños y personas que padecen asma bronquial.
  - El aumento del ausentismo escolar y laboral.
  - Los cambios en la estructura del pulmón y en los mecanismos naturales de defensa.
  - Los efectos cancerígenos u oncogénicos.
  - Los efectos sobre el sistema nervioso, entre otros.

- Las evidencias experimentales y poblacionales en seres humanos han demostrado una asociación entre contaminación ambiental, sea por gases irritantes, como por material particulado, y daño respiratorio. Esta asociación es significativamente mayor en grupos de riesgo tales como sujetos asmáticos y los portadores de Enfermedades Bronquiales Obstructivas y, si bien no es posible por razones éticas demostrar una causalidad inequívoca de la contaminación sobre estas enfermedades, no hay dudas razonables que impidan afirmar que la contaminación atmosférica las agrava, causando no sólo un

deterioro en su calidad de vida, sino también un importante impacto económico en las comunidades afectadas por esta situación de deterioro ambiental. 000300

- Es necesario reconocer que los efectos sobre la salud de las personas varían en función de la composición del material particulado, aumentando el riesgo de ocurrencia de enfermedades crónicas y cánceres a menor diámetro de las partículas y mayor toxicidad del material particulado.

- La División de Salud Ambiental del Ministerio de Salud en su documento "Salud y Contaminación Atmosférica" de 31 de marzo de 1999, indica lo siguiente:

> "Además, la División de Salud Ambiente del Ministerio de Salud ha estudiado el efecto de las concentraciones de contaminantes en la Región Metropolitana sobre la salud, utilizando los registros de la Red de Vigilancia de calidad del aire y las consultas totales y por Infecciones Respiratorias Agudas recolectados por la red de Centros Centinelas de Consultorios de Atención Primaria de la Región Metropolitana, creados y desarrollados por el programa "Campaña de Invierno" del Ministerio. Esto con el propósito de establecer la cuantía en exceso de enfermos que prevendría la extensión, por 24 horas, de los períodos de pre-emergencia y emergencia.
>
> También para informar del proceso del establecimiento de las normas primarias de calidad del aire en relación al PM10 promedio anual y el PM 2.5, que conforman con los IBM, el mandato del Comité de Ministro de la CONAMA, de la reunión regular de agosto de 1998, en relación al tema del Plan de Descontaminación de la Región Metropolitana.
>
> Los antecedentes de las investigaciones bibliográficas y el análisis de regresión múltiple y de comparación de dos y tres días de niveles altos de contaminación por partículas y su efecto en salud, son aportes nuevos sobre el tema, que han demostrado:
>
> a) En la Región Metropolitana existe el efecto acumulativo de daño respiratorio significativo en población de niños sanos.
> b) Los datos recientes (1998) de la Región Metropolitana indican que se produce un efecto agudo, en que por cada 10% de aumento de nivel de **contaminación promedio** diario por PM10, se asocia a un aumento en las IRAs Bajas diarias en un 0,6%, y, a largo plazo, se produce un efecto acumulado de morbilidad respiratoria por la contaminación atmosférica, de tal manera que por cada 10% de aumento de la contaminación por PM10, las IRAs Bajas aumentan 7,6%.
> c) Como efecto agudo de la exposición a **concentraciones máximas diarias** de PM10, por cada 10% de aumento, se observa igualmente un 0,6% de aumento de IRAs bajas y en efecto acumulado, por cada 10% de aumento del nivel de contaminación, las IRAs Bajas aumentan diariamente en 6%.
> d) Se ha establecido que la proporción de consultas por IRAs Bajas en relación al total de consultas pediátricas es un Indicador Biomédico adecuado para estimar los efectos de la contaminación de PM10 sobre la salud de las personas, llegándose a establecer que un valor de un 40% de este indicador representa el límite inferior a partir del cual se requiere la adopción de medidas adicionales para proteger la salud de la población.
> e) En cuanto a la proporción de Bronconeumonías como IBM, su uso no ha sido demostrado como eficaz en el presente estudio.

f) Recientemente, Ostro y colab. Utilizando la misma información de 2 años de los sitios centinelas ha mostrado que en niños menores de 2 años un cambio de 50UG/m3 de PM10 se asocia con un aumento de 4 a 12% de los síntomas de IRA baja; en niños mayores de 3 años, este aumento varía entre 3 y 9%, siendo de igual magnitud ante aumentos del Ozono en 50 ppb. En la población de mayor edad, el aumento de PM10 se asocia a un aumentos de IRA alta y el aumento de Ozono se asocia a aumento de IRAs altas y bajas en este grupo etario.

g) La comparación de tres días sucesivos de altas concentraciones de PM10 y su rezago y su efecto sobre la morbilidad en relación a dos días sucesivos de alta contaminación (mayor de 150 µg/m3 de PM10) y su rezago, establece una diferencia de 3,3 puntos de IRAs Bajas. Esta diferencia de 3,3 puntos si se aplica a toda la población de menores de 15 años de la Región Metropolitana, establece un riesgo potencial probabilístico de un número de cerca de 40.000 jóvenes menores de 15 años de verse afectados por síntomas de IRA Baja. Si este riesgo se aplica a los menores de 15 años que son atendidos en el sector público, se trata de miles de casos de niños de verse afectados por IRA Baja, sin contar con un aumento del mismo orden en otras poblaciones de alto riesgo como la de adultos mayores. Todo esto implica un costo social de más de cuatro millones de dólares por cada evento, en el primer caso y de 500.000 dólares en el segundo. Estos riesgos podrían prevenirse, si se implementa el uso de los IBM y adopta el criterio de 40%, detectado por los Centros Centinela del Ministerio de Salud, como límite a partir del cual es necesario prolongar los períodos de emergencia durante 24 horas.

Finalmente, se observa claramente en las curvas obtenidas en la Región Metropolitana que relacionan la concentración promedio de PM10 con el Indicador IRAs Bajas, se aprecia que los valores de este Indicador iguales o mayores a 40% se presentan en forma sostenida a partir de concentraciones superiores a 100 ug/m3 y que la probabilidad de que se observen estos valores superiores a 40% es relativamente alta (38%) en el rango de concentraciones promedio semanales que van en un rango de 150 a 250 ug/m3."

II. OBJETIVOS DE LA NORMA

Proteger la salud de la población del país de aquellos efectos agudos, crónicos y acumulativos generados por niveles de concentración de material particulado respirable MP10 en el aire en el corto y el largo plazo.

Los Indicadores Biomédicos propuestos permitirán prevenir que un grupo importante de niños que están con un leve proceso de hiperreactividad, puedan desarrollar una obstrucción bronquial al tercer día de exposición a partículas y otros contaminantes.

Evitar sobresaturar los recursos de salud al incorporarse niños con casos de obstrucción bronquial que suman un tercer día de alta exposición.

III. DEFINICIONES

a) *Efectos agudos*: aquellos producidos por la exposición a elevadas concentraciones de contaminantes por cortos períodos de tiempo. Estos incluyen irritación de mucosas, faringitis, laringitis, neumonias, bronquitis, entre otros.

b) *Efectos crónicos*: aquellos producidos por la acción de concentraciones variables de contaminantes por largos períodos. Se manifiestan por un aumento de la incidencia

y la gravedad de asma bronquial, bronquitis obstructiva crónica, enfisema pulmonar y cánceres. 000902

c) *Efectos acumulativos*: aquellos que aparecen debido a la exposición prolongada a contaminantes y pueden expresarse después de años de exposición, independientemente de si la exposición se mantienen o ha cesado.

d) *Material particulado respirable* MP10: material particulado con diámetro aerodinámico menor o igual que 10 micrones.

e) *Concentración:* El valor promedio temporal detectado en el aire expresado en microgramos por metro cúbico normal ($\mu g/m^3N$) de material particulado respirable.

f) *Concentración de 24 horas:* corresponde a la media aritmética de los valores efectivamente medidos de concentración en cada estación monitora en 24 horas consecutivas. En caso de utilizarse monitores con resolución temporal inferior a 24 horas, o de pérdida parcial de información horaria, el número de valores a considerar en el cálculo de la media será mayor o igual que el equivalente a 18 horas.

g) *Concentración mensual*: corresponde a la media aritmética de los valores efectivamente medidos de concentración de 24 horas en cada estación monitora en un mes calendario. Solo se considerará como valor de concentración mensual válido aquel que contemple mediciones de al menos el 75% de los valores programados para el mes de acuerdo a la periodicidad de monitoreo previamente definida.

h) *Concentración anual*: corresponde a la media aritmética de los valores efectivamente medidos de concentración mensual en cada estación monitora en un año calendario. Solo se considerará como valor de concentración anual válido aquel que contemple mediciones de al menos 11 de los meses del año calendario. En el caso que se disponga valores para más de 8 meses, pero menos de 11 meses del año calendario, se considerarán como valores mensuales en aquellos meses faltantes, hasta completar los 11 meses necesarios para el cálculo, el promedio entre el valor asociado al mes que precede y el mes que sigue de aquel que se hubiese obtenido información válida de concentración mensual. Si se dispone de valores para 8 o menos meses, no se podrá calcular un valor de concentración anual para la estación de monitoreo correspondiente.

i) *Estación de monitoreo de material particulado respirable MP10 con representatividad poblacional (EMRP):* una estación de monitoreo podrá clasificarse como *EMRP* si se cumplen simultáneamente los siguientes criterios: i) que exista al menos un área edificada habitada en un radio de 2km, contados desde la ubicación de la estación; ii) que esté colocada a más de 15m de la calle o avenida más cercana, y a más de 50m de la calle o avenida más cercana que tenga un flujo igual o superior a 2500 vehículos/día; iii) que esté colocada a más de 50m de la salida de un sistema de calefacción (que utilice carbón, leña o petróleo equivalente a petróleo-2 o superior) o de otras fuentes fijas similares.

Una *EMRP* tendrá un área de representatividad para la población expuesta consistente en un radio de 2km, contados desde la ubicación de la estación.

j) *Año calendario*: aquel período temporal que se inicia el 1 de enero, y culmina el 31 de diciembre del mismo año.

k) *Indicador Biomédico*: Valor cuantitativamente medible o cualitativamente descriptible que sirve para evaluar una situación determinada y que en este caso represdentan los efectos sobre la salud humana, relacionados con la calidad del

ambiente. Sirve para medir cambios y evaluar acciones. Un tipo de Indicador Biomédico es el porcentaje de consultas nuevas en niños menores de 14 años diagnosticadas como Infecciones Respiratorias Agudas Bajas en relación a las Atenciones pediátricas en niños menores de 14 años.

l) *Consultas nuevas en niños menores de 14 años*: Episodios nuevos de consultas por morbilidad en menores de 14 años en un día de atención del conjunto de centros centinelas, con exclusión de las consultas de control de un mismo menor de 14 años, por un mismo cuadro de morbilidad.

m) *Atenciones pediátricas en niños menores de 14 años*: *E*l número total de consultas ocurridas en un día de atención del conjunto de centros centinelas, en menores de 14 años adscritos a dichos centros, con exclusión de las consultas de control de un mismo menor de 14 años, por un mismo cuadro de morbilidad.

n) *Infección Respiratoria Aguda Baja (IRA baja):* Proceso inflamatorio que afecta a la laringe, tráquea, bronquios o parénquima pulmonar. Los diagnósticos asociados son:

- Sindrome bronquial obstructivo: SBO (sindrome bronquial obstructivo), SBOA (sindrome bronquial obstructivo agudo), SBOR (sindrome bronquial obstructivo recidivante), BOR (bronquitis obstructiva recidivante), AB (asma bronquial), Bronquitis obstructiva, Bronquiolitis;
- Bronconeumonía: BN (bronconeumonia), BRN (bronconeumonia), neumonia, sindrome neumónico, neumopatía aguda
- Otras IRA bajas: laringitis aguda, laringitis obstructiva aguda, laringitis catarral, rinofaringolaringitis, sindrome laringeo, epiglotitis, traqueitis, laringotraqueitis, bronquitis aguda, faringobronquitis, laringobronquitis, traqueobronquitis, bronquitis catarral, peribronquitis, neumonitis, sindrome coqueluchoideo, coqueluche, tos convulsiva, gripe, estado gripal, influenza, atelectasia.

o) *Centros Centinela*: Consultorios de Atención Primaria de Salud, dotados a través del Programa de Infecciones Respiratorias Agudas del Ministerio de Salud de infraestructura, materiales, equipos, y personal adiestrado. Para efectos de la presente norma, la definición y clasificación de un Consultorio de Atención Primaria de Salud como Centro Centinela, la realizará el Servicio de Salud correspondiente, previo informe de autorización del Programa de Infecciones Respiratorias Agudas del Ministerio de Salud, expresado a través de una Resolución Exenta del Ministerio de Salud.

p) *Hospitalización Abreviada*: técnica que se encuentra implementada en todos los Consultorios, Servicios de Atención Primaria de Urgencia (SAPU) y Servicios de Urgencia de la Región Metropolitana, que permite el tratamiento adecuado de los niños obstruidos, no importando lo grave que sea su obstrucción.

q) *Plan operacional de episodios críticos*: Instrumento de gestión ambiental establecido por un plan de descontaminación. Dicho plan operacional se pone en marcha al sobrepasarse los niveles que determinan situaciones de emergencia establecidas en la presente norma de calidad primaria.

IV. DEFINICIÓN DEL NIVEL DE LA NORMA DE CALIDAD PRIMARIA DIARIA PARA MATERIAL PARTICULADO RESPIRABLE MP10

La norma de calidad del aire primaria diaria para el contaminante Material Particulado Respirable MP10, será de ciento veinte microgramos por metro cúbico normal (120 $\mu g/m^3N$) como concentración de 24 horas.

V. DEFINICIÓN DE LOS NIVELES QUE DETERMINAN LAS SITUACIONES DE EMERGENCIA AMBIENTAL PARA MATERIAL PARTICULADO RESPIRABLE MP10

Defínense como niveles que originan situaciones de emergencia ambiental para material particulado respirable MP10, aquellos que de acuerdo al valor calculado para la calidad del aire, en concentración de 24 horas, se encuentre en el respectivo rango señalado en la Tabla siguiente:

Nivel	**Material Particulado Respirable MP10 (ug/m3N) en 24 horas)**
Nivel 1° (Alerta)	165-209
Nivel 2° (Preemergencia)	210-299
Nivel 3°(Emergencia)	300 o superior

VI. DEFINICIÓN DEL NIVEL DE LA NORMA DE CALIDAD PRIMARIA ANUAL PARA MATERIAL PARTICULADO RESPIRABLE MP10

La norma de calidad del aire primaria anual para el contaminante Material Particulado Respirable MP10, será cincuenta microgramos por metro cúbico normal (50 $\mu g/m^3N$) como concentración anual.

Se considerará sobrepasada la norma primaria anual de calidad del aire para material particulado respirable cuando la concentración anual calculada como promedio aritmético de tres años calendario consecutivos en cualquier estación monitora clasificada como *EMRP*, sea mayor o igual a 50 $\mu g/m^3N$.

VII. DEFINICIÓN DEL NIVEL DEL INDICADOR BIOMÉDICO

El Indicador Biomédico Infecciones Respiratorias Agudas bajas (IRA bajas), se considerará sobrepasado cuando su nivel porcentual sea mayor que 40%, nivel calculado como la media aritmética de los resultados diarios obtenidos en los Centros Centinelas y entregados para este indicador, por el Servicio de Salud respectivo en los últimos 7 días.

VIII. METODOLOGÍA DE MEDICIÓN DE LA NORMA

a) Monitoreo directo de material particulado respirable MP10
Para efectos del monitoreo de material particulado respirable MP10, los métodos de medición serán los indicados en el Artículo 7° del Decreto Supremo 59 de 1998 del Ministerio Secretaría General de la Presidencia.

Condiciones que deben considerarse en los datos para ser usados en el cálculo de concentraciones:
Aquellos datos de monitoreo resultantes de eventos no controlables o excepcionales, tales como incendios, erupciones volcánicas, terremotos o temporales de viento, podrán no ser considerados para efectos del cálculo de concentraciones de 24 horas, o concentraciones mensuales, según determine la ocurrencia de estos fenómenos la Comisión Regional del Medio Ambiente respectiva, en base a información objetiva presentada a dicha Comisión.

b) Uso de Indicador Biomédico
Para efectos del cálculo del nivel porcentual del indicador biomédico, se calculará diariamente un valor como el porcentaje de consultas nuevas en niños menores de 14 años diagnosticadas como infección respiratoria aguda baja, sobre las atenciones pediátricas en niños menores de 14 años en el conjunto de los Centros Centinela. Se

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tomarán promedios aritméticos de los valores así calculados de los últimos 7 días, para compararlos con el nivel indicado en el punto VII.

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Condiciones para poner en operación el Sistema de Indicadores Biomédicos:
El Servicio de Salud respectivo, mediante Resolución fundada publicada en el Diario Oficial, deberá aprobar la forma en que se aplicará la metodología de uso del Indicador Biomédico en su territorio jurisdiccional. Esta Resolución tendrá como antecedente un informe de carácter técnico realizado por expertos independientes, nacionales o internacionales, en el que se constate los resultados de la metodología de uso de los Indicadores Biomédicos informados por el Programa de Infecciones Respiratorias Agudas del Ministerio de Salud.

Características del Sistema de Indicadores Biomédicos que debieran indicarse en la Resolución:
- período de uso del indicador en el año calendario;
- centros centinela adscritos;
- descripción del cálculo para obtener el valor final del indicador;
- requerimientos de calidad de los datos y su recopilación para utilizarlos en el cálculo del valor final del indicador;
- hora de comunicación de los resultados;
- estimación y caracterización del error esperado y controles desarrollados para tomar en consideración posibles variaciones del indicador por cambios importantes en el número de consultas totales;
- método utilizado de estandarización de los diagnósticos;
- periodicidad de revisión del Sistema de Indicadores Biomédicos

IX. EFECTO DE LA OPERACIÓN DEL SISTEMA DE INDICADORES BIOMÉDICOS EN LA APLICACIÓN DE MEDIDAS DE PREEMERGENCIA Y EMERGENCIA

En el plan operacional de episodios críticos, si se constatare por los sistemas de monitoreo directo de calidad del aire que se han superado los niveles 2º o 3º (preemergencia o emergencia ambiental respectivamente indicados en la presente norma), y el Indicador Biomédico calculado según se indica en los puntos VII y VIII, se encuentra por sobre el nivel porcentual indicado en el punto VII, las medidas de preemergencia o emergencia declarada serán prolongadas por las siguientes 24 horas, de acuerdo al siguiente detalle:

Evento declarado	Nivel del Indicador Biomédico IRA Baja	Medida a tomar
Nivel 2º (Preemergencia)	Menor o igual que 40%	--
Nivel 2º (Preemergencia)	Mayor que 40%	Extensión por las siguientes 24 horas de las medidas correspondientes al Nivel 2º que indica el Plan operacional de episodios críticos del Plan de Descontaminación o Prevención respectivo
Nivel 3º(Emergencia)	Menor o igual que 40%	--
Nivel 3º(Emergencia)	Mayor que 40%	Extensión por las siguientes 24 horas de las medidas correspondientes al Nivel 3ºque indica el Plan operacional de episodios críticos del Plan de Descontaminación o Prevención respectivo

Si no existe un evento declarado Nivel 2° o 3° (Preemergencia o Emergencia), la sola ocurrencia de valores del Nivel del Indicador Biomédico IRA Baja superiores a 40% no será condición suficiente para que se adopten las medidas señaladas en el cuadro precedente.

X. PLAZOS DE CUMPLIMIENTO DE LA NORMA

Para efectos de declaración de zonas latentes o saturadas en virtud de la norma anual de material particulado respirable MP10, la presente norma comenzará a regir a partir del día 1 de enero del año 2003.

En el caso del sistema de indicadores biomédicos, para efectos de aplicación de las medidas citadas en el punto IX, la presente norma comenzará a regir una vez que se publique, en el Diario Oficial, la Resolución que se indica en el punto VIII.b.

XI. PRIORIZACIÓN DEL ESTABLECIMIENTO DE REDES DE MONITOREO DE CALIDAD DEL AIRE VÁLIDAS PARA EVALUAR EL CUMPLIMIENTO DE LA PRESENTE NORMA

Para efectos de determinar los lugares prioritarios dentro del país en que se deberá instalar redes de monitoreo a fin de evaluar el cumplimiento de la presente norma, deberán considerarse los siguientes antecedentes, en el siguiente orden de importancia:

1. Composición química del material particulado respirable MP10 en términos de su toxicidad, al que está expuesta la población y la cantidad de población urbana expuesta en la zona en estudio;
2. Valores absolutos de concentraciones de material particulado respirable MP10 medido, y tendencias históricas, positivas o negativas, de dichos valores;
3. Presencia de desarrollos industriales significativos que produzcan un impacto por emisiones de material particulado respirable sobre la zona en estudio y volumen del parque automotor existente en la zona en estudio.

XII. FISCALIZACIÓN DE LA NORMA

Corresponderá a los Servicios de Salud del país y, en la Región Metropolitana al Servicio de Salud del Ambiente de la Región Metropolitana, fiscalizar el cumplimiento de las disposiciones de la presente norma.

XIII. VIGENCIA

La presente norma entrará en vigencia el día 1 de enero del año siguiente al de su publicación en el Diario Oficial

2.- Sométase a consulta el presente anteproyecto de Revisión de la Norma de Calidad Primaria para Material Particulado Respirable MP10.

Para tales efectos:

a.- Remítase copia del expediente al Consejo Consultivo de la Comisión Nacional del Medio Ambiente, así como también al Consejo Consultivo de la Comisión Nacional del Medio Ambiente - Región Metropolitana, para que emitan su opinión sobre el anteproyecto de revisión de norma. Tales Consejos dispondrán de 60 días contados desde la recepción de la copia del expediente, para el despacho de su opinión. Las opiniones que se emitan serán fundadas, y en ella se dejará constancia de los votos disidentes.

b.- Dentro del plazo de 60 días, contados desde la publicación en el Diario Oficial, del extracto de la presente resolución, cualquier persona, natural o jurídica, podrá formular observaciones al contenido del anteproyecto de norma. Dichas observaciones deberán formularse por escrito, en la Comisión Regional del Medio Ambiente correspondiente al domicilio del interesado, y deberán ser acompañadas de los antecedentes en los que se fundan, especialmente los de naturaleza técnica, científica, social, económica y jurídica.

Anótese, publíquese en extracto, comuníquese y archívese.

COMISION NACIONAL * DIRECTOR EJECUTIVO *

RODRIGO EGAÑA BARAONA
Director Ejecutivo
CONAMA

22 JUL 1999

Lo que transcribo a Ud.
para su conocimiento
saluda atentamente a U
RODRIGO A. GONZALE
Oficial de Partes
Comisión Nacional
Medio Ambiente (CON

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- Archivo.

estreo y al método de refe-
n el anexo.
deberá tener por resultado
ecta o indirecta de la aplica-
de concentración fijado en
ulo 2.°

rea un Comité para la adap-
Directiva al progreso cien-
sucesivo denominado «Co-
mpuesto por representantes
ros y presidio por un repre-
ión.
tablecerá su reglamento in-

el caso en que se haga refe-
nto definido en el presente
te someterá las cuestiones al
iativa propia bien a instancia
un Estado miembro.
nte de la Comisión someterá
to de las medidas que deberán
té emitirá su dictamen sobre
plazo que el Presidente podrá
u cia que revista la cues-
Se nunciará por mayoría
os de los Estados miembros se
reglo al apartado 2 del artícu-
. El Presidente no participará

adoptará las medidas previs-
atengan al dictamen del Co-

das previstas no se atengan al
té, o a falta de dictamen, la
sin demora al Consejo una
a las medidas que deberán
ejo decidirá por mayoría cua-

un plazo de tres meses a partir
le la propuesta al Consejo, éste
unciado, las medidas propues-
por la Comisión.

os Estados miembros aplica-
nes legales, reglamentarias y
esarias para cumplir la presen-
plazo de veinticuatro meses a
ción e informarán de ello inme-
misión.
s miembros comunicarán a la
le l posiciones de Derecho
n en el ámbito regulado por la

destinatarios de la presente
Estados miembros.

§ 201. DIRECTIVA DEL CONSEJO 77/312/CEE, DE 29 DE MARZO DE 1977, SOBRE LA VIGILANCIA BIOLOGICA DE LA POBLACION CONTRA EL PELIGRO DEL SATURNISMO

(«DOCE» núm. L 105, de 28 de abril de 1977)

EL CONSEJO DE LAS COMUNIDADES EUROPEAS

Visto el Tratado constitutivo de la Comunidad Económica Europea y, en particular, su artículo 235,

Vista la propuesta de la Comisión,

Visto el dictamen del Parlamento Europeo (1),

Visto el dictamen del Comité Económico y Social (2),

Considerando que una de las tareas esenciales de la Comunidad Económica Europea consiste en promover un desarrollo armónico de las actividades económicas en el conjunto de la Comunidad y una expansión continua y equilibrada, objetivos que no pueden concebirse con independencia de la lucha contra la contaminación y los agentes nocivos, ni sin la mejora de la calidad de la vida y la protección del entorno;

Considerando que las diversas utilizaciones del plomo originan actualmente la contaminación saturnina de numerosas áreas del entorno;

Considerando que las múltiples fuentes difusoras de plomo presentes en el entorno hacen difícil la determinación de la exposición global de un individuo a este contaminante y, en consecuencia, que la protección de la salud del hombre requiere un control tan estricto como sea posible de la impregnación saturnina global del individuo;

Considerando que es preciso poner en obra una vigilancia biológica de la población contra el riesgo del saturnismo, y evaluar los resultados de esta vigilancia con el fin de elaborar, llegado el caso, nuevas propuestas;

Considerando que hace falta definir las normas técnicas y los niveles de referencia biológica para realizar esta vigilancia;

Considerando que la determinación de la plombemia constituye actualmente el mejor medio de evaluar la dosis de plomo absorbida recientemente por un individuo como consecuencia de su exposición al plomo presente en el entorno, y que la actividad enzimática de la dehidratasa del ácido deltaaminolevulehico (ALAD) puede servir de análisis indicativo o complementario para la determinación de la exposición al plomo:

Considerando que el programa de acción de las Comunidades Europeas en materia de medio ambiente (3) prevé la coordinación de los programas nacionales encaminados a mejorar la calidad de vida, así como una acción prioritaria en cuanto al plomo,

Ha adoptado la presente Directiva:

Artículo 1.° Los Estados miembros tomarán las medidas necesarias para aplicar un procedimiento común de vigilancia biológica con el objeto de evaluar la exposición de la población al peligro del saturnismo fuera de los lugares de trabajo.

Art. 2.° Este procedimiento común, cuya aplicación se limitará a cuatro años, se basa en la medición de la plombemia.

A modo de análisis indicativo o complementario, podrá utilizarse también la medición del ALAD con arreglo a las normas especificadas en los anexos II y III.

Art. 3.° 1. Las condiciones en que se efectuará esta vigilancia biológica estarán determinadas por:

- Las modalidades de muestreo y de análisis.
- La frecuencia del muestreo.

2. Las extracciones de sangre destinadas al muestreo se practicarán en voluntarios.

Art. 4.° El muestreo se realizará sobre:

- Grupos de 100 personas, por lo menos, en las regiones urbanas de más de 0,5 millones de habitantes.
- Grupos de 100 personas, por lo menos, en la medida en que esta cifra pueda alcanzarse, elegidas entre las poblaciones expuestas a fuentes significativas de contaminación por plomo.
- Grupos críticos determinados por las autoridades competentes de los Estados miembros.

En cada Estado miembro y a lo largo de cada campaña, se efectuarán al menos 50 análisis por cada millón de habitantes.

Art. 5.° El muestreo de los grupos mencionados en el artículo 4.° se efectuará a lo largo de dos campañas, por lo menos, en cada zona estudiada, durante la aplicación del programa, separadas por un intervalo de veinticuatro meses como mínimo. En la segunda campaña no se examinará necesariamente a los mismos individuos que en la primera campaña.

Art. 6.° Para evaluar los resultados de la vigilancia biológica y adoptar, si hace falta, las medidas previstas en el artículo 8.°, las tasas de plombemia siguientes, fijadas teniendo en cuenta las relaciones dosis-efecto que figuran en el anexo I, serán simultáneamente consideradas como niveles de referencia:

- 20 microgramos de plomo por 100 mililitros de sangre, como máximo, para el 50 por 100 del grupo de población examinado.
- 30 microgramos de plomo por 100 mililitros de sangre, como máximo, para el 90 por 100 del grupo de población examinado.

(1) «DOCE» núm. C 28, de 9 de febrero de 1976, p. 31.
(2) «DOCE» núm. C 50, de 4 de marzo de 1976, p. 9.
(3) «DOCE» núm. C 112, de 20 de diciembre de 1973, p. 3.

§ 201

— 35 microgramos de plomo por 100 mililitros de sangre, como máximo, para el 98 por 100 del grupo de población examinado.

Art. 7.º Para la determinación de la plombemia:

— Los Estados miembros comunicarán a la Comisión los nombres de los laboratorios que participen en el programa de vigilancia biológica y los métodos de análisis utilizados.
— La Comisión, en colaboración con los Estados miembros, organizará programas de intercomparación, en los que participarán los laboratorios anteriormente mencionados.
— La Comisión, en colaboración con los Estados miembros, estudiará los resultados de estos programas con el fin de mejorar la comparabilidad de los métodos de análisis.

Art. 8.º Cuando el resultado de los análisis acuse uno o varios rebasamientos de los niveles de referencia indicados en el artículo 6.º, los Estados miembros:

— Comprobarán la validez de los resultados.
— Buscarán las fuentes de exposición que provocan esos rebasamientos; esta acción afectará igualmente a todos los individuos que tengan una plombemia superior a 35 microgramos por 100 mililitros.
— Tomarán las medidas apropiadas, según el criterio de sus autoridades nacionales competentes.

Art. 9.º 1. Los Estados miembros designarán, dentro de los seis meses siguientes a la notificación de la presente Directiva, la autoridad nacional competente, que comunicará a la Comisión:

— Los datos relativos a la vigilancia biológica de los grupos de población mencionados en el artículo 4.º, incluyendo indicaciones sobre los métodos de análisis, los grupos de población examinados y las zonas en que las muestras se han tomado; estos datos deberán ofrecer toda garantía en lo que atañe al respeto del anonimato de las personas examinadas; las modalidades y la forma de transmisión de estos datos serán fijadas por común acuerdo entre la Comisión y los Estados miembros.
— Las informaciones sobre las causas o los factores que presumiblemente ocasionan el rebasamiento de los niveles de referencia indicados en el artículo 6.º

2. La autoridad nacional competente notificará, además, a la Comisión las medidas adoptadas en virtud del tercer guión del artículo 8.º

Art. 10. La Comisión reunirá por lo menos dos veces al año a los representantes de los Gobiernos de los Estados miembros, con el objeto, principalmente, de:

— Garantizar la ejecución armonizada de la vigilancia biológica y, en particular, de las disposiciones contenidas en los artículos 4.º y 5.º
— Velar por la comparabilidad de los análisis efectuados.
— Examinar las informaciones y facilitar el intercambio de informaciones entre los Estados miembros sobre los resultados obtenidos por la vigilancia biológica, así como sobre las medidas adoptadas en virtud del artículo 8.º

Art. 11. Sobre la base de las informaciones recogidas en virtud del artículo 9.º, la Comisión elaborará, en colaboración con las autoridades nacionales competentes:

— Una Memoria anual de conjunto sobre la ejecución de este programa, que será remitida a los Estados miembros, así como al Consejo y al Parlamento Europeo.
— Un informe general al término de este programa que sirva de fundamento para la eventual preparación de nuevas propuestas en las que también se tendrían en cuenta los progresos conseguidos en los conocimientos científicos y técnicos.

Art. 12. Los Estados miembros adoptarán las medidas necesarias para ajustarse a la presente Directiva dentro de un plazo de doce meses, contado a partir de su notificación e informarán de ello inmediatamente a la Comisión.

Art. 13. Los destinatarios de la presente Directiva serán los Estados miembros.

ANEXO I

Relación dosis-efecto

Las tasas de plombemia tomadas en consideración para estimar los resultados de la vigilancia biológica se desprenden del análisis de los datos científicos referentes a los diversos efectos tóxicos del plomo. Este análisis que toma en cuenta las variaciones normales de los valores biológicos de la población permite establecer relaciones quasi cuantitativas entre dosis y efecto. La relación dosis-efecto que sigue sirve de base de referencia para la puesta en marcha de la presente Directiva.

Un crecimiento de la actividad ALAD en los glóbulos rojos, como consecuencia de la exposición al plomo, mientras no dé lugar a una alteración en la hematopoiesis, puede ser aceptada para la población. En lo que se refiere a las plombemias inferiores a 15-20 µg/100 ml., se considera en la actualidad que el decrecimiento de la actividad ALAD no provoca alteraciones en la hematopoiesis.

El aumento de las protoporfirinas eritrocitarias (PPE) en la sangre es señal de una interferencia con la utilización del hierro, que inhibe de este modo la síntesis del hema. El aumento de los PPE interviene para tasas de plombemia superiores a 20-30 µg/100 ml. Un aumento de los PPE puede ser debido, sin embargo, a otras causas.

La interferencia con la síntesis del glutatión sólo puede ser tolerada para una pequeña fracción de la población y solamente si es débil y no produce otras manifestaciones subclínicas. Esta interferencia inaceptable con la síntesis del glutatión no tiene

lugar siempre que la tasa de plombemia sea inferior a 30 µg/100 ml.

El aumento significativo de la excreción urinaria del ácido delta-aminolevulínico (ALAU) es síntoma de alteraciones importantes del metabolismo de las porfirinas y, por tanto, de salud débil. El aumento estadísticamente significativo del ALAU se manifiesta sólo para tasas de plombemia superiores a 35 µg/100 ml.

ANEXO II

Correspondencia entre tasas de plombemia y actividad enzimática

Con arreglo al artículo 2.° de la presente Directiva, la correspondencia entre la tasa de plombemia y la actividad enzimática del ALAD, medida según el método europeo estandarizado (anexo III), es la siguiente:

Plombemia (µg/100 ml. sangre)	ALAD (unidades/litro)
35	20
30	25
20	35

Mientras los valores medidos del ALAD sean superiores de forma significativa a los límites indicados más arriba para las diferentes fracciones de población, no es necesario realizar nuevas mediciones de tasas de plombemia a modo de confirmación.

ANEXO III

Prescripciones técnicas referentes a la determinación de la actividad ALAD

Método europeo estandarizado para la determinación de la actividad de la dehidratasis del ácido delta-aminolevúlnico

El principio del método adoptado para determinar la actividad de la dehidratasis del ácido delta-aminolevúlnico es bien conocido. Se basa en la incubación del enzima con un exceso de sustrato de ácido delta-aminolevúlnico. El porfobilinógeno formado tras un tiempo determinado es mezclado con el reactivo de Ehrlich modificado y el color conseguido se mide mediante un fotómetro contra un blanco. La cantidad de porfobilinógeno producido es indicativa de la actividad del ALAD.

METODO

Efecto de la luz

Recientes experimentos han demostrado que el porfobilinógeno es, sobre todo, muy sensible a la luz. Cualquier análisis debería realizarse en ausencia total de luz solar directa en el laboratorio (y no sólo en el lugar donde se efectúa el análisis).

Primera fase.—Muestras de la sangre y conservación antes del análisis.

- Realizar una toma de sangre de 2 ml. de sangre venosa mediante una jeringa de plástico (no estabilizada al plomo) en presencia de heparina secada (< 5 mg.).
- Preparar inmediatamente cuatro extracciones de 0,2 ml. distribuidas en tubos de plástico (no estabilizados al plomo) y enfriarlas a 4° C. El pipetaje se debe efectuar con pipetas graduadas de tipo Marbourg.
- Si el análisis se realiza en un plazo de tres horas, el enfriamiento de las extracciones no es necesario.
- La duración máxima de conservación de las muestras a 4° C es de veinticuatro horas.
- Inmediatamente antes del análisis, hay que colocar todas las muestras en un baño de agua helada durante diez minutos.

Nota: Entre las materias plásticas a considerar, podemos citar el polietileno, el polistireno y el polipropileno. La duración de conservación de veinticuatro horas a 4° C resulta de una estimación prudente. Este período de tiempo basta para permitir el transporte de la muestra desde el lugar de la toma de sangre hasta un laboratorio central para su análisis. De las cuatro extracciones de sangre, tres deben ser utilizadas para la determinación del ALAD y una para el test testigo.

Segunda fase.—Determinación de la hematócrita.

Esta determinación se debe efectuar:

- Al mismo tiempo que la toma de sangre.
- Por un método capilar que utilice dos muestras.

Centrifugación después del cierre de un extremo a una velocidad de 30.000 revoluciones por minuto como mínimo (no menos de cinco minutos).

Nota: Esta determinación se debe realizar preferentemente en el mismo lugar, pero no más de veinticuatro horas después de la toma de sangre. Utilizar si es posible una centrifugadora microhematócrita.

Legislación comunitaria vigente

Documento 396L0062

Capítulos del Repertorio donde puede consultarse este documento:
[15.10.20.30 - Control de la contaminación atmosférica]

396L0062
Directiva 96/62/CE del Consejo de 27 de septiembre de 1996 sobre evaluación y gesti
Diario Oficial
n°
L 296 de 21/11/1996 P. 0055 - 0063

Texto:

DIRECTIVA 96/62/CE DEL CONSEJO de 27 de septiembre de 1996 sobre evaluación y
EL CONSEJO DE LA UNIÓN EUROPEA,
Visto el Tratatado constitutivo de la Comunidad Europea y, en particular, el apartado 1 de
Vista la propuesta de la Comisión (1),
Visto el dictamen del Comité Económico y Social (2),
De conformidad con el procedimiento establecido en el artículo 189 C del Tratado (3),
Considerando que el quinto Programa de acción de 1992 sobre el medio ambiente, cuyo p
Considerando que, para proteger el medio ambiente en su totalidad así como la salud hum
Considerando que, para tener en cuenta los mecanismos específicos de formación de ozor
Considerando que los valores numéricos de los valores límite, los umbrales de alerta, y, r
Considerando que la Comisión debe realizar estudios para analizar los efectos de la acció
Considerando que la calidad del aire ambiente debe evaluarse en relación con valores lím
Considerando que, para poder comparar las evaluaciones de la calidad del aire ambiente b
Considerando que, para poner en cuenta otras técnicas de estimación de la calidad del aire
Considerando que las medidas generales establecidas en la presente Directiva deben com
Considerando que se deben adoptar estas medidas específicas lo antes posible para dar cu
Considerando que se deben recoger los primeros datos significativos sobre los niveles de
Considerando que para proteger el medio ambiente en su totalidad, así como la salud hum
Considerando que las medidas que adopten los Estados miembros deben tener en cuenta l
Considerando que, como lleva tiempo aplicar estas medidas y que resulten eficaces puede
Considerando que puede haber zonas de los Estados miembros en las que los niveles sean
Considerando que los Estados miembros deben consultarse mutuamente en caso de que el
Considerando que la determinación de umbrales de alerta a partir de los cuales se deben t
Considerando que en las zonas y aglomeraciones urbanas en que los niveles de contamina
Considerando que, para facilitar el manejo y la comparación de los datos recibidos, la Co
Considerando que la puesta en práctica de una política de gestión y evaluación de la calid
Considerando que es necesario evitar que aumente innecesariamente la cantidad de inform
Considerando que puede ser conveniente la necesaria adaptación de los criterios y técnica
Considerando que, para fomentar el intercambio recíproco de información entre los Estad
Considerando que deben tratarse prioritariamente las sustancias ya incluidas en la Directi
HA ADOPTADO LA PRESENTE DIRECTIVA:

Artículo 1

Objetivos

El objetivo general de la presente Directiva es definir los principios básicos de una estrate
- definir y establecer objetivos de calidad del aire ambiente en la Comunidad para evitar,
- evaluar, basándose en métodos y criterios comunes, la calidad del aire ambiente en los E
- disponer de información adecuada sobre la calidad del aire ambiente y procurar que el p
- mantener una buena calidad del aire ambiente y mejorarla en los demás casos.

Artículo 2

Definiciones
A efectos de la presente Directiva se entenderá por:
1) «aire ambiente»: el aire exterior de la troposfera, excluidos los lugares de trabajo;
2) «contaminante»: cualquier sustancia introducida directa o indirectamente por el hombr
3) «nivel»: la concentración de un contaminante en el aire ambiente o su depósito en supe
4) «evaluación»: se refiere a cualquier método utilizado para medir, calcular, predecir o e
5) «valor límite»: un nivel fijado basándose en conocimientos científicos, con el fin de ev
6) «valor de referencia objetivo»: un nivel fijado con el fin de evitar más a largo plazo efe
7) «umbral de alerta»: un nivel a partir del cual una exposición de breve duración supone
8) «límite de exceso tolerado»: el porcentaje del valor límite en el que éste puede sobrepa
9) «zona»: la porción de su respectivo territorio delimitada por los Estados miembros;
10) «aglomeración»: un áera que se caracteriza por una concentración de población de má

Artículo 3

Aplicación y responsabilidades
Para la aplicación de las disposiciones de la presente Directiva, los Estados miembros des
- la aplicación de la presente Directiva,
- la evaluación de la calidad del aire ambiente,
- la autorización de los dispositivos de medición (métodos, aparatos, redes, laboratorios),
- asegurar la calidad de la medición efectuada por los dispositivos de medición, comprob
- analizar los métodos de evaluación,
- coordinar en su respectivo territorio los programas comunitarios de garantía de calidad
Cuando los Estados miembros suministren a la Comisión la información contemplada en

Artículo 4

Definición de los valores límite y de los umbrales de alerta correspondientes al aire ambi
1. En el caso de los contaminantes de la lista del Anexo I, la Comisión presentará al Cons
- a más tardar el 31 de diciembre de 1996 para las sustancias contaminantes 1 a 5;
- de acuerdo con el artículo 8 de la Directiva 92/72/CEE para el ozono;
- el 31 de diciembre de 1997 a más tardar para las sustancias contaminantes 7 y 8;
- tan pronto como sea posible y a más tardar el 31 de diciembre de 1999 para las sustanci
Para fijar los valores límite y, de forma adecuada, los umbrales de alerta, se tendrán en cu
En lo que respecta al ozono, dichas propuestas tendrán en cuenta los mecanismos específi
En el caso de que se rebase un valor de referencia objetivo fijado para el ozono, los Estad
En cuanto a otros contaminantes, la Comisión presentará al Consejo propuestas de establ
2. La Comisión velará por revisar, teniendo en cuenta los datos más recientes de la invest
3. En el momento de establecer los valores límite y los umbrales de alerta, se determinará
a) las mediciones que deberán utilizarse en aplicación de la legislación a que se refiere el
- la localización de los puntos de toma de muestras,
- el número mínimo de puntos de toma de muestras,
- las técnicas de medición de referencia y de toma de muestras;
b) el uso de otras técnicas de evaluación de la calidad del aire ambiente, en particular la m
- la resolución espacial para la modelización y los métodos de evaluación objetiva,
- técnicas de referencia para la modelización.
Estos criterios y técnicas se establecerán con respecto a cada contaminante y con arreglo a
4. Para tener en cuenta los niveles efectivos de un contaminante dado en el momento de e
Este margen se reducirá con arreglo a modalidades que se determinarán específicamente p
5. El Consejo adoptará la legislación prevista en el apartado 1 y en las normas previstas en
6. Cuando un Estado miembro tome medidas más estrictas que aquéllas a que se refiere el
7. Cuando un Estado miembro tenga la intención de fijar valores límite o umbrales de aler

Artículo 5

Evaluación preliminar de la calidad del aire ambiente
Los Estados miembros que no dispongan de mediciones representativas de los niveles de

Artículo 6

Evaluación de la calidad del aire ambiente
1. Una vez definidos los valores límite y los umbrales de alerta, la calidad del aire ambien
2. De conformidad con los criterios que se mencionan en el apartado 3 del artículo 4, y en
- las aglomeraciones definidas en el punto 10 del artículo 2,
- las zonas en que los niveles se hallen comprendidos entre los valores límite y los niveles
- las demás zonas en que los niveles superen los valores límite.
Las medidas previstas podrán completarse mediante técnicas de modelización para facilit
3. Para la evaluación de la calidad del aire ambiente podrá utilizarse una combinación de
4. Cuando los niveles sean inferiores a un nivel por determinar en las disposiciones previ
5. En los casos en que haya que medir contaminantes, las mediciones se harán en lugares

Artículo 7

Mejora de la calidad del aire ambiente

Requisitos generales
1. Los Estados miembros tomarán las medidas necesarias para garantizar el respeto de los
2. Las medidas que se adopten para alcanzar los objetivos de la presente Directiva deberá
a) tener en cuenta un enfoque integrado para la protección del aire, el agua y el suelo;
b) no contravenir la legislación comunitaria relativa a la protección de la seguridad y de l
c) no tener efectos negativos y significativos sobre el medio ambiente de los demás Estad
3. Los Estados miembros elaborarán planes de acción que indiquen las medidas que deba

Artículo 8

Medidas aplicables en las zonas en las que los niveles rebasen el valor límite
1. Los Estados miembros establecerán la lista de las zonas y aglomeraciones en que los ni
Cuando no se haya fijado un margen de exceso tolerado para un contaminante determinad
2. Los Estados miembros establecerán la lista de las zonas y aglomeraciones en las que lo
3. En las zonas y aglomeraciones contempladas en el apartado 1, los Estados miembros tc
Dicho plan o programa, que deberá estar a disposición del público, especificará al menos
4. En las zonas y aglomeraciones contempladas en el apartado 1 en que el nivel de más de
5. La Comisión controlará regularmente la aplicación de los planes o programas presentad
6. Cuando el nivel de un contaminante sea superior o amenace con ser superior al valor lí

Artículo 9

Requisitos aplicables a las zonas en que los niveles sean inferiores al valor límite
Los Estados miembros establecerán la lista de las zonas y aglomeraciones en las que los r
Los Estados miembros mantendrán los niveles de los contaminantes en dichas zonas y ag

Artículo 10

Medidas aplicables cuando los niveles sobrepasan los umbrales de alerta
Cuando se rebasen los umbrales de alerta, los Estados miembros garantizarán que se tome

Artículo 11

Transmisión de las informaciones e informes
Tras la adopción por el Consejo de la primera propuesta contemplada en el primer guión d
1) los Estados miembros informarán a la Comisión sobre las autoridades competentes, lab
a) en las zonas contempladas en el apartado 1 del artículo 8:
i) le señalarán la aparición de niveles superiores al valor límite incrementado por el marg
Cuando no se haya fijado el margen de exceso tolerado de un contaminante determinado,

ii) le señalarán los motivos de cada caso registrado, dentro de los nueve meses siguientes
iii) los planes o programas mencionados en el apartado 3 del artículo 8, transmitiéndoselo
iv) la marcha del plan o programa, cada tres años;
b) le transmitirán cada año, y a más tardar nueve meses después del final de cada año, la l
c) le transmitirán, cada tres años, en el marco del informe sectorial al que hace referencia
d) le transmitirán los métodos utilizados para la evaluación preliminar de la calidad del ai
2) La Comisión publicará:
a) cada año, una lista de las zonas y aglomeraciones contempladas en el apartado 1 del art
b) cada tres años, un informe sobre la calidad del aire ambiente en la Comunidad. Este inf
3) La Comisión utilizará, en caso necesario, los conocimientos disponibles en la Agencia

Artículo 12

Comité y funciones del Comité
1. Las modificaciones necesarias para adaptar al progreso científico y técnico los criterios
Dicha adaptación no deberá suponer una modificación directa o indirecta de los valores lí
2. La Comisión estará asistida por un Comité compuesto por los representantes de los Est
El representante de la Comisión presentará al Comité un proyecto de medidas. El Comité
La Comisión adoptará las medidas previstas cuando sean conformes al dictamen del Com
Cuando las medidas previstas no se conformen al dictamen del Comité, o en ausencia de d
Si tres meses después de haberse presentado una propuesta al Consejo éste no se hubiere p

Artículo 13
1. Los Estados miembros pondrán en vigor las disposiciones legales, reglamentarias y adı
Cuando los Estados miembros adopten dichas disposiciones, éstas harán referencia a la pı
2. Los Estados miembros comunicarán a la Comisión el texto de las principales disposici

Artículo 14
La presente Directiva entrará en vigor el día de su publicación en el Diario Oficial de las C

Artículo 15
Los destinatarios de la presente Directiva serán los Estados miembros.

Hecho en Bruselas, el 27 de septiembre de 1996.
Por el Consejo
El Presidente
M. LOWRY

(1) DO nº L 377 de 31. 12. 1991, p. 48.

ANEXO I

LISTA DE LOS CONTAMINANTES ATMOSFÉRICOS QUE DEBEN TENERSE EN C
I. Contaminantes que deberán ser examinados en la fase inicial, incluidos los contaminant
1. Dióxido de azufre
2. Dióxido de nitrógeno
3. Partículas finas, como los hollines (incluido PM 10)
4. Partículas en suspensión
5. Plomo
6. Ozono
II. Otros contaminantes atmosféricos
7. Benceno
8. Monóxido de carbono
9. Hidrocarburos policíclicos aromáticos
10. Cadmio
11. Arsénico
12. Níquel
13. Mercurio

ANEXO II

FACTORES QUE DEBERÁN TENERSE EN CUENTA AL ESTABLECER LOS VAL
Cuando se fije el valor límite y, de forma adecuada, el umbral de alerta, podrán tenerse es
- grado de exposición de las poblaciones y, en particular, de los subgrupos sensibles,
- condiciones climáticas,
- sensibilidad de la fauna, de la flora y de sus hábitats,
- patrimonio histórico expuesto a los contaminantes,
- viabilidad económica y técnica,
- transporte a larga distancia de los contaminantes, con inclusión de los contaminantes sec

ANEXO III

DIRECTRICES PARA LA SELECCIÓN DE LOS CONTAMINANTES ATMOSFÉRIC
1. Posibilidad, gravedad y frecuencia de los efectos; por lo que toca a la salud humana y a
2. Presencia generalizada y concentración elevada del contaminante en la atmósfera.
3. Transformaciones medioambientales o alteraciones metabólicas que puedan dar lugar a
4. Persistencia en el medio ambiente, en particular si el contaminante no es biodegradable
5. Impacto del contaminante:
- importancia de la población expuesta, de los recursos vivos o de los ecosistemas,
- organismos receptores particularmente vulnerables en la zona afectada.
6. Podrán utilizarse también métodos de evaluación del riesgo.
Deberán tenerse en cuenta para la selección de los contaminantes los criterios pertinentes

ANEXO IV

INFORMACIÓN QUE DEBE INCLUIRSE EN LOS PROGRAMAS LOCALES, REGI
Información que debe facilitarse en virtud del apartado 3 del artículo 8:
1) Localización del rebasamiento:
- región,
- ciudad (mapa),
- estación de medición (mapa, coordenadas geográficas).
2) Información general:
- tipo de zona (ciudad, área industrial o rural),
- estimación de la superficie contaminada (km2) y de la población expuesta a la contamin
- datos climáticos útiles,
- datos topográficos pertinentes,
- información suficiente acerca del tipo de organismos receptores de la zona afectada que
3) Autoridades responsables:
nombres y direcciones de las personas responsables de la elaboración y ejecución de los p
4) Naturaleza y evaluación de la contaminación:
- concentraciones observadas durante los años anteriores (antes de la aplicación de las me
- concentraciones medidas desde el comienzo del proyecto,
- técnicas de evaluación utilizadas.
5) Origen de la contaminación:
- lista de las principales fuentes de emisión responsables de la contaminación (mapa),
- cantidad total de emisiones procedentes de esas fuentes (t/año),
- información sobre la contaminación procedente de otras regiones.
6) Análisis de la situación:
- detalles de los factores responsables del rebasamiento (transporte, incluidos los transpor
- detalles de las posibles medidas de mejora de la calidad del aire.
7) Detalles de las medidas o proyectos de mejora que existían antes de la entrada en vigor
- medidas locales, regionales, nacionales o internacionales,
- efectos observados de estas medidas.
8) Información sobre las medidas o proyectos adoptados para reducir la contaminación tra
- lista y descripción de todas las medidas previstas en el proyecto,
- calendario de aplicación,
- estimación de la mejora de la calidad del aire que se espera conseguir y del plazo previst
9) Información sobre las medidas o proyectos a largo plazo previstos o considerados.

10) Lista de las publicaciones, documentos, trabajos, etc. que completen la información s

Fin del documento

Documento enviado el: 27/07/1998

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Lead

General Description

Lead is a bluish or silvery-grey soft metal with a melting-point of 327.5 °C and a boiling-point at atmospheric pressure of 1740 °C. It has four naturally occurring isotopes (atomic weights: 208, 206, 207 and 204 in order of abundance). The isotopic ratios differ for various mineral sources. This property has been used in nonradioactive-tracer studies to investigate the environmental and metabolic pathways of lead.

The usual oxidation state of lead in inorganic compounds is +2. Apart from nitrate, chlorate and, to a much lesser degree, chloride, most of the inorganic salts of lead (II) have a poor solubility in water.

Organic lead compounds such as tetraethyl lead and tetramethyl lead are of great importance due to their extensive use as fuel additives. Tetraethyl lead and tetramethyl lead are colourless liquids with boiling-points of 110 °C and 200 °C respectively. Since their volatility is lower than that of most petrol components, the evaporation of petrol tends to concentrate tetraethyl lead and tetramethyl lead. Both compounds are decomposed at boiling-point as well as by ultraviolet light and trace chemicals in air, such as halogens, acids, or oxidizing agents.

Sources

The combustion of alkyl lead additives in motor fuels accounts for the major part of all lead emissions into the atmosphere. An estimated 80–90% of lead in ambient air derives from the combustion of leaded petrol. The degree of pollution from this source differs from country to country, depending on motor vehicle density and the efficiency of efforts to reduce the lead content of petrol.

The mining and smelting of lead ores create pollution problems in some areas. The level of contamination of the surrounding air and soil depends on the amount of lead emitted, the height of the stack, topography, and other local features. Secondary lead smelters, the refining and manufacture of compounds and goods containing lead, and refuse incineration also give rise to lead emissions.

Since coal, like many minerals, rocks and sediments, usually contains low concentrations of lead, a number of other industrial activities such as iron and steel production, copper smelting and coal combustion must be regarded as additional sources of lead emissions into the atmosphere.

The presence of lead water-pipes in old houses can be an important source of lead exposure for humans, particularly in soft-water areas. In certain areas lead-containing paint in old houses can be an additional source of exposure.

Occurrence in air

Current "baseline" levels of lead in the atmosphere are estimated to be in the range of $5 \times 10^{-5} \mu g/m^3$ *(1)*. Whether or not this level is wholly natural or a composite of natural and anthropogenic sources can be determined by analysing the isotopic composition. Even in the remotest sites, human activity has raised the lead concentration in the air considerably higher than natural levels. In nonurban sites located near urban areas, air lead levels average around $0.5 \mu g/m^3$, while in rural areas, levels in the range of 0.1 to $0.3 \mu g/m^3$ are found.

High concentrations of lead in ambient air are found in urban areas with high traffic density. At present, urban air lead levels are in the range of 0.5 to $3.0 \mu g/m^3$ (annual means) in most European cities. However, owing to decreases in the lead content of petrol, there is a trend towards lower air lead values *(2)*. High air lead levels are found in the vicinity of lead smelters.

Most of the lead in the air is in the form of fine particles with a mass median equivalent diameter of less than $1 \mu m$. The fraction of organic lead (predominantly lead alkyls that escaped combustion) is generally below 10% of the total atmospheric lead, the majority (>90%) of lead from leaded petrol emission being emitted as inorganic particles (e.g. PbBrCl). In the immediate vicinity of smelters, the particle size distribution usually shows a predominance of larger particles. However, these particles settle at distances of a few hundred metres or 1–2 km, so that further away the particle size distribution is indistinguishable from that of other urban sites.

Since people spend much of their time indoors, ambient air data may not accurately indicate actual exposure to airborne lead. Studies on indoor/outdoor air lead levels show that the indoor concentrations, in general, are lower than the corresponding outdoor values. Overall, the data suggest that indoor/outdoor ratios in the range of 0.6–0.8 are typical for airborne lead in houses without air-conditioning. Lower indoor/outdoor ratios have been observed during winter, when windows and doors are tightly closed *(3)*.

Lead is removed from the atmosphere by dry or wet deposition. The residence time of lead-containing particles in the atmosphere varies according to a number of factors, such as particle size, wind currents, rainfall and height of emission. Soil and water pollution from car emission fallout is predominantly limited to the immediate urban area. Fallout from the emission of industrial sources, such as smelters, is likewise limited mainly to the immediate vicinity. However, strong evidence indicates that a fraction of airborne lead is transported over long distances. As a result, a long-term global accumulation of lead has occurred in recent decades. This has been

demonstrated convincingly by analyses of glacial ice and snow deposits in remote areas *(1)*.

Routes of Exposure

Air

Most of the lead in ambient air is in the form of submicron-sized particles. Some 30–50% of these inhaled particles are retained in the respiratory system. Virtually all of this retained lead is absorbed into the body. Particles in the size range of 1–3 μm are also efficiently deposited in the lungs. Larger particles are deposited with variable efficiency, mainly in the upper respiratory tract with incomplete absorption. All lead particles that are cleared by the lung can be swallowed and result in further lead absorption in the gastrointestinal tract.

Drinking-water

Lead concentrations in drinking-water and groundwater vary from 1 to 60 μg/litre. In most European countries, the levels of lead in domestic tap-water are relatively low, i.e. normally below 20 μg/litre. Consequently, man's exposure to lead through water is generally low in comparison with exposure through food *(4,5)*. However, in some places (areas with soft water, where lead water-pipes and lead plumbing are common), lead in drinking-water can contribute significantly to the total lead intake *(6)*.

Food

Most people receive the largest portion of their daily lead intake via food. Most lead enters food during storage and manufacture, e.g. in canned food and in alcoholic drinks. The most important pathway whereby atmospheric lead enters the food chain is thought to be direct foliar contamination of plants. This contamination depends on the rate of fallout of lead in the districts where food is grown; it tends to be higher in heavily industrialized areas. Additionally, air deposits raise the level of lead in soil, which, in the course of decades and centuries, may result in an increased uptake of lead through the roots.

The concentrations of lead in various food items are highly variable. Several studies have reported average lead intakes in the range of 100–500 μg/day for adults, with individual diets covering a much greater range. More recent data indicate total daily intakes of about 100 μg or less *(2,7)*. For young children, estimates of total daily intakes are about one half the figures for adults. Recent data suggest that levels of lead in the diet appear to have been falling in the last few years.

Additional exposures

An individual's lead exposure may be increased by choice, habit or unavoidable circumstances, in addition to the "normal" environmental exposure through food, drinking-water and air. These additional exposures can be categorized as being either due to lead in the ambient air or independent of lead in the ambient air.

The former category includes high lead levels in dustfall and soil in residential areas near smelters or refineries, high-density traffic, and the consumption of vegetables and fruit grown on high-lead soils or near sources of lead emissions (smelters, roadways with high traffic density).

The latter category includes occupational exposures; secondary occupational exposures of members of the families of lead workers; contamination of house dust in houses with interior lead paint; contamination of tap-water in houses with lead water-pipes or lead plumbing; use of improperly glazed earthenware vessels; tobacco smoke *(8)* and alcohol consumption (in particular, wine) *(8,9)*.

Lead in dust, indoors as well as outdoors, is an important potential source of intake by ingestion, particularly for young children living in contaminated areas, e.g. lead smelter areas and central urban areas *(10–14)*.

Relative significance of different routes of exposure

Exposure to lead from water, food, air and other sources can vary significantly for different individuals and population groups. Since the relative contribution of each of these sources can also vary substantially, comprehensive information covering a wide range of circumstances cannot be provided. To give some idea of possible situations, a few simplified calculations are presented in Table 1.

Contributions from occupational exposures, cigarette-smoking and various other sources were not taken into account. Regarding the absorbed dose of lead, the contribution of inhaled airborne lead is in the range of 15–70% in adults and 2–17% in children. As far as adults are concerned, these figures are consistent with the results of isotopic tracing studies, which indicate that the percentage of petrol lead contributing to total human blood lead can be in the order of 25% or somewhat higher *(15)*.

An important group omitted in Table 1 is that of infants (up to 1 year old). At present, insufficient information is available on the lead content of their diet and its absorption for reasonable estimates to be made. However, the contribution of drinking-water in this group is likely to be high, probably higher than that for the children aged 1–5 years referred to in Table 1.

It should be emphasized that preschool children represent the most important risk group. For this group the contribution of air lead to blood lead by way of inhalation alone, as estimated in Table 1, clearly underestimates the contribution of environmental lead to blood lead, as air lead can only be taken as an indicator of general multimedia lead pollution. Because of the breathing behaviour of preschool children, outdoor lead deposition is the most important single explanation of differences between inner city and suburban areas in the blood lead of children *(13)*. Table 2 therefore illustrates some possible situations based on various assumptions of dust intake by children along with intake of lead in air, food and water. It is obvious that lead in dust can make a substantial contribution to absorbed lead, sometimes up to 80% of the total amount.

Table 1. Estimates of lead (μg/day) absorbed by adults and by children aged 1–5 years from air, food and drinking-water at different air lead levels

Mean air lead level (μg/m³)	Source			Total	Air/total (%)
	Air	Food	Water		
Adults					
0.3	2.4	10	2	14.4	17
0.5	4.0	10	2	16	25
1.0	8.0	10	2	20	40
2.0	16.0	10	2	28	57
3.0	24.0	10	2	36	67
Children (1–5 years old)					
0.3	0.6	25	5	30.6	2.0
0.5	1.0	25	5	31	3.2
1.0	2.0	25	5	32	6.3
2.0	4.0	25	5	34	11.8
3.0	6.0	25	5	36	16.7

Note. The estimates are based on the following assumptions:

Air: respiratory volume:
- adults: 20 m³/day
- children: 5 m³/day

respiratory absorption: 40%

Food: intake:
- adults: 100 μg/day; absorption 10%
- children: 50 μg/day; absorption 50%

Water: concentration 20 μg/litre:
- adults: 1 litre/day; absorption 10%
- children: 0.5 litre/day; absorption 50%

Kinetics and Metabolism

Absorption

Absorption through the respiratory tract is influenced by the particle size distribution and the ventilation rate. For adults the retention rates of airborne particulates range from 20% to 60%. Although lead salts differ widely in terms of water solubility, the chemical form of lead is not considered an important factor for respiratory absorption.

The proportion of lead absorbed from the gastrointestinal tract is about 10% in adults, whereas levels of 40–50% have been reported in children *(16,17)*. Gastrointestinal absorption is highly dependent on dietary or

Table 2. Estimates of lead (μg/day) absorbed by children from ingested dust, inhaled air, food and drinking-water, assuming different amounts of dust intake

Source				Total	Dust/total (%)
Dust	Air	Food	Water		
0	2	25	5	32	0
12.5	2	25	5	44.5	28.1
25	2	25	5	57	43.9
50	2	25	5	82	61.0
100	2	25	5	132	75.8

Note. The estimates are based on the following assumptions:

Dust:	lead concentration: 1 mg/g
Dust intake:	0, 25, 50, 100, 200 mg/day; absorption 50%
Air concentration:	1 μg/m³; respiratory volume 5 m³/day; respiratory absorption 40%
Food intake:	50 μg/day; absorption 50%
Water concentration:	20 μg/litre; water intake 0.5 litre/day;absorption 50%

nutritional factors *(5)*: both milk and fasting enhance absorption. Diets with low levels of calcium, vitamin D and iron have been shown to increase lead absorption in laboratory animals.

Distribution

The nonexcreted fraction of absorbed lead is distributed among three compartments: blood, soft tissues and the mineralizing tissues (bones, teeth). About 95% of the lead body-burden in adults is located in the bones, compared with about 70% in children *(18)*. Ninety-nine per cent of the lead in the bloodstream is bound to erythrocytes. The biological half-time of lead in blood can be as short as 20–40 days (isotopic tracer data), although longer half-time values have been reported in lead workers, and these may depend on the lead body-burden *(19,20)*.

Lead concentration in bones increases with age and this increase is more noticeable in males in the more dense tibial bones *(21)*. Lead stored in bones was shown to have a biological half-time of some years *(22)*.

Lead may be released from the bones in decalcification processes related to elderly people, pregnancy, acidosis, thyrotoxicosis or active remodelling processes in the bones of children. Animal experiments have shown mobilization of lead in pregnancy *(23)*. The evidence for the release of lead from bone in disease states involving febrile illness or altered metabolic activity in humans is at present speculative and more information is required.

Elimination

Nonabsorbed lead passes through the gastrointestinal tract and is excreted in the faeces. Of the absorbed fraction, 50–60% is removed by renal and biliary excretions. Intestinal clearance is about 50% of the renal clearance. (These figures relate to adult subjects.) Surprisingly little information exists about the age dependency of lead retention and excretion. Some data indicate that children, particularly infants, retain a higher amount of lead *(16,17)*.

Health Effects

The toxicity of lead may to some extent be explained by its interference with different enzyme systems: lead inactivates these enzymes by binding to SH-groups of its proteins or by displacing other essential metal ions. For this reason, almost all organs or organ systems may be considered potential targets for lead, and a wide range of biological effects of lead has been documented. These include effects on haem biosynthesis, the nervous system (neurotoxic effects), the kidneys, reproduction, the immune system, and also cardiovascular, hepatic, endocrinal and gastrointestinal effects. In conditions of low-level and long-term lead exposure such as are found in the general population, the most critical effects are those on haem biosynthesis, erythropoiesis, the nervous system and blood pressure.

Effects on experimental animals and *in vitro* test systems

Toxicological effects

Effects on both haem biosynthesis and the nervous system have been studied in laboratory animals. Inhibition of the activity of delta-aminolaevulinic acid dehydrase (ALAD), an enzyme involved in haem biosynthesis, is among the earliest biological effects of lead. Delta-aminolaevulinic acid dehydrase inhibition has also been observed in brain tissue. Comparative studies in rodents suggest that the brain of suckling rodents is more vulnerable to lead-induced ALAD inhibition than the adult brain *(24,25)*.

Neurobehavioural models of learning and memory have been used to study the effects of low-level lead exposure on the nervous system of rodents and monkeys. The more recent work in this field has been selectively reviewed *(26)*: learning and memory deficit has been found in rats after prenatal and postnatal lead exposure at blood lead levels below 0.2 μg/ml. Similar effects have been observed in monkeys at blood lead levels in excess of 0.4 μg/ml. There is some evidence that, if exposure occurred during the early stages of brain maturation, learning and memory deficit persists into adulthood long after the cessation of lead exposure.

Mutagenic and carcinogenic effects

There is no evidence that lead acetate or lead chloride induce mutations in bacteria. However, some chromosomal aberration tests in mammalian systems (either *in vitro* or *in vivo*) have given positive results.

According to IARC *(27)*, there is sufficient evidence that lead acetate, lead subacetate and lead phosphate are carcinogenic in rats and that lead subacetate is carcinogenic in mice. These compounds induce benign and malignant tumours of the kidney following oral or parenteral administration. Moreover, gliomas were observed in rats given lead subacetate by the oral route.

There are insufficient data to evaluate the carcinogenicity and mutagenicity of organometallic lead compounds.

Effects on humans

Toxicological effects

As far as long-term and low-level lead exposure is concerned, the following effects have to be considered in relation to the general population:

(*a*) effects on haem biosynthesis;

(*b*) effects on the nervous system; and

(*c*) effects on blood pressure.

The present discussion is, therefore, limited to these aspects of lead toxicity.

Effects on haem biosynthesis and erythropoiesis. The normal process of haem biosynthesis and its disturbance by lead is well understood. On the cellular level, the initial and final steps of haem formation are mitochondrial, whereas the intermediate steps take place in the cytoplasm.

Essentially, lead interferes with the activity of three enzymes:

(*a*) it indirectly stimulates the mitochondrial enzyme delta-aminolaevulinic acid synthetase (ALAS);

(*b*) it directly inhibits the activity of the cytoplasmatic enzyme delta-aminolaevulinic acid dehydrase (ALAD);

(*c*) it interferes with the normal functioning of intramitochondrial ferrochelatase, which is responsible for the insertion of iron (II) into the protoporphyrin ring.

Delta-aminolaevulinic acid synthetase stimulation has been found in lead workers at blood lead levels of about 0.4 μg/ml *(28)*. In contrast, ALAD in the erythrocytes is inhibited at very low blood lead levels. According to Hernberg & Nikkanen *(29)*, activity inhibition in urban adults was 50% at a blood lead level of 0.16 μg/ml. Roels et al. *(30)* were unable to determine a threshold for ALAD inhibition in children.

Delta-aminolaevulinic dehydrase inhibition results in an accumulation of its substrate, ALA, in blood, plasma and urine. Although the threshold for urinary ALA elevation is widely accepted as being 0.4 μg/ml, some studies demonstrated blood lead/urinary ALA correlations at an even lower blood lead value *(28)*.

Lead's interference with the formation of haem from protoporphyrin is apparent from increased levels of erythrocyte protoporphyrin or zinc protoporphyrin in blood. Studies based on pooled data from various authors point to a threshold of about 0.2 μg/ml in adults *(31)*. In children the threshold at which an increase of erythrocyte protoporphyrin occurs is in the range of 0.1–0.2 μg/ml *(32,33)*. There are some data suggesting that the no-response level in children is even lower than 0.1 μg/ml *(33)*.

Effects of lead on erythropoiesis and erythrocyte physiology represent more direct signs of damage to the haemopoietic system than haem precursors in blood or urine. Anaemia is a frequent outcome of chronic lead intoxication. Calculations by Piotrowski & O'Brien *(31)* using pooled data of various authors suggest that the threshold in children is about 0.25 μg/ml. A corresponding level of 0.5 μg/ml has been found in adult lead workers *(34)*.

In young children, lead exposure is associated with a decrease in the biosynthesis of the important hormonal metabolite of vitamin D: 1,25-dihydroxy-vitamin D *(35)*. This effect is correlated with blood lead in a group of 177 children over the blood lead range of 0.12–1.2 μg/ml. While this finding is based on limited information, its potential significance may be considerable.

Effects on the nervous system. Encephalopathy has been observed in adults at blood lead levels exceeding 1.2 μg/ml, and in children at levels of 0.8–1.0 μg/ml. The outcome is frequently fatal in children, and those who survive often present irreversible neurological and neuropsychological sequelae *(36,37)*. This aspect of lead toxicity is noncontroversial. Controversy exists as to whether lead exposure associated with blood lead levels below 0.7 μg/ml may be detrimental to the structure and function of the peripheral nervous system and/or CNS.

With regard to the peripheral nervous system, a decreased sensory and motor nerve conduction velocity has been found in lead-exposed workers at blood lead levels between 0.3 and 0.8 μg/ml *(38,39)*. The effect is reversible after discontinuation of exposure *(40)*. However, the effect is usually small, i.e. between 4 and 9 m/second for blood lead levels ranging from 0.05 to 0.6 μg/ml. Some negative findings *(41–43)* do exist, however, and the clinical significance has been questioned *(44)*.

Children as a risk group for CNS effects have received particular attention in studies dealing with lead-induced neuropsychological deficits at blood lead levels between 0.15 and 0.7 μg/ml. The earlier work describing cognitive dysfunctions, namely IQ deficit, impairment of eye–hand coordination and attention deficits, as well as behavioural abnormalities such as hyperactivity, has been critically reviewed *(45,46)*. The reviewers concluded that these earlier studies presented no convincing evidence of cognitive deficits associated with blood lead levels below 0.4 μg/ml in children; at higher levels, concern is warranted.

More recent work extends the range of concern to lower blood lead levels of about 0.2 μg/ml. This work also has recently been reviewed *(47)*. In the absence of prospective studies, final conclusions cannot be drawn from

these largely retrospective approaches, but extending the area of concern for lead-related neuropsychological deficits to blood lead levels below 0.3 μg/ml seems justified.

In addition to neuropsychological data, recent electrophysiological findings in children exposed to blood lead must be considered. A significant linear relationship between slow-wave voltage and blood lead levels ranging from 0.06 to 0.59 μg/ml has been found *(48)*. A 2-year follow-up *(49)* essentially confirmed these findings, thus demonstrating a persistence of the effect over at least this time-span. Although the clinical significance of these findings is not established, they clearly indicate altered CNS function at blood lead levels below 0.3 μg/ml.

Effects on blood pressure. Epidemiological and animal studies indicate that lead increases blood pressure. Data obtained from the National Health Assessment and Nutritional Evaluation Survey II in the USA showed that, after adjustment for age, body mass index, nutritional factors and blood biochemistries in a multiple regression model, the relationship of systolic and diastolic blood pressures to blood lead levels was statistically significant ($P < 0.01$) in white males aged 20–74 years *(50,51)*. A threshold blood lead level for this relationship was not evident. These findings were essentially confirmed in an epidemiological study on 7735 men aged 40–59 years in the United Kingdom *(52)*; however, the dose–response relationship was much weaker than in the United States survey. Although these data alone do not prove a causal relationship between low blood lead levels and blood pressure, the findings are consistent with results from animal studies, indicating that a causal relationship is probable. Considering the fact that high blood pressure is a major health problem in many industrialized countries and that a reduction of blood lead would decrease the number of hypertensive subjects and the cardiovascular and cerebrovascular risk implications associated with high blood pressure, it is highly desirable to keep the blood lead level of the general population as low as possible.

Carcinogenic effects

According to IARC *(53)*, evidence of the carcinogenicity of lead and lead compounds in humans is inadequate.

Evaluation of Human Health Risks

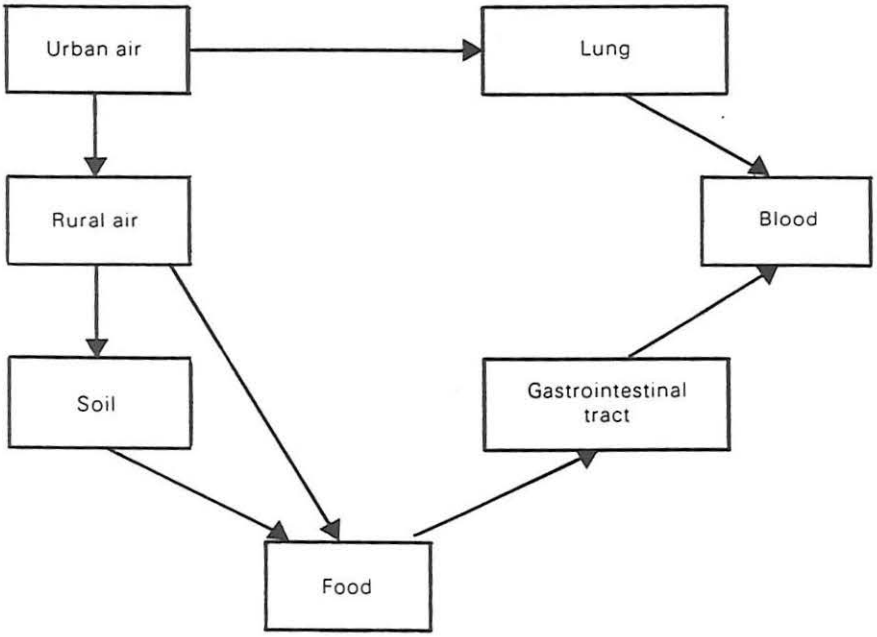
Exposure

Average lead levels are usually below 0.5 μg/m³ at nonurban sites. Urban air lead levels at sites close to streets are between 0.5 and 3 μg/m³ (annual means) in most European cities. Additional routes of exposure must not be neglected, e.g. lead in dust, a cause of special concern for children.

Quantitative relationships of multimedia exposure to blood lead

As shown in Fig. 1, air lead not only enters the body directly through the lungs but will also pass into other media which, in turn, are routes of human exposure.

Fig. 1. Air lead pathways contributing to blood lead levels



The relationships between air lead exposure and blood lead have been shown to exhibit downward curvilinearity if the range of exposures is sufficiently large (i.e. if it includes high levels). At lower levels of exposure the deviation from linearity is negligible and linear models of the relationship between intake and blood lead are as good as nonlinear model fits. For these lower ranges of the relationship (lead below $3\mu g/m^3$ in air and below $0.3\mu g/ml$ in the blood) various data show that a change of $1\mu g/m^3$ in air lead is associated with a blood lead change of $0.01–0.02\mu g/ml$ *(1)*. However, this relationship is only valid for adults. On the basis of published studies it is not possible to make a reliable quantitative estimate of the relationship between air lead and children's blood lead.

Quantification of the relationship between lead in other media affected by air lead and blood lead is not well understood, but a multimedia modelling approach may be employed to obtain rough estimates of such inputs.

Dose–effect relationships

It is generally accepted that the level of lead in blood is the best indicator of current exposure and a reasonably good indicator of the lead body-burden. It is therefore useful to relate the biological effects of lead to blood lead as an indicator of internal exposure.

Table 3 summarizes the threshold effect levels for haematological and neurological parameters in adults. Whereas clinically defined and severe anaemia does not occur at blood lead levels below 0.8 μg/ml, elevated erythrocyte protoporphyrin levels have been observed at blood lead levels of above 0.2 μg/ml in men and of 0.15–0.2 μg/ml in women. Neurological and neuropsychological effects of questionable validity have been found in lead workers at blood lead levels of 0.4–0.6 μg/ml, whereas in one study a decrease of nerve conduction velocity has been found to start at blood lead levels of about 0.3 μg/ml.

Table 4 summarizes threshold effect levels for haematological and neurological parameters in children. Whereas anaemia has not been observed at

Table 3. Summary of lowest-observed-effect levels for lead-induced health effects in adults

Lowest-observed-effect levels of blood lead	Haem synthesis and haematological effects	Effects on nervous system
1.0 –1.2 μg/ml		Encephalopathic signs and symptoms
0.8 μg/ml	Frank anaemia	
0.6 μg/ml		---------- ↑
0.5 μg/ml	Reduced haemoglobin production	Overt subencephalopathic neurological symptoms
0.4 μg/ml	Increased urinary ALA and elevated coproporphyrin	↓ ---------- ↑
0.3 μg/ml		Peripherical nerve dysfunction (slowed nerve conduction velocities)
0.2 –0.3 μg/ml	Erythrocyte protoporphyrin elevation in males	↓ ?
0.15–0.2 μg/ml	Erythrocyte protoporphyrin elevation in females	
0.1 μg/ml	ALAD inhibition	

Table 4. Summary of lowest-observed-effect levels for lead-induced health effects in children

Lowest-observed-effect levels of blood lead	Haem synthesis and haematological effects	Effects on nervous system
0.8 -1.0 μg/ml		Encephalopathic signs and symptoms
0.7 μg/ml	Frank anaemia	
0.6 μg/ml		
0.5 μg/ml		
0.4 μg/ml	Increased urinary ALA and elevated coproporphyrin	
0.25-0.3 μg/ml	Reduced haemoglobin synthesis	Cognitive CNS deficit Peripheral nerve dysfunction (slowed nerve conduction velocities)
0.1 -0.2 μg/ml	Erythrocyte protoporphyrin elevation	CNS electrophysiological changes
0.1 μg/ml	ALAD inhibition	----- ? -----

blood lead levels below 0.7 μg/ml, haemoglobin reduction was found at blood lead levels of about 0.25 μg/ml, and erythrocyte protoporphyrin elevation at blood lead levels between 0.15 and 0.2 μg/ml. Neuropsychological effects, although not universally agreed upon, have been observed at blood lead levels between 0.2 and 0.5 μg/ml, and electrophysiological findings of unknown clinical significance as well as attentional deficits have been found at blood lead levels below 0.3 μg/ml. Dose–response relationships have been established for erythrocyte protoporphyrin elevation in children *(32)*, elevation of free erythrocyte protoporphyrin in children and adults (females and males) *(30)*, urinary ALA increase and ALAD inhibition in children *(31)*.

No comparable dose–response relationships can as yet be given for measures of neurological and/or neuropsychological outcome.

Health risk evaluation

The decisive parameter upon which the guidelines for lead in air should be based is the concentration of lead in blood. After a review of the array of

toxic effects of lead, weight was given to the elevation of erythrocyte protoporphyrin. In selecting the appropriate limit value for lead in blood, a value of 0.2 μg/ml may be regarded as the borderline dividing the no-adverse-effect level from the lowest-adverse-effect levels. At this level, the increase of free erythrocyte protoporphyrin starts in a small percentage of adult subjects. At concentrations slightly higher, the whole chain of effects may become apparent, including haemoglobin decrease (in children only), subtle neurological changes, and disturbances in vitamin D levels.

On the other hand, at levels lower than 0.2 μg/ml the most sensitive effect is manifested (inhibition of ALAD activity); this, however, was not accepted as the basis, since it was judged to be a subcritical effect of no clear biological significance.

Certain lead compounds have been found to be carcinogenic in rats and mice after oral exposure. There is inadequate evidence of lead being carcinogenic in humans. Accordingly, IARC has classified lead in Group 3.

The following assumptions are made in constructing the guideline value.

Adult urban population

1. Ninety-eight per cent of the adult population should have blood lead levels below 0.2 μg/ml.

2. From the frequency distribution of blood lead levels in the general population it can be calculated that the median value, corresponding to a 98th percentile of 0.2 μg/ml, is approximately 0.1 μg/ml.

3. There is a "baseline" level of blood lead, which can be taken as either nonanthropogenic in origin or as resulting from relatively minimal anthropogenic lead exposure. Based on the population data of three large cities in which low values of blood lead were reported *(54)* and data from rural areas in Japan *(55)*, the "baseline" level of blood lead seems to be, on average, around 0.04–0.06 μg/ml. It is somewhat lower in women than in men. The distance between the upper limit of tolerable median (0.1 μg/ml) and the average "baseline" level of blood lead is thus 0.04–0.06 μg/ml.

4. As shown in Table 1, it can be assumed that, at a typical urban concentration of lead in air of the order of 1 $\mu g/m^3$, the contribution of direct inhalation to the total lead absorption of adults is approximately 40%. This percentage indicates that inhalation of airborne lead is an important route of exposure in adult humans.

5. The WHO Environmental Health Criteria document on lead *(5)* explains that in experimental conditions, where one deals with direct inhalation only, 1 μg per m^3 air contributes to 0.01–0.02 μg per ml blood. The upper figure is used hereafter as a more conservative one. By simple conversion 0.04–0.06 μg/ml corresponds to 2–3 $\mu g/m^3$.

6. It must then be taken into account that in real-life situations an increase of lead in air also contributes to increased lead uptake by indirect

ways, through other environmental pathways. To allow for uptake by other routes also, a recalculation factor of 5 : 1 is introduced (i.e. 1 μg/m^3 would contribute to 0.05 μg/ml). This relationship, then, already includes a certain protection factor.

7. In order to ensure that any anthropogenic input of lead into the blood should be limited so as not to exceed a value of 0.2 μg/ml in 98% of the adult population and the corresponding median value of 0.1 μg/ml, it would be reasonable that air lead, measured as a long-term average value, should not exceed 1 μg/m^3.

8. Taking into account some uncertainties that are inherent in the above evaluation, a guideline in a range of values 0.5–1.0 μg/m^3, measured as long-term average values in urban areas, is recommended.

Children of preschool age

Children up to 6 years of age are a population at increased risk for lead exposure as well as for adverse health effects, for the following reasons.

1. Children have behavioural characteristics (outdoor activity, less concern for hygienic conditions, mouth activities or even pica), which increase the risk of lead exposure.

2. Children eat and drink more per unit of body weight than adults, so that their relative lead intake is increased.

3. Lead absorption in the gastrointestinal tract is substantially higher in children (about 50%, compared with about 10% in adults) *(9, 10)*.

4. Among children there is a greater prevalence of nutritional deficiencies (e.g. iron and vitamin D), which enhance absorption of lead from the gastrointestinal tract.

5. The blood–brain barrier is not yet fully developed in young children.

6. Haematological and neurological effects of lead occur at lower thresholds in children than in adults.

Since the placenta is no effective biological barrier, pregnant women represent a second group at increased risk because of exposure of the fetus to lead.

In establishing guidelines for the critical group of children of preschool age, the following reasoning can be applied.

1. As seen from Table 1, the contribution of direct inhalation to total lead absorption, at an air concentration of 1 μg/m^3, is smaller than in adults (approximately 6% only). Most of the absorbed dose can be accounted for by other pathways, mainly food. In addition, the specific behaviour of children entails an additional input in the form of dust (dirt) passing by hand to the mouth.

2. Uncertainties connected with numerical values of transfer coefficients are too great to calculate alternative pathways of lead uptake from other sources and the relationships between lead in other media affected by air lead on the one hand and blood lead on the other.

3. Since in children the estimated contribution of direct inhalation is smaller compared with adults, decreases in air lead will have correspondingly little direct effect. Therefore, guideline values for adults should be used for the protection of the whole population, including children.

Because of the limited direct influence of atmospheric lead on blood lead levels in children, the air guidelines for lead should not be regarded as an adequate way of protecting this population. Other measures may need to be taken in parallel. These should specifically take the form of monitoring the lead content of dust and soils arising from air lead fallout. The normal behaviour of children with regard to dust and soil defines these media as potentially serious exposure sources. An additional source may be lead from paint. A specific monitoring value is not recommended. It should be mentioned, however, that the Greater London Council has selected an external dust lead value of 500 μg per gram dust as a basis for further evaluation and a value of 5000 μg per gram dust as the basis for implementing control strategies. In the Federal Republic of Germany, the lead fallout has been limited to 250 $\mu g/m^2$ per day. According to a recent study *(14)*, this limit is sufficient to prevent excessive intake of lead from environmental dust by children.

Guidelines

A guideline in the range of 0.5–1.0 μg lead per m^3 (long-term average, e.g. annual mean) is recommended; this incorporates a protection (safety) factor close to 2.

This guideline value is based on the assumption that 98% of the general population will be maintained below a blood lead level of 0.2 μg/ml, predicated on the selection of elevated erythrocyte protoporphyrin as the effect. This particular blood level was carefully considered, but it was recognized that other starting-points in terms of both effects and statistical limits might be chosen. In that case, another air lead guideline might result.

Because of the limited influence of atmospheric lead on the overall exposure of children, air quality guidelines are not sufficient to protect this population, and other initiatives may be necessary.

The monitoring of lead deposition in dust and soil in areas near point emissions and in high-density traffic areas is advisable in order to protect young children from excessive exposure and consequent adverse health effects.

References

1. **Nriagu, J.O.** Lead in the atmosphere. *In:* Nriagu, J.O., ed. *The biogeochemistry of lead in the environment.* Amsterdam, Elsevier-North Holland, 1978, Part A, pp. 137–184.

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Introduction

Human beings need a continuous supply of food, air and water to exist. The requirements for air and water are relatively constant (10–20 m^3 and 1–2 litres per day respectively). Air and water are also used in industrial processes of energy conversion, in manufacturing, and for the removal of waste products, some of which may be injurious to human health. In a comprehensive set of guidelines for drinking-water developed by WHO *(1)*, guideline values were recommended for specified contaminants, a consistent process of assessment being used.

The WHO Regional Office for Europe has subsequently developed the present air quality guidelines for the European Region. The task of developing such guidelines is more difficult than that of drawing up drinking-water guidelines, since for air, unlike water, there is no centrally supplied and controlled source. The development of consistent rules for assessing 28 chemical air contaminants also posed a challenge.

These air quality guidelines should be seen as a contribution to the target on air pollution contained in WHO's regional strategy for health for all. This target states that "by 1995, all people of the Region should be effectively protected against recognized health risks from air pollution" *(2)*. Accordingly, "the achievement of this target will require the introduction of effective legislative, administrative and technical measures for the surveillance and control of both outdoor and indoor air pollution, in order to comply with criteria to safeguard human health" *(2)*.

Various chemicals are emitted into the air from both natural and man-made (anthropogenic) sources. The quantities may range from hundreds to millions of tonnes annually. Natural air pollution stems from various biotic and abiotic sources (e.g. plants, radiological decomposition, forest fires, volcanoes and other geothermal sources, emissions from land and water), leading to a natural background concentration that varies according to local sources or specific weather conditions. Anthropogenic air pollution has existed at least since people learned to use fire, but it has increased rapidly since industrialization began. The increase in air pollution as a consequence of the expanding use of fossil energy sources and the growth in the manufacture and use of chemicals has been accompanied by mounting public awareness of and concern about its detrimental effects on

health and the environment. Moreover, knowledge of the nature, quantity, physicochemical behaviour and effects of air pollutants has greatly increased in recent years. Nevertheless, more needs to be known. Certain aspects of the health effects of air pollutants require further assessment; these include newer scientific areas such as developmental toxicity. The proposed guideline values will undoubtedly be changed as future studies lead to new information.

The impact of air pollution is broad. In man, the pulmonary deposition and absorption of inhaled chemicals can have direct consequences for health. However, public health can also be indirectly affected by the deposition of air pollutants in plants, animals and the other environmental media, resulting in chemicals entering the food chain or being present in drinking-water and thereby constituting additional sources of human exposure. Furthermore, the direct effects of air pollutants on plants, animals and soil can influence the structure and function of ecosystems, including their self-regulation ability, thereby affecting the quality of life.

Although in recent decades major efforts have been made to reduce air pollution, the situation in the European Region is still not satisfactory. While air pollution has decreased and peak concentrations have been reduced in many larger cities and urban areas, the overall pollution in terms of the amounts of pollutants released into the atmosphere has often been only slightly reduced or has remained unchanged, and concentrations have even increased in some areas and for some pollutants *(2–5)*.

Many countries of the European Region encounter rather similar air pollution problems, partly because pollution sources are comparable, and in any case air pollution does not respect national frontiers. The subject of the transboundary medium- and long-range transport of air pollution has received increasing attention in Europe in recent years. International efforts to combat its consequences are under way, for instance within the framework of the Convention on Long-range Transboundary Air Pollution established by the United Nations Economic Commission for Europe *(6)*.

The task of reducing levels of exposure to air pollutants is a complex one. It begins with an analysis to determine which chemicals are present in the air, at what levels, and whether these levels of exposure are hazardous to human health and the environment. It must then be decided whether an unacceptable risk is present. When a problem is identified, mitigation strategies are developed and implemented so as to prevent excessive risk to public health in the most efficient way.

Analyses of air pollution problems are exceedingly complicated. Some are national in scope (e.g. definition of actual levels of exposure of the population, determination of acceptable risk, identification of the most efficient control strategies), while others are of a more basic character and are applicable in all countries (e.g. analysis of the relationships between chemical exposure levels, doses and their effects). The latter form the basis of the present guidelines.

The most direct and important source of air pollution affecting the health of many people is tobacco smoke. Even those who do not smoke may inhale the smoke produced by others ("passive smoking"). Indoor pollution

in general and occupational exposure in particular also contribute substantially to overall human exposure: indoor concentrations of nitrogen dioxide, carbon monoxide, respirable particulates, formaldehyde and radon are often higher than outdoor concentrations *(7)*.

Outdoor air pollution can originate from a single point source which may affect only a relatively small area. More often, outdoor air pollution is caused by a mixture of pollutants from a variety of diffuse sources, such as traffic and heating, and from point sources. Finally, in addition to those emitted by local sources, pollutants transported over medium and long distances contribute further to the overall level of air pollution.

The relative contribution of emission sources to human exposure to air pollution may vary according to regional and lifestyle factors. Although indoor air pollution will be of higher relevance than outdoor pollution as far as certain air pollutants are concerned, this does not diminish the importance of outdoor pollution. In terms of the amounts of substances released, the latter is far more important and may have deleterious effects on animals, plants and materials as well as adverse effects on human health.

Nature of the Guidelines

The primary aim of the air quality guidelines[a] is to provide a basis for protecting public health from adverse effects of air pollution and for eliminating, or reducing to a minimum, those contaminants of air that are known or likely to be hazardous to human health and wellbeing.

The guidelines are intended to provide background information and guidance to governments in making risk management decisions, particularly in setting standards, but their use is not restricted to this. They also provide information for all who deal with air pollution. The guidelines may be used in planning processes and various kinds of management decision at community or regional level. When guideline values are indicated, this does not necessarily mean that they must take the form of general countrywide standards, monitored by a comprehensive network of control stations. In the case of some agents, guideline values may be of use mainly for carrying out local control measures around point sources.

It should be emphasized that when air quality guideline values are given, these values are not standards in themselves. Before standards are adopted, the guideline values must be considered in the context of prevailing exposure levels and environmental, social, economic and cultural conditions *(1)*. In certain circumstances there may be valid reason to pursue policies which will result in pollutant concentrations above or below the guideline values.

Ambient air pollutants can cause several significant effects which require attention: irritation, odour annoyance, acute and long-term toxic effects (including carcinogenic effects). Air quality guidelines either indicate levels combined with exposure times at which no adverse effect is expected concerning noncarcinogenic endpoints, or they provide an estimate of lifetime

[a] Guidelines in the present context are not restricted to suggested numerical values, but also include any kind of recommendation or guidance in the relevant field.

cancer risk arising from those substances which are proven human carcinogens or carcinogens with at least limited evidence of human carcinogenicity (see p. 12).

The guidelines represent the current best scientific judgement, but there is a need for periodic revision, since much remains to be determined regarding the toxicity of air pollutants for humans.

It is believed that inhalation of an air pollutant in concentrations and for exposure times below a guideline value will not have adverse effects on health and, in the case of odorous compounds, will not create a nuisance of indirect health significance (see definition of health, *Constitution of the World Health Organization*). Compliance with recommendations regarding guideline values does not guarantee the absolute exclusion of effects at levels below such values. For example, highly sensitive groups especially impaired by concurrent disease or other physiological limitations may be affected at or near concentrations referred to in the guideline values. Health effects at or below guideline values can also result from combined exposure to various chemicals or from exposure to the same chemical by multiple routes.

It is important to note that guidelines have been established for single chemicals. Chemicals, in mixture, can have additive, synergistic or antagonistic effects; however, knowledge of these interactions is still rudimentary. With a few exceptions, such as the combined effect of sulfur dioxide and particulates, there is insufficient information at present to establish guidelines for mixtures. An adequate margin of safety should exist between the guideline values and concentrations at which toxic effects will occur.

Risk estimates for carcinogens do not indicate a safe level; they are presented so that the carcinogenic potencies of different carcinogens can be compared and an assessment of overall risk made.

Although health effects were the major consideration in establishing the guidelines, ecologically based guidelines for preventing adverse effects on terrestrial vegetation were also considered and guideline values were recommended for a few substances. These ecological guidelines for vegetation have been established because, in the long term, only a healthy total environment can guarantee human health and wellbeing (see p. 17). Ecological effects on species other than plants have not been discussed, since they are outside the scope of this book.

The guidelines do not differentiate between indoor and outdoor exposure (with the exception of exposure to mercury) because, although the sites influence the type and concentration of chemicals, they do not directly affect the basic exposure–effect relationship. Occupational exposure has been considered in the evaluation process, but it was not a main focus of attention as these guidelines relate to the general population. However, it should be noted that occupational exposure may add to the effects of environmental exposure.

The guidelines do not apply to very high short-term concentrations which may result from accidents or natural disasters.

The health effects of tobacco smoking have not been assessed here, the carcinogenic effects of smoking having recently been evaluated by IARC (*8* and see Annex 1). Neither have the effects of air pollutants on climate

been considered, since too many uncertainties remain to allow an evaluation of the possible adverse health and environmental effects. However, possible changes of climate have to be investigated very seriously by the appropriate bodies because their overall consequences, for example the "greenhouse effect", may go beyond direct adverse effects on human health or ecosystems.

Procedures used in Establishing the Guidelines

The first step in the process of establishing air quality guidelines was the selection of pollutants. Air pollutants of special environmental and health significance to countries of the European Region were identified and selected on the basis of the following criteria suggested by a WHO Scientific Group *(9)*:

(*a*) severity and frequency of observed or suspected adverse effects on human health, where irreversible effects are of special concern;

(*b*) ubiquity and abundance of the agent in man's environment, with emphasis on air pollutants;

(*c*) environmental transformations or metabolic alterations, as these alterations may lead to the production of chemicals with greater toxic potential;

(*d*) persistence in the environment, particularly if the pollutant would resist environmental degradation and accumulate in humans, the environment or food chains; and

(*e*) population exposed (size of exposed population and special groups at risk).

Other factors affecting the selection were the timetable of the project and the fact that only those substances could be considered for which sufficient documentation was available (such as the WHO *Environmental health criteria* documents). On the basis of these criteria, the following 28 pollutants were selected for evaluation.

Organic air pollutants

Acrylonitrile
Benzene
Carbon disulfide
1,2-Dichloroethane
Dichloromethane
Formaldehyde
Polynuclear aromatic hydrocarbons (carcinogenic fraction)
Styrene
Tetrachloroethylene
Toluene
Trichloroethylene
Vinyl chloride

Inorganic air pollutants

Arsenic
Asbestos
Cadmium
Carbon monoxide
Chromium
Hydrogen sulfide
Lead
Manganese
Mercury
Nickel
Nitrogen oxides
Ozone/photochemical oxidants
Particulate matter
Radon
Sulfur oxides
Vanadium

After a planning meeting in early 1984 that offered suggestions on content, format, workplan and timetables for the air quality guidelines project, a series of nine meetings involving more than 130 experts took place to evaluate various air pollutants (see Annex 2).

Before the meeting of each working group, scientific background documents were prepared as a basis for discussion and for establishing the guidelines. After each meeting, a text on the individual pollutant or pollutant group was drafted on the basis of the amended background documents, incorporating the working group's conclusions and recommendations. The drafts were then circulated to all participants of the meetings for their comments and corrections. An editorial consultation group of scientists was then convened to review the documents for clarity of presentation, adequacy of description of the rationale supporting each guideline and consistency in the application of criteria. Certain sections in which inconsistencies were noted were again submitted for review, whereupon the final draft was prepared and submitted for extramural review; it was sent to the governments of Member States of the Region, and to organizations and individuals engaged in air quality research or management. The process concluded with a review in a final meeting, at which the recommendations and conclusions of all the working groups were submitted for final appraisal.

References

1. *Guidelines for drinking-water quality. Vol. 1. Recommendations.* Geneva, World Health Organization, 1984.
2. *Targets for health for all.* Copenhagen, WHO Regional Office for Europe, 1985.
3. **United Nations Economic Commission for Europe.** *Airborne sulphur pollution: effects and control.* New York, United Nations, 1984 (Air Pollution Series, No. 1).
4. **International Register of Potentially Toxic Chemicals.** *List of environmentally dangerous chemical substances and processes of global significance: scientific monographs.* Geneva, United Nations Environment Programme, 1984 (UNEP Report No. 2).
5. *Pilot compendium of environmental data.* Paris, Organisation for Economic Co-operation and Development, 1984.
6. **United Nations Economic Commission for Europe.** Convention on Long-range Transboundary Air Pollution. *International digest of health legislation,* **30**(4): 965 (1979).
7. *Estimating human exposure to air pollutants. GEMS: Global Environmental Monitoring System.* Geneva, World Health Organization, 1982 (WHO Offset Publication, No. 69).
8. *Tobacco smoking.* Lyon, International Agency for Research on Cancer, 1986 (IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans, Vol. 38).
9. Background and purpose of the WHO environmental health criteria programme. *In: Mercury.* Geneva, World Health Organization, 1976 (Environmental Health Criteria, No. 1).

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Criteria used in establishing guideline values

Relevant information on the pollutants was carefully considered during the process of establishing guideline values. Ideally, guideline values should represent concentrations of chemical compounds in air that would not pose any hazard to the human population. However, the realistic assessment of human health hazards necessitates a distinction between absolute safety and acceptable risk. To aim at achieving absolute safety, one would need a detailed knowledge of dose–response relationships in individuals in relation to all sources of exposure, the types of toxic effect elicited by specific pollutants or their mixtures, the existence or nonexistence of "thresholds" for specified toxic effects, the significance of interactions and the variation in sensitivity and exposure levels within the human population. However, such comprehensive and conclusive data on environmental contaminants are not always available. Very often the relevant data are scarce and the quantitative relationships uncertain; scientific judgement and consensus therefore play an important role in establishing acceptable levels of population exposure. Value judgements are unavoidable, because terms such as "adverse" and "sufficient evidence" are not in themselves totally objective, their meaning being based on generally agreed judgements.

Although it may be accepted that a certain risk can be tolerated or is simply unavoidable, the risk within a population may not be equally distributed. There may be subpopulations which are at considerably higher risk from the same exposure. Therefore, groups at special risk in the general population must be taken specifically into account in the risk management process. Even if knowledge about groups with specific sensitivity is available, unknown factors may exist that change the risk in an unpredictable manner.

Criteria Common to Carcinogens and Noncarcinogens

Sources, levels and routes of exposure

Available data are provided on the current levels of human exposure to pollutants from all sources, including the air. Special attention is given to atmospheric concentrations in urban and in nonpolluted rural areas and in the indoor environment. Where appropriate, concentrations in the workplace are also indicated for comparison with environmental levels. To provide

information on the contribution from air in relation to all other sources, data on uptake by inhalation, ingestion from water and food, and dermal contact are given where relevant. However, for most chemicals, data on total human exposure are lacking to some extent.

Kinetics and metabolism

Available data on the toxicokinetics of distribution in humans and experimental animals are indicated for inter- and intraspecies extrapolation, especially to assess the magnitude of body-burden from long-term, low-level exposures and to characterize better the mode of toxic action. Data concerning the distribution of an agent in the body are important in determining the molecular or tissue dose to target organs. High-to-low-dose and interspecies extrapolations are more easily carried out using equivalent tissue doses. Metabolites are mentioned, particularly if they are known or believed to exert a greater toxic potential than the original agent. Additional data of interest include the rate of excretion and the biological half-life.

Criteria for Endpoints other than Carcinogenicity

For those compounds reportedly without carcinogenic effects (or for which data on carcinogenicity were lacking or insufficient), the starting-point for the derivation of guideline values was to define the lowest concentration at which effects are observed in humans, animals and plants. In doing so, an attempt was made to define a lowest-observed-adverse-effect level. The question whether the lowest-observed-effect level or the no-observed-effect level should be used instead is mainly a matter of availability of data. If a series of data fixes the lowest-observed-effect level and the no-observed-effect level, either of those levels might be used. The gap between the lowest-observed-effect level and the no-observed-effect level is among the factors included in judgements concerning the appropriate margin of protection. However, a single, free-standing no-observed-effect level which is not defined in reference to a lowest-observed-effect level or a lowest-observed-adverse-effect level is not conclusive. Opinions on this subject differ, but the working consensus was that the level of concern in terms of human health is more relatable to the lowest-observed-adverse-effect level; this level was therefore used whenever possible. In the case of irritant and sensory effects on humans, it is desirable where possible to determine the no-observed-effect level.

On the basis of the evidence concerning adverse effects, judgements about the protection factors (safety or uncertainty factors) needed to minimize health risks were made. Averaging times were included, since the time of exposure is critical in determining toxicity. Criteria applied to each of these key factors are described below.

Criteria for selection of a lowest-observed-adverse-effect level

The distinction between adverse and nonadverse effects poses considerable difficulties *(1)*. Any observable biological change may be considered an adverse effect under certain circumstances. The definition of an adverse

effect has been given as "any effect resulting in functional impairment and/or pathological lesions that may affect the performance of the whole organism or which contributes to a reduced ability to respond to an additional challenge" *(2)*. Even with such a definition, a significant degree of subjectivity and uncertainty remains. Ambient levels of major air pollutants frequently cause subtle effects that are typically detected only by sensitive methods. This makes it exceedingly difficult, if not impossible, to achieve a broad consensus as to which effects are adverse. To resolve this difficulty, data should be ranked in three categories.

1. Observations, even of potential health concern, which are single findings that have not been verified by other groups. Because of the lack of verification by other investigators, such data could not readily be used as a basis for guideline values. They do, however, indicate the need for further research and may be considered in evaluating a margin of protection.

2. A lowest-observed-effect level: such a level is represented by data which have been supported by other scientific information. When the results are in a direction that might result in pathological change, there is a higher degree of health concern. Scientific judgement based on all available health information is used to determine how effects in this category can be used in determining the pollutant level that is to be avoided so that excessive risk can be prevented.

3. A substantial change in the direction of pathological effects: these findings have had a major influence on guideline considerations.

Criteria for selection of protection factors

In previous evaluations by WHO, protection factors, usually called safety factors, have been applied to derive guidelines from accepted criteria for adverse effects on health *(3,4)*. The rationale has been that such a factor allows for a variety of uncertainties, for example, about possibly undetected effects on particularly sensitive members of the population, synergistic effects of multiple exposures, and the adequacy of existing data. Traditionally, the safety factor has been used to allow for uncertainties in extrapolation from animals to humans and from a small group of individuals to a large population *(1)*.

In these guidelines, the terms "protection factor" and "margin of protection" have been used in preference to "safety factor" or "margin of safety" because the word "safety" may convey to the public the impression of absolute freedom from risk; this goes beyond what is intended by scientists when they refer to safety factors. These factors are applied in guidelines for the protection of human health. They are not applied to ecological guidelines, because they already include a kind of protection factor with regard to the variety of species covered.

A wide range of factors for protecting human health is used in this book, based on scientific judgements concerning the interplay of various criteria. The decision process for developing protection factors has been complex, involving the transformation of mainly nonquantitative information into a single number expressing the judgement of a group of scientists.

Some of the factors which are taken into account in deciding the margin of protection can be grouped under the heading of scientific uncertainty. Uncertainty occurs because of limitations in the extent or quality of the data base. One can confidently set a lower margin of protection (i.e. use of a smaller number), when a large number of high quality, mutually supportive scientific experiments in different laboratories using different approaches clearly demonstrate the dose–response, including a lowest-observed-effect level and a no-observed-effect level. In reality, difficulties inherent in studying air pollutants and the failure to perform much needed and very specific research usually preclude this situation.

Where a protection factor was adopted in the air quality guidelines, the reasoning behind this factor is given in the scientific background information. As previously mentioned, exceeding a guideline value with an incorporated protection factor does not necessarily mean that adverse effects will result. However, the risk to public health will increase, particularly in situations where the most sensitive population group is exposed to several pollutants simultaneously. It is therefore necessary to exercise some kind of judgement regarding the size of the protection factor.

Groups within a population respond differently to pollutants *(5)*. Individuals with pre-existing lung disease, for instance, can be at higher risk from exposure to air pollutants than healthy people. Differences in response can be due to factors other than pre-existing health factors, such as age, sex, level of exercise taken, or to unknown factors. Thus, the population must be considered very heterogeneous in respect of response to air pollutants. Existing information does not allow adequate assessment of the proportion of the population that has enhanced response. However, an estimate of even a few per cent of the total population entails a large number of people.

Effects observed in laboratory animals in the absence of human studies generally require a larger protection factor, because humans may be more susceptible than laboratory animal species. Negative data from human studies will tend to reduce the magnitude of the protection factor. Also of importance are the nature and reversibility of the reported effect. A pollutant level producing slight alterations in physiological parameters requires a smaller protection factor than a pollutant level producing a clearly adverse effect. Scientific judgement about protection factors will also take into account the toxicology of pollutants, including the type of metabolites formed, variability in metabolism or response in humans suggesting hypersusceptible groups, and the likelihood that the compound or its metabolites will accumulate in the body. It is also important to consider the exposure level used in health studies and to make appropriate conversions to environmental situations.

It is obvious, therefore, that diverse factors must be taken into account in proposing a margin of protection. The protection factor cannot be assigned by a simple mathematical formula; it requires experience, wisdom and judgement.

Criteria for selection of averaging times

The development of toxicity is a complex function of the interaction between concentration and time of exposure. A chemical may cause acute,

minor, reversible effects after brief exposure and irreversible or incapacitating effects after prolonged exposure. Our knowledge is usually insufficient to delineate these concentration–time interrelationships. Therefore, expert judgement must be applied, based on the weight of the evidence available *(6)*. Generally, when short-term exposures lead to adverse effects, short-term averaging times are recommended. The use of a long-term average under such conditions would be misleading, since the typical pattern of repeated peak exposures is averaged over time and the risk manager has difficulty in deciding upon effective strategies. In other cases, exposure–response knowledge is sufficient to recommend a long-term average. This frequently occurs for chemicals that accumulate in the body over time, thereby resulting in adverse effects. In such cases, the integral of exposure can have more impact than the pattern of peak exposure.

It should be noted that these averaging times are based on effects. Therefore, if the guidelines are used as a basis for regulation, the regulator needs to select the most appropriate and practical standards in relation to the guidelines, without necessarily using the guidelines directly.

A similar situation occurs for effects on vegetation. Plants are generally damaged by short-term exposures to high concentration as well as by long-term exposures to low concentration. Therefore, both short- and long-term guidelines to protect plants are proposed.

Criteria for consideration of sensory effects

Some of the substances selected for evaluation have malodorous properties at concentrations far below those at which toxic effects occur. Although odour annoyance cannot be regarded as an adverse health effect in a strict sense, it affects the quality of life *(7)*. Therefore, odour threshold levels for such chemicals have been indicated where relevant and used as a basis for separate guideline values.

For practical purposes, the following aspects and respective levels were considered in the evaluation of sensory effects:

(*a*) intensity, where the *detection threshold level* is defined as the lower limit of the perceived intensity range (by convention the lowest concentration that can be detected in 50% of the cases in which it is present);

(*b*) quality, where the *recognition threshold level* is defined as the lowest concentration at which the sensory effect, e.g. odour, can be recognized correctly in 50% of the cases;

(*c*) acceptability and annoyance, where the *nuisance threshold level* is defined as the concentration at which not more than a small proportion of the population (less than 5%) experiences annoyance for a small part of the time (less than 2%); since annoyance will be influenced by a number of psychological and socioeconomic factors, a nuisance threshold level cannot be defined on the basis of concentration alone.

Criteria for Carcinogenic Endpoint

Cancer risk assessment is basically a two-step procedure, involving a qualitative assessment of how likely it is that an agent is a human carcinogen, and a quantitative assessment of the cancer rate the agent is likely to cause at given levels and durations of exposure *(8)*.

Qualitative assessment of carcinogenicity

The decision to consider a substance as a carcinogen is based on the qualitative evaluation of all available information on carcinogenicity, ensuring that the association is unlikely to be due to chance alone. Here the classification criteria of the International Agency for Research on Cancer have been applied *(9)*. These classify chemicals for carcinogenicity in the following way.

Group 1 — Proven human carcinogens. This category includes chemicals or groups of chemicals for which there is sufficient evidence from epidemiological studies to support a causal association between the exposure and cancer.

Group 2 — Probable human carcinogens. This category includes chemicals and groups of chemicals for which, at one extreme, the evidence of human carcinogenicity is almost sufficient, and those for which, at the other extreme, it is inadequate. To reflect this range, the category is divided into two subgroups according to higher (Group 2A) and lower (Group 2B) degrees of evidence.

Group 2A. This group is usually used for chemicals for which there is at least limited evidence of carcinogenicity in humans and sufficient evidence for carcinogenicity in animals.

Group 2B. This group is usually used for chemicals for which there is inadequate evidence of carcinogenicity in humans and sufficient evidence of carcinogenicity in animals. In some cases the known chemical properties of a compound and the results of short-term tests have allowed its transfer from Group 3 to Group 2B, or from Group 2B to Group 2A.

Group 3 — Unclassified chemicals. This group includes chemicals or groups of chemicals which cannot be classified as to their carcinogenicity in humans.

With regard to this classification scheme, it is likely that in the future some chemicals in Group 3 can be classified as noncarcinogenic.

It was concluded that the qualitative evaluation applied by IARC should serve as the baseline in establishing the air quality guidelines for carcinogens, and that the IARC categorization scheme should be used if no new divergent evidence was available. In this respect, it was decided that all chemicals categorized in Groups 1 and 2A, i.e. proven human carcinogens

and carcinogens with at least limited evidence of human carcinogenicity, should be treated as human carcinogens, and guidelines should be formulated accordingly, indicating only risk estimates. For chemicals classified in Group 2B (inadequate evidence in humans, sufficient evidence in animals) it was decided that, until new evidence appeared, guidelines would point out the carcinogenic effects in laboratory animals and cite risk estimates (if such estimates could be reasonably obtained) in the health risk evaluation part of the scientific background information. However, these risk estimates, based on animal data only, would not be incorporated in the guideline recommendations because of various uncertainties in this connection. Guideline values based on noncarcinogenic endpoints would be given for these pollutants.

Quantitative assessment of carcinogenic potency

The aim of risk assessment is to apply information available from very specific study situations (mainly occupational studies) to the general population in order to calculate the possible risk to the latter. Therefore, quantitative risk assessment or, more specifically, dose–response assessment generally includes the extrapolation of risk from relatively high dose levels (characteristic of animal experiments or occupational exposures), where cancer responses can be measured, to relatively low dose levels, which are of concern in environmental protection and where such risks are too small to be measured directly, either in animal studies or in epidemiological studies *(10)*.

The choice of the extrapolation model depends on the current understanding of the mechanisms of carcinogenesis *(11)*. No single mathematical procedure can be regarded as fully appropriate for low dose extrapolation. Methods based on a linear, nonthreshold assumption have been used at the international level *(10,12)* and the national level (various health assessment documents produced by the US Environmental Protection Agency (EPA) and the National Institute of Public Health, Netherlands) more frequently than models which assume a safe or virtually safe threshold.

In these guidelines the risk associated with lifetime exposure to a certain concentration of a carcinogen in the air has generally been estimated by linear extrapolation and the carcinogenic potency expressed as the incremental unit risk estimate. The incremental unit risk estimate for an air pollutant is defined as "the additional lifetime cancer risk occurring in a hypothetical population in which all individuals are exposed continuously from birth throughout their lifetimes to a concentration of 1 $\mu g/m^3$ of the agent in the air they breathe" *(13)*.

Calculations expressed in unit risk estimates provide the opportunity to compare the carcinogenic potency of different agents and can help to set priorities in pollution control according to the existing exposure situation. By using unit risk estimates, any reference to the "acceptability" of risk is avoided. The decision on the acceptability of a risk should be made by national authorities in the framework of risk management.

For those substances for which appropriate human studies are available, the method known as the "average relative risk model" *(14)* has been generally used and is therefore described in more detail below.

For animal cancer bioassays several methods have been used to estimate the incremental risks. Two general approaches have been proposed. A strictly linearized estimate has been used by US EPA generally *(11)*. Nonlinear relations have been proposed by others where either the concentration–tumour response was found experimentally or where metabolism is of limited capacity. Accordingly, risk estimates based on animal bioassays are considered separately.

Quantitative assessment of carcinogenicity based on human data

The quantitative assessment using the average relative risk model includes four steps: (*a*) selection of studies; (*b*) standardized description of study results in terms of relative risk, exposure level and duration of exposure; (*c*) extrapolation towards zero dose; and (*d*) application to a general (hypothetical) population.

First, a reliable human study must be identified, where the exposure of the study population can be estimated and the excess of cancer incidence is statistically significant. If several studies exist, the best representative study should be selected or several risk estimates evaluated.

When a study is identified, the relative risk (R) as a measure of response must be calculated. It is important to note that the 95% confidence limits around the central value for the relative risk can be wide and should be specifically stated and evaluated. The relative risk is then introduced in the following formula (average relative risk model) which combines steps (*c*) and (*d*) and allows the unit lifetime risk (UR) (i.e. risk associated with a lifetime exposure to 1 $\mu g/m^3$) to be calculated:

$$UR = \frac{P_o(R - 1)}{X}$$

where: P_o = background lifetime risk; this is taken from age/cause-specific death or incidence rates found in national vital statistics tables using the life table methodology, or it is available from a matched control population

R = relative risk, being the ratio between the observed (O) and expected (E) number of cancer cases in the exposed population; the relative risk is sometimes expressed as the standardized mortality ratio SMR = (O/E) × 100

X = lifetime average exposure (standardized lifetime exposure for the study population on a lifetime continuous exposure basis); in the case of occupational studies, X represents a conversion from the occupational 8-hour, 240-day exposure over a specific number of working years and can be calculated as X = 8-hour TWA × 8/24 × 240/365 × (average exposure duration [in years])/(life expectancy [70 years]), where TWA is the time-weighted average ($\mu g/m^3$).

It should be noted that the unit lifetime risk depends on P_o (background lifetime risk), which is determined from national age-specific cancer incidence or mortality rates. Since these rates are also determined by exposures other than the one of interest and may vary from country to country, it follows that the UR may also vary from one country to another.

Necessary assumptions for average relative risk method

Before any attempt is made to assess the risk in the general population, numerous assumptions are needed at each phase of the risk assessment process to fill in various gaps in the underlying scientific data base. Therefore, as a first step in any given risk assessment, an attempt should be made to identify the major assumptions that have to be made, indicating their probable consequences. These assumptions are as follows.

1. *The response (measured as relative risk) is some function of cumulative dose or exposure.*

2. *There is no threshold dose for carcinogens.*

Many stages in the basic mechanism of carcinogenesis are not yet known or are only partly understood. However, taking available scientific findings into consideration, several scientific bodies *(3,10,12,15–17)* have concluded that there is no scientific basis for assuming a threshold or no-effect level for chemical carcinogens. This view is based on the fact that most agents that cause cancer also cause irreversible damage to deoxyribonucleic acid (DNA). The assumption applies for all nonthreshold models.

3. *The linear extrapolation of the dose–response curve towards zero gives an upper-bound conservative estimate of the true risk function if the unknown (true) dose–response curve has a sigmoidal shape.*

The scientific justification for the use of a linear nonthreshold extrapolation model stems from several sources: the similarity between carcinogenesis and mutagenesis as processes which both have DNA as target molecules; the strong evidence of the linearity of dose–response relationships for mutagenesis; the evidence for the linearity of the DNA binding of chemical carcinogens in the liver and skin; the evidence for the linearity in the dose–response relationship in the initiation stage of the mouse 2-stage tumorigenesis model; and the rough consistency with the linearity of the dose–response relationships for several epidemiological studies *(10)*. This assumption applies for all linear models.

4. *There is constancy of the relative risk in the specific study situation.*

In a strict sense, constancy of the relative risk means that the background age/cause-specific rate at any time is increased by a constant factor. The advantage of the average relative risk method is that this needs to be true only for the average.

Advantages of the method

The average relative risk method was selected in preference to many other more sophisticated extrapolation models because it has several advantages,

the main one being that it seems to be appropriate for a fairly large class of different carcinogens, as well as for different human studies. This is possible because averaging doses, i.e. averaging done over concentration and duration of exposure, give a reasonable measure of exposure when dose rates are not constant in time. This may be illustrated by the fact that the use of more sophisticated models *(13, 14, 18, 19)* results in risk estimates very similar to those obtained by the average relative risk method.

Another advantage of the method is that the carcinogenic potency can be calculated when estimates of the average level and duration of exposure are the only known parameters besides the relative risk. Furthermore, the method has the advantage of being simple to apply, allowing non-experts in the field of risk models to calculate a lifetime risk from exposure to the carcinogens.

Limitations of the method

As pointed out earlier, the average relative risk method is based on several assumptions which appear to be valid in a wide variety of situations. However, there are specific situations in which the method cannot be recommended, mainly because the assumptions do not hold true.

The cumulative dose concept, for instance, is inappropriate when the mechanism of the carcinogen suggests that it cannot produce cancer throughout all stages of the cancer development process. Also, specific toxicokinetic properties, such as a higher excretion rate of a carcinogen at higher doses or a relatively lower production rate of carcinogenic metabolites at lower doses, may diminish the usefulness of the method in estimating cancer risk. Furthermore, supralinearity of the dose–response curve or irregular variations in the relative risk over time which cannot be eliminated would reduce the value of the model. However, evidence concerning these limitations either does not exist or is still too preliminary to make the average relative risk method inappropriate for carcinogens evaluated here.

A factor of uncertainty, rather than of methodological limitation, is that data on past exposure are nearly always incomplete *(12, 17)*. Although it is generally assumed that in the majority of studies the historical dose rate can be determined within an order of magnitude, there are possibly greater uncertainties, even of more than two orders of magnitude, in some studies. In the risk assessment process it is of crucial importance that this degree of uncertainty be clearly stated. This is often done simply by citing upper and lower limits of risk estimates *(12)*. Duration of exposure and the age- and time-dependence of cancer caused by a particular substance are less uncertain parameters, although the mechanisms of relationship are not so well understood *(8)*.

Risk estimates from animal cancer bioassays

Animal bioassays of chemicals provide important information on the human risk of cancer from exposure to chemicals. These data enhance our confidence in assessing human cancer risks on the basis of epidemiological data.

Several chemicals considered in this volume have been studied using animal cancer bioassays. The process is continuing and new information on

the potential carcinogenicity of chemicals is rapidly appearing. Consequently, the status of chemicals is constantly being reassessed.

During the preparation of this book, dichloromethane was classified by IARC as showing sufficient evidence of carcinogenicity in animals, on the basis of studies in rats and mice *(20)*. Detailed studies of the kinetics of metabolism of dichloromethane have also recently been completed, indicating that the capacity of mammals to metabolize dichloromethane is limited. Thus, the extent of metabolism of dichloromethane at high doses where cancer bioassays are conducted is less than at levels of environmental exposure. The linearized cancer risk models may therefore represent considerable overestimates of the carcinogenic potential of dichloromethane in humans at levels likely to occur in the environment. As with dichloromethane, there are also considerable uncertainties in establishing human risk estimates derived from animal data for formaldehyde and 1,2-dichloroethane. Therefore, the significance of such estimates is still very problematic.

There is little doubt of the importance of animal bioassay data in reaching an informed decision on a chemical. The collection and use of data such as those on saturation mechanisms, absorption, deposition and metabolic pathways, as well as on interaction with other chemicals, is important and should be continued. Regrettably, these data were not available for the above-mentioned chemicals at the time of this evaluation of guidelines for air pollutants. The process of evaluating guidelines and the impact of human exposure to these chemicals should continue and be revised as new information becomes available.

Interpretation of risk estimates

The risk estimates presented in this book should *not* be regarded as being equivalent to the true cancer risk. Quantitative risk estimates can provide policy-makers with rough estimates of risk which may serve well as a basis for setting priorities, balancing risks and benefits, and establishing the degree of urgency of public health problems among subpopulations inadvertently exposed to carcinogens *(11)*.

Ecological Effects

The importance of taking an integrated view of both health and ecological effects in air quality management was recognized from the beginning of the project. Ecological effects may have a significant indirect influence on human health and wellbeing. For example, most of the major urban air pollutants are known to have adverse effects at low levels on plants, including food crops. A consultation group was therefore convened to consider ecological effects of sulfur oxides, nitrogen oxides and ozone/photochemical oxidants on terrestrial vegetation. These substances are important both because of the high anthropogenic amounts produced and because of their wide distribution. They deserve special attention because of significant adverse effects on ecological systems in concentrations far below those known to be harmful to humans.

The pollutants selected for consideration here form only part of the vast range of air pollutants that have ecological effects. The project timetable permitted only an evaluation of adverse effects on terrestrial plant life, although effects on animal and aquatic ecosystems are also of great concern in parts of Europe. Nevertheless, even this limited evaluation clearly indicates the importance attached to the ecological effects of such pollutants in the European Region.

References

1. *Principles and methods for evaluating the toxicity of chemicals. Part I.* Geneva, World Health Organization, 1978 (Environmental Health Criteria, No. 6).
2. **US Environmental Protection Agency.** Guidelines and methodology used in the preparation of health effect assessment chapters of the consent decree water quality criteria. *Federal register,* **45**: 79347-79357 (1980).
3. *Guidelines for drinking-water quality. Vol. 1. Recommendations.* Geneva, World Health Organization, 1984.
4. **Vettorazzi, G.** *Handbook of international food regulatory toxicology. Vol. 1. Evaluations.* New York, SP Medical and Scientific Books, 1980.
5. *Guidelines on studies in environmental epidemiology.* Geneva, World Health Organization, 1983 (Environmental Health Criteria, No. 27).
6. WHO Technical Report Series, No. 506, 1972 (*Air quality criteria and guides for urban air pollutants:* report of a WHO Expert Committee).
7. *Sulfur oxides and suspended particulate matter.* Geneva, World Health Organization, 1979 (Environmental Health Criteria, No. 8).
8. **Peakall, D.B. et al.** Methods for quantitative estimation of risk from exposure to chemicals. *In:* Vouk, V.B. et al., ed. *Methods for estimating risk of chemical injury: human and non-human biota and ecosystems.* New York, John Wiley & Sons, 1985.
9. *Chemicals, industrial processes and industries associated with cancer in humans. IARC Monographs, Volumes 1 to 29.* Lyon, International Agency for Research on Cancer, 1982 (IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans, Supplement 4).
10. *Arsenic.* Geneva, World Health Organization, 1981 (Environmental Health Criteria, No. 18).
11. **Anderson, E.L.** Quantitative approaches in use in the United States to assess cancer risk. *In:* Vouk, V.B. et al., ed. *Methods for estimating risk of chemical injury: human and non-human biota and ecosystems.* New York, John Wiley & Sons, 1985.
12. *Some industrial chemicals and dyestuffs.* Lyon, International Agency for Research on Cancer, 1982 (IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans, Vol. 29).
13. *Health assessment document for nickel.* Research Triangle Park, NC, US Environmental Protection Agency, 1985 (Final report No. EPA-600/8-83-012F).

14. *Health assessment document for acrylonitrile.* Washington, DC, US Environmental Protection Agency, 1983 (Final report No. EPA-600-8-82-007F).
15. **National Research Council.** *Drinking water and health.* Washington, DC, National Academy of Sciences, 1977.
16. **Anderson, E.L. et al.** Quantitative approaches in use to assess cancer risk. *Risk analysis,* **3**: 277–295 (1983).
17. *Risk assessment and risk management of toxic substances. A report to the Secretary, Department of Health and Human Services.* Washington, DC, US Department of Health and Human Services, 1985.
18. *Health assessment document for chromium.* Research Triangle Park, NC, US Environmental Protection Agency, 1984 (Final report No. EPA-600-8-83-014F).
19. *Health assessment document for inorganic arsenic.* Research Triangle Park, NC, US Environmental Protection Agency, 1984 (Final report No. EPA-600-8-83-021F).
20. *Some halogenated hydrocarbons and pesticide exposures.* Lyon, International Agency for Research on Cancer, 1986 (IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans, Vol. 41).