Methods of approximation and determination of human vulnerability for offshore major accident hazard assessment

Contents

Introduction

1 This appendix provides a summary of information relating to the effects of the hazards offshore personnel may be exposed to in the event of an incident and is intended for use in the preparation and evaluation of risk assessments.

2 In the assessment of survivability or fatality probability, during a major accident on an offshore or onshore installation, it is important to take into account the following factors:

- Information prior to fire (alarms)
- Development of incidents
- Reaction times of personnel
- Emergency procedures and preparedness
- Escape time and distance to safety
- Type of hazard (toxic gas, thermal radiation, blast etc.)
- Protection and attenuation effects (i.e. shielding or reflection)
- Harm levels as a function of time (dose)
- Total exposure time (accumulated dose)
- Other critical aspects like visibility, toxic gases, explosion loads etc

Estimation of harm

3 In order to estimate the level of harm from a hazardous agent it is necessary to provide a means to quantify the exposure in terms of the intensity, duration of exposure and consequences of effect. This is usually achieved by an estimation of the received dose and a comparison of this against, statistically manipulated, experimental data to determine the probability of harm to an exposed population or individual. Vulnerability criteria can be established to determine dose levels that result in specific consequences. In this guidance the indicative criteria provides:

- The threshold of harm above which, protection is required to prevent impairment of the functions an individual requires for escape or to avoid becoming a fatality (i.e. survivability) and,
- A means for the estimation of fatality probability should dose levels exceed the harm threshold and adequate protection is not present.

4 There are two main approaches for the determination of the effects of received dose: the use of Probit Functions and the Determination of Harmful Dose (typically applied to toxic or thermal hazards)

Probits (probit functions)

5 Probits account for the variation in tolerance to harm for an exposed population. The fatality rate of personnel exposed to harmful agents over a given period of time can be calculated by use of probit functions that typically take the form:

$$\mathbf{Y = k_1 + k_2(\ln V)} \qquad \textbf{(Equation 1)}$$

Where:

Y = probit, (value range 2.67 – 8.09 representing 1 – 99.9% fatality) a measure of the percentage of the vulnerable resource that might sustain damage. Fatality probability can then be determined by evaluation of Y on a probit transformation chart such as that provided by Finney 1971 (see Annex 2).

$k_1 + k_2$ = Constants

V = the product of intensity or concentration of received hazardous agent to an exponent “n” and the duration of exposure in seconds or minutes

For thermal radiation, $V = I^{4/3}t$ and is called the thermal dose, with units $(kW/m^2)^{4/3}$ **seconds**.

6 A modified thermal dose unit, V’, may be provided where account is taken of additional protection or other mitigation.

When applied to thermal radiation hazards V' (= Φ. V) and is called the effective dose. Φ equals a factor that may be included to account for such issues as the variation in skin area exposed to thermal radiation. (i.e. 0.5 for normally clothed population and 1.0 if clothing has been ignited (Lees 1994).

7 For toxic gas V equals the product of gas concentration to an exponent "n" and time in **minutes**. Concentration can be reported in parts per million (ppm) or milligrams per cubic metre (mg/m^3), thermal radiation probits may use radiation units of watts or kilowatts so care is needed to ensure the correct probit is applied with the correct units.

Probits may be obtained for almost any hazardous agent but are typically available for thermal radiation, toxic gas and blast overpressure effects.

For example, several probit functions have been developed for NH_3, SO_2, and HF. The following are examples commonly used for each gas where the probit equation described earlier takes the form:

$$Y = k_1 + k_2 \ln(C^n x t) \qquad \textbf{(Equation 2)}$$

Where;

C = hazard concentration (ppm) and t = time in minutes

Table 1: Probits for hazardous gases taken from NORSOK Z013 (DNV / Scanpower)

Substance	**K_1**	**K_2**	**n**	**LC_{50} 5 min exposure**
Ammonia NH_3	-9.82	0.71	2	15240
Sulphur Dioxide SO_2	-15.67	2.1	1	3765
Hydrogen Fluoride HF	-48.33	4.853	1	11845

8 There are many published probits for estimating fatality levels from exposure to harmful agents for example the values listed in Table 2 are taken from Lees (2005) using the Perry & Articola (1980) values for Ammonia, Chlorine and Hydrogen Fluoride. Many probit sets have been produced by various sources including Louvar J.F. and Louvar B.D. (1998) and the "Green Book" (1992) and there can be significant variation in the dose effect estimates for each probit equation for the same hazard.

Table 2: Sample probits taken from Lees (2005) and estimated dose effects.

				LC_1 (ppm)		**LC_{50} (ppm)**	
Material	**K_1**	**K_2**	**n**	**5 min**	**30 min**	**5 min**	**30 min**

Acrolein	-9.93	2.05	1.0	93	16	291	48
Ammonia	-35.9	1.85	2.0	15057	6147	28264	11539
Benzene	-109.78	5.3	2.0	18096	7388	22545	9204
Carbon monoxide	-37.98	3.7	1.0	11810	1968	22169	3695
Chlorine	-8.29	0.92	2.0	173	71	613	250
Hydrogen chloride	-16.85	2.0	1.0	3464	577	11106	1851
Hydrogen sulphide	-31.42	3.008	1.43	897	256	1543	441
Nitrogen dioxide	-13.79	1.4	2	160	65	367	150
Phosgene	-19.27	3.686	1.0	77	13	145	24
Sulphur dioxide	-15.67	2.1	1.00	1241	207	3764	627
Toluene	-6.794	0.41	2.5	5352	2614	51965	25377
Hydrogen Fluoride	-35.87	3.354	1	19652	3260	39184	6531
Hydrogen Cyanide	-29.42	3.008	1.43	564	161	969	277

9 Probit Analysis is an approximate methodology but it does allow quantification of consequence resulting from exposure. However, care must be taken in probit equation selection as the estimate fatality probability can vary significantly for the same hazardous agent depending on the probit selected. This is demonstrated later [see para 51]. Care must also be taken to ensure the units of concentration are appropriate for the probit equation used.

Specified level of toxicity (SLOT) or significant likelihood of death (SLOD)

10 The probit approach is one means of estimating the level of fatality for exposure to a hazardous agent and an alternative is the application of the Specified Level of Toxicity (SLOT) or Significant Likelihood Of Death (SLOD) approaches proposed by HSE.

11 The SLOT approach is described by Turner and Fairhurst (1993) and involves the use of the most relevant toxicity data available that is then extrapolated for use on man. In its usual application the estimated dose is termed SLOT Dangerous Toxic Load (or SLOT DTL) .The concept of SLOD for use in group risk analysis is proposed by Franks, Harper and Bilio (1996) and this estimates the dose required to produce a “Significant Likelihood Of Death”.

12 The consequences of exposure such as inhalation of toxic chemicals, exposure to blast over pressure or exposure to thermal radiation are usually derived from available information, preferably human data or that obtained from animal experiments. The uncertainties in translating animal data to data relevant for humans are large and therefore "safety factors" may be included in the modelling. In general animals have a higher adsorption rate and humans have a higher respiratory rate in accident situations. For pragmatic reasons the SLOT DTL is usually taken to be equivalent to the $LC_{1\text{-}5}$ derived from animal experiments.

The SLOT DTL and SLOD approaches extrapolate toxicity data to determine a dangerous toxic load (A) that gives a specific level of harm for a certain received dose. These values are the product of exposure and time and usually take the form:

$$C^n t = A \qquad \textbf{(Equation 3)}$$

Where:

t = exposure time in minutes

C = concentration in ppm

n = an exponent for “C”

A = Dangerous Toxic Load (for SLOT DTL or “SLOD”)

The exponent “n” is derived from the extrapolation of the toxicity data used. The Units of “A” are **ppm**$^{\textbf{to an exponent “n”}}$ **x minutes**.

13 SLOT DTL is usually defined as the dose that results in highly susceptible people being killed and a substantial portion of the exposed population requiring medical attention and severe distress to the remainder exposed. And as such it represents the dose that will result in the onset of fatality for an exposed population (commonly referred to as LD_1 or $LD_{1\text{-}5}$)

14 SLOD is defined as the dose to typically result in 50% fatality (LD_{50}) of an exposed population and is the value typically used for group risk of death calculation onshore, typical values are shown in Table 3.

Table 3: Typical values of SLOT & SLOD DTL’s (see Eqn 3 for units)

Substance	SLOT	SLOD	“n”
Carbon monoxide	40125	57000	1
Carbon dioxide	1.5×10^{40}	1.5×10^{41}	8
Hydrogen sulphide	2×10^{12}	1.5×10^{13}	4
Ammonia	3.78×10^{8}	1.03×10^{9}	2

Sulphur dioxide	4.66×10^6	7.45×10^7	2
Hydrogen fluoride	1.2×10^4	2.1×10^4	1
Oxides of Nitrogen (as nitrogen dioxide)	9.6×10^4	6.24×10^5	2

(Further information for other substance values is available)

Considering Hydrogen Sulphide and the application of SLOT the table above provides the following:

$A = 2 \times 10^{12}$ for HSE "dangerous dose" ($LD_{1\text{-}5}$) or 1.5×10^{13} for SLOD (LD_{50})

And the exponent, n = 4
Application of the DTL equation provides the following exposure times:

Table 4: Hydrogen sulphide concentrations based on SLOT

Exposure time (minutes)	Average concentration to produce the effect	
	Dangerous dose	SLOD
5	795	1316
10	669	1107
15	604	1000
30	508	841
60	427	707

In the case of thermal radiation the dose unit is calculated as $(kW/m^2)^{4/3}$ **seconds**, dangerous dose and significant likelihood of death are set at 1000 and 2000 thermal dose units (tdu) respectively based on HSE report FS/03/04 (2004).

Immediate Dangerous to Life or Health (IDLH) concept

15 The acronym IDLH refers to a concentration, formally specified by a regulatory value, and defined as the maximum exposure concentration for a given chemical in the workplace from which one could escape within 30 minutes without any escape-impairing symptoms or any irreversible health effects. Exposure levels (as airborne concentrations) are specified by the U.S. National Institute for Occupational Safety and Health (NIOSH). As these criteria represent an exposure limit, beyond which impairment may be expected to occur, their use in risk assessment is to be expected.

16 Table 5 describes these limits for some hazardous substances typically encountered offshore, and compares them against the values expected to result in fatalities of the more vulnerable members of an exposed population estimated from the use of SLOT and the probit approaches. All values are presented for a 30-minute exposure interval. (For further details see Centers for Disease Control and Prevention)

Table 5: Comparison of IDLH and onset of fatality for probit and SLOT values

Substance	30 minute exposure period maximum exposure (ppm)			
	IDLH (NIOSH)	Probit (1%) fatality		SLOT for HSE Dangerous Dose (1% fatality)
		Lees 2005	NORSOK Z013	
Acrolein	2	16	N/a	14
Ammonia	300	1882	1206	3550
Carbon monoxide	1,200	1968	N/a	1338
Carbon Dioxide	40,000 (4%)	N/a	N/a	68,766
Hydrogen sulphide	100	256	N/a	508
Nitrogen dioxide	20 (as NO_2)	65	N/a	57
Sulphur dioxide	100	207	207	394
Hydrogen chloride	50	577	N/a	790
Hydrogen cyanide	50	161	N/a	80
Hydrogen Fluoride	30 (as F)	165	1221*	400

N/a: not available at the time of preparation

*: Probit values obtained appear very high compared with Lees & Dangerous dose values.

It can be seen from above that the use of IDLH as the limiting value for the onset of fatality has several consequences:

- Its use is limited to exposure of 30 minutes or less, use for a greater period of exposure would err in a non-conservative direction.
- It appears to produce results that are significantly conservative when compared with the probit or SLOT approaches.
- IDLHs are more suitable for use as a workplace risk management tool rather than in a major accident risk assessment.
- In all cases it does describe a lower limit for survivability that is reasonably conservative.

Note: The above suggestions and review does not take account of the relatively new US system of toxicity limits applicable to accidental short-term exposures. These are Acute Exposure Guideline Levels (AEGLs). AEGLs specify exposure concentrations for time periods ranging from 10 minutes to 8 hours that would not cause three levels of harm: discomfort, disability and death. AEGL values for several hundred compounds are now reported. These are intended to apply to the general population, which includes vulnerable groups such as the young and old. Consequently, AEGLs are rather conservative and may be unsuitable for the purpose of offshore risk assessment; AEGL-3 values in ppm (most severe of the categories) for 30 minute exposure to the substances listed in Table 5 are: acrolein (14), ammonia (1600), carbon monoxide (600), carbon dioxide (not listed), hydrogen sulphide (59), nitrogen dioxide (25), sulphur dioxide (30), hydrogen chloride (210), hydrogen cyanide (21) and hydrogen fluoride (62). Further information is available.

It should also be noted that there was a similar project underway to develop the methodology for similar acute exposure levels, known as Acute Exposure Threshold Levels (AETLs) in Europe. The methodology for setting AETLs was published in 2006. It is anticipated that these will incorporate a similar level of conservatism as the AEGLs. Draft AETLs were developed for about 20 chemicals as part of ACUTEX (Trainor et al, 2006), but these were never subject to peer review or finalised and the technical supporting documents are not publicly available. There has been no further progress since the ACUTEX project finished in 2006 and it is probable that this initiative is permanently stalled.

Blast overpressure

17 An assessor will meet a variety of blast and pressure units and the following table provides conversion between the most commonly used units:

Table 6: Conversion from one bar

Conversion from 1 bar pressure					
Pascal (pa)	**KiloPascal (Kpa)**	**Lbs / sq inch**	**Std Atmosphere**	**Newtons/ sq metre**	**Torr**
100,000	100	14.5038	0.986923	100,000	750.062

Direct effects of blast

18 The rapid compression and decompression of a blast wave on the human body results in transmission of pressure waves through the tissues. Resulting damage is primarily at junctions between tissues of different densities (bone and muscle), or at the interface between tissue and airspace. Lung tissue and the gastrointestinal system (both contain air) are particularly susceptible to injury. The tissue disruptions can lead to severe haemorrhage or to air embolism; either can be rapidly fatal. Direct overpressure effects do not extend out as far from the point of detonation as other effects and are often masked by the drag force effects. In the event of a vapour cloud

explosion, the overpressure levels necessary to cause injury to the public are typically defined as a function of peak overpressure, without regard to exposure time. Persons who are exposed to explosion overpressures have no time to react or take shelter; thus, time does not enter into the relationship. The HSC report into the transportation of dangerous goods by road & rail suggests Equation 4 as the probit relationship for blast over pressure fatality:

$Y = 1.47 + 1.37 \ln (P)$	Pressure in psig units	**(Equation 4a)**
$Y = 5.13 + 1.37 \ln (P)$	Pressure in barg units	**(Equation 4b)**

P = peak overpressure (barg)	1% fatality	0.17 barg (2.4 psig)
	50% fatality	0.90 barg (13.1 psig)
	95% fatality	3.00 barg (43.5 psig)

19 The main parts of the body directly susceptible to the damaging effects of overpressure are the eardrums and lungs. Lung damage can be fatal and an example of the consequences in terms of probability of injury or fatality, as suggested by the Australian Petroleum Production & Exploration Association Limited (APPEA) is shown in Table 7.

Table 7: Explosion overpressure effects

Overpressure (barg)	Consequence
0.210	20% probability of fatality to personnel inside 0% probability of fatality in the open
0.350	50% probability of fatality inside 15% probability of fatality in open
0.70	100% probability of fatality inside or in unprotected structures

20 The significance of the data provided in Table 7 is that the human body is relatively resistant to static overpressure compared to rigid structures such as buildings. For example, an un-reinforced cinder block panel will shatter at 0.1 to 0.2 atmospheres. While personnel offshore are typically out of doors, or inside the TR it would be expected that a lower fatality expectation exists than for a domestic situation. However, there are significant items of plant and equipment that would be available to provide missiles in the event of an explosion and consideration of data provided the following criteria is suggested for blast assessment of personnel outside the TR:

- Maximum survivable blast 0.17 - 0.21 barg
- 1% fatality 0.25 - 0.35 barg
- 50% fatality 0.5 - 1.0 barg

21 Overpressures lower than those in Table 7 can cause non-lethal injuries such as lung damage and eardrum rupture. Lung damage is a relatively serious injury, usually

requiring hospitalisation, even if not fatal; whereas eardrum rupture is a minor injury, often requiring no treatment at all.

The threshold level of overpressure for an un-reinforced, un-reflected blast wave that can cause lung damage is about 1.0 atmosphere.

A blast wave in the order of 0.25 bar (25 kPa) to 0.5 bar (50 kPa) is considered to be the range for the threshold for eardrum perforation. The overpressure associated with a 50 percent probability of eardrum rupture is about 1.0 bar.

22 The direct effect of explosion over pressure is normally displayed in the form of lethality as a function of overpressure and duration of the blast wave. Depending on the orientation of a body to a blast wave or a reflective surface the overpressure effects can increase or decrease. Casualties requiring medical treatment from direct blast effects are typically produced by overpressures between 1.0 and 3.4 bar. However, other effects (such as indirect blast injuries and thermal injuries) are so predominant that casualties with only direct blast injuries make up a small part of an exposed group.

Indirect effects

23 People can survive fairly strong blast waves and in accidents involving explosion there are very few cases in which the blast effect has directly caused fatality. Typical injuries following an explosion are caused by:

- Burn
- Impacting fragments
- Buildings or other structures falling down or being disintegrated
- Persons falling or "flying" and subsequently hitting a solid object (Whole body displacement).

Important parameters for determining the effects and the risk from an explosion include:

- Maximum overpressure
- Time to reach the maximum overpressure
- Indoor or outdoor exposure of people
- Possibility of flying fragments
- Designed pressure sustainability of building (damage resistance)

In risk analysis the most important effects are:

- Flying fragments hitting personnel
- Whole body displacement resulting in impact damage
- Damage from impact caused by collapsed structures

Fragments

24 The drag forces of the blast winds produced by a vapour cloud explosion may be sufficient to result in the break up of structure, plant or equipment resulting in fragmentation and missile formation. Thus, multiple and varied missile injuries may result, increasing their overall severity and significance.

25 Flying fragments from an explosion are usually more dangerous than the overpressure per se. Fragments may be debris from demolished equipment or structures caused by the explosion, or loose equipment. Fragments from glass breakage are very common and extremely dangerous. The possibility of harm from glass fragments must be determined during an analysis of explosion effects.

Estimates of the pressure needed for breakage of conventional glass by DNV / Statoil (NORSOK Z013) provide:

- 1% level glass breakage ppeak = 0.017 bar (1.7 kPa)
- 90% level glass breakage ppeak = 0.062 bar (6.2 kPa)

The velocity to which missiles are accelerated is the major factor in causing injury. The probability of a penetration injury increases with increasing velocity, particularly for small, sharp missiles such as glass fragments. Small, light objects are accelerated to speeds approaching the maximum (blast) velocity.

26 Other missiles are also produced as a result of explosion and their effects should also be addressed. DNV / Statoil (NORSOK Z013) suggestions are provided in Table 8 for the expected effects from missiles produced as the result of explosion:

Table 8: Injuries from missiles

Injury	Peak overpressure (bar / kPa)	Impact velocity (m/s)	Impulse (Ns/m^2)
Skin laceration threshold	0.07 – 0.15 / 7-15	15	512
Serious wound threshold	0.15 – 0.2 / 15- 20	30	1024
Serious wounds near 50% probability	0.25 – 0.35 / 25- 35	55	1877
Serious wounds near 100% probability	0.5 – 0.55 / 50- 55	90	3071

In addition The American Military (NATO Field Manual 1993) provides the following data on impact from glass fragments in the event of explosion:

Table 9: Probability of penetration of glass fragments into abdominal cavity

Mass of glass fragments (g)	Impact velocity (metres per second)		
	1%	50%	99%
0.1	78	136	243
0.6	53	91	161
1	46	82	143
10	38	60	118

Whole body displacement

27 During the whole-body displacement, blast overpressure and impulses interact with the body in such a manner that it is essentially picked up and translated. In such events the head is the most vulnerable part of the body from the effects of displacement and subsequent impact on to a solid surface. This displacement (acceleration) is a function of the size, shape and mass of the person and the blast forces.

The following conclusions have been drawn by DNV / Statoil:

- 50 % of the people being picked up and translated with a speed more than 0.6 m/s will suffer minor injuries.
- One percent of those with a speed of about 4 m/s will suffer injuries like ruptured organs and bone fractures.
- If thrown against a solid wall about 40 % will suffer major injuries.

The expected effects from whole body displacement can be estimated from data consistent with Table 10:

Table 10: Injuries from whole body displacement

Total body impact tolerance	Related impact velocity (m/s)
Most "Safe"	3.05
Lethality Threshold	6.40
Lethality 50 %	16.46
Lethality Near 100 %	42.06

The NATO field manual (1993) provides further information on the probability of injuries and fractures

Table 11: Blunt injuries and fractures (fatal & non fatal)

Probability of injury	Velocity (M/SEC)

	Non Fatal	Fatal
1%	2.6	6.6
50%	6.6	17
99%	16.5	13.9

Overpressure of 0.21 bar (3 psi) can throw the human body, causing 1% fatality. For external gas explosions, overpressures above 0.35 bar (5 psi) have been considered to blow personnel who are outside into the sea and trap personnel who are inside under debris. A simple assumption can be made that 50% of people inside the 0.35 bar region are fatalities and none outside it (OCB/Technica, 1988); a more conservative approach is to use 100% fatalities within 0.2 – 0.3 bar or the gas cloud LFL, whichever is greater (Spouge, 1999).

Thermal radiation

Introduction

28 Continuous, low level incident heat fluxes or high air temperature, such as those experienced some distance from large to medium sized jet or pool fire, firstly result in physiological effects that may cause pathology as the exposure (dose) increases. Events such as fireball, BLEVE or large jet fire produce high intensity fluxes that may result in rapid pathology. Short duration events can have significant effects in the near to medium field but little or no physiological effects in the far field. In the assessment of thermal hazards both should be evaluated.

29 The impact criteria contained in this section relate to the thermal radiation exposures that may progressively cause pain, first, second, third degree burns or possibly an immediately fatal outcome.

Physiological effects of thermal radiation

Heat stress / exhaustion

30 Deviations of body core temperature beyond normal limits can soon cause severe pathological effects. An excessive Body Heat Storage of >50Wh/m^2 body core temperature ultimately results in heat exhaustion.

31 The body's ability to regulate its temperature depends on its ability to shed excess heat gained from metabolism, a process dependent on the temperature and humidity of the surroundings. Short and long duration of high air temperature may cause heat stress resulting in a fatal outcome.

32 Inside living quarters, control rooms or other compartments where personnel should be safe in a fire situation, the air temperature may become too high leading to physiological effects on humans such as:

- Difficulty with breathing resulting in incapacitation.
- High pulse or core temperature leading to collapse.

33 In most cases the air temperature inside the enclosures will not be sufficiently high for the pathological effects such as skin burns to be dominant. However, during escape or at evacuation stations personnel may be directly exposed to the fire and thermal radiation may be more critical than the air temperature and pathological effects will be dominant.

34 Most physiological effects of thermal radiation on man involve voluntary exposures that are relatively lengthy, possibly several or tens of minutes. Where the exposure is of low thermal radiation level, high air temperature may become the most critical parameter. This is typically the case inside living quarters, control rooms or other types of compartments exposed to fire where personnel may stay for a period of time.

35 Table 12 indicates some physiological effects of elevated ambient temperatures on the human individual based on full-scale fire tests and information from Hadjisophocleous et al (1998) and Bryan (1986). The National research Council of Canada (NRCC) fire tests indicate that 149 ºC is the maximum survivable breathing air temperature, but only for short periods and in the absence of moisture.

Table 12: Elevated temperature response on human individuals (Norsok Z013)

Temperature (°C)	Physiological Response
127	Difficult breathing
140	5-min tolerance limit
149	Mouth breathing difficult, temperature limit for escape
160	Rapid, unbearable pain with dry skin
182	Irreversible injury in 30 seconds
203	Respiratory systems tolerance time less than four minutes with wet skin

36 NORSOK Z013 reports that elevated temperatures have influence on the pulse rate. The pulse rate climbs steadily with time and air temperature. The pulse jumps from normal 84 to 120 beats a minute when the air temperature increases to 100ºC. It further increases to 150 beats/minute after 10 minutes at an air temperature of 113ºC.

37 Table 12 indicates that the maximum air temperature tolerance of the human respiratory tract is approximately 203ºC. It has been suggested that occupants of aircraft fires have been exposed to an upper limit of 309ºC causing third degree burns within 20 seconds and rendering escape impossible. Above air temperatures of 150ºC, impact is dominated by pain from skin burns, occurring in less than 5 minutes exposure. Up to an air temperature of 140ºC, the impact is dominated by difficulties to breath.

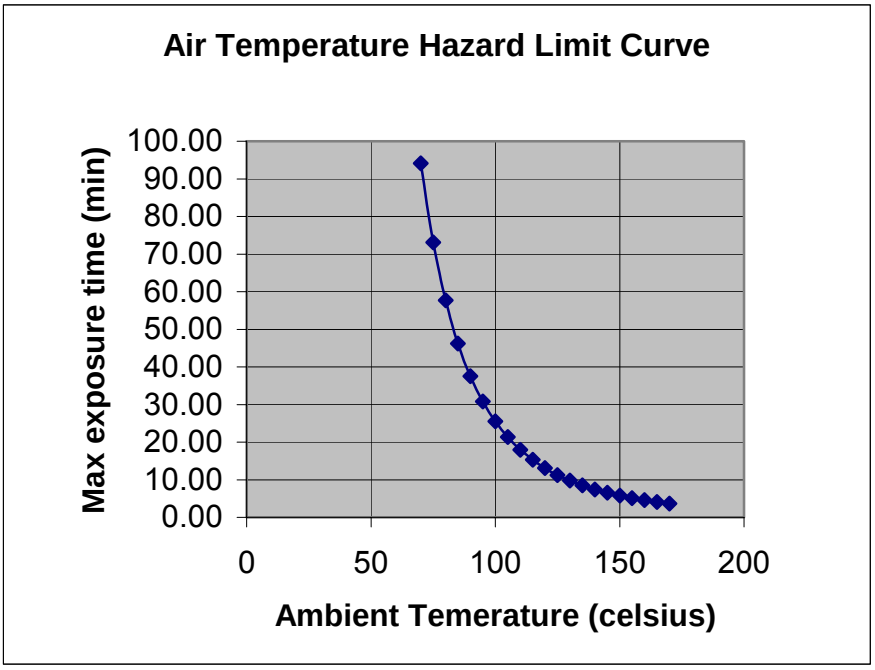
38 It is assumed that below 70 ºC the situation inside a compartment will be uncomfortable but not fatal. No probit functions have been found for exposure to high ambient temperatures. To enable assessment several accident investigators have proposed various air temperature hazard curves. For example NORSOK Z013 provides equation 5 for the average time to incapacitation for temperatures between 70 - 150 ºC.

$$t = 5.33 \times 10^{8}/[(T)^{3.66}] \qquad \textbf{(Equation 5)}$$

t = exposure time to incapacitation (minutes)
T = ambient temperature (ºC)

A plot of the equation (figure 1) provides the Air Temperature Hazard Limit Curve.

Figure 1: Air temperature hazard limit curve



39 With temperatures of 70 ºC and 150 ºC inside a compartment, time to incapacitation may be 94 minutes and 6 minutes respectively based on the above presented equation and curve.

40 An example of methodology and acceptance criteria for heat exhaustion is based on ISO 7933 that sets the warning level for body heat storage at 50Wh/m^2 and the danger level at 60Wh/m^2. ISO 7933 also defines the danger level in terms of four parameters as described in Table 13.

Table 13: Body heat danger parameters and limiting values

Thermal stress parameter	Danger level	Equivalence

Skin wettedness	85%	
Sweat rate in grams/hour for 1.8m^2 body surface ("standard body")	390	
Heat storage in Wh/m^2	600	1^{o}C increase in body core temperature 4^{o}C increase in mean skin temperature
Water loss in grams for a "standard body"	3250	4 – 6% of Body mass

Pathological effects of thermal radiation

41 The pathological effects, of thermal radiation, on humans are most relevant in the immediate vicinity of an incident. This includes occupancy on unshielded escape ways, evacuation stations or inside enclosures if radiation becomes a dominant factor (i.e. above 140ºC ambient temperature in flame line of sight). In such situations the progressive effects resulting are:

- Pain
- First degree burns
- Second degree burns
- Third degree burns
- Fatality.

42 These effects are commonly linked to the intensity of the incident thermal radiation and Table 14 provides the typical consequences of exposure to various levels of intensity and the expected time to each effect, values have been approximated to reflect uncertainty in calculation and represent "cautious best estimate" values.

Table 14: Thermal radiation exposure effects

Thermal Radiation kW/m^2	Effect
1.2	Received from the sun at noon in summer
2	Minimum to cause pain after 1 minute
Less than 5	Will cause pain in 15-20 seconds and injury after 30 seconds exposure
Greater than 6	Pain within approximately 10 seconds rapid escape only is possible
12.5	Significant chance of fatality for medium duration exposure. * Thin steel with insulation on the side away from the fire may reach thermal stress level high enough to cause

	structural failure.
25	* Likely fatality for extended exposure and significant chance of fatality for instantaneous exposure. * Spontaneous ignition of wood after long exposure. * Unprotected steel will reach thermal stress temperatures that can cause failure.
35	* Cellulosic material will pilot ignite within one minute's exposure. * Significant chance of fatality for people exposed instantaneously.

Severity and consequences of exposure

43 It would be expected that an individual either in pain from thermal dose received or suffering from 1º burns will escape rapidly as the injury is not sufficient to impede movement, yet the pain will be too uncomfortable to bear standing still.

44 An individual with 2º burns will have even greater motivation to escape, commonly referred to as the fight or flight response. However at this level of injury, the skin affected will be very uncomfortable to use in contact with another surface. Simple tasks, such as turning door handles or dressing in survival equipment will take longer. Depending on the location and extent of injury, more difficult tasks, such as operating control panels or turning valves may be impossible. Depending on the burn location escape will probably incur further injury as skin may fall away from the wound and dirt and microorganisms may be picked up.

45 With 3º burns an individual will be in severe pain and will certainly realise that they are in immediate danger of loss of life. Individual response is hard to predict. However fine control of injured extremities will be impossible and other functions will be severely impaired. Escape will probably incur further injury and dirt and microorganisms may be picked up in any wounds. Individuals with 3º burns should be considered as casualties who cannot evacuate unaided.

The thermal dose unit

46 The level of intensity, duration of exposure, percentage of exposed bare skin result in a received **thermal radiation dose (tdu)** that can be calculated and used to estimate these progressive effects from the following equation:

$$\text{Dose} = (I^{4/3}) \times t \qquad \textbf{(Equation 6)}$$

$$\text{Thermal Dose Unit} = 1\ (kW/m^2)^{4/3}.s,$$

"I" incident thermal flux (kW/m^2)

"t" time of exposure (seconds).

Table 15 (produced by Rew 1996) demonstrates the range of data available for estimating the dose effect relationship of thermal radiation

Table 15: Ranges of thermal doses required causing pain, burns and fatal outcome

Effect	Dose $(kW/m^2)^{4/3}.s$			
	Ultraviolet	**Reference**	**Infrared**	**Reference**
Pain	108 - 127			
Significant injury	290	Tsao & Perry (1979)	c.80	Mehta et al (1973)
Level/First degree burns	260-440	Eisenberg et al (1975)	130	Tsao & Perry (1979)
	300-440	Glasstone & Dolan 1977)		
Second degree burns/ 1% lethality level for average clothing	670-960	Glasstone & Dolan (1977)	240	Stoll & Green (1958)
	810-950	Eisenberg et al (1975)	270-310	Stoll & Green (1958)
	c.1000	Mixter (1954)	c.350	Mehta et al (1973)
	1100	Hinshaw (1957)	290-540	Williams et al (1973)
			730	Arnold et al (1973)
Third degree burns/ 50% lethality level for average clothing	1220-1790	Glasstone & Dolan (1977)	c.500	Mehta et al (1973)
	3100	Hinshaw (1957)		

Note: The data sources listed are referenced by Rew (1996).

47 It should be noted that a significant proportion of burn data is based on studies from nuclear explosions (i.e. Eisenberg's estimate). The thermal dose required for a given lethality level is in general lower for hydrocarbon fires than for nuclear explosions, because of the difference in wavelength and skin absorption

characteristics. Infrared radiation from hydrocarbon fires is of a longer wavelength than ultra-violet radiation from nuclear explosions.

48 Tsao & Perry (1979) suggested that a dosage of infrared radiation 2.23 times less than that for ultraviolet radiation will produce the same degree of skin burn.

49 A review by HSE (Rew 1996) also suggests that there is an under estimate of the consequences of exposure from the Eisenberg probit. This work suggests a value of 1800 – 2000 tdu, higher than that suggested by TNO, to be more representative of 50% fatality. However, the calculated fatality rates should be used as guidance in the fatality assessment more than as absolute values.

50 Table 16 is based on information provided by DNV / Statoil (2001) and demonstrates the range of data available for estimating the dose / effect relationship of thermal radiation.

Table 16: Ranges of thermal doses required causing pain, burns and fatal outcome (DNV / Statoil (2001))

Effect	Thermal dose ($s*[kW/m^2]^{4/3}$)	Comments
Pain	108 – 127	bare skin
	85 - 129	bare skin
Significant injury Level/First degree burns	600 - 800	bare skin
	250 – 350	bare skin
	210 – 700	bare skin
Second degree burns/ 1% lethality level for average clothing	900 – 1300	bare skin
	500 – 3000	bare skin
Third degree burns/ 50% lethality level for average clothing	2000 – 3000	bare skin

Probit functions for thermal dose estimation

51 Several probit functions have been developed based on experiments carried out on animals and humans. These are commonly used in consequence analysis amongst the most commonly used are those developed by Eisenberg, Lees, Tsao & Perry and TNO. These include function for naked skin and others where some protection is present. The more commonly used probits are shown in Table 17.

Table 17: Commonly referenced probit functions

Source	Probit equation	Lethal dose	
		1%	50%
Eisenberg et al (1975)	Y = -14.9 + 2.56 ln V	960	2380

Tsao & Perry (1979)	Y = -12.8 + 2.56 ln V	420	1046
Lees (1994)	Y = -10.7 + 1.99 ln V	828	2670
TNO	Y = -15.3 + 3.02 ln V	389	841

Note: "V" in the above probits is the thermal dose where $V = I^{4/3} \times t$

52 For example, DNV / Statoil provide calculated fatality rates for different thermal incident fluxes and exposure times by use of the TNO probit function presented above are shown in Table 18.

Table 18: Fatality rate as a function of radiation level and exposure time from the Tsao & Perry probit model for naked human skin

Exposure Time (s)	Fatality Rate (%)		
	10 kW/m^2	20 kW/m^2	30 kW/m^2
10	0	5	39
20	1	53	93
30	11	87	100
40	31	97	100
50	53	99	100
60	71	100	100

53 Annex 3 provides radiation dose curves for differing levels of incident thermal radiation and a comparison of the probit fatality estimates from the probits described in table 17. A significant variation in harm prediction is noted in that:

- Compared to the probit function from Eisenberg the TNO model for naked human skin also provides a **much** higher fatality rate.
- The TNO model is based on the Eisenberg probit function adjusted for experiments carried out at hydrocarbon fires.
- The Eisenberg probit function is based on experiments carried out at nuclear explosions where UV radiation dominates.
- The Lees probit provides the most optimistic consequences.

Effects of clothing

54 API (API 521) has defined a criterion for workers exposed to flare radiation who are assumed to be wearing heavy industrial clothes. For continuously manned locations a limit of 1.58 kW/m^2 is considered acceptable. For this level of radiation pain would be felt after 60 seconds, but clothing is deemed to provide sufficient protection.

55 In addition API RP 510:1990 Suggests 4.7kW/m^2 represents the maximum level of thermal radiation where emergency actions lasting up to several minutes may be required without shielding but with protective clothing.

56 A suitable probit function may also be used to estimate radiation levels for 100 % fatality of lightly clothed personnel. For example NORSOK Z013 through application of the TNO probit provides the following exposure time intervals:

- 16 kW/m2 - Exposure time less than 0.5 minute
- 10 kW/m2 - Exposure time from 0.5 minute to 1 minute
- 4 kW/m2 - Exposure time from 1 minute to 2 minutes
- 2 kW/m2 - Exposure time from 2 minutes to 10 minutes

57 NORSOK Z013 provides an alternative approach to application of the “Neisser curve” and provided the following criteria based on the approach:

- 25 kW/m2 - Exposure time less than 0.5 minute
- 13 kW/m2 - Exposure time from 0.5 to 1 minute
- 8 kW/m2 - Exposure time from 1 minute to 2 minutes
- 4 kW/m2 - Exposure time from 2 minutes to 10 minutes

58 The criteria are recommended for clothed personnel for a 100% fatality probability for the time exposed.

59 The approach assumes a constant heat load over the exposure period. In reality, most fires will initially expand and then decay with time, and thus the radiation received at any given point will also be a function of time. **A full integration of the dose received may be performed if greater detail is required.** This is considered unlikely for a typical offshore risk assessment.

60 More recent information in the TNO “Green Book” provides probits for protected (p) and unprotected (up) targets. The values of fatality probability against dose are provided in Table 19.

Table 19: Fatality probability for protected and unprotected targets.

Probit	Fatality probability	
	1%	50%
Unprotected Dose (UP) level (tdu)	421	1047
Protected (tdu)	587	1459

The TNO Green Book method treats clothing by assuming that no burn damage occurs to clothed skin unless the clothing ignites. If clothing does ignite, the probability of death is assumed to be 100%. Some of the assumptions in the TNO method are overly optimistic.

61 Type of fire, the distance from the fire and the time of exposure are very important parameters in the assessment of fatalities. On an offshore platform it is believed that personnel will be exposed to a fire for a longer period because of the time needed to escape from the seat of the fire or evacuate the platform. Typically average escape speeds will not be expected to exceed 1.5 m/s because of the complexity of the installation lay out and presence of equipment.

62 In general offshore personnel are more protectively clothed than onshore personnel, making them more resistant against thermal radiation. The majority of the data available is given for lightly clothed personnel and is typically representative of onshore personnel. However, some data is also presented for well-clothed personnel, which is representative for offshore situations.

63 The effects of fire on humans depend on the rate at which heat is transferred from the fire to the person, and the time the person is exposed to the fire. Even short-term exposure to high heat flux levels may be fatal. This situation could occur to persons wearing ordinary clothes that are inside a flammable vapour cloud (defined by the lower flammable limit) when it is ignited. In risk analysis studies, it is common practice to make the simplifying assumption that all persons inside a flammable cloud at the time of ignition are killed and those outside the flammable zone are not [Cox, 1993].

Suggested thermal dose fatality criteria

64 Following consideration of the pathological and physiological effects of thermal radiation Table 20 provides suggested harm criteria.

Table 20: Suggested thermal dose fatality criteria

Thermal Dose Units (tdu): $((kW/m^2)^{4/3})$ sec	Effect
1000	1% Fatality
2000	50% Fatality
3200	100% Fatality

65 These values have been selected as they are consistent with current HSE criteria where thermal doses required to produce second and third degree burns are approximately the same doses as 1% fatality (1000tdu) and 50% fatality (1800 tdu for a normal member of the public and 2000 tdu for a typical offshore worker) respectively, for exposed personnel dressed in a typical manner for on and offshore.

66 On the basis of the information provided in paragraphs 43 – 45 and Table 20, the radiation criteria defined in Table 21 is recommended for use in risk assessment.

Table 21: Recommended thermal radiation flux criteria

Consequences	Maximum Radiation Level

Immediate Fatalities to all Personnel Local to the Fire (escape not possible):	35 kW/m^2
Impairment of Escape Routes:	6 kW/m^2
Impairment of TEMPSC Embarkation Areas:	4 kW/m^2

67 Excessive thermal radiation levels or flame engulfment can render escape routes, evacuation routes and embarkation areas impassable to personnel not wearing special protective clothing. Under such conditions it is not unreasonable to apply a 100% fatality probability to personnel in this predicament.

Hydrocarbon combustion products

68 Smoke and other combustion products provide a greater potential risk to life on an offshore installation than those resulting from the immediate exposure to heat, fire or explosion. This is demonstrated by the Piper Alpha disaster where less than 5% fatalities resulted from burns while smoke inhalation accounted for over 80%. Also, on shore it is expected that building occupants are twice as likely to be killed by smoke inhalation than exposure to flame or heat. This may well be an under-estimate, since smoke inhalation is known to worsen the prognosis for burn injury victims. Smoke, especially the carbon monoxide component, is known to cause the majority of deaths in a fire. Apart from direct toxic effects, it impedes escape by reducing visibility and also increases the mortality rates of burn victims (Ramsdale et al).

69 In the event of a hydrocarbon fire the constituents will vary depending on the hydrocarbon (or other combustibles) and fire type. Generally it is the amount of oxygen available for combustion that has the most marked effect on smoke composition. A poorly ventilated fire may contain up to 5 volume percent of carbon monoxide and it is not unreasonable, for assessment purposes, to combine this with an assumption of 10 volume percent carbon dioxide. For well-ventilated fires, oxygen depletion appears to have the main effect (Spouge, 1999).

Other significant products of combustion include:

- Oxides of Nitrogen (NO_x)
- Hydrogen Sulphide (H_2S)
- Ammonia (NH_3)
- Sulphur Dioxide (SO_2)
- Hydrogen Fluoride (HF)

70 Smoke can also contain significant quantities of vaporized but un-combusted hydrocarbons and other highly toxic substances such as:

- Phosgene ($COCl_2$)

- Acrolein
- Hydrogen Chloride (HCl)
- Carbon Disulphide (CS_2)
- Hydrogen Cyanide (HCN)
- Hydrogen Bromide (HBr)

As the combustion process consumes atmospheric oxygen, and asphyxiant gases exclude it, then these effects must also be considered when evaluating exposure to smoke and combustion fume.

Suggested harm criteria for exposure to hydrocarbon combustion products

71 Exposure to the products of hydrocarbon combustion is described in the following section and from the information presented the criteria presented in Table 22 is suggested.

Table 22: Suggested harm criteria for exposure to hydrocarbon combustion products

Hazard	Note	5 Minute Exposure		30 Minute Exposure	
		Incapacitation	Death	Incapacitation	Death
Carbon Monoxide Only		4000 ppm	8000 ppm	1000 ppm	2000 ppm
Low oxygen		<14%	< 10%	<17%	<12%
Carbon Dioxide Only		>7%	>10%	>5%	>7%
Carbon Monoxide (with 3% CO_2)	1	1500 ppm	5000 ppm	450 ppm	1400 ppm
	2	2000 ppm	6000 ppm	620 ppm	1800 ppm
Smoke Obscuration		3m for primary compartment escape		10m for escape routes	

In this case incapacitation is considered to be the limit where an exposed worker becomes incapable of making rational decisions or carrying out action to promote escape or evacuation. On this basis a high degree of fatality probability may be assigned to exposures lower than those considered to directly result in fatality.

Notes: 1 - High workload breathing rate (30l/min), 2 - Medium workload breathing rate (18 l/min)

Carbon monoxide

72 The toxicity of carbon monoxide is due to the formation of blood carboxyhaemoglobin. This results in a reduction of the supply of oxygen to critical body organs and is referred to as anemic anoxia. The affinity of haemoglobin for CO is extremely high (over 200 times higher than O_2), so that the proportion of haemoglobin in the form of carboxyhaemoglobin (COHb) increases steadily as CO is inhaled. There is little doubt that CO is the most important toxic agent formed in hydrocarbon fires because:

- It is always present in fires, often at high concentrations.
- It causes confusion and loss of consciousness thus impairing, or, preventing, escape.

Visual impairment has been reported at 10-20% COHb %, but according to the toxicology summary in EH64 (which represents HSE established position) the onset occurs at concentrations above 30%.

Effects of carbon monoxide

73 Evidence suggests that relatively low levels of COHb may have adverse effect on reaction time, an important factor in escape from fire, and the toxic effect of carbon monoxide may be modified by heat stresses. Experiments on test animals under heat stress showed that blood carboxyhaemoglobin concentrations at the time of death were much lower than in animals not stressed by heat.

74 The physiological effects on human individuals from carbon monoxide estimated from data collected are shown in Table 23 (Norsok Z013) and include:

Table 23: Concentration vs. effect for carbon monoxide exposure

CO (conc)	Effects
1500 ppm	Headache after 15 minutes, collapse after 30 minutes, death after 1 hour
2000 ppm	Headache after 10 minutes, collapse after 20 minutes, death after 45 minutes
3000 ppm	Maximum "safe" exposure for 5 minutes, danger of collapse in 10 minutes
6000 ppm	Headache and dizziness in 1 to 2 minutes, danger of death in 10 to 15 minutes
12800 ppm	Immediate effect, unconscious after 2 to 3 breaths, danger of death in 1 to 3 minutes

Alternatively it should be noted that effects table in EH64 displays a few slight differences. It should be noted that the EH64 version represents an agreed HSE interpretation of the available data, and is compatible with the World Health Organisation review of CO.

A CO concentration of 1500 ppm is taken as the limit for impairment of an escape route in 30 mins (Spouge, 1999). This is consistent with the IDLH value from which escape is considered possible in 30 mins without any escape-impairing or irreversible effects (NIOSH, 1990). A CO concentration of 10,000 ppm is taken as the limit for escape actions lasting a few seconds. If escape is not possible in a few seconds death is assumed to occur. When carbon monoxide levels in air exceed 3% (30,000 ppm) death occurs almost at once. Initial CO concentrations in smoke range from less than 0.1% for well-ventilated fires to 3% for under-ventilated fires. Thus only under-ventilated fires have potential for causing fatalities by asphyxia (Spouge, 1999).

75 Table 23 contents may be compared with the effects of percentage COHb in the blood stream as collected from several sources and shown in Table 24.

Table 24: Effects of %COHB in blood

% COHb in Blood	Physiological and subjective symptoms
2.5-5	No Symptoms
5-10	Visual light threshold slightly increased
10-20	Tightness across forehead and slight headache Breathlessness Dyspnoea on moderate exertion Occasional headache Signs of abnormal vision
20-30	Definite headache, Easily fatigued, Impaired judgment, Possible dizziness and dim vision, Impaired manual dexterity
30-40	Severe headache with dizziness, Nausea and vomiting
40-50	Headache, Collapse, Confusion, fainting on exertion
60-70	Unconsciousness, Convulsions, respiratory failure and death
80 80+	Rapidly fatal Immediately fatal

It should be noted that smokers might have up to 5% COHb and are expected to cross effect thresholds sooner than non-smokers. From Table 24 it can be concluded that COHb levels in the range 10-20 % represent a range of values where there is a reduced potential of ability to escape or carry out functions requiring dexterity or conscious effort; well-trained subjects engaging in heavy exercise in polluted indoor environments can increase their COHb levels quickly up to these levels (Air Quality Guidelines, 2000). Impaired coordination, tracking, vigilance and cognitive performance have been reported at COHb levels as low as 5.1–8.2% and at COHb levels of 7% and 10% visual tracking performance is significantly impaired if the subjects engage in heavy exercise (Air Quality Guidelines, 2000). It is suggested that the upper limit for survivability without significant impairment is 15% COHb with a

cautious best estimate of 10% COHB to be used where exposure is followed by intense physical activity such as escape or evacuation under harsh conditions. Above 20% COHb impairment and death become more certain within a relatively short period and recovery may not be possible.

Use of probits

76 Several probit functions have been developed based on experimental data from animals and the following probit function is taken from the TNO Green Book as an example of that used in the assessment of fatality assessment:

$$\mathbf{Pr = -38.8 + 3.7\ln(C^{*}t)} \qquad \textbf{(Equation 7)}$$

Where

C = concentration (mg/m^3) (1mg/m^3 = 0.862 ppmV for CO M.W. 28)
t = exposure time (min)

77 The following lethality levels for different CO concentrations and exposure times from the above probit (equation 7) are presented in Table 25. The CO concentrations and exposure time necessary for a 50 % lethality level are included.

Table 25: Concentration / time consequences from the example probit function for carbon monoxide exposure

Concentration (ppm)/ approximate exposure time for % lethality			
1- 5		**50**	
1100	60 min	2000ppm	60 min
2200	30 min	4000ppm	30 min
3200	20 min	6000ppm	20 min
6400	10 min	12000ppm	10 min
13000	5 min	24000ppm	5 min

78 Based on a 1-5 % lethality level it can be concluded that the probit function is more or less consistent with the thresholds of effect presented in Table 23.

79 The HSE “SLOT DTL” approach suggests 1-5 % and 50% fatalities at 4000 and 5700 ppm respectively for a 10 minute exposure (40,000 ppm.min & 57000 ppm.min see Table 3) which is more conservative than the values obtained from the probit described in Equation 7. Evidence suggests that a rule of thumb relationship exists that if carbon monoxide dose exceeds 30,000 ppm.min it is likely to be fatal.

80 Another example for the estimation of COHb blood concentration is by application of the Coburn, Forster & Kane correlation as described in Equation 8.

In this case:

$$C_{body} = (((C_{air}(1-\delta))/1316) - \delta(\beta V_{co} - \alpha D) + \beta V_{co})/\alpha \quad \textbf{(Equation 8)}$$

Where C_{body} is the final COHb blood concentration (ml/CO/ml blood)

C_{air} is the CO concentration breathed

D is the initial COHb blood concentration (usually taken as 0.0015 ml CO/ml blood)

V_{co} is the rate of endogenous CO production, taken as 0.007 ml/min

α is a constant of value 2.29

β also a constant of 0.04

$$\delta = \exp(-t\alpha/V_b\beta)$$

where V_b is the blood volume typically 5500ml for an average male
t is the time of exposure in minutes.

Again this equation may be used to determine the CO exposure criteria to result in 15 % COHb that is proposed as the criterion for impairment and death. If a more cautious approach is to be used this COHb concentration may be reduced to 10% for the limit of survivability. Again it is recommended that assessors confirm the equations to be used by examination of the authors original publications.

Note:

1 The EH 40/2005 exposure limits are 200 ppm for 15 minutes exposure; 30 ppm for 8-hour exposure and these values are significantly below the thresholds for the acute effects considered in major accident hazard analysis.

2 The Coburn-Foster-Kane equation tends to over-predict COHb levels but a conservative approach is favoured given possible synergistic effects of other toxic agents present in fires.

3 Previously HSE has used the following updated (by Smith et al 1996) version of the CFK equation:

$$\%COHb_t = \%COHb_0 (e^{-t/A}) + 218 (1 - e^{-t/A}) [1/B + ppm\ CO/1403] \quad \textbf{(Equation 9)}$$

where:

$\%COHb_t$ = %COHb at time t

$\%COHb_0$ = %COHb at beginning of exposure

(suggested value for background %COHb, reflecting endogenous CO production is 1%)

t = exposure time in mins

218 = Haldane constant, describes relative affinity of CO for Hb compared with O_2.

A and B are constants which take into account alveolar ventilation rates, lung diffusing capacity, and corrections for standard temperature pressure dry (STPD). There are different values for A and B for different degrees of work effort; values of A and B for light work are 175 and 1958 respectively

Carbon dioxide (CO2)

Effects of CO2

81 While carbon dioxide is not considered to be particularly toxic, at levels normally observed in fires, a moderate concentration does stimulate the rate of respiration. This would be expected to cause accelerated uptake of any toxic and/or irritant gases present during an incident involving fire and fume as breathing rate increases 50 % for 20,000 ppm (2% v/v) carbon dioxide and doubles for 30,000 ppm (3% v/v) carbon dioxide in air. At 50,000 ppm (5%v/v) breathing rate triples and breathing becomes laboured and difficult for some individuals as it represents a significant level of oxygen depletion, although it can be sustained for up to 1 hour without serious after-effects (Spouge, 1999).

Typical Carbon Dioxide responses used by NORSOK Z013 are illustrated in table 26.

Table 26: Carbon dioxide concentration vs. effect and time to unconsciousness

Concentration of carbon dioxide (ppm) / % v/v	Responses
45000 / 4.5 %	Reduced concentration capability for more than 8 hours exposure, adaptation possible
55000 / 5.5%	Breathing difficulty, headache and increased heart rate after 1 hour
65000 / 6.5%	Dizziness, and confusion after 15 minutes exposure
70000 / 7.0%	Anxiety caused by breathing difficulty effects becoming severe after 6 minutes exposure
100 000 / 10%	Approaches threshold of unconsciousness in 30 minutes

120 000 / 12%	Threshold of unconsciousness reached in 5 minutes
150 000 / 15%	Exposure limit 1 minutes
200 000 / 20%	Unconsciousness occurs in less than 1 minute

Based on the "probit" for carbon dioxide from the HSE, where DTL = $C^n t$ and n = 8, SLOT = 1.5×10^{40} and SLOD = 1.5×10^{41}, concentrations and exposure times for 1% and 50% lethality levels are illustrated in Table 27

Table 27: Concentration / time consequences from the HSE probit function for carbon dioxide exposure

Concentration / approximate exposure time for % lethality			
1-5		**50**	
63,000 ppm	60 min	84,000 ppm	60 min
69,000 ppm	30 min	92,000 ppm	30 min
72,000 ppm	20 min	96,000 ppm	20 min
79,000 ppm	10 min	105,000 ppm	10 min
86,000 ppm	5 min	115,000 ppm	5 min
105,000 ppm	1 min	140,000 ppm	1 min

Table 27 illustrates a significant danger with carbon dioxide exposure, that of toxicity increasing rapidly for only small changes in concentration above a certain level - there is not a large difference between the SLOD and SLOT values and the value of n = 8 suggests a steep exponential curve. Differences in CO_2 concentration between different lethality levels and exposure times are relatively small; concentrations for lethality levels 1-5% and 50% for a given exposure time differ by only 33%. This means that, unlike with some of the more toxic gas, when physiological symptoms of intoxication occur it is usually too late for the subject and only a further small increase in concentration brings about rapid impairment and death, a situation often seen in diving fatalities.

Based on Table 26 NORSOK Z013 recommends the following 100 % fatal limits for CO_2 exposure times:

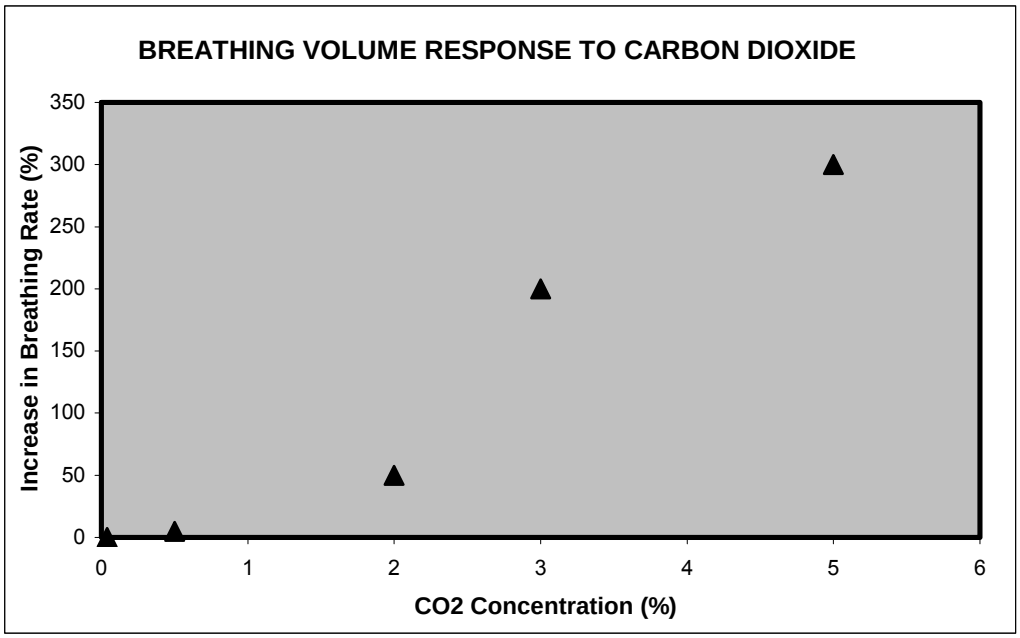
- 150 000 ppm of CO_2 Exposure time < 5 minutes
- 120 000 ppm of CO_2 Exposure time 5 - 30 minutes
- 100 000 ppm of CO_2 Exposure time > 30 minutes

82. The maximum safe level for carbon dioxide on its own has been suggested as 40000 ppm (4%) as this relates to a 17% oxygen concentration; this is the IDLH for

carbon dioxide from which escape is considered possible in 30 minutes without any escape-impairing or irreversible effects. However, as carbon dioxide induces increased respiration rate at above 2% (50% increase at this concentration and rate doubling at 3%) produces oxygen depletion and can increase the uptake of other toxics present, it is suggested that the maximum safe level of carbon dioxide in smoke produced from an emergency is established at 30,000ppm or 3%.

Human breathing rate response to increase in carbon dioxide concentration is illustrated in Figure 2.

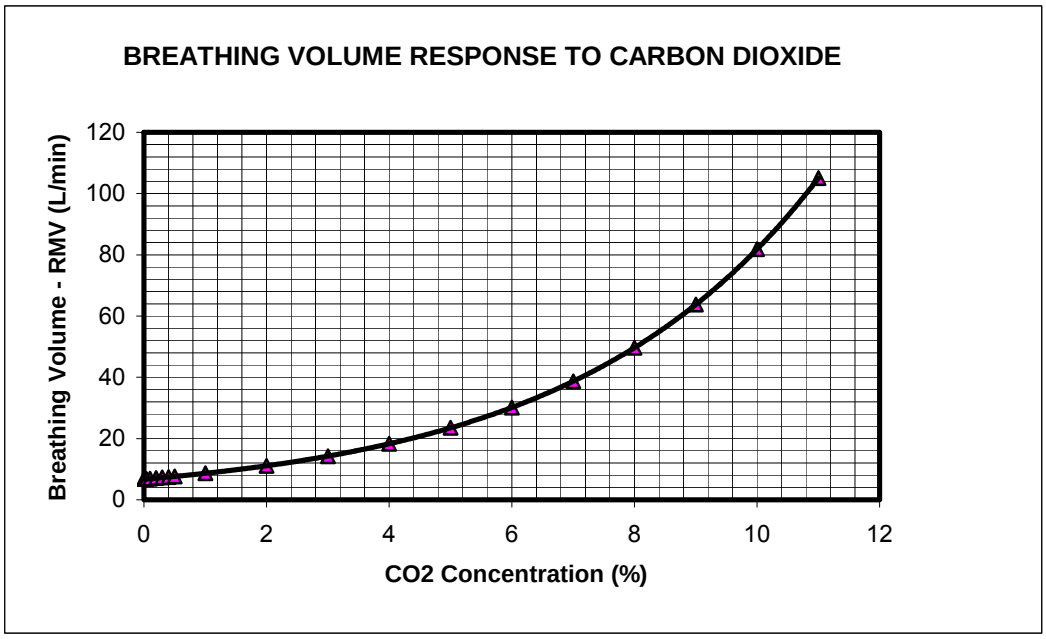
Figure 2: Change in human breathing volume response to carbon dioxide (data from ref. 17, 28, 32 and 33)



The following regression equation has been described in Fire Protection Engineering, Third Edition, for change in breathing volume as a function of carbon dioxide concentration:

$$RMV\ (L/min) = \exp(0.2496 \times \%CO_2 + 1.9086) \quad \textbf{(Equation 10)}$$

Figure 3: Change in human breathing rate response to carbon dioxide as described by equation 10.



83 The ACGIH (2001) summary on carbon dioxide provides additional data to NORSOK Z013 and can be summarised as follows:

Exposures to the WEL of 5000 ppm (0.5%) appear to be well tolerated over 8-hours, provided that normal oxygen levels are present (19-20%).

The STEL of 15,000 ppm should also be well tolerated.

However, when CO_2 reaches 30,000 respiratory and metabolic changes may develop, and effects will be more severe if oxygen levels decline and/or when exercise is strenuous. Even wearing an air-fed respiratory may be difficult. The ACGIH STEL is 30,000 ppm however the WEL STEL of 15,000 ppm is considered more appropriate.

At 30,000 ppm (3%) there is a stimulation of respiration; this is because the dissolved carbon dioxide in the bloodstream increases the acidity of the blood and this stimulates respiratory centres in the brain. If oxygen levels decline even by a few percent (15-17%), then at 30,000 ppm carbon dioxide, workers are likely to feel unwell; blood pressure and pulse rate will go up and hearing acuity will suffer. Mild narcotic effects may well occur so there could be safety issues.

During physical exercise, exposures to carbon dioxide at around 30,000 ppm will cause an increase in the resistance to airflow (probably due to some bronchoconstriction due to the acidity of this soluble gas) making air-fed respirators more difficult to wear - ACGIH makes a note of caution on this point.

Carbon dioxide build-up due to normal respiration

83 As the normal breathing function produces carbon dioxide (21% oxygen inhaled produces 4.5% CO_2 exhaled) CO_2 will build up in a "closed" environment. A typical breathing rate of 500ml 15-20 times a minute will give a CO_2 production rate of almost 0.4 litres / min average. Assuming normal human performance can continue until oxygen is depleted to less than 18% (see section 5 below). This means that carbon monoxide levels may reach 2.9% (29000ppm) before the onset of performance impairment. On this basis the time taken to reach such a condition will be less than 80 minutes per person per m^3 of free air in the closed system.

84 In the event of exposure of the TR to smoke and fume the HVAC system will be shut down and isolated and the residual air change rate will either import clean air or combustion gases, (the latter is most likely and an increase in CO_2 and CO will result). The import of additional asphyxiant gases will combine with the internal effects (including oxygen depletion) in the TR and the time to impairment will decrease. Where ventilation occurs with clean air, the time to impairment due to CO_2 build up will increase the time to expected TR impairment.

85 It must also be noted that 3%v/v CO_2 increases respiration rate by 100% and a TR will typically have a closed in ventilation rate of 0.1 air changes per hour (ach) so the nature and effect of CO_2 build up due to respiration is complex. It is expected that a detailed analysis of such circumstances would be appropriate for places of high-density occupation where low or no air change rate is present.

Oxygen depletion

Effects of oxygen depletion

86 The first effect of oxygen depletion noticeable to a victim is reduced capacity for exercise as the level of oxygen-saturated haemoglobin reduces.

Table 28 provides values suggested by Kimmerle (1974), to indicate the responses of humans to different reduced levels of oxygen in air, and the data is considered comparable with that provided in Table 26:

Table 28: Effects of oxygen depletion

Percent of Oxygen in Air	Symptoms
21-20	Normal
18	Night vision begins to be impaired

17	Respiration volume increase, muscular coordination diminishes, attention and thinking clearly requires more effort
12 to 15	Shortness of breath, headache, dizziness, quickened pulse, effort fatigues quickly, muscular coordination for skilled movement lost
10 to 12	Nausea and vomiting, exertion impossible, paralysis of motion
6 to 8	Collapse and unconsciousness occurs
6 or below	Death in 6 to 8 minutes

87 Oxygen constitutes approximately 21% v/v in clean air (20.9%). Down to about 15 % v/v the body counteracts decreases in oxygen concentration by increasing the flow of blood to the brain and only minor effects on motor coordination are apparent. These effects become noticeable at less than 17% v/v oxygen

88 Oxygen concentrations below 15 % by volume produce oxygen starvation (hypoxia) effects such as increased breathing, faulty judgment and rapid onset of fatigue. Concentrations below 10 % cause rapid loss of judgment and comprehension followed by loss of consciousness, leading to death within a few minutes. This is taken to be the limiting oxygen concentration where escape needs only a few seconds. If escape is not possible within few seconds, incapacitation and death is assumed to occur. The effects of oxygen depletion are described by the British Cryogenics Council as the four stages of asphyxiation and are shown in Table 29.

Table 29: The four stages of asphyxiation

Asphyxiation stage	Oxygen concentration (% v/v) / effects
1st	21 to 14% Reducing: Increased pulse and breathing rate with disturbed muscular coordination
2nd	14 to 10%: Faulty judgement, rapid fatigue and insensitivity to pain
3rd	10 to 6%: Nausea and vomiting, collapse and permanent brain damage
4th	Less that 6%: Convulsion, breathing stopped and death

No probit functions are found in the literature describing the lethality level for personnel when exposed to different concentrations of oxygen in the air and exposure time.

Oxygen in the blood oxygen (saturation)

89 The concentration of oxygen in the blood is logarithmically related to the concentration inhaled. A reduction in the arterial saturation of oxygen can have a range of effects that have been shown to be dependant on the work rate of those affected. Typically the most noticeable consequences are a reduced ability to think clearly and a reduction in the time to exhaustion during intense physical activity. For an individual at rest the maximum reduction of oxygen saturation in the blood (SaO_2) due to the reduction in ambient oxygen has been determined by Dripps & Comroe (1947) and can be calculated from Equation 11:

$$\text{Drop in } SaO_2 = e^{(6.8-0.298 \times (\text{Inhaled Oxygen concentration}))} \quad \textbf{(Equation 11)}$$

For example, an ambient oxygen concentration of 15% results in a maximum drop in blood saturation of 10%.

The mean reduction relationship provided by Dripps & Comroe is of the form:

$$\text{Drop in } SaO_2 = e^{(6.8-0.288 \times (\text{Inhaled Oxygen concentration}))} \quad \textbf{(Equation 12)}$$

In this case an ambient oxygen concentration of 13.5% is required to give the same 10% drop in blood saturation.

90 However during escape or emergency action physical activity increases workload and based upon the 95th percentile prediction interval calculated from the Torre-Bueno study for light to moderate exercise, **in the absence of carbon monoxide**, gives the relationship described by equation 11.

$$\text{Drop in } SaO_{2(95\% \text{ percentile})} = e^{(10.5-0.455 \times (\text{Inhaled Oxygen concentration}))} \quad (\textbf{Equation 13})$$

91 In this event an ambient oxygen concentration of 18% results in a 10% drop in blood saturation while 17% ambient oxygen results in a 15% reduction. Typically maximal oxygen consumption is reduced by a similar percentage of blood COHb level. On this basis it is suggested that a similar criteria is adopted for SaO_2 where:

Cautious recoverable SaO_2 reduction = 10%

Threshold of SaO_2 reduction leading to harm and fatality = 15%

92 On the basis of Equation 13 and the proposed SaO_2 reduction criteria the limiting oxygen depletion levels for survivability can be established at 18% and 17% respectively.

93 Typically, a lowered oxygen saturation results in a reduction in the time to physical exhaustion during high workload. Ekblom & Huot (1972) showed that the

time taken to reach exhaustion during maximum physical effort was reduced to 55% when COHb was 12.8 to 15.8%.

Combined effects of carbon monoxide, carbon dioxide and oxygen depletion

94 The combined effects of CO, CO_2 and oxygen depletion are the main causes of fatalities in smoke. Depending on the degree of ventilation of a compartment or location during a fire, the nature and composition of the smoke can change and produce significantly differing consequences. In the event of an under ventilated fire, CO hazards will be dominant. For a well-ventilated fire CO production is much less and oxygen depletion may be the dominant hazard. There are a lot of uncertainties in the calculation of amount of smoke produced in a fire situation and amount of toxic gases in the smoke. This depends on type of burning fuel and ventilation conditions.

95 The proportion of toxic gases in smoke depends on the chemical structure of the burning materials and the degree of ventilation to the fire. The differences in the toxic gases produced by burning different hydrocarbons are small, and ventilation has the main effect. Typically, ventilation restriction occurs only for fires in modules or compartments. These fires will either be fuel controlled or ventilation controlled. In general, reduced ventilation greatly increases the ratio of CO, while the O_2 and the CO_2 remain more or less unaffected as shown in Table 30.

Table 30: Typical gas concentrations close to a fire (Norsok Z013)

Substance	Gas Concentration in smoke (%)			
	Well ventilated fire		Under ventilated fire	
	Gas fire	Liquid fire	Gas fire	Liquid fire
CO	0.04	0.08	3.0	3.1
CO_2	10.9	11.8	8.2	9.2
O_2	0	0	0	0

96 For an onshore installation the possibilities to escape from an accident is greater than on an offshore installation where personnel may be exposed to toxic gases over a longer time period. In this event tolerable concentration estimates should account for the increased exposure where it occurs.

97 In the event of exposure to smoke containing both CO and CO_2 increase respiration rate can significantly reduce time to impairment and death for an exposed population.

98 An example of the calculation of the blood level of COHb in the presence of carbon dioxide, as applied to offshore workers exposed to smoke from a major accident, is shown from the equation suggested by Forbes *et al* as modified by Clark *et al* 1980) **(Equation 14)**:

%COHB = Kc x %CO inhaled x Exposure time (minutes) (Equation 14)

Where:

K = a constant related to breathing rate that is dependant on work rate or effort.

c = factor to account for the presence of Carbon dioxide as shown in Table 31.

99 It is suggested that values between 8 and 11 are most appropriate for individuals in an emergency situation. Eight is selected as equivalent to a light workload (50 Watts), 18 l/min ventilation rate and pulse of 110 /min for muster and evacuation or occupancy of the TR during an emergency. It is suggested that in the event of escape or other emergency action the value should be increased to 11 to represent heavy work (100 Watts) 30l/min respiration and 135-beats/min pulse.

Table 31: Value of constant "c" for differing CO_2 levels and expected CO concentration to give 15% COHb after 30 minutes exposure

C	**K value**	**1**	**1.2**	**1.5**	**1.8**
%CO_2		0.0	1	2	3
ppm CO giving 15% COHb after 30 minute	8	620	520	420	170
	11	450	380	300	250

Note: % CO = ppm level / 10,000
Maximum CO ppm inversely proportional to exposure time and to %COHb

Toxic agents (gases, liquids or solids)

Toxic gas effects (mainly NOx, NH3, SO2 and HF)

100 **Warning**: It should be noted that the consequences of exposure to hazardous substances, such as toxic gases, are the subject of continuous research and review. As more and better data becomes available the exposure levels and harm criteria are continuously modified. While every attempt has been made to ensure the values used in this note are appropriate and accurate, risk assessors should ensure the harm criteria used is appropriate and reflects the most recent established values.

101 Toxic gases may be present as the result of fire, in the well fluids or part of the hydrocarbon transportation and treatment process. Effects of toxic gases can be divided into two categories:

- Local irritants: these may cause incapacitation mainly by effects on the eyes and the upper respiratory tract that may impair escape capability and sometimes cause delayed death due to lung damage.

- Systematically acting agents: these cause damage to the body via the blood and distribution to other organs and include so-called narcotic gases (note: not all systemic agents are narcotics).

102 The main toxic gases of fire effluents have been described earlier but to recap they include:

- Carbon monoxide, (CO),
- Carbon dioxide, (CO2),
- Hydrogen sulphide, (H2S),
- Nitrogen oxides, (NOx),
- Ammonia, (NH3),
- Sulphur dioxide, (SO2)
- Hydrogen fluoride, (HF).
- Hydrogen cyanide (HCN).

CO, Nitrous Oxide, Hydrogen Cyanide and CO2 are classified as narcotic gases, while the other are classified as irritants or highly irritant.

Although CO is not the most toxic of the above-mentioned gases, it is present in relatively high concentrations in smoke, and so its effects are usually dominant.

103 The issue of toxic gas generation from fire impingement has been addressed in the specification of firewalls designed to the IMO FTP code (1998 resolution MSC61(67)) the limits, as shown in Table 32, have been set for hazardous vapour generation from firewalls exposed to radiant heat;

Table 32: IMO resolution MSC 61(67) limits for fume generation from firewalls exposed to radiant heat

Substance	CO	HBr	HCl	HCN	HF	NOx	SO_2
Maximum conc (ppm)	1450	600	600	140	600	350	120

Toxicological effects of NOx, NH3, SO2 and HF:

- NOx: Strong pulmonary irritant capable of causing immediate death as well as delayed injury

- NH3: Pungent, unbearable odour; irritant to eyes and nose

- SO2: A strong irritant of the eyes, mucous membranes, and upper respiratory tract and skin, intolerable well below lethal concentrations

- HF: Respiratory irritant

The lethal concentration predictions for humans provided in NORSOK Z013 are given in table 33.

Table 33: Predicted lethal concentrations for expected toxic gases (Norsok Z013)

Toxicant	Human LC50 (ppm) predicted from metabolic rate		Human lethal concentrations (ppm)
	5 minute exposure	30 minute exposure	
NH_3	55000	----------	2000
SO_2	17000	8000	600-800 (few mins)
HF	44000	4600	
NO_x	410	180	250 (few min)

104 The following probits can be obtained from the TNO green book but will not necessarily provide the same LC50 values as presented in Table 33 or as the probit values provided by NORSOK Z013 as described in Table 1.

Ammonia	$Pr = -15.8 + \ln(C^{2.0}*t)$ 30 min LC_{50} 6164 mg/m^3	**(Equation 15)** (8650 ppm)
Sulphur Dioxide	$Pr = -19.2 + \ln(C^{2.4}*t)$ 30 min LC_{50} 5784 mg/m^3	**(Equation 16)** (2180ppm)
Hydrogen Fluoride	$Pr = -8.4 + \ln(C^{1.5}*t)$ 30 min LC_{50} 802 mg/m^3	**(Equation 17)** (970 ppm)
Nitrogen Dioxide	$Pr = -18.6 + \ln(C^{3.7}*t)$ 30 min LC_{50} 235 mg/m^3	**(Equation 18)** (123 ppm)

Combination of hazardous exposures and estimation of impairment

105. One approach to consider the combinational effects of impairment due to exposure to hazards is described by information prepared for HSE and yet to be published. In this approach the fractional sum for each hazard exposed is estimated. It is suggested that this approach may also take in to account the effects of elevated ambient temperature as it takes the form of the sum of the simple fractions of each exposure when measured against the limiting value estimated for the period under evaluation (i.e. the fraction of the incapacitating dose).

106. For example, the calculation for the additive effects of respiratory irritation takes the form described by equation 19.

$$\frac{C_1}{L_1} + \frac{C_2}{L_2} + \frac{C_3}{L_3} \ldots\ldots\ldots\ldots\ldots \frac{C_n}{L_n} = D_{tot} \qquad \textbf{(Equation 19)}$$

For impairment to occur $\mathbf{D_{tot}}$ must be greater than unity

107 Although this approach has often been used by Occupational Hygienists (who refer to it as the additive equation) it is considered to have two significant issues:

- It has only really been demonstrated (in animals) for acute effects.
- The approach is only usually considered valid if the harmful agents considered bring about the same end point, attack the same organ or have a similar mode of action.

Despite this the approach is probably conservative.

108. The additive equation is considered best suited for the estimation of time to impairment of the TR. The exposure limit for each hazardous agent should be selected on the basis of time to impairment. They should also take into account any significant sensitivities of the exposed population such as elevated COHb in smokers and increased sensitivities due to chronic medical conditions such as respiratory irritation (i.e. Asthmatics). Table 34 highlights the significance of these considerations:

Table 34: Suggested 60 minute exposure limits for Individual harmful agents to minimize respiratory irritation and concentrations leading to severe effects on the respiratory tract

Agent	60 Minute Limit (ppm)		Severe effects (ppm)
	Asthmatics	Normal	
Acrolein	No data	0.2	2
Nitrogen dioxide	0.2	1.0	25
Sulphur dioxide	0.1	1.0	10
Hydrogen chloride	0.5	5.0	50

109 The combined effects of CO, oxygen depletion and the additional effects of other hypoxic agents can be estimated over a specific period (i.e. one hour for TR impairment studies) by using the concept of the Permissible Hypoxic Dose (PHD). In this approach each toxic agent provides a Fraction towards the PHD (FPHD).

110 To calculate this fraction (FPHD) it is first necessary to establish an impairment threshold for each of the hypoxic agents. The sum of all fractions, from each hypoxic agent, will determine the reduction of absorbed oxygen in the blood stream caused

by inhalation of the toxins present. In the event of the sum of all FPHD's exceeding unity impairment occurs.

111 Information prepared for HSE and yet to be published proposes an impairment criterion of a 10% reduction in SaO_2. By their estimation this represents an ambient concentration of 18% oxygen or a carboxyhaemoglobin level of 10%. In addition, equivalent concentrations for Hydrogen Cyanide, Hydrogen Sulphide and Nitrogen Oxides that give a 10% reduction in SaO_2, and their basis, have been suggested for a 60 minute exposure period (Table 35). In the event of exceeding a Permissible Hypoxic Dose individuals so exposed will be considered to have exceeded the threshold of survivability resulting in impairment (ability to perform emergency tasks with proper dexterity) and death as the dose received increases.

In the case of NO_2, a WATCH conclusion was that asthmatics were not more sensitive than non-asthmatics to respiratory tract irritation. For hydrogen chloride, there is not really any data to show whether or not asthmatics are more sensitive than non-asthmatics; but there is information showing that exposure of mild asthmatics to 1.8 ppm for 45 mins is without effect, so the value of 0.5 ppm may be conservatively high.

Table 35: Suggested equivalent concentrations of toxins to reduce SaO_2 10% for use in permissible hypoxic dose estimations.

Toxin	10% SaO_2 reduction equivalent	Mode of action
Oxygen depletion	3%	Simple asphyxia
Hydrogen cyanide	20 ppm	Cytochrome oxidase inhibition
Hydrogen sulphide	60 ppm	Cytochrome oxidase inhibition
Nitrogen oxides	50 ppm	Methaemoglobinaemia

The total FPHD is then estimated from the following relationship:

$$FPHD = \frac{\text{Drop in } SaO_2}{10\%} + \frac{\%\ COHb}{10\%} + \frac{\text{ppm HCN}}{20} + \frac{\text{ppm } H_2S}{60} + \frac{\text{ppm NOx}}{50}$$

(Equation 20)

For impairment to be achieved FPHD > 1.0

Where impairment is defined as excedence of the level of exposure that produces no acute effects over the period of consideration

112 Note, in this example the effects of HCN and H_2S are assumed to be entirely concentration dependent without any cumulative effect. It is assumed the effects of CO and NO_x, are cumulative and will not be reversible over the evaluation period (one hour in this case). This equation can be extended to include other hypoxic agents but the FPHD must not equal or exceed 1.0.

113 The selection of appropriate impairment criteria for this approach is important to avoid gross over or under estimation of the consequences of a combined exposure. In the example shown above it may be considered that the impairment criteria is overly pessimistic when compared against the values described previously (SLOT, Probit and IDLH values). The selection of a 10% drop in SaO_2 is considered appropriate to produce a risk assessment applying using a "Cautious Best Estimate Approach". Where the use of conservative criteria results in the determination of a tolerable risk a robust demonstration is obtained. Should the analysis fail to provide such a demonstration it may be necessary to apply a greater degree of rigor to vulnerability criteria but this must also be supported with appropriate references to support the modified criteria.

114 Also as the complexity of assessment increases and the criteria conservatism is reduced it is essential that claim limits are clearly stated, adequately referenced and used appropriately.

Effects of other gases

115 Toxic gases (e.g. Hydrogen chloride) or the generation of toxic fumes, due to thermal degradation of chemicals or construction materials, could quickly render atmospheres un-breathable.

116 The suggested performance standard is, therefore, that if these should be present in the TR at levels above the IDLH value, then impairment of the area has occurred.

Hydrogen sulphide

117 Other than toxic products from combustion of hydrocarbons the most likely toxic gas present in well fluids is Hydrogen Sulphide (H_2S). The effects likely to be experienced by humans exposed to various concentrations of H_2S are described in Table 36.

Table 36: Effects of exposure to hydrogen sulphide

Concentration (ppm)	Effect
20 - 30	Conjunctivitis
50	Objection to light after 4 hours exposure. Lacrimation
150 - 200	Objection to light, irritation of mucous membranes, headache
200 - 400	Slight symptoms of poisoning after several hours
250 - 600	Pulmonary edema and bronchial pneumonia after prolonged exposure

500 - 1000	Painful eye irritation, vomiting.
1000	Immediate acute poisoning
1000 - 2000	Lethal after 30 to 60 minutes
> 2000	Rapidly lethal

Hydrogen sulphide gas can be detected at concentrations lower than 1 ppm (Amoore, 1985), but the sense of smell is lost after 2-15 min exposure at that concentration, thus making it impossible to detect dangerous concentrations (Lees, 2005). At concentrations exceeding 50 ppm (70 mg/m³), olfactory fatigue prevents detection of H_2S odour. Moreover, the odour of hydrogen sulphide can be masked by the presence of other chemicals; tests show that concentrations below 1 ppm could be detected by odour in air, whereas in the presence of light hydrocarbons such as propane or butane even 5-10 ppm could not be smelt (Lees, 2005).

A recent WATCH review suggest that a no-effect level for eye irritation was around 80-100 ppm. Also, it was concluded that concentrations in excess of 500 ppm could be fatal to humans. A lethal exposure was documented for a worker exposed to approximately 600 ppm H_2S for 5-15 minutes (Simson and Simpson, 1971). Inhalation of 1,000 ppm (1,400 mg/m³) is reported to cause immediate respiratory arrest (ACGIH, 1991).

Probits

Several probit functions have been developed based on experiments data from animals and the probit functions defined by equations 21a to 21c can be obtained from the TNO Green Book:

$$Pr = -32.92 + 3.01\ln(C^{1.43}*t) \quad \textbf{(Equation 21a)}$$

$$Pr = -42.6 + 2.36\ln(C^{2.17}*t) \quad \textbf{(Equation 21b)}$$

$$Pr = -44.7 + 2.9\ln(C^{2.0}*t) \quad \textbf{(Equation 21c)}$$

Where C is concentration in mg/m^3
t is exposure in minutes

$1mg/m^3 = 0.71$ ppm

Alternatively the dangerous dose approach may be utilized and is the preferred approach by HSE.

Note: The EH40/2002 exposure limits for hydrogen sulphide are 5ppm for 8-hour exposure, and 10 ppm for 15 minutes.

Exposure to hydrocarbon vapours

118 Hydrocarbons are not specified as toxic but exposure to elevated levels can produce narcosis and ultimately death. Also, as mentioned below, the hydrocarbons can cause asphyxiation at very high concentrations. While the existence of more acute hazards, such as ignition of a hydrocarbon cloud within its flammable range, typically dominates, the consequence assessment of exposure to hydrocarbon vapour should be carried out for offshore installation major accidents. This is of greater significance where personnel are located in poorly ventilated areas, including confined spaces such as supporting structures (i.e. hollow columns or supporting legs), drainage systems or process equipment (large vessels). Hydrocarbon vapours are typically heavier than air and will tend to accumulate in low spots where they have a range of significant effects depending on the hydrocarbon, the concentration and the time of exposure. In addition to the toxicity of certain hydrocarbons they can produce narcotic effects that will impair thought processes and manual dexterity thus reducing ability to escape.

119 Hydrocarbons of C5 upwards demonstrate pre-anaesthetic effects (dizziness, confusion, inappropriate behaviour) that can start after 10 minutes exposure to 3000 – 5000 ppm. Inhalation of high concentrations may cause central nervous system depression such as dizziness, drowsiness, headache, and similar narcotic. The higher molecular weight (“heavier”) alkanes have a proportionally higher potency as anaesthetics (e.g. n-heptane is around 10 times more potent than n-pentane).

Typically, the lower “gaseous” alkanes (methane to butane) are generally regarded as having low toxicity, but they act as simple asphyxiates by displacing oxygen. Signs of asphyxiation will be noticed when oxygen is reduced to below 16-18%, and may occur in several stages (as described earlier).

120 Gas Dispersion modelling may not account for localized flows and time varying concentrations of gas. Where flammable gas exposure may occur the physiological effects of exposure may be overtaken by a more acute hazard, i.e. explosion or flash fire. Under these circumstances it is typical to establish a concentration isopleth that provides the establishment of an exposure limit of <50% LEL. At concentrations much lower than this value (2.5% v/v or 25000ppm) oxygen depletion and / or the anaesthetic effects may be significant for medium duration exposure where ignition does not occur and evacuation to a “safe haven” such as the TR is not possible. The 5000ppm isopleth from a hydrocarbon release would be expected to have a much larger footprint than that estimated for ½ LFL.

Smoke / obscuration of vision

121 The absence of vision may delay or prevent escape from fires and cause people to be exposed to the fire gases for an unacceptable long period of time. While the exposure to high concentrations of toxic and hot gases usually will be significant only in the vicinity of the fire, the effect of reduced visibility may also be significant far away from the fire source.

122 For example, in multi-compartment buildings, the smoke blocking effect may be significant in rooms far away from the room of fire origin. Moreover, the smoke blocking effect is reported to be the first condition becoming critical of the three hazardous conditions of fires i.e. heat stresses, obscuration of vision, toxic effects. The hazard of smoke is characterized by three factors:

- The threat of reduced visibility due to soot.
- Hot smoke can cause pain and injuries
- A concentration of toxic and irritating components can lead to incapacitation or death.

The relative order of these factors can be found by comparison of threshold values with actual exposure in a fire scenario.

123 A visibility of 4-5 m is about the threshold of diminished performance, and this is the smoke level that should be considered in smoke ventilation system design. It is suggested that there should be a minimum of 3m vision for escape from a primary compartment and at least 10m for an escape route. Important factors to consider in a risk analysis with regard to obscuration of vision (and time to escape) are:

- Exposure to smoke
- Arrangements of escape ways (layout, sign, illumination, railing, etc.)
- Training of personnel
- Familiarization with the installation.

Where an escape way is well laid out and provided with high visibility marking or illumination then the 3m criterion may be applied.

Hypothermia

124 OTO 95 038 “Review of Possible Survival times for Immersion in the North sea” (Robertson & Simpson 1996 see Annex 4) draws the following conclusions:

> The dominant threat to an immersed person is drowning. However much of the past effort has focused upon death from hypothermia. While the study of human thermal physiology is amenable to scientific analysis, survival times based upon cooling of the vital organs alone is irrelevant if the victim is unable to breathe due to inhalation of water. Nevertheless the effects of cold are debilitating and increase the probability of drowning. Due to this contributory effect, data on the early stages of hypothermia are an important input factor in the overall estimation of survival times in cold water.
>
> Within a group of survivors a proportion of them are likely to succumb to cold shock upon initial immersion in the water. Others within the group may be injured prior to immersion and hence be incapable of self-help once in the water. The estimated survival time ranges presented in this paper therefore assume death within minutes for these most vulnerable individuals. The upper bounds of the survival time ranges are for a thin, reasonably fit and uninjured individual. The estimate for survival in winter for such people, wearing a typical UKCS immersion suit and lifejacket combination, is within half an hour. It is considered that survival time estimates which are significantly longer than this period probably include an unjustified degree of optimism.

125 On this basis assessors are recommended to use the detail provided in OTO 95 038 unless they can demonstrate that any alternative data used is suitably robust and more suitable for their particular assessment.

Table 37: Predicted survival times for complete immersion of an average individual in light and heavy sea conditions at 5°C (taken from Prediction of Sea Survival Time, Defense and Civil Institute of Environmental Medicine: document – DCIEM No. 96-R-12).

Exposure Description	Survival Times (hrs)	
	Light Sea	Heavy Sea
Survival coverall + 2	13.4	5.5
Fisherman's work suit + 2 + 3	13.4	5.5
Aviation coverall + cotton-ribbed ug + 4	5.9	3.4
Aviation coverall + single pile + 4	13.8	5.7
Aviation coverall + double pile + 4	29.3	9.2
Quick-don (dry) suit + 1 + 2 + 3	19.3	7.0
Quick-don (dry) suit + double pile + fg	30.7	9.5
Dry immersion suit + 2 + 4 + 5	13.8	5.5
Dry immersion suit + single pile + 4	32.1	9.8
Dry immersion suit + double pile + 4	> 36	14.2
4 mm Neoprene wet suit	5.5	3.5
7 mm Neoprene wet suit	8.3	4.9

(1) – t-shirt; (2) – long-sleeved shirt; (3) – heavy sweater; (4) – vest; (5) – Work jacket; (ug) – undergarment; (fg) – flying suit.

Table 38: Stages of hypothermia and clinical features (L. McCullough and S. Arora, Diagnosis and Treatment of Hypothermia, American Family Physician, 70 [12] (2004) 2325-32).

Hypothermia zone	Body temperature	Clinical features
Mild	32.2 to 35	* Initial excitation phase to combat cold: Hypertension, Shivering, Tachycardia, Tachypnea, Vasoconstriction * With time and onset of fatigue: Apathy, Ataxia, Cold diuresis kidneys lose concentrating ability, Hypovolemia, Impaired judgment
Moderate	28 to 32.2	Atrial dysrhythmias, Decreased heart rate, Decreased level of consciousness, Decreased respiratory rate, Dilated pupils, Diminished gag reflex, Extinction on shivering, Hyporeflexia, Hypotension
Severe	< 28	Apnea, Coma, Decreased or no activity on electroencephalography, Nonreactive pupils, Oliguria, Pulmonary edema, Ventricular

		dysrhythmias/asystole

Annex 1
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Annex 2

Probit Table

Percentage affected - probit transformation (taken from Finney, D.J., 1971. probit analysis, p25).

%	0	1	2	3	4	5	6	7	8	9
0	-	2.67	2.95	3.12	3.25	3.36	3.45	3.52	3.59	3.66
10	3.72	3.77	3.82	3.87	3.92	3.96	4.01	4.05	4.08	4.12
20	4.16	4.19	4.23	4.26	4.29	4.33	4.36	4.39	4.42	4.45
30	4.48	4.50	4.53	4.56	4.59	4.61	4.64	4.67	4.69	4.72
40	4.75	4.77	4.80	4.82	4.85	4.87	4.90	4.92	4.95	4.97
50	5.00	5.03	5.05	5.08	5.10	5.13	5.15	5.18	5.20	5.23
60	5.25	5.28	5.31	5.33	5.36	5.39	5.41	5.44	5.47	5.50
70	5.52	5.55	5.58	5.61	5.64	5.67	5.71	5.74	5.77	5.81
80	5.84	5.88	5.92	5.95	5.99	6.04	6.08	6.13	6.18	6.23
90	6.28	6.34	6.41	6.48	6.55	6.64	6.75	6.88	7.05	7.33
%	0.0	0.1	0.2	0.3	0.4	0.5	0.6	0.7	0.8	0.9
99	7.33	7.37	7.41	7.46	7.51	7.58	7.65	7.75	7.88	8.09

Examples:

1% is equivalent to 2.67 probits. (“y” in equation xxx)

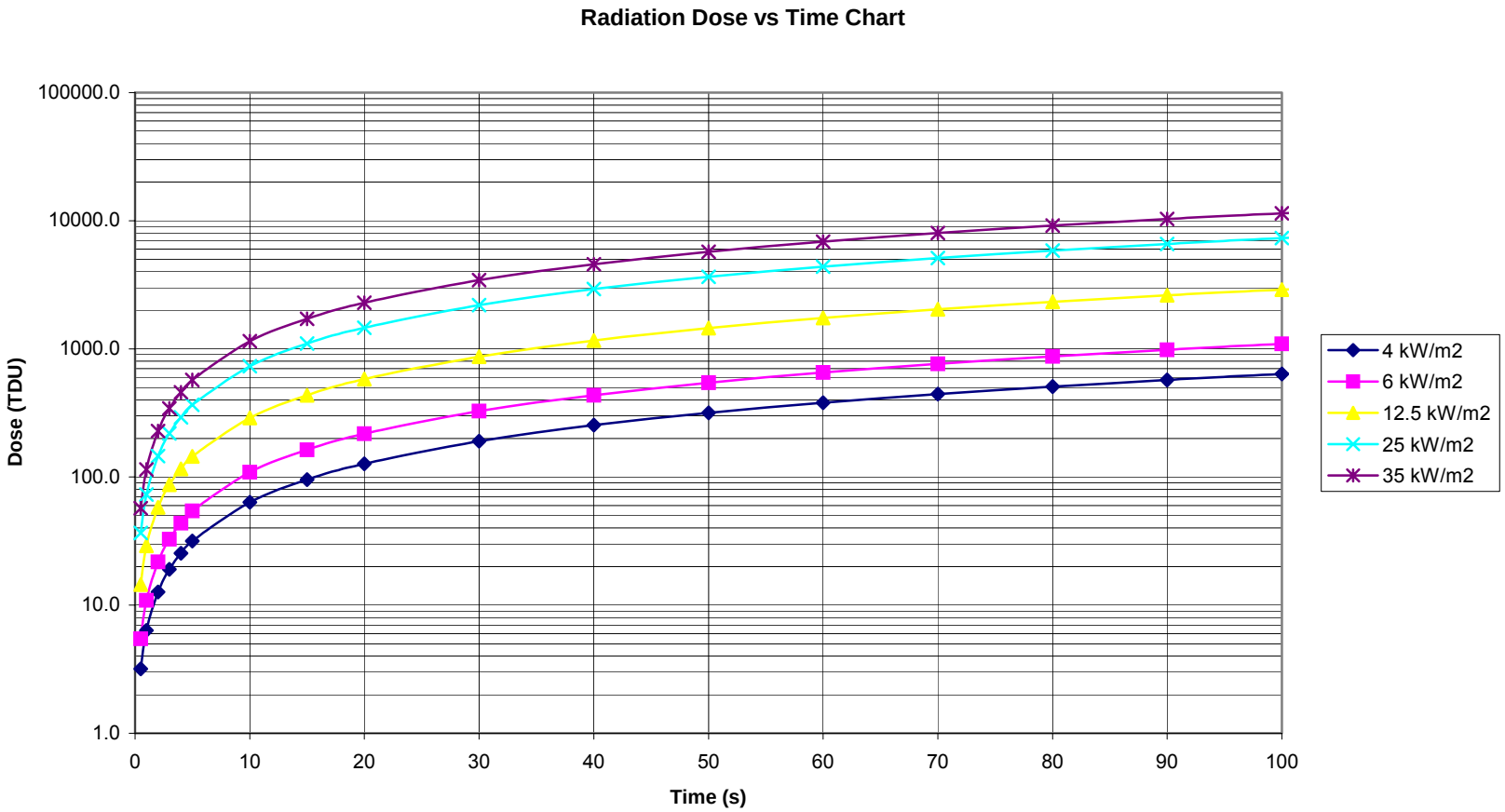
42% is equivalent to 4.80 probits.

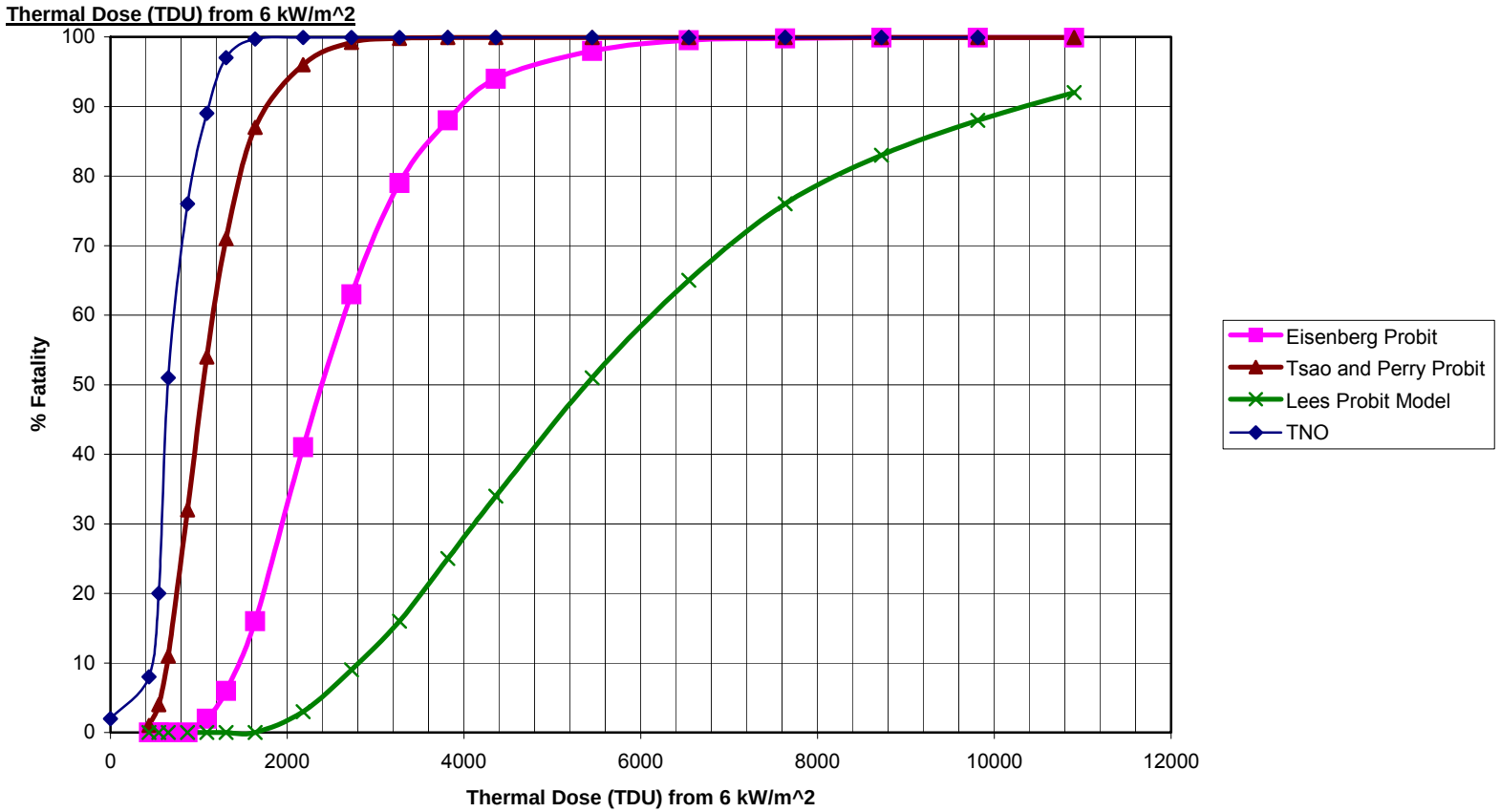
50% is equivalent to 5.00 probits.

75% is equivalent to 5.67 probits.

99.9% is equivalent to 8.09 probits.

Annex 3
Thermal radiation dose charts




Thermal Dose (TDU) from 6 kW/m^2
% Fatality
100
90
80
70
60
50
40
30
20
10
0
0
2000
4000
6000
8000
10000
12000
Thermal Dose (TDU) from 6 kW/m^2
Eisenberg Probit
Tsao and Perry Probit
Lees Probit Model
TNO

Annex 4
Predicted survival times against sea temperature (OTO 95038)

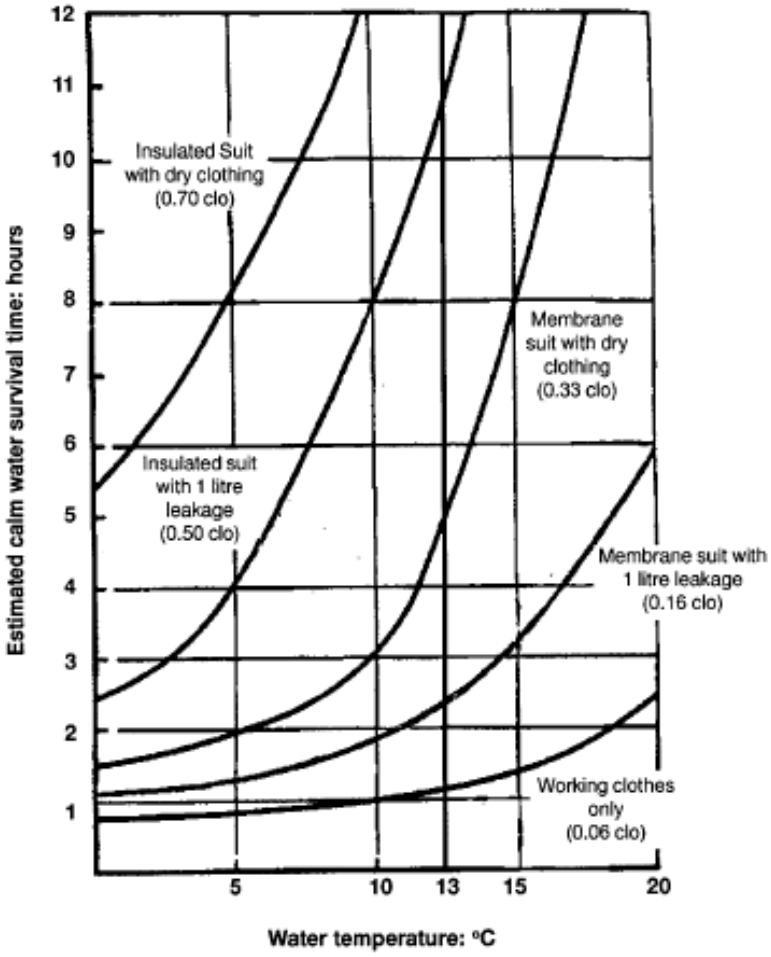


Figure 2
Predicted Survival Time Against Sea Temperature for Different Levels of Immersed Clothing Insulation - As Derived from Wissler Model, Modified by Hayes, 1987